Comprehensive Care Project

Bruna M. Rett #2429

Department of Dental Hygiene

West Los Angeles College

Advanced Seminar and Periodontology in Dental Hygiene

March 8th, 2024

I. Patient Identification.

1. Age: 59.
2. Sex: Male.
3. Race and/or Ethnicity: Asian American.
4. Marital Status: Single.
5. Occupation: Actor.

II. Chief Concern.

1. Based on discussion with the patient.
    a. No chief concern.

III. Medical History.

1. Past Medical History (include last physical examination).
    a. Client is currently under the care of a physician in order to get clearance for hepatitis B carrier.
    b. May need a hip replacement soon since his hip has been bothering him within the past year.
    a. Last comprehensive physical examination was in 9/29/2022.

2. Past medication or drug use.
    b. No past medications or drug use.

3. BMI (Body Mass Index).
    a. 33.4
    b. A BMI of 30 or greater is considered obesity.
    c. BMI is a screening measure that uses the client's weight and height to determine a healthy weight range.
        i. It is not intended to diagnose disease or illness but maintaining a healthy BMI range is one way to support overall health as the client ages.
    d. Having excess weight may increase the risk for chronic conditions such as heart disease, high blood pressure, high cholesterol and diabetes type II.
        i. Increasing physical activity can lower the risk for heart disease.
        ii. Eating a healthy diet is key to prevent heart disease.

4. Family History
    a. No known history of disease in the family.

5. General
    a. Client is hepatitis B carrier.
    b. Possibly having high cholesterol since last physical checkup, but never followed up nor is taking medications for this particular condition.

6. Hospitalizations
    a. Client has not had any serious illness, operation, nor been hospitalized in the last 5 years.

7. Review of Systems
    a. Upon thorough extraoral and intraoral examinations that were completed at each dental visit:
        i. The client's neurological system appears to be functioning appropriately when observing his gait from the moment he walked into the office until he walked out.
        ii. Upon tracking his eye movement by using index finger, the patient had no issues when moving his eyes.
        iii. The patient's psychological and functional systems are within normal limits.
        iv. When analyzing his mental health and personal safety, even though the client stated that he has occasional stress, it does not seem to be an issue in which he seriously thinks about hurting himself.
        v. Upon taking the patient's vital signs (such as blood pressure/temperature/respiration/oxygen saturation) and checking the throat and oral cavity, the client's respiratory and cardiovascular systems appears to be in good condition.
        vi. The client's dermatological and GI systems seems to be within normal limits, despite occasionally taking medication for gout issue.
        vii. When it comes to the patient's sexual and endocrine systems, it appears to be within a normal range that is expected for his age, since occasional gout problems may lead to erectile dysfunction.
        viii. The patient's hematological and immunological systems are in healthy conditions given the fact that the patient did not bleed excessively during scaling and has not complained of sore gingiva/tissues after receiving treatment.
        ix. Patient states that has no past or current implications with bone or healing.

8. Current Medication (Implication and Dental concerns)
    a. Indomethacin 25mg prn for gout issues.
        i. Not enough information on dental implications and concerns.
        ii. Side effects: drowsiness – used caution when raising dental chair.
    b. Propecia 1mg once daily.
        i. Not significant dental implications and concerns reported.

9. Baseline Vital Signs
    a. Monitoring the client's vital signs is extremely important in order to record the progress of treatment and to detect any anomalies that may rise, and the patient may not be aware of.
    b. Making sure that the patient has a healthy pulse, blood pressure, oxygen, respiration and temperature decreases the likelihood of having to deal with medical emergencies in the dental office.

c. In case of any abnormal vital signs, referrals may be needed in order to continue to monitor the progress of disease.
d. An average of the client's baseline vital signs was collected over a total of five appointments:
    i. Blood Pressure: 116/73 mmHg – healthy range.
    ii. Oxygen Saturation: 98% - healthy range.
    iii. Respiration: 14 breaths per minute – healthy range.
    iv. Temperature: 98.0 °F – healthy range.
    v. Pulse: 73 beats per minute – healthy range.

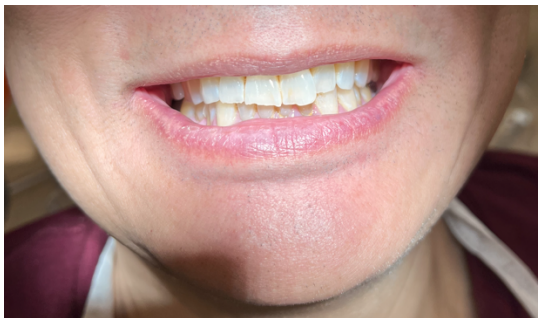
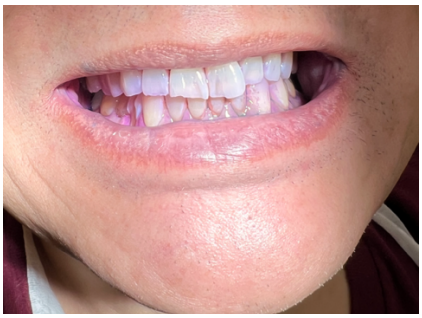
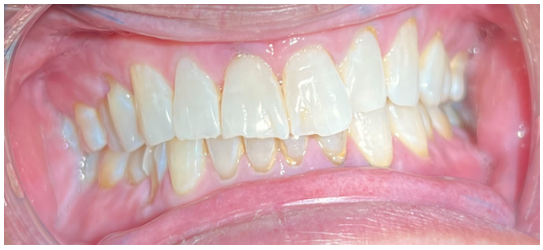
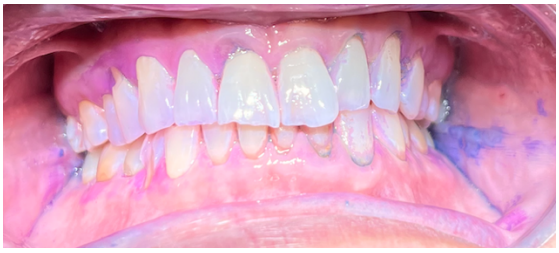
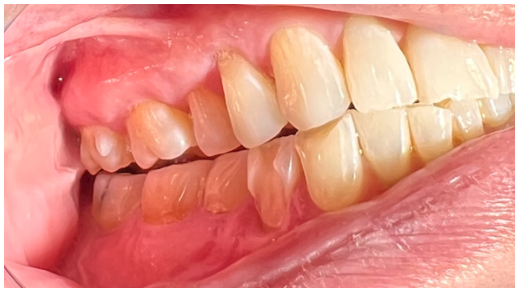
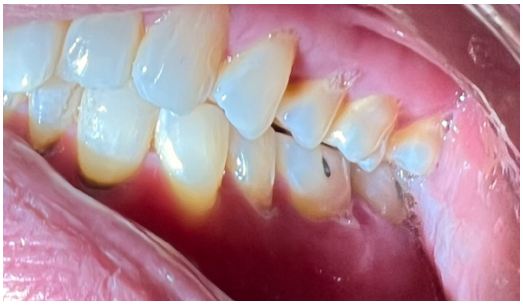
10. ASA status with rationale.
    a. The physiological status for this client is ASA II.
        i. A patient with mild systemic disease such as hepatitis B carrier and occasional gout issues.
            - No significant side effects due to medications were reported by the patient.
        ii. Mild disease without substantive functional limitations such as obesity and well-controlled systemic conditions.
    b. The American Society of Anesthesiologists (ASA) classification system does not predict the perioperative risks.
        i. But when used with other factors can be helpful in predicting perioperative risks.
    c. The final assignment of Physical Status classification is made on the day of anesthesia care by the anesthesiologist after evaluating the client prior to the procedure.

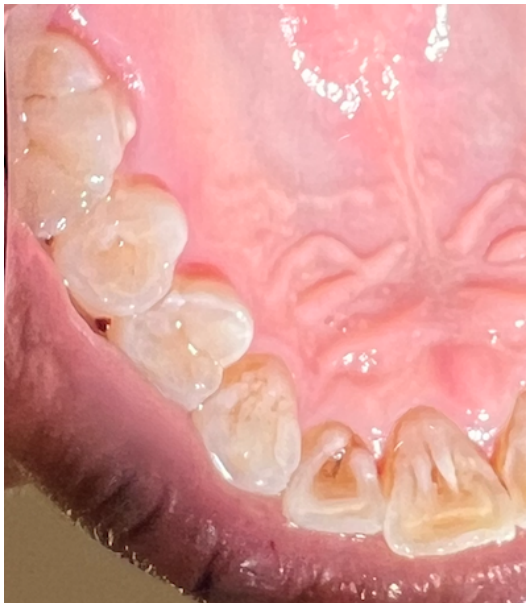
IV. Clinical Examination (Pre-Treatment)

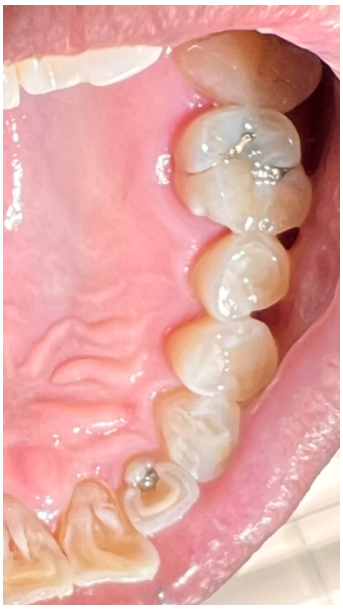
1. Extra-and-Intra Oral Examination
    a. Extraoral
        i. Asymptomatic clicking and popping upon opening and closing on the right side.
    b. Intraoral
        i. Missing #1, #16, #17, and #32.
        ii. Left palatal tonsil redness.
        iii. Palatal tori approximately 9x6 mm.
        iv. Keratinized tissue on buccal mucosa on the right side.
        v. Lingual erosion from #7-#9.
        vi. Tooth #28 missing buccal enamel with dentin exposure.
        vii. Tooth #29F abfraction.
        viii. Lingual erosion #7-#9.
        ix. Tooth #19B has a very prominent CEJ (per Dr. David Nguyen).
        x. Recession 1-2mm on maxillary and mandibular canines.
        xi. Crown to root ratio: generalized 2:1
        xii. Generalized attrition – most prominent on mandibular anterior incisal edges.
        xiii. Generalized BOP on molars.
        xiv. Chipped incisal edges #8-#9.
        xv. Cusp of Carabelli present on the mesiopalatal surface of maxillary first molars.

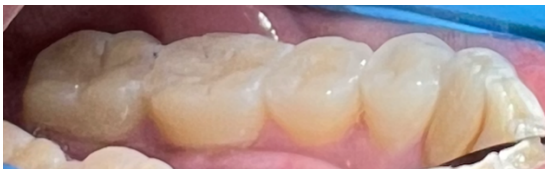
xvi. Cust of Carabelli present on the mesiobuccal surface of tooth #2.
xvii. Prominent masseter muscles
xviii. Right molar relationship: Class I.
xix. Right canine relationship: Class I.
xx. Left molar relationship: Class I.
xxi. Left canine relationship: Class I.
xxii. Overjet: 5mm.
xxiii. Overbite: 2mm.

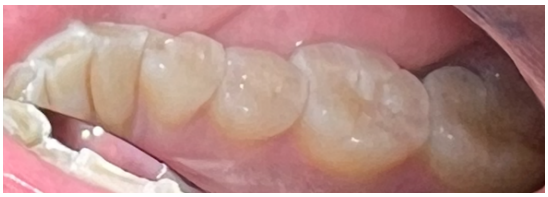
Pre-treatment photos

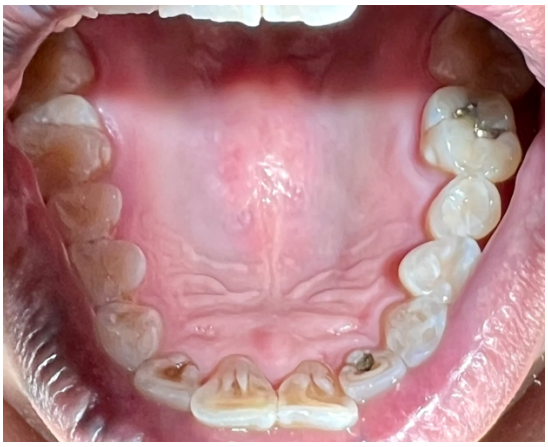







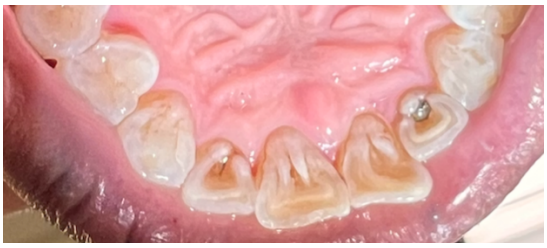


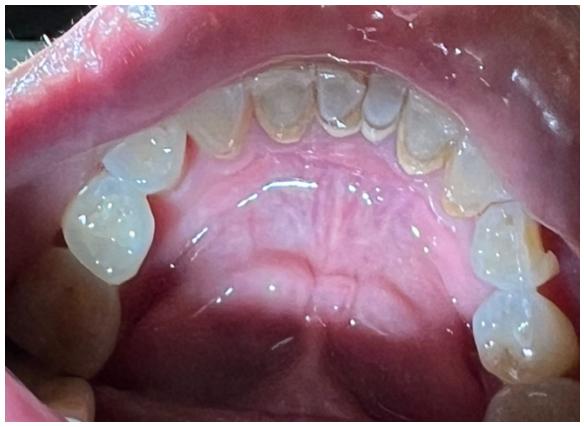


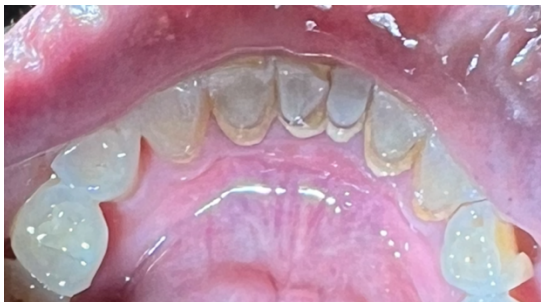


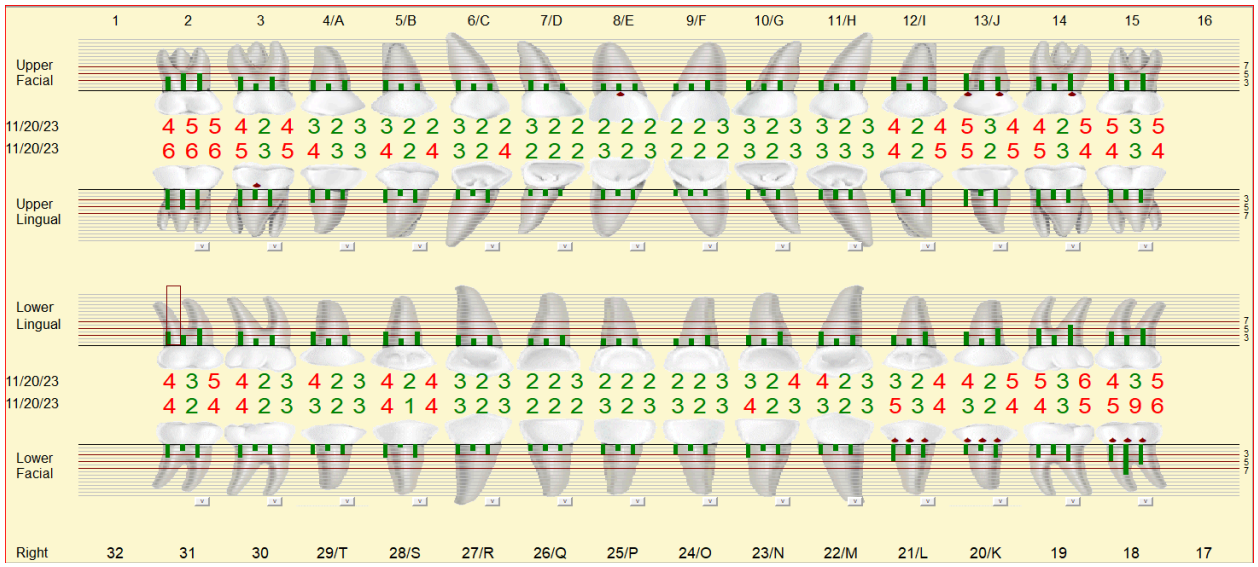











1 2 3 4/A 5/B 6/C 7/D 8/E 9/F 10/G 11/H 12/I 13/J 14 15 16
Upper Facial
11/20/23 4 5 5 4 2 4 3 2 3 3 2 2 3 2 2 3 2 2 2 2 2 2 3 3 2 3 3 2 3 4 2 4 5 3 4 4 2 5 5 3 5
11/20/23 6 6 6 5 3 5 4 3 3 4 2 4 3 2 4 2 2 2 3 2 3 2 2 2 3 2 3 3 3 3 4 2 5 5 2 5 5 3 4 4 3 4
Upper Lingual
Lower Lingual
11/20/23 4 3 5 4 2 3 4 2 3 4 2 4 3 2 3 2 2 3 2 2 2 2 2 3 3 2 4 4 2 3 3 2 4 4 2 5 5 3 6 4 3 5
11/20/23 4 2 4 4 2 3 3 2 3 4 1 4 3 2 3 2 2 2 3 2 3 3 2 3 4 2 3 3 2 3 5 3 4 3 2 4 4 3 5 5 9 6
Lower Facial
Right 32 31 30 29/T 28/S 27/R 26/Q 25/P 24/O 23/N 22/M 21/L 20/K 19 18 17

West Los Angeles College
Health Science Division
Department of Dental Hygiene
Process Evaluation Form

Student Name/Number: Bruna Rett #2429
Date: 11/6/23
Instructor: Bonner
Attempt: Feedback (1st) 2nd 3rd

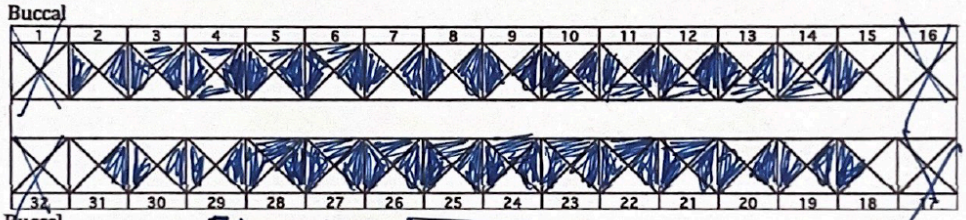
PLAQUE INDEX

Process Evaluation	Pass	No Pass	Notes
- PE form ready / Student name, date, and attempt filled in	✓		
- Discusses rationale, benefit, and outcome of disclosing and plaque index for patient	✓		
- Prepares all necessary armamentarium	✓		
- Neatly discloses patient's teeth	✓		
- Identifies plaque on similar teeth surfaces as instructor	✓		
- Calculates plaque index identified by student and instructor correctly - Student's calculation is within 10% difference of instructor's	✓		2%
- Demonstrates professional behavior and uses dental terminology - Demonstrates professional patient management	✓		
- ***Maintains infection control throughout entire evaluation	✓		

*** = *Indicates critical criteria. If competency is not met, student will receive a summary evaluation grade of "0".*

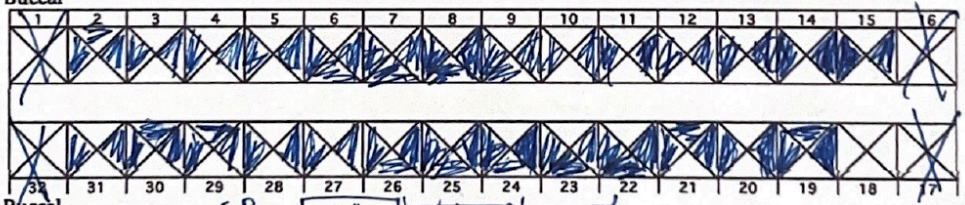
STUDENT FINDINGS

Buccal



Buccal
Show your calculation here: 71 / 28×4 ×100 = 63%

Buccal



Buccal
Show your calculation here: 68 / 28×4 = 61%

4	3	2	1	0
Student demonstrated correct sequence and proper technique without guidance.	Student demonstrated 1-2 minor errors and required minimal guidance.	Student demonstrated 3 minor errors, required guidance but corrected with verbal feedback.	Student required constant guidance, demonstrated 4 errors, difficulty completing task.	Student required constant guidance, unable to complete task. Student made more than 4 errors or ONE critical error.

Instructor Signature

4.0
Grade

West Los Angeles College
Department of Dental Hygiene
Process Grading Form
Periodontal Evaluation

Student Name Bruna Reh #2429
Date 11/6/23
Instructor Bonner

Grade: 4.0

Clinical Assessment	Pass	No Pass	Notes
Discusses the patient's periodontal history	✓		
**Assesses attachment loss; recession, probing, furcation, mobility, mucogingival involvement	✓		
**Assesses disease activity; MBI, BOP's, tissue description (marginal and attached gingival, alveolar mucosa)	✓		
Identifies all local contributory factors (anatomic and traumatic factors)	✓		
Identifies all host response factors and systemic risk factors	✓		

Interpret radiographs	Pass	No Pass	Notes
Correlates radiographic findings with clinical findings	✓		
Identifies the type and extent of bone loss	✓		
Identifies and correlates furcation invasion areas	✓		
Determines crown to root ratio	✓		
Identifies the periodontal ligament and correlates to clinical findings	✓		
Correlates bone appearance with disease activity	✓		
Notes and discusses pathology or other radiographic findings	✓		

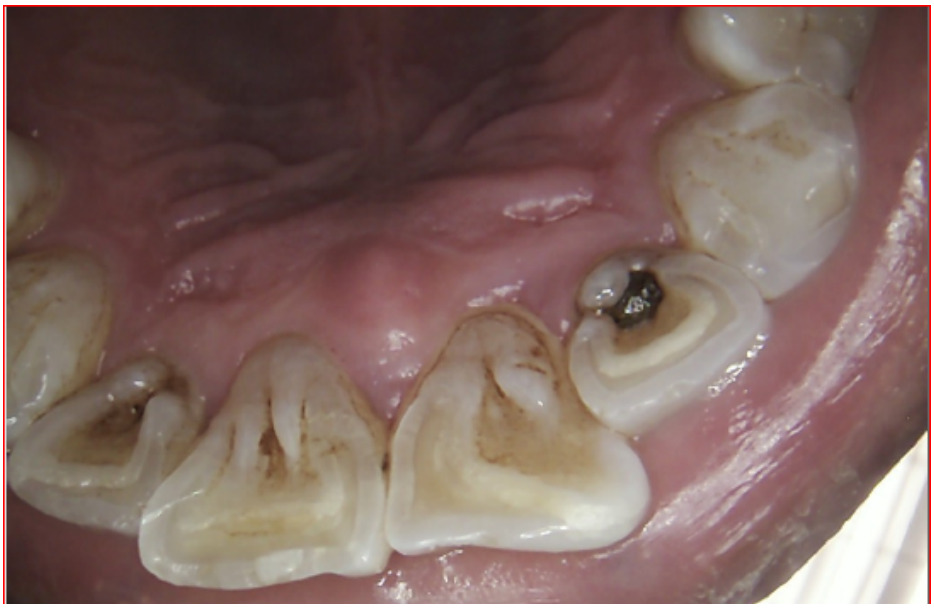
Dental Hygiene Diagnosis and Treatment Plan	Pass	No Pass	Notes
Assesses an appropriates periodontal diagnosis and AAP classification of Stages with Rationales.	✓		
Determines an appropriates treatment plan	✓		
Identifies the need for consultations or referrals	✓		
Determines an appropriates Supportive Periodontal Therapy interval	✓		
Discusses the treatment prognosis	✓		
Uses appropriate professional terminology	✓		
Manages patient care and complete assessment in a timely manner	✓		
**Communicates with patient regarding periodontal status	✓		

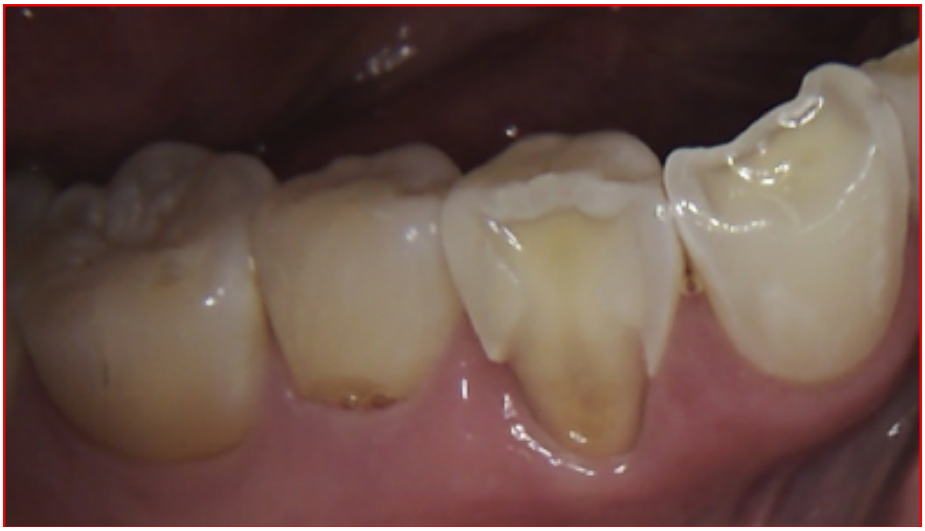
** These asterisks indicate critical criteria. When asterisk criteria are not performed correctly, summary evaluation grade will be "0".

Summary Evaluation

4	3	2	1	0
Student *demonstrated* correct sequence and proper technique without guidance.	**Student demonstrated *1-2 minor errors and* required minimal guidance.**	**Student demonstrated 3 *minor errors required* guidance but corrected with verbal feedback**	**Student required *constant guidance*, demonstrated 4 errors, difficulty completing task**	**Student required *constant guidance*, unable to complete task. Student made more than 4 errors or ONE critical error.**

Faculty Signature: [signature]





Palatal erosion of anterior teeth:

- Dental erosion may cause overtime gradual loss of tooth surface.
- If not treated, acid will continue to dissolve tooth structure until it becomes irreversible.
    - Dietary counseling was provided about minimizing soda drink consumption, which can help reduce erosion.
        - Sodas contain phosphoric and citric acids, which are added for flavor enhancement and preservative, binding to the calcium present in the teeth surface, leading to chemical decomposition of tooth structures.

Abfraction of tooth #29F may have been caused by:

- Parafunctional habits such as asymptomatic bruxism that occurs heavier on the right side of the TMJ.
    - Result of secondary occlusal forces that occurs on an already compromised periodontium.

- OHI was emphasized about the use of soft bristle toothbrush, which the client reported to have used hard bristles most of his life.
- Patient presented with prominent masseter muscles.
    - Which may be indication of strong bite and persistent bruxism overtime.

Severe wearing of enamel on tooth #28F:
- Yellow dentin exposure.
- Able to see thru root canal.
- Tooth #27 adjacent to it also has signs of severe attrition.
    - Dentin exposed coronally.
- May also be a result of secondary occlusal forces that occurs on an already compromised periodontium.

V. Dental History and Caries Examination

1. History of dental exam, treatment, and hygiene visits.

- Last dental exam was in 2015.
    - Possibly received a cleaning as well.
- Patient has had orthodontic treatment when a teenager.

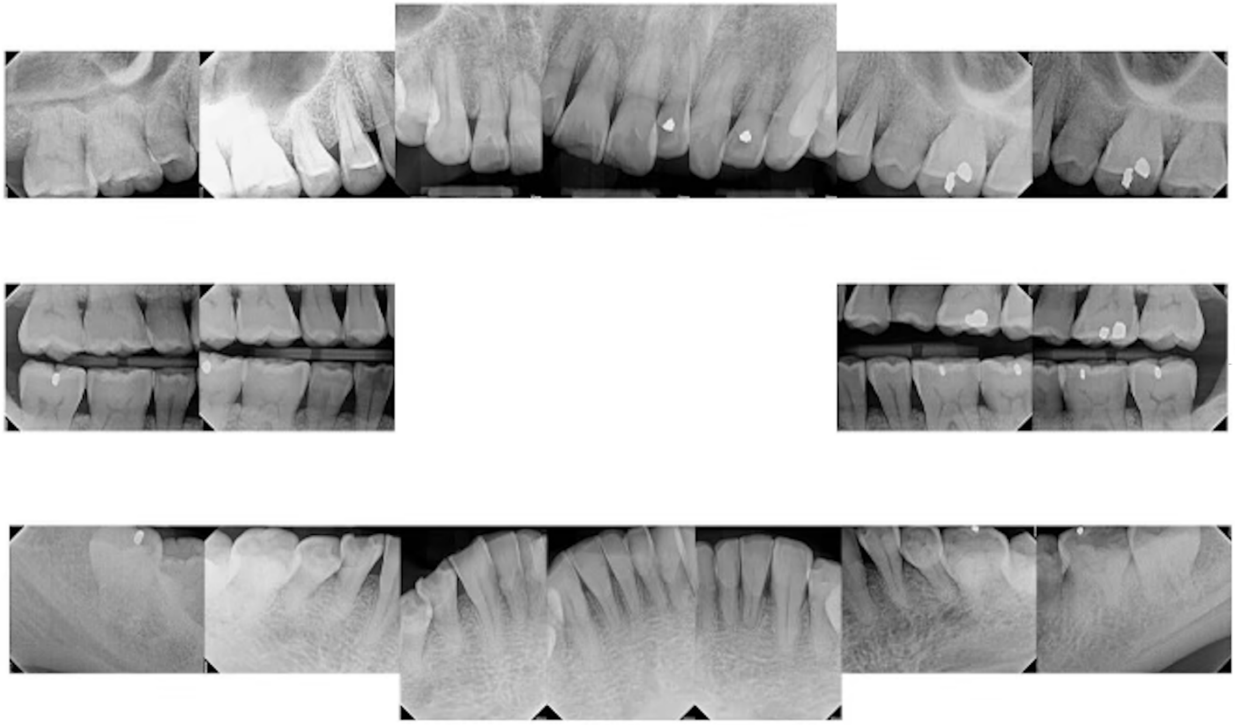
2. Present Status

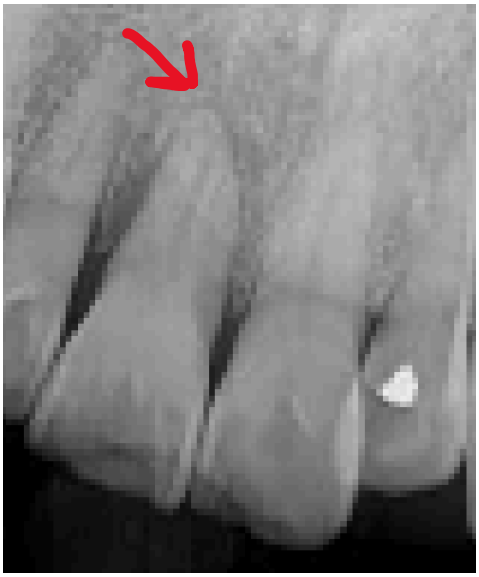
- Patient appears to be in good health and able of tolerating treatment well.

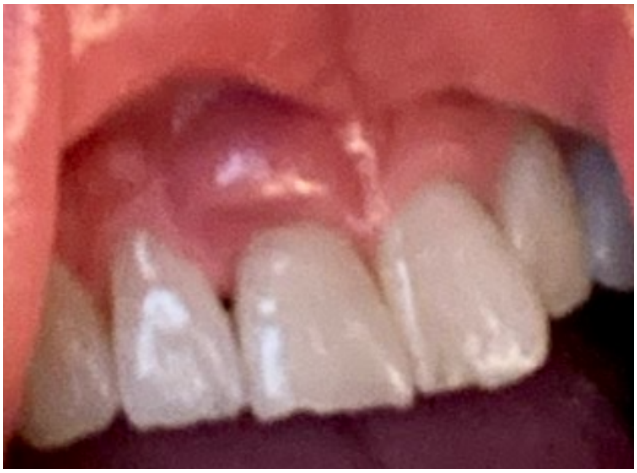
3. Existing caries and quality of restorations (type and location), Caries Index (DMF)

- All restorations seem to be sound and in good condition, with no open margins:
    - Class I amalgam restoration on #10 and #31.
    - Class II amalgam restoration on #18 and #19.
    - Tooth #14 occlusal amalgam.
- Decayed: 0.
- Missing: none due to periodontal disease.
- Filled: 5.
- D+M+F Index = 5
    - This measurement gives the sum of an individual's teeth surfaces.
    - For an adult, DMFT ranges from 0 to 28.
    - It does not indicate the number of teeth that are at risk.
    - Third molars that have been extracted are not counted as missing.

4. Evaluation of radiographs for caries and restorative needs.







PA radiograph of tooth #8 was taken during intake appointment in 6/2023.

A few days prior to the last appointment (URQ) on 12/2023, the client sent me a photo of what it appeared to be an abscess on tooth #8F:

- The entire appointment on 12/07/2023 was spent scaling and root planning, making sure that all local factors such as calculus were removed from all surfaces of tooth #8.
    - To promote tissue healing and hopefully improve the overall situation.
- Client came back on 1/13/2024 to complete URQ scaling and root planning.

VI. Oral Hygiene Evaluation (Pre-Treatment)

1. Patient's skill level.
    a. Patient's manual dexterity was good, as he possessed the ability to make coordinated hand and finger movements when showing his tooth brushing skills during OHI.
        a. It is assumed that the client's skeletal, muscle, and neurologic functions are capable of producing precise movements and reach specific areas of the oral cavity.

2. Patient's knowledge and awareness of dental and periodontal diseases.
    a. Patient was aware about the importance of keeping home hygiene care.
    b. Patient was not fully aware about the clenching and grinding that has been causing damage mainly to the right side of his mandible.
        - Gave information about the importance of using a night guard or other types of treatment to prevent destruction of tooth structures such as abfraction and attrition.
            i. Temporomandibular muscle relaxation and self-massage therapy were also discussed with the client.
    c. Client understands the importance of visiting the dentist twice a year and the dental hygienist more often.
        - He did not know how often he needed to receive his dental cleaning, which we figured it out by the end of the treatment during the re-evaluation appointment.
    d. Patient was not surprised about periodontal condition but was unaware of the fact that there are multiple stages an individual may belong to depending on several factors.

3. Objectives developed during OHI (based on patient's needs and current dental status)
    a. To use soft bristle toothbrush.
        - Patient for most of his life used hard brittles toothbrush because he thought "they clean better".
            i. Further counseling on how to properly adjust was given.
        - Hard bristle toothbrush will further damage the periodontium tissues and contribute to abfraction and recession.
            i. Such scientific research about the importance of using soft bristle was discussed.
    b. To use gentle pressure when brushing and not push the bristles down too hard.
            i. Angle the toothbrush at a 45-degree angle and make small circle movements.
                1. Modified Bass Technique.
            ii. Move to a different quadrant every 30 seconds and brush for a total of 2 minutes, twice a day.
    c. Flossing.
        - Discussed the importance of cleaning interproximal spaces.
            i. They are more prone to the accumulation of plaque, calculus and bacteria.
        - Had the patient pull out floss at an arm length, and floss using c-shape while I held the mirror and gave instructions when appropriate.

4. Oral Hygiene Instruction

West Los Angeles College
Department of Dental Hygiene
Process Grading Form

CCP

Student Name Bruna Dett #2429
Date 1/13/25
Instructor Velasco

Oral Hygiene Instruction - Caries Risk Assessment (CAMBRA) assessment **Grade:** 4.0

Preparation	Pass	No Pass
Explains the benefits of oral hygiene instruction and caries risk assessment to patient	✓	
Establishes appropriate patient/operator positioning	✓	
Uses verbal and nonverbal communication skills to establish patient/provider relationship	✓	

Assessment	Pass	No Pass
Assesses the patient's current oral hygiene status and caries risk by interview, clinical observation and assessment form.	✓	
Uses saliva test if available	~	
Disclose and determine the Plaque Index if applicable. It allows the patient to self-evaluate.	✓	
Asks the patient to demonstrate brushing, flossing, and other oral care techniques	✓	
Assesses patient's oral structure, occlusion, tissue contour, types of the embrasure and manual dexterity	✓	
Determines dental knowledge of the patient	✓	

Interpretation and Plan	Pass	No Pass
Interprets appropriate risk level using a risk assessment form	✓	
Customizes the oral health care plan based on the patient's needs and status	✓	
Selects appropriate preventive aids and products for the risk level (Include options for antimicrobials, fluoride, xylitol, sealants, and frequency of radiographs and dental hygiene services) Electric TB, waterpik (flosspick)	✓	

Implement and Instruction	Pass	No Pass
Discusses disease concepts and engages patient	✓	
Discusses goal and technique to improve oral hygiene status and caries risk status	✓	
Demonstrates appropriate tooth brushing method, flossing method, Interproximal cleaning *method, and other preventive homecare agents*	✓	
Demonstrates by using the tell-show-do approach	✓	
Allows patient to give feedbacks and evaluates the patient's ability to perform recommended instruction	✓	
Professionalism	**Pass**	**No Pass**
Provides instruction and demonstration in a caring manner	✓	
Manages patient care and provides service in a timely manner within 15 minutes.	✓	
Documents patient's oral hygiene status and specific OHI instruction given to patient in patient's record.	✓	
**Appropriate protective wear and infection control throughout the process.	✓	

** These asterisks indicate critical criteria. When asterisk criteria are not performed correctly, summary evaluation grade will "0".

Summary Evaluation

4	3	2	1	0
Sudent demonstrated correct sequence and proper technique without guidance.	**The student demonstrated 1-2 minor errors and required minimal guidance.**	**Student demonstrated 3 minor errors required guidance but corrected with verbal feedback**	**Student required constant guidance, demonstrated 4 errors, difficulty completing the task**	**Student required constant guidance, unable to complete task. Student-made more than 4 errors or ONE critical error.**

Form #13 Oral Hygiene Instruction/CAMBRA 2021

Velasco

VII. Nutritional Analysis

1. Three-day dietary analysis

Food Diary for Ming Lo **(Name of patient) DATE:** 12/5/2023

DAY 1, 2, or 3 (circle one)

- Please list EVERYTHING you eat and drink on 3 consecutive and typical days. (if possible, at least include 1 weekend day)
- Please include extras such as chewing gum, sugar and cream in coffee, or mustard on sandwich.
- Please put down your guess for nutritional facts such as calories, sodium content, carbohydrates. Your dental hygienists will look up and provide the correct information later.

Time (How long?)	Food Eaten or Beverages Drank (List each food item separately)	Amount Eaten or Drank
Example: 6:00 a.m. to 6:15 a.m. (15 minutes)	Egg McMuffin	1
6:00 a.m. to 6:15 a.m. (15 minutes)	Orange Juice	1 large cup (22 oz)
1100am-1115am	Shredded Beef and Tofu	1 large plate
	diet coke	40 oz
200pm-215pm	1 pack trail mix	6 oz
915pm-930pm	Avocado Chicken Salad	1 large plate
915pm - 1200pm	diet coke	44 oz

1. Any Exercises? Please List your exercise of the day

Food Diary for Ming Lo **(Name of patient) DATE:** 12/6/2023

DAY # 1, 2, or 3 (circle one)

- Please list EVERYTHING you eat and drink on 3 consecutive and typical days. (if possible, at least include 1 weekend day)
- Please include extras such as chewing gum, sugar and cream in coffee, or mustard on sandwich.
- Please put down your guess for nutritional facts such as calories, sodium content, carbohydrates. Your dental hygienists will look up and provide the correct information later.

Time (How long?)	Food Eaten or Beverages Drank (List each food item separately)	Amount Eaten or Drank
Example: 6:00 a.m. to 6:15 a.m. (15 minutes)	Egg McMuffin	1
6:00 a.m. to 6:15 a.m. (15 minutes)	Orange Juice	1 large cup (22 oz)
1030am-1040am	3 Chicken Tenders	Large
	Diet Coke	44 Oz
	Apple	1
200pm-215pm	Apple with Peanut Butter	Half Apple
415pm - 430pm	Corn, Peas and Carrots	1.5 cups
900pm-915pm	Chicken Tostada Salad	1 Large Plate

2. Any Exercises? Please List your exercise of the day

Food Diary for Ming Lo **(Name of patient) DATE:** 12/7/2023

DAY # 1, 2, or 3 (circle one)

- Please list EVERYTHING you eat and drink on 3 consecutive and typical days. (if possible, at least include 1 weekend day)
- Please include extras such as chewing gum, sugar and cream in coffee, or mustard on sandwich.
- Please put down your guess for nutritional facts such as calories, sodium content, carbohydrates. Your dental hygienists will look up and provide the correct information later.

Time (How long?)	Food Eaten or Beverages Drank (List each food item separately)	Amount Eaten or Drank
Example: 6:00 a.m. to 6:15 a.m. (15 minutes)	Egg McMuffin	1
6:00 a.m. to 6:15 a.m. (15 minutes)	Orange Juice	1 large cup (22 oz)
1030am-1045am	Chicken Tenders	3 Large Pieces
1045am-1100am	Pizza Pastries with Cream Cheese	2
	Diet Coke	44 oz
400pm-415pm	Apple with Peanut Butter	1
	Diet Coke	30 oz
900pm-930pm	Ceasar Salad with Sausage	1 Serving Salad, 2 Sausages
	Diet Coke	30 oz

3. Any Exercises? Please List your exercise of the day

2. Complete analysis of carbohydrate intake.
    a. A carbohydrate analysis is found by identifying the intake of liquid or solid forms of sugar in the client's 3-day dietary analysis.
        - The grand total number of exposures of sugar in liquid form is 6.
        - The grand total number of exposures of sugar in solid form is 2.
            i. 2 x 20 minutes = a total of 40 minutes of average daily acid production is below 5.5 pH acid exposure.
            ii. The oral cavity has been exposed to carbohydrates for 40 minutes in 3 days, therefore placing the client at a high caries risk.
    b. The recommendation is to decrease the solid carbohydrate intake such as diet soda and drink more water instead.
        - Water is a healthier choice not only for the oral cavity but for the entire body.
    c. The goal is to place the patient's teeth at a low caries risk.
    d. The nutritional findings related to this patient does not currently have any issues linked to any systemic diseases yet.
        - However, if patient is not able to reduce the amount of daily calories intake, the risk of cardiovascular or systemic diseases will be increased.
    e. A person's daily nutrition is directly correlated to periodontal disease.
        - If the patient keeps on ingesting acidic foods, added sugars, saturated fats and sodium, it leads to an increased risk of periodontal disease.

3. Nutritional focus and recommendations, including rationale.
    a. The 5 food groups recommended by MyPlate plan is:
        - 1 ½ cups of fruits.
        - 2 ½ cups of vegetables.
        - 6 oz of grains.
        - 5 oz of protein.
        - 3 cups of dairy.
    b. The limit should be:
        - <45g a day of added sugars.
        - <20g a day of saturated fat.
        - <2,300 mg a day of sodium.
    c. It is recommended at least 2 ½ hours per week of physical activities.
        - Client did not report having any type of physical exercise during the 3-day dietary analysis.
    d. Patient fails to meet recommendations when it comes to fruits and vegetables.
    e. Patient exceeds daily recommendations when it comes to added sugars and saturated fat.
    f. Nutritional counseling regarding eating more fruits, vegetables and exercise more was discussed and implemented.

VIII. Fluoride Analysis

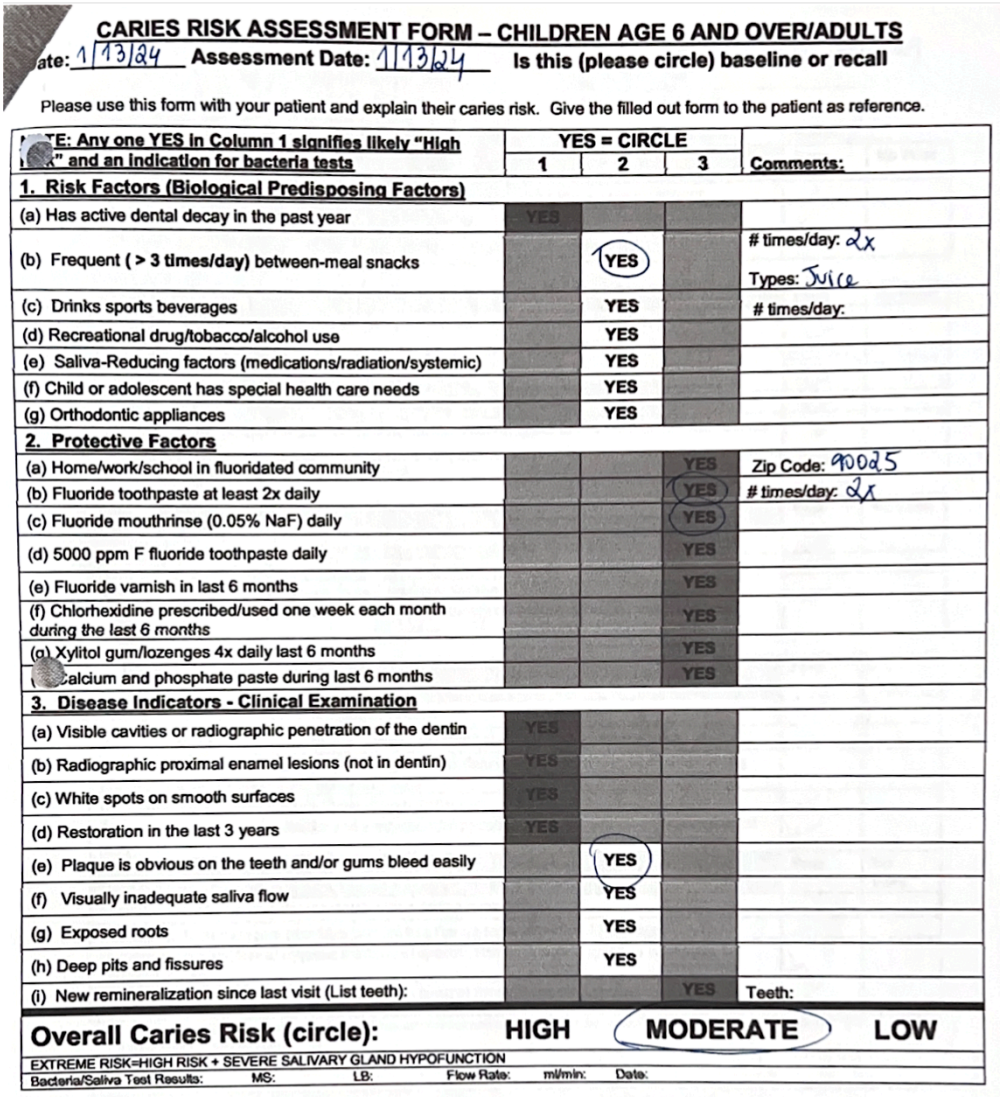
1. Current usage of fluoride.
    a. The fluoride concentration of water in the patient's city according to Westlake Village is 0.8mg/L.

- Moderate drinking water fluoride level.
    - Fluoride. California Water Service

2. Identification and rationale of fluoride focus.
    a. Focusing in fluoride treatment is one of the main goals of periodontal therapy.
        - Client is at moderate risk for caries.
    b. Fluoride products promote cosmetic, hygiene and therapeutic factors.
    c. It helps to remove food debris, dental plaque and extrinsic stains.
    d. It prevents the formation of supragingival calculus.
    e. It reduces the risks for caries and sensitivity.
    f. Fluoride benefits focus on the remineralization process.
        - Fluoride acts as a catalyst that speeds up the enamel strengthening mechanism of the teeth.

IX. Caries Risk Assessment

1. CAMBRA Assessment (Must be graded)



CARIES RISK ASSESSMENT FORM – CHILDREN AGE 6 AND OVER/ADULTS

Date: 1/13/24 **Assessment Date:** 1/13/24 **Is this (please circle) baseline or recall**

Please use this form with your patient and explain their caries risk. Give the filled out form to the patient as reference.

NOTE: Any one YES in Column 1 signifies likely "High Risk" and an indication for bacteria tests	YES = CIRCLE 1	2	3	Comments:
1. Risk Factors (Biological Predisposing Factors)				
(a) Has active dental decay in the past year	YES			
(b) Frequent (> 3 times/day) between-meal snacks		YES		# times/day: 2x Types: Juice
(c) Drinks sports beverages		YES		# times/day:
(d) Recreational drug/tobacco/alcohol use		YES		
(e) Saliva-Reducing factors (medications/radiation/systemic)		YES		
(f) Child or adolescent has special health care needs		YES		
(g) Orthodontic appliances		YES		
2. Protective Factors				
(a) Home/work/school in fluoridated community			YES	Zip Code: 90025
(b) Fluoride toothpaste at least 2x daily			YES	# times/day: 2x
(c) Fluoride mouthrinse (0.05% NaF) daily			YES	
(d) 5000 ppm F fluoride toothpaste daily			YES	
(e) Fluoride varnish in last 6 months			YES	
(f) Chlorhexidine prescribed/used one week each month during the last 6 months			YES	
(g) Xylitol gum/lozenges 4x daily last 6 months			YES	
(h) Calcium and phosphate paste during last 6 months			YES	
3. Disease Indicators - Clinical Examination				
(a) Visible cavities or radiographic penetration of the dentin	YES			
(b) Radiographic proximal enamel lesions (not in dentin)	YES			
(c) White spots on smooth surfaces	YES			
(d) Restoration in the last 3 years	YES			
(e) Plaque is obvious on the teeth and/or gums bleed easily		YES		
(f) Visually inadequate saliva flow		YES		
(g) Exposed roots		YES		
(h) Deep pits and fissures		YES		
(i) New remineralization since last visit (List teeth):			YES	Teeth:

Overall Caries Risk (circle): HIGH MODERATE LOW

EXTREME RISK=HIGH RISK + SEVERE SALIVARY GLAND HYPOFUNCTION

Bacteria/Saliva Test Results: MS: LB: Flow Rate: ml/min: Date:

2. Caries risk prognosis (discuss reasoning).
    a. The prognosis is fair, given the fact that the patient is motivated to improve health and apply healthy habits to his daily life.
        - But if client does not significantly decrease the amount of diet soda consumption, the prognosis can become questionable.
    b. If home care is followed as discussed, the client could have risk factors such as visible plaque reduced.

3. Provide recommendations based on patient's assessment.
    a. Brushing twice daily with soft bristle toothbrush along with fluoridated toothpaste and mouthwash.
    b. Soft bristles are the least abrasive, hence preventing damage to the tooth surfaces when compared with hard bristles.
    c. Fluoridated products aids in stain removal, prevents dental plaque biofilm accumulation as well as the formation of supragingival calculus

4. Provide education and written information on caries control and management.
    a. Tooth decay is caused by certain types of anaerobic bacteria that do not need oxygen to survive.
        i. Dental plaque occurs when these bacteria attach themselves to the teeth, causing damage.
    b. Bacteria feeds off of what we eat, especially starch and sugars.
        i. This process happens very fast, usually within 5 minutes after eating and drinking, the bacteria starts to produce acid as a by-product of the bacteria's own digestion.
        ii. These acids are harmful and can penetrate the enamel and dissolve minerals such as calcium and phosphate,
    c. Saliva has the role of repairing the damage that is caused by this acidic environment.
    d. It neutralizes acids as well as providing fluoride and minerals that can replace the loss caused by bacteria.
    e. The start of tooth decay mainly includes frequently snacking unhealthy foods and drinks.

X. Dental Hygiene Treatment Plan

1. Recommendation of sealant application (provide rational)
    a. Patient may benefit from sealant treatment on teeth #31 and #30.
        - Other molars and premolars already have existing sealants.
    b. Sealant is recommended for deep pits and fissures in posterior teeth as a way to prevent caries.
        - It seals the margins of the crevices in the occlusal surface of teeth, acting as a protective barrier against bacteria, plaque and debris.

2. Consideration of possible implications of systemic conditions.

a. Hepatitis B may influence periodontitis since this condition affects liver by eliciting inflammation response.

3. Consideration of possible implications of local factors/conditions
   a. Implications occurring in the periodontium is related to parafunctional habits that exceeds the capacity of the periodontium.
      a. Secondary occlusal trauma on an already compromised periodontium tissues.

4. Possible implications of medications on oral health.
   a. Medication for gout may cause xerostomia.
      - However, patient has excellent salivary flow.

5. Physical limitation or disability
   a. None.

6. Referral to a DDS and other medical discipline.
   a. Referral to an endodontic specialist was given by Dr. David Nguyen.
      - His diagnose was a necrotic tooth #8 with the possibility or a root canal in order to save the tooth.
   b. Patient seems interested in taking care of a front tooth, since it has an important role in smile.

7. Rationale for the treatment plan and patient needs (provide reasoning)
   a. Client has deep pockets in some areas (>6mmm).
   b. WLAC Calculus code is 4M with localized 5M on the mandibular anterior.
   c. Patient has limited access to dental care in the past decade and it interested in continuing his care with WLAC faculty and students.
   d. Scaling and root planning will benefit his condition, or at least arrest his Periodontal Classification Stage III, grade B.

8. Goals and Objectives of the dental hygiene treatment
   a. The goal is to provide the patient with the tools and knowledge to continue to improve his home care oral hygiene routine.
   b. The objectives are to make sure that the patient continues motivated and does not skip dental visits for another 10 years ever again.

9. Treatment plan: Complete Dental Hygiene Diagnosis and Care Plan.
   a. The following was based on keeping half mouth cleaned so the patient can feel the difference:
      i. Appointment 1: LLQ 11/20/2023.
      ii. Appointment 2: ULQ 11/27/2023.
      iii. Appointment 3: LRQ 12/4/2024
      iv. Appointment 4: URQ 12/7/2024.
      v. Appointment 5: Continued URQ 1/13/2024.
      vi. Appointment 6: Re-evaluation 2/24/2024.

11. Vitals at each appointment.
    a. Appointment 1:
        i. BP: 120/73.
        ii. P: 73.
        iii. Respiration: 14.
        iv. SpO2: 98%.
        v. Temperature: 97.6.
    b. Appointment 2:
        i. BP: 115/70.
        ii. P: 75.
        iii. Respiration: 14.
        iv. SpO2: 99%.
        v. Temperature: 97.0
    c. Appointment 3:
        i. BP: 117/73.
        ii. P: 78.
        iii. Respiration: 14.
        iv. SpO2: 98%.
        v. Temperature: 98.1
    d. Appointment 4:
        i. BP: 112/70.
        ii. P: 81.
        iii. Respiration: 14.
        iv. Temperature: 96.7
    e. Appointment 5:
        i. BP: 105/70.
        ii. P: 65.
        iii. Respiration: 14.
        iv. Temperature: 97.0
    f. Appointment 6:
        i. BP: 105/70.
        ii. P: 63.
        iii. Respiration: 14.
        iv. Temperature: 97.3

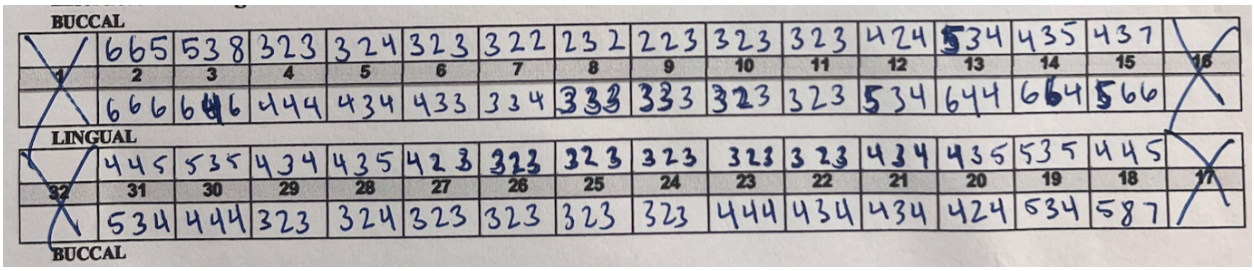
12. Oral hygiene instruction at each appointment (be specific, include rationale)
    a. Provided oral hygiene instructions on:
        i. Brush using modified bass technique.
        ii. Floss using c-shape method.
        iii. Use fluoridated products such as toothpaste and mouth wash to prevent caries.

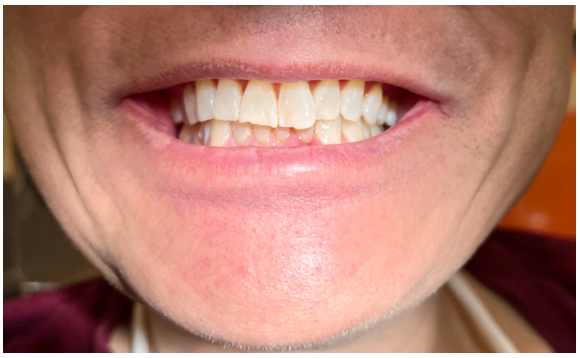
14. Rationale for periodontal maintenance appointment interval and follow up visits
    a. Re-care routine should be 2-3 months for periodontal maintenance.
    b. This has been discussed and decided between the dental hygiene care team.

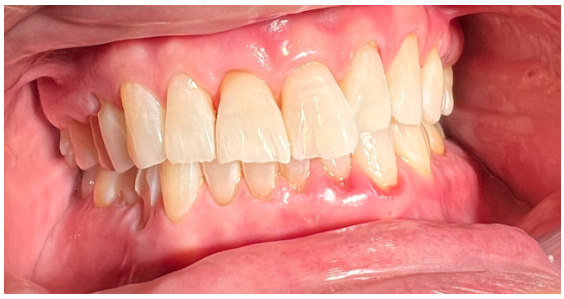
c. Patient was opened to the suggestions.
d. Allowed patient to provide feedback and answered his questions.
    a. Listened to his concerns.

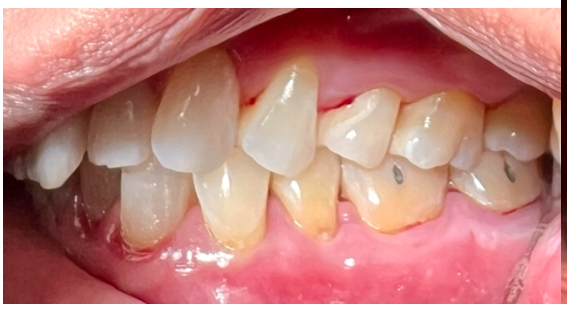
XI. Re-Evaluation & Post Instructions Status

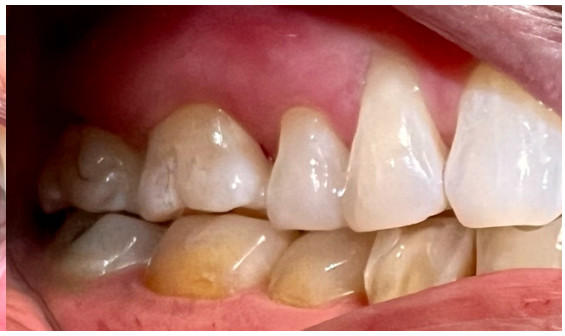
1. Periodontal Re-Evaluation

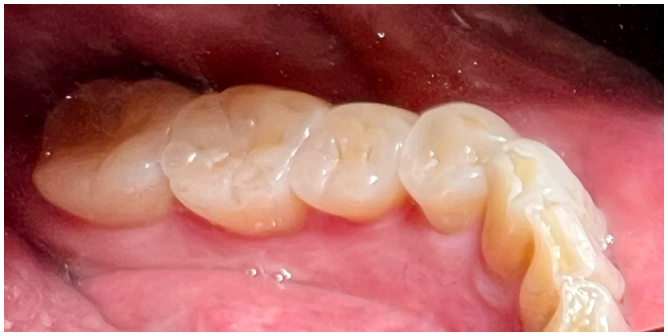


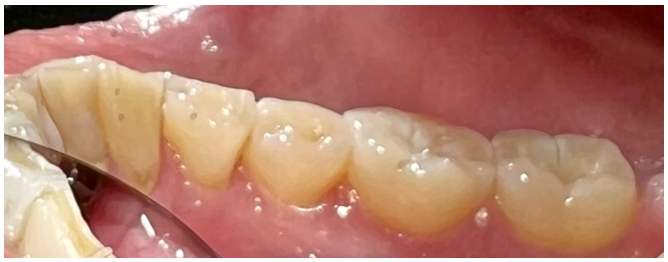


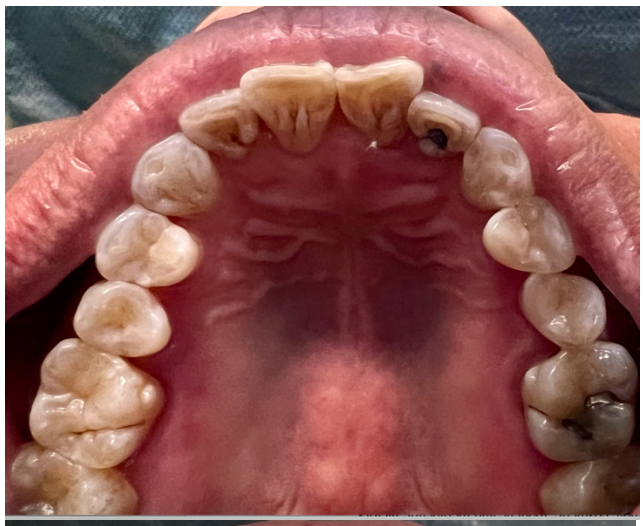


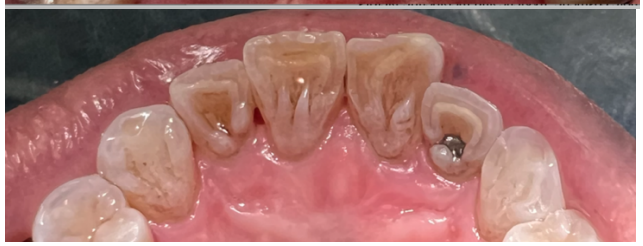


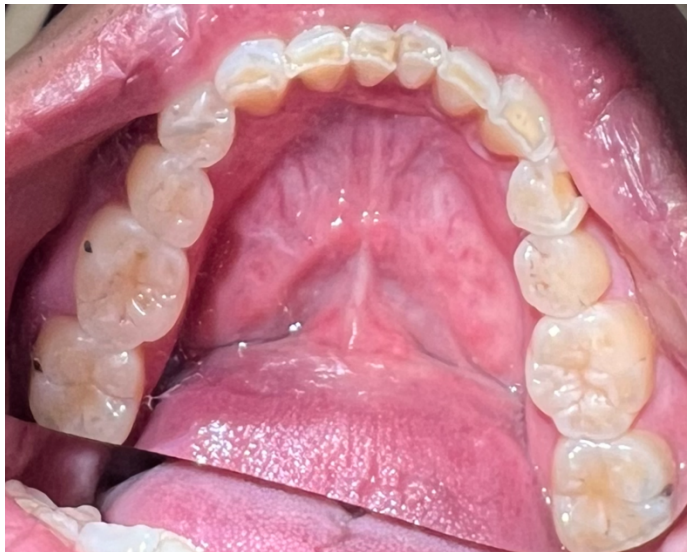


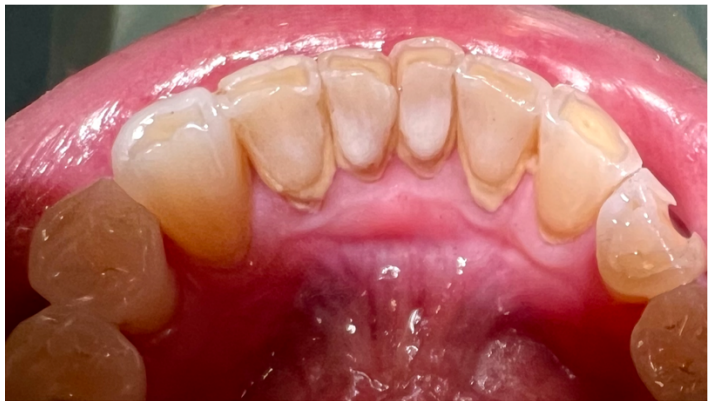


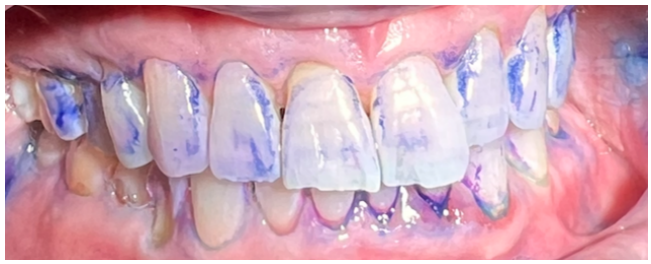


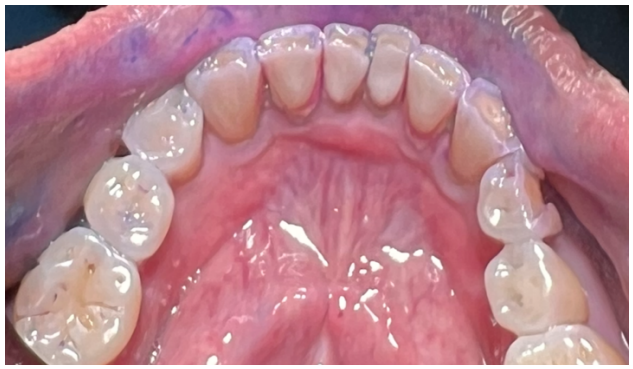












3. Patient compliance with recommended home care (oral hygiene/prevention instructions/nutrition/referral).
    a. Plaque Index only improved by 3%.
        a. Calculated to be at 63% pre-treatment.
        b. Calculated to be at 60% post-treatment.
        c. Needs to work more on proper OHI and home care.

XII. Discussions

The overall outcome of this project was not as expected, for each time the patient came back for an appointment, it was noticed that calculus and plaque had already formed. The client's understandings on nutrition is acceptable, for he agrees that he should reduce diet coke consumption. Moreover, carbonated drinks contribute to erosion, consequently placing the teeth at a higher risk for caries. The patient had a mirror and was able to visualize the erosion occurring on the palatal surface of maxillary anterior teeth, as well as the attrition, abfraction and dentin exposure present on the LRQ.

In regard to patient participation, the client seemed to be motivated to become a permanent and frequent patient of the WLAC Dental Clinic, and he expresses the desire to continue to be treated by dental hygiene students. He got to know some of the instructors well, and had a great experience talking to them. Patient was always on time, stayed curious throughout the appointments by actively asking questions, follow ups and exited to meet his goals and objectives. However, oral hygiene instructions must be emphasized in every visit since his plaque index could have been better.

The goals and objectives were mostly met, in which was to restore his oral health and improve quality of life. The patient said that he would try to follow-up with an endodontic specialist regarding the necrosis diagnosis he received from Dr. David Nguyen for tooth #9. It is interesting to see in real life the effects of parafunctional habits on an already compromised periodontium, aka secondary occlusal trauma. Parafunctional habits and abfraction.

The final prognosis for this patient would be poor since the patient appears to have difficulty maintaining good oral hygiene home care. Due to plaque index post-treatment having very little improvement, poor diet is also a great contributor to plaque formation by creating an

acidic environment in the saliva that benefits bacteria that causes calculus and cavities. Focusing on OHI in subsequent appointments would be the ideal.

XIII. Conclusions (Not outline format, research writing format at least 2 pages)

Hepatitis B is a highly infectious virus that damages the liver, and it can be transmitted through contaminated blood or bodily fluids. For this reason, hepatitis B is a major global health concern that can cause serious inflammation of the liver, which can fatally evolve into cirrhosis. Moreover, studies have found that the dental and periodontal status of patients with hepatitis B could have significant increase risk for periodontal disease (Gheorghe et. al. , 2022). Since inflammation can also be signs of periodontal disease, having hepatitis B in conjunction to periodontitis suggests that an integration between medical doctors and dental professionals should be proposed.

A study done in the US population, published by the Journal of Dental Research, says that nonalcoholic fatty liver disease such as hepatitis B were significantly associated with tooth loss and moderate to severe periodontitis (Weintraub et. al., 2019). This research supports previous studies about emphasizing the importance of understanding the connections between diseases that occur in the oral cavity and other systemic diseases that affects one or more organs such as Hepatitis B. The seriousness of making sure that our clients are in good overall health, especially if they're carriers or certain systemic conditions, is the key to providing safe and comprehensive care that sees the individual as a whole.

When it comes to parafunctional habits, the patient already has a systemic disease that as mentioned above, may negatively impact their oral health condition and prognosis. The enamel and dental lesions present on tooth #29F tend to be progressive, with damage to the soft tissues of the tooth very close to entering the dental pulp. According to the Current Health Sciences Journal, abfraction lesions in combination with other factors such as parafunctional habits, may lead to loss of protection of the natural tissues (Rusu Olaru, 2019).

In order to prevent abfraction lesions from progressing even further, early diagnosis and recognition of the trauma must be addressed. It is common to have secondary occlusal trauma on an already compromised periodontium caused by forces that exceeds the maximum capacity of the oral tissues and structures. But making sure that the client is healthy and esthetically confident is part of the dental hygiene plan, which aims to provide care that is measurable, achievable and realistic. The use of aids such as mouth guards, splinters, therapy and others may help stop or at least arrest any oral conditions that is aggressive to the oral cavity, head, neck and TMJ issues.

Ultimately, making sure that the patient is receiving appropriate care in a timely manner is the ultimate goal that healthcare providers should aspire to achieve. Regardless of the condition but especially if an individual has any type of immune compromised diseases that affects oral health, the client has the right to receive care in order to prevent further damage. It is part of the ethics of a professional to be unbiased while providing care to patient with hepatitis B. Everyone deserves respect and the autonomy to make informed decisions about their own personal health.

XIV. Summary

My learning experience with this particular patient has been extremely humbling. It was frustrating to see that all of the calculus has already built up again, leading me to believe that the client has fast healing, poor home hygiene care, or a mixture of both. I thought that emphasizing OHI in each appointment and providing him with an Oral B toothbrush would improve the situation significantly, but he has not been to the dentist in about 10 years, so I'm assuming that it will take a few more months to completely arrest his periodontal condition. I believe this patient should be treated on a periodontal office, but his socioeconomic status does not allow him to pursue this type of care. I do plan on seeing this patient one more time before I graduate, and it will be interesting to see if more progress will be achieved.

XV. References

Fluoride. California Water Service. (2021, September 24). https://www.calwater.com/waterquality/fluoride/

Gheorghe DN, Bennardo F, Popescu DM, Nicolae FM, Ionele CM, Boldeanu MV, Camen A, Rogoveanu I, Surlin P. Oral and Periodontal Implications of Hepatitis Type B and D. Current State of Knowledge and Future Perspectives. J Pers Med. 2022 Sep 26;12(10):1580. doi: 10.3390/jpm12101580. PMID: 36294719; PMCID: PMC9604856.

Learn how to eat healthy with myplate. MyPlate. (n.d.). https://www.myplate.gov/

Rusu Olaru A, Popescu MR, Dragomir LP, Rauten AM. Clinical Study on Abfraction Lesions in Occlusal Dysfunction. Curr Health Sci J. 2019 Oct-Dec;45(4):390-397. doi: 10.12865/CHSJ.45.04.07. Epub 2019 Dec 30. PMID: 32110441; PMCID: PMC7014982.
Weintraub, J. A., Lopez Mitnik, G., & Dye, B. A. (2019). Oral Diseases Associated with Nonalcoholic Fatty Liver Disease in the United States. *Journal of Dental Research*, *98*(11), 1219–1226. https://doi.org/10.1177/0022034519866442

XVI. Appendices

1. Copy of patient's consent form

West Los Angeles College
DENTAL HYGIENE PROGRAM

CLIENT CONSENT FORM

Name of Clinical Facility West Los Angeles College

Client Name [redacted]

I hereby authorize the dental hygiene student named below to provide preventive and dental hygiene service. I was informed about the protocol and clinical development of the Comprehensive Care Project, as well as its benefits and possible risks. I agree to be enrolled in the project. I was able to ask questions about the project and my participation to the student and instructors. I agree to have my dental chart to be reviewed, and have copies made of it, for the sole purpose of the project. I also agree and understand that the project requires multiple appointments.

PHOTO RELEASE AGREEMENT

I hereby consent to and authorize the use and reproduction by student, or anyone authorized by the West Los Angeles College Department of Dental Hygiene, of any and all photographs that have been taken of me solely for educational purposes, without financial compensation to me.

Client Name: Ming Lo Signed: [redacted]

I have informed my pa[redacted]equirements and I will conduct th[redacted] the parameters of the Standards of Clinical Dental Hygiene Practice.

Student Name: Bruna Rett Signed: Bruna Rett

INSTRUCTOR'S APPROVAL

I have reviewed all the assessment records and approved to have this patient enrolled in the Comprehensive Care Project.

WLAC Instructor's Name: Mariah Bonner

Instructor Signature: [redacted] Date: 11-6-23

2. Copy of Human Needs and Treatment Plan Form

DENTAL HYGIENE DIAGNOSIS AND CARE PLAN

IV. Dental Hygiene Human Needs Assessment

Dental Hygiene Human Needs	Patient's Needs	Goal and Care Plan	Evaluation
Chief Complaint			
(2) (1) Protection from health risks, anxiety, fear and stress			
(3) Wholesome Facial Image			
(5) Skin and Mucous Membrane Integrity of Head and Neck	BOP Probing depths ≥ 6 mm.	Provide adjunct therapy such as Arestin if SRP alone is not able to resolve deep pockets.	
(4) Biologically Sound and functional dentition	Signs of disease on tooth #8. Erosion. Tooth #28 has signs of trauma.	To remove any locals factors that may complicate healing.	
(7) Conceptualization and problem solving			
(6) Freedom from Head and Neck Pain			
(8) Responsibility for Oral health	Inadequate plaque control and self-monitoring of health status. No dental exams within past 2 years.	Emphasize OHI and the importance of it. Make sure client comes back for perio maintanence q-3 months.	

V. Appointment Plan:

Appointment	Date/Interval	Procedures:
1 LLQ	11/20/23	SRP with Lido
2 ULQ	11/27/23	SRP with Lido
3 LRQ	12/04/23	SRP with Lido
4 URQ	1/8/24	SRP with Lido
5 Re-eval	2/6/24	SRP with Lido
6		
Maintenance	3 months	

Signatures: Patient [redacted] Date 11/6/2023 Faculty [signature] Date 11-6-23 Student Bruna Petit

Dental Hygiene Diagnosis and Tx Plan – Kamibayashi - for Comprehensive Care Project

3. Copy of the medical history form

Health History Form

E-mail: [redacted] Today's Date: 6/20/2023

As required by law, our office adheres to written policies and procedures to protect the privacy of information about you that we create, receive, or maintain. Your answers are for our records and will be kept confidential subject to applicable laws. You will be asked some questions about your responses to this questionnaire and there may be additional questions concerning your health. This information is vital to allow us to provide appropriate care for you. This office does not use this information to discriminate.

Name: Last [redacted] First [redacted] Middle

Pronouns: *Circle all that apply* She/her/hers He/him/his They/them/theirs Other: Please specify

Phone: *Include area code* (310) [redacted] Business Phone: *Include area code* ( )

Address (Mailing address): [redacted] City: LA State: CA Zip: 90025

Occupation: Actor Height: 5'8" Weight: 220 lbs Date of Birth: 10/30/1964

Current gender identity: Please circle

Male Female Transgender male/Transman/FTM Transgender Female/Transwomen/MTF Additional category: _____ Decline to Answer

Emergency Contact: [redacted] Relationship: [redacted] Phone number: *Include area code* [redacted]

If you are completing this form for another person, what is your relationship to that person?

Your name: Relationship:

Do you have any of the following disease or problems? (Check DK if you Don't know)	Yes	No	DK
Active Tuberculosis	☐	X	☐
Persistent cough greater than 3 weeks	☐	X	☐
Cough that produces blood	☐	X	☐
Been exposed to anyone with tuberculosis	☐	X	☐

If you answered yes to any of the above items, please stop and return to the receptionist.

Dental Information

What is the reason for your dental visit today? cleaning

Date of your last dental exam 2015?

What was done at your last dental appointment? cleaning

Date of last dental x-rays: 2015?

	Yes	No	DK
Are you currently experiencing dental pain or discomfort?	☐	X	☐
Do your gums bleed when you brush or floss?	☐	X	☐
Are your teeth sensitive to cold, hot, sweets, or pressure?	☐	X	☐
Is your mouth dry?	☐	X	☐
Have you had any periodontal (gum) treatments?	☐	X	☐
Have you ever had orthodontic (braces) treatment?	✓	☐	☐
Have you had any problems associated with dental treatment?	☐	✓	☐
Is your home water supply fluoridated?	☐	☐	✓

	Y	N	DK
Do you have any earaches or neck pain?	☐	X	☐
Do you ever have discomfort in the jaw?	☐	X	☐
Do you grind or brux your teeth?	☐	X	☐
Do you have sore or ulcers in your mouth?	☐	X	☐
Do you wear dentures or partials?	☐	X	☐
Do you participate in active recreational activities?	☐	X	☐
Have you ever had a serious injury to your head or mouth?	☐	X	☐

Medical Information

Are you under the care of a physician now? yes

Physician Name: Dr. Norman Silverman Phone: *Include area code* 310 996-9355

Address/city/ state/ zip: 1950 Sawtelle Blvd. #130, LA, CA 90025

Has there been any change in your general health within the past year? Hip replacement necessary

If yes, what conditions are being treated? occasional gout, ED, hip pain

Date of last physical exam: 9/29/2022

Have you had a serious illness, operation, or been hospitalized in the last 5 years? no

If yes, please explain: _____

Are you taking or have you recently taken any prescription or over the counter medication? indomethacin (gout) 25mg occasional

If so, please list all, including vitamins, natural or herbal preparations and/or diet supplements: Propecia (1mg once daily)

Mental Health and Personal Safety

Is stress a problem for you? no

Do you feel depressed? no

Do you panic when stressed no

Do you have any problems with eating or your appetite? no

Do you cry frequently? no

Have you ever seriously thought about hurting yourself? no

Do you have trouble sleeping? no

Have you ever been to counselor? no

Do you live alone? yes

Do you have vision or hearing loss? ~~no~~ slight vision loss, no hearing loss

Physical and/or mental abuse have also become major public health issues in this country. This often takes the form of verbally threatening beh... or actual physical or sexual abuse. Would you like to discuss this issue with your provider? no

Medical Information

Please mark (X) your response to indicate if you have not had any of the following diseases or problems

	Yes	No	DK
Do you wear contact lenses?		✓	
Joint replacement. Have you had an orthopedic total joint replacement (hip, knew, finger)? no			
If yes, Date: _____ If yes, any complications? _____			
Are you taking or scheduled to begin taking an antiresorptive agent (like Fosamax, Actonel, Atelvia, Boniva, Reclast, Prolia) for osteoporosis or Paget's disease?		✓	
Since 2001, were you treated or are you presently scheduled to begin treatment with an antiresorptive agent (like Aredia, Zometa, XGEVA) for bone pain, hypercalcemia or skeletal complications resulting from Paget's disease, multiple myeloma or metastatic cancers?		✓	
Date Treatment began: _____			

Allergies. Are you allergic to or have had a reaction to:
To all yes responses, specify type of reaction.

Local anesthetics		✓	
Aspirin		✓	
Penicillin or other antibiotics		✓	
Barbiturates, sedatives, or sleeping pills		✓	
Sulfa drugs		✓	
Codeine or other narcotics		✓	

Please mark (X) your response to indicate if you have not had any of the following diseases or problems

	Yes	No	DK
Artificial (prosthetic) heart valve		✓	
Previous infective endocarditis		✓	
Damaged valves in transplanted heart		✓	
Congenital heart disease (CHD)			
Unrepaired, cyanotic CHD		✓	
Repaired (completely) in last 6 months		✓	
Repaired CHD with residual defects		✓	

Except for the conditions listed above, antibiotic prophylaxis is no longer recommended for any other form of CHD

	Yes	No	DK		Yes	No	DK
Cardiovascular disease	✓			Mitral valve prolapse		✓	
Angina	✓			Pacemaker		✓	
Arteriosclerosis	✓			Rheumatic fever		✓	
Congestive heart failure	✓			Rheumatic heart disease		✓	
Damaged heart valves	✓			Abnormal bleeding		✓	
Heart attack	✓			Anemia		✓	
Heart murmur	✓			Blood transfusion		✓	
Low blood pressure	✓			Hemophilia		✓	
High blood pressure	✓			AIDS or HIV infection		✓	
Other congenital heart defects	✓			Arthritis		✓	

	Yes	No	DK
Do you use controlled substances (drugs)?		✓	
Do you use tobacco (smoking, snuff, chew, bidis)?		✓	
If so, how interested are you in stopping?			
Circle one: VERY / SOMEWHAT / NOT INTERESTED			
Do you drink alcoholic beverages?		✓	
If yes, how much alcohol did you drink in the last 24 hours? 0			
If yes, how much do you typically drink in a week? _____			

WOMEN ONLY Are you:

Pregnant?			
Number of weeks: _____			
Taking birth control pills or hormonal replacement?			
Nursing?			

Allergies. Are you allergic to or have had a reaction to:
To all yes responses, specify type of reaction.

Metals		✓	
Latex(rubber)		✓	
Iodine		✓	
Hay fever/seasonal		✓	
Animals		✓	
Food		✓	
Other		✓	

Please mark (X) your response to indicate if you have not had any of the following diseases or problems

	Yes	No	DK		Yes	No	DK
Autoimmune disease		✓		Glaucoma		✓	
Rheumatoid arthritis		✓		Hepatitis, jaundice or liver disease		✓	
Systemic lupus erythematosus		✓		Epilepsy		✓	
Asthma		✓		Fainting spells or seizures		✓	
Bronchitis				Neurological disorders		✓	
Emphysema	✓			If yes, specify			
Sinus trouble	✓			Sleep disorder		✓	
Tuberculosis		✓		Do you snore?			
Cancer/Chemotherapy/ Radiation Treatment		✓		Mental health disorders ,Specify :		✓	
Chest pain upon exertion		✓		Recurrent Infections		✓	
Chronic pain		✓		Type of infection		✓	
Diabetes Type I or II		✓		Kidney problems		✓	
Eating disorder		✓		Night sweats		✓	
Malnutrition		✓		Osteoporosis	✓		
Gastrointestinal disease		✓		Persistent swollen glands in neck		✓	
G.E. Reflux/ persistent heartburn		✓					
Ulcers	✓			Severe headaches migraines		✓	
Thyroid problems	✓			Severe or rapid Weight loss		✓	
Stroke	✓			STDs		✓	
				Excessive urination		✓	

Has a physician or previous dentist recommended that you take antibiotics prior to your dental treatment? ✓

Name of physician or dentist making recommendation: Phone number include area code ( )

Do you have any disease, condition, or problem not listed above that you think I should know about? ✓

Please, explain: possible hepatitis B ~~carrier~~ carrier, high cholesterol

NOTE: Both doctor and patient are encouraged to discuss any and all relevant patient health issues prior to treatment.

I certify that I have read and understand the above and that the information given on this form is accurate. I understand the importance of a truthful health history and that my dentist and his/her stuff will rely on this information for treating me. I acknowledge that my questions, if any, about inquires set forth above have been answered to my satisfaction. I will not hold my dentist, or any other member of his/her staff, responsible for any action they take or do not take because of errors or omissions that I may have made in the completion of this form.

Signature of Patient/Legal Guardian: [redacted]

Signature of Dentist 12/14/17

FOR COMPLETION BY DENTIST

Comments:

4. Copy of the oral hygiene progress form

West Los Angeles College
Health Science Division
Department of Dental Hygiene
Process Evaluation Form

Student Name/Number: Bruna Rett #2429
Date: 11/6/23
Instructor: Bonner
Attempt: Feedback (1st) 2nd 3rd

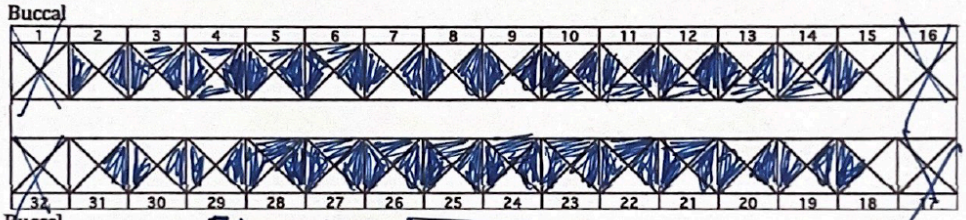
PLAQUE INDEX

Process Evaluation	Pass	No Pass	Notes
- PE form ready / Student name, date, and attempt filled in	✓		
- Discusses rationale, benefit, and outcome of disclosing and plaque index for patient	✓		
- Prepares all necessary armamentarium	✓		
- Neatly discloses patient's teeth	✓		
- Identifies plaque on similar teeth surfaces as instructor	✓		
- Calculates plaque index identified by student and instructor correctly - Student's calculation is within 10% difference of instructor's	✓		2%
- Demonstrates professional behavior and uses dental terminology - Demonstrates professional patient management	✓		
- ***Maintains infection control throughout entire evaluation	✓		

*** = *Indicates critical criteria. If competency is not met, student will receive a summary evaluation grade of "0".*

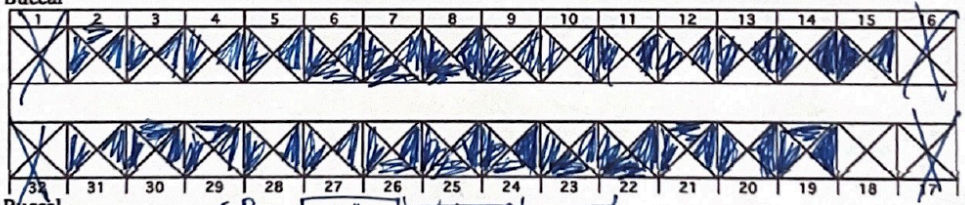
STUDENT FINDINGS

Buccal



Buccal
Show your calculation here: 71 / 28×4 ×100 = 63%

Buccal



Buccal
Show your calculation here: 68 / 28×4 = 61%

4	3	2	1	0
Student demonstrated correct sequence and proper technique without guidance.	Student demonstrated 1-2 minor errors and required minimal guidance.	Student demonstrated 3 minor errors, required guidance but corrected with verbal feedback.	Student required constant guidance, demonstrated 4 errors, difficulty completing task.	Student required constant guidance, unable to complete task. Student made more than 4 errors or ONE critical error.

Instructor Signature

4.0
Grade

Food Diary for Ming Lo **(Name of patient) DATE:** 12/5/2023

DAY # 1, 2, or 3 (circle one)

- Please list EVERYTHING you eat and drink on 3 consecutive and typical days. (if possible, at least include 1 weekend day)
- Please include extras such as chewing gum, sugar and cream in coffee, or mustard on sandwich.
- Please put down your guess for nutritional facts such as calories, sodium content, carbohydrates. Your dental hygienists will look up and provide the correct information later.

Time (How long?)	Food Eaten or Beverages Drank (List each food item separately)	Amount Eaten or Drank
Example: 6:00 a.m. to 6:15 a.m. (15 minutes)	Egg McMuffin	1
6:00 a.m. to 6:15 a.m. (15 minutes)	Orange Juice	1 large cup (22 oz)
1100am-1115am	Shredded Beef and Tofu	1 large plate
	diet coke	40 oz
200pm-215pm	1 pack trail mix	6 oz
915pm-930pm	Avocado Chicken Salad	1 large plate
915pm - 1200pm	diet coke	44 oz

1. Any Exercises? Please List your exercise of the day

Food Diary for Ming Lo **(Name of patient) DATE:** 12/6/2023

DAY # 1, 2, or 3 (circle one)

- Please list EVERYTHING you eat and drink on 3 consecutive and typical days. (if possible, at least include 1 weekend day)
- Please include extras such as chewing gum, sugar and cream in coffee, or mustard on sandwich.
- Please put down your guess for nutritional facts such as calories, sodium content, carbohydrates. Your dental hygienists will look up and provide the correct information later.

Time (How long?)	Food Eaten or Beverages Drank (List each food item separately)	Amount Eaten or Drank
Example: 6:00 a.m. to 6:15 a.m. (15 minutes)	Egg McMuffin	1
6:00 a.m. to 6:15 a.m. (15 minutes)	Orange Juice	1 large cup (22 oz)
1030am-1040am	3 Chicken Tenders	Large
	Diet Coke	44 Oz
	Apple	1
200pm-215pm	Apple with Peanut Butter	Half Apple
415pm - 430pm	Corn, Peas and Carrots	1.5 cups
900pm-915pm	Chicken Tostada Salad	1 Large Plate

2. Any Exercises? Please List your exercise of the day

Food Diary for Ming Lo **(Name of patient) DATE:** 12/7/2023

DAY # 1, 2, or 3 (circle one)

- Please list EVERYTHING you eat and drink on 3 consecutive and typical days. (if possible, at least include 1 weekend day)
- Please include extras such as chewing gum, sugar and cream in coffee, or mustard on sandwich.
- Please put down your guess for nutritional facts such as calories, sodium content, carbohydrates. Your dental hygienists will look up and provide the correct information later.

Time (How long?)	Food Eaten or Beverages Drank (List each food item separately)	Amount Eaten or Drank
Example: 6:00 a.m. to 6:15 a.m. (15 minutes)	Egg McMuffin	1
6:00 a.m. to 6:15 a.m. (15 minutes)	Orange Juice	1 large cup (22 oz)
1030am-1045am	Chicken Tenders	3 Large Pieces
1045am-1100am	Pizza Pastries with Cream Cheese	2
	Diet Coke	44 oz
400pm-415pm	Apple with Peanut Butter	1
	Diet Coke	30 oz
900pm-930pm	Ceasar Salad with Sausage	1 Serving Salad, 2 Sausages
	Diet Coke	30 oz

3. Any Exercises? Please List your exercise of the day

USDA
Food and Nutrition Service
United States Department of Agriculture

Start simple with MyPlate Plan

The benefits of healthy eating add up over time, bite by bite. Small changes matter. Start Simple with MyPlate.

A healthy eating routine is important at every stage of life and can have positive effects that add up over time. It's important to eat a variety of fruits, vegetables, grains, protein foods, and dairy or fortified soy alternatives. When deciding what to eat or drink, choose options that are full of nutrients. Make every bite count.

Food Group Amounts for 1,800 Calories a Day for Ages 14+ Years

Fruits	Vegetables	Grains	Protein	Dairy
1½ cups	**2½ cups**	**6 ounces**	**5 ounces**	**3 cups**
Focus on whole fruits Focus on whole fruits that are fresh, frozen, canned, or dried.	Vary your veggies Choose a variety of colorful fresh, frozen, and canned vegetables—make sure to include dark green, red, and orange choices.	Make half your grains whole grains Find whole-grain foods by reading the Nutrition Facts label and ingredients list.	Vary your protein routine Mix up your protein foods to include seafood; beans, peas, and lentils; unsalted nuts and seeds; soy products; eggs; and lean meats and poultry.	Move to low-fat or fat-free dairy milk or yogurt (or lactose-free dairy or fortified soy versions) Look for ways to include dairy or fortified soy alternatives at meals and snacks throughout the day.

Limit

Choose foods and beverages with less added sugars, saturated fat, and sodium. Limit:

- Added sugars to <**45 grams** a day.
- Saturated fat to <**20 grams** a day.
- Sodium to <**2,300 milligrams** a day.

Activity

Be active your way:
Children 6 to 17 years old should move **60 minutes** every day. Adults should be physically active at least **2½ hours** per week.

WLAC Dental Hygiene Clinic -- Periodontal Treatment Plan

Patient Name: [redacted]

Student Name: ~~Ling Zeng~~ Bruna RDH

Calculus Code & Additional Notes			
Student			*Instructor*
UR: 4m		UL: 4m	Sara Coppola
LR: 4m		LL: 4m	

AAP Classification	
Student Stage II Grade B	*Instructor* Sara Coppola

Appt #	Treatment Plan	*Instructor*
Student		
1	LLQ with Lido	Sara Coppola
2	ULQ with Lido	
3	LRQ with Lido	
4	URQ with Lido	
5	Re-eval	
6		

The proposed treatment, benefits, and risks have been described to me. I have had the opportunity to ask questions and have had all my questions answered. I hereby acknowledge that I am consenting to all aspects of treatment.

Patient Signature [redacted]

Student Signature Bruna RDH

RDH Signature Sara Coppola

Date: 11/20/23

WEST LOS ANGELES COLLEGE
DEPARTMENT OF DENTAL HYGIENE
DENTAL HYGIENE DIAGNOSIS AND CARE PLAN

Student Name: Bruna Reff Patient Initial : ML

I. **Medical Assessment:** Patient is hep B carrier since child. Patient stated that he was born in Thailand and never had any issues with hep B. Patient takes Idomethacin 25 mg prn for gout. Finasteride 1 mg once daily for hair growth.

II. **Dental History:**

a. Caries Previous amalgam restoration on teeth ~~#30~~ #7 Class I, Occlusal #14, #18 Class II, #19, #31.

b. Periodontal Generalized recession, attrition and horizontal bone loss.

c. Last dental visit: 2015 Reason: Cleaning

d. Last radiographs: FMX's 6/21/23 BW's PA;s Other

III. **Oral Assessment:**

a. Extra/Intra Oral Exam Findings: Lingual erosion #7-#9. Tooth #28 is missing buccal enamel with dentin exposure. Tooth #29 F abfraction.

b. Occlusal Classification (I, II, and III): Rt C I Rt M I Lt C I Lt M I

Overbite 2mm Overjet 3mm Crossbite none

Maximum Opening 60mm TMD Asymptomatic clicking on right side

c. Special Dental Prostheses (Dentures, Implants etc.) none

e. Caries Risk Assessment/Defective Restorations: none

f. Periodontal Assessment:

Gingival description (Color, Consistency, Contour, Texture)

Maxillary Free Gingiva: pink, firm, ~~stippled~~.

Maxillary Attached Gingiva: pink, firm, stippled.

Mandibular Free Gingiva: pink, firm, s

Mandibular Attached Gingiva: pink, firm, stippled.

MBI, Probing, BOP's and Exduate BOP on molars and premolars.

Recession and mucogingival involvement: Recession on mand. + max. canine.

Furcations: none

Mobility: none

Periodontal Classification: AAP class ~~Class~~ Stage III

WLAC Calculus Code: Generalized 4 medium. Localized 5 medium-heavy on lower anteriors.

Journal of *Personalized Medicine*

MDPI

Review

Oral and Periodontal Implications of Hepatitis Type B and D. Current State of Knowledge and Future Perspectives

Dorin Nicolae Gheorghe [1,†], Francesco Bennardo [2,*], Dora Maria Popescu [1], Flavia Mirela Nicolae [1], Claudiu Marinel Ionele [3], Mihail Virgil Boldeanu [4], Adrian Camen [5,†], Ion Rogoveanu [3] and Petra Surlin [1,†]

[1] Department of Periodontology, Faculty of Dental Medicine, University of Medicine and Pharmacy of Craiova, 200349 Craiova, Romania
[2] School of Dentistry, Department of Health Sciences, Magna Graecia University of Catanzaro, Viale Europa, 88100 Catanzaro, Italy
[3] Department of Gastroenterology, Faculty of Medicine, University of Medicine and Pharmacy of Craiova, 200349 Craiova, Romania
[4] Department of Immunology, Faculty of Medicine, University of Medicine and Pharmacy of Craiova, 200349 Craiova, Romania
[5] Department of Oral Surgery, Faculty of Dental Medicine, University of Medicine and Pharmacy of Craiova, 200349 Craiova, Romania
* Correspondence: francesco.bennardo@unicz.it or fbennardo92@gmail.com
† These authors contributed equally to this work.

Abstract: Periodontitis is characterized by low-grade inflammation of the periodontal tissues, the structures that support and connect the teeth to the maxilla and mandible. This inflammation is caused by the accumulation of subgingival bacterial biofilm and gradually leads to the extensive damage of these tissues and the consequent loss of teeth. Hepatitis B is a major global health concern; infection with the hepatitis B virus causes significant inflammation of the liver and the possibility of its gradual evolution to cirrhosis. Hepatitis D, caused by infection with the delta hepatitis virus, is manifest only in patients already infected with the type B virus in a simultaneous (co-infected) or superimposed (superinfected) manner. The dental and periodontal status of patients with hepatitis B/D could exhibit significant changes, increasing the risk of periodontitis onset. Moreover, the progression of liver changes in these patients could be linked to periodontitis; therefore, motivating good oral and periodontal health could result in the prevention and limitation of pathological effects. Given that both types of diseases have a significant inflammatory component, common pro-inflammatory mediators could drive and augment the local inflammation at both a periodontal and hepatic level. This suggests that integrated management of these patients should be proposed, as therapeutical means could deliver an improvement to both periodontal and hepatic statuses. The aim of this review is to gather existing information on the proposed subject and to organize significant data in order to improve scientific accuracy and comprehension on this topic while generating future perspectives for research.

Keywords: periodontitis; hepatitis B; hepatitis D; pathogenesis; relationship; link; influence

check for updates

Citation: Gheorghe, D.N.; Bennardo, F.; Popescu, D.M.; Nicolae, F.M.; Ionele, C.M.; Boldeanu, M.V.; Camen, A.; Rogoveanu, I.; Surlin, P. Oral and Periodontal Implications of Hepatitis Type B and D. Current State of Knowledge and Future Perspectives. *J. Pers. Med.* **2022**, *12*, 1580. https://doi.org/10.3390/jpm12101580

Academic Editor: Luca Testarelli

Received: 7 September 2022
Accepted: 20 September 2022
Published: 26 September 2022

Publisher's Note: MDPI stays neutral with regard to jurisdictional claims in published maps and institutional affiliations.

1. Introduction

The oral cavity hosts over 700 bacterial species, which usually co-exist in a harmonious state, called eubiosis when commensal bacteria do not allow harmful ones to trigger diseases [1]. These bacteria can also be found inside the gingival groove, or sulcus, a narrow space delimited by the tooth's surface and the gingiva [2]. If the gingival sulcus is not properly and periodically cleaned by professional and at-home methods, this will allow the emergence of highly pathogenic bacteria. Consequently, this subgingival pathogenic bacterial biofilm will cause periodontal inflammation (periodontitis) [3]. In other words, if the subgingival biofilm is left undisturbed for lengthy periods of time, allowing highly

pathogenic bacteria to colonize, the conditions for the onset of periodontitis are met [4]. As a result, these bacteria and their toxins reach the gingival tissues, causing the inflammatory response that is characteristic of periodontitis [3]. This low-grade, local inflammation usually has a gradual evolution, generating a damaging setting due to acidosis and enzyme activation for crucial elements of the periodontium, such as collagen fibers [5]. These fibers are the main component of the periodontal ligament, the structure of which connects the tooth to the alveolar bone. If damaged, the ligament will contribute to the formation of periodontal pockets (deep areas along the tooth's root), which provide the ideal environment for more pathogenic bacteria [5]. Eventually, the alveolar bone itself is targeted by these bacteria and, under the effect of their collagenolytic enzymes and cellular damage, will begin to lose its normal size [6]. Consequently, the teeth will lose their support, increase their mobility, and finally be extracted, with significant consequences on the patient's life quality and general health [6]. In addition, it is important to highlight that it is not only natural teeth that can be affected by the inflammation of their supporting tissues but dental implants too. In this case, the disease is called "peri-implantitis" and can lead to increased implant mobility, poor bone integration and, in some cases, implant removal [7].

Extensive research performed during the last few decades has shown that the consequences of periodontitis go beyond the disruption of normal dental functions [8]. The periodontium is linked to the rest of the body by blood and lymphatic means [9]. As a result, every pathologic alteration in general homeostasis has the potential to affect periodontal health [5]. Periodontitis, on the other hand, can affect a patient's overall health, as well as the clinical presentation of specific conditions [5]. Researchers have investigated the bi-directional relationship between periodontitis and systemic health and sickness, leading to the formation of the "periodontal medicine" concept [10]. This concept incorporates and discusses the mutually influencing interactions that occur between periodontitis and systemic illnesses such as diabetes and cardiovascular disease [11,12]. Other significant correlations have been highlighted between periodontitis and autoimmune diseases such as rheumatoid arthritis and psoriasis [13,14]. In 2018, with the new classification of periodontal diseases, this concept gained clinical relevance, as certain systemic conditions were found to significantly modify the severity and rate of progression of periodontal diagnosis [15].

Hepatitis B is an infectious illness that damages the liver, caused by the hepatitis B virus (HBV) [16]. The virus is spread by contact with infected blood or bodily fluids [17]. In locations where the illness is widespread, infection around the time of birth or when in contact with other people's blood throughout infancy are the most typical ways of contracting hepatitis B [17]. In locations where the illness is uncommon, the most common sources of transmission are intravenous drug use and sexual contact. Working in healthcare, blood transfusions, dialysis, living with an infected person, and traveling to countries with high infection rates are also considered to be significant risk factors [18]. HBV is capable of causing both acute and chronic infection [19]. Many people have no symptoms when they first become infected [20]. During an acute infection, some people may experience vomiting, yellowish skin, weariness, black urine, and abdominal discomfort [18,20]. These symptoms usually last a few weeks, and the first infection is seldom fatal [20]. Once infected, symptoms may develop from 30 to 180 days later [20,21]. If entering a chronic phase, the infection can lead to life-threatening complications, including cirrhosis or hepatocellular carcinoma [22].

Hepatitis D is caused by infection with the hepatitis D virus (HDV) and only occurs in individuals who are already infected with the HBV type [23]. HDV transmission can occur either concurrently with HBV infection (co-infection) or is superimposed on chronic hepatitis B or hepatitis B carrier status (superinfection) [23]. Because of the severity of its effects, an HDV infection in a person with chronic hepatitis B (superinfection) is considered the most dangerous kind of viral hepatitis [24]. In acute infections, these problems include an increased chance of liver failure and a rapid development of liver cirrhosis, as well as an increased risk of developing liver cancer in chronic infections [25]. Hepatitis D has the

greatest mortality rate of any hepatitis infection, at 20% when combined with the hepatitis B virus [26]. According to a 2020 prediction, 48 million people are now afflicted with this virus [27].

Previous research was performed on the analysis of possible pathogenic connections existing between periodontitis and chronic hepatitis C (CHC) caused by the infection with the hepatitis C virus (HCV) [28,29]. Thus, it was highlighted that patients with CHC could exhibit significantly more severe oral health challenges, including periodontal ones, caused by the clinical manifestations of periodontitis (gingival bleeding, pocket depth, attachment loss) when compared to healthy controls. Local periodontal inflammation has been shown to have increased strength in CHC patients, as depicted by the immunological quantitative assessment of relevant pro-inflammatory mediators in gingival crevicular samples (GCF) [29]. These mediators include interleukins (IL, such as IL-1α, IL-1β, IL-18), inflammasomes (NLRP3 inflammasome), collagenolytic enzymes (Caspase-1) and pentraxins (PTX, such as PTX-3 and C-reactive protein). All of these pro-inflammatory markers were shown to express more elevated GCF levels in patients with periodontitis and CHC than in periodontitis patients with no CHC or with healthy controls [28]. Interestingly, these markers have also been shown to express serum-elevated levels in CHC patients, suggesting an additional periodontal risk in such patients [29]. Conversely, the implementation of non-surgical periodontal therapy in these patients has delivered less significant improvements in the intensity of local periodontal inflammatory reaction than in non-CHC periodontitis patients, suggesting a limited efficiency of the therapy in their case. Thus, an additional therapeutical focus should be given to these types of patients when seeking periodontal or dental care [28,29].

Given the results generated by our previous research on the topic of possible pathogenic connections existing between HCV infection and periodontitis, we aim to expand the project on HBV and HDV infection and periodontal links. Thus, we performed a review of the existing relevant scientific literature in order to gather data and assess the current state of the art. The aim of this review is to extract and compile available information on the subject so as to set future development of this topic and to generate the scientific background needed for the onset of future projects on possible pathogenic connections existing between periodontitis and HBV/HDV infection.

2. Materials and Methods

This review followed the criteria and guidelines of the Preferred Reporting Items for Systematic Review and Meta-Analyses (PRISMA) (Figure 1).

2.1. PICO Question

"Is there currently relevant scientific data on the possible significant pathogenic connections between oral implications and HBV/HDV infection that could offer the background for future development of the subject?" (Population: patients with hepatitis B/D infection; Intervention: oral health status assessment; Comparison: relevant background information; Outcome: development of complementary studies).

2.2. Search Strategy

Relevant scientific databases were electronically searched for English in-extenso papers and abstracts for this review. These included Medline (via PubMed), Web of Science, and Scopus. The time bracket for the search was set between 1st January 1970 and 1st July 2022. The keywords used during the search were: "periodontitis", "hepatitis B", and "hepatitis D", "oral", "oral health", "oral status", "periodontal disease", "hepatitis B virus", "hepatitis D virus", "pathogenic", "connection", "patients", "gingival fluid", "dental status", "transmission", "saliva" and "blood", using the Boolean operators "AND", "OR".

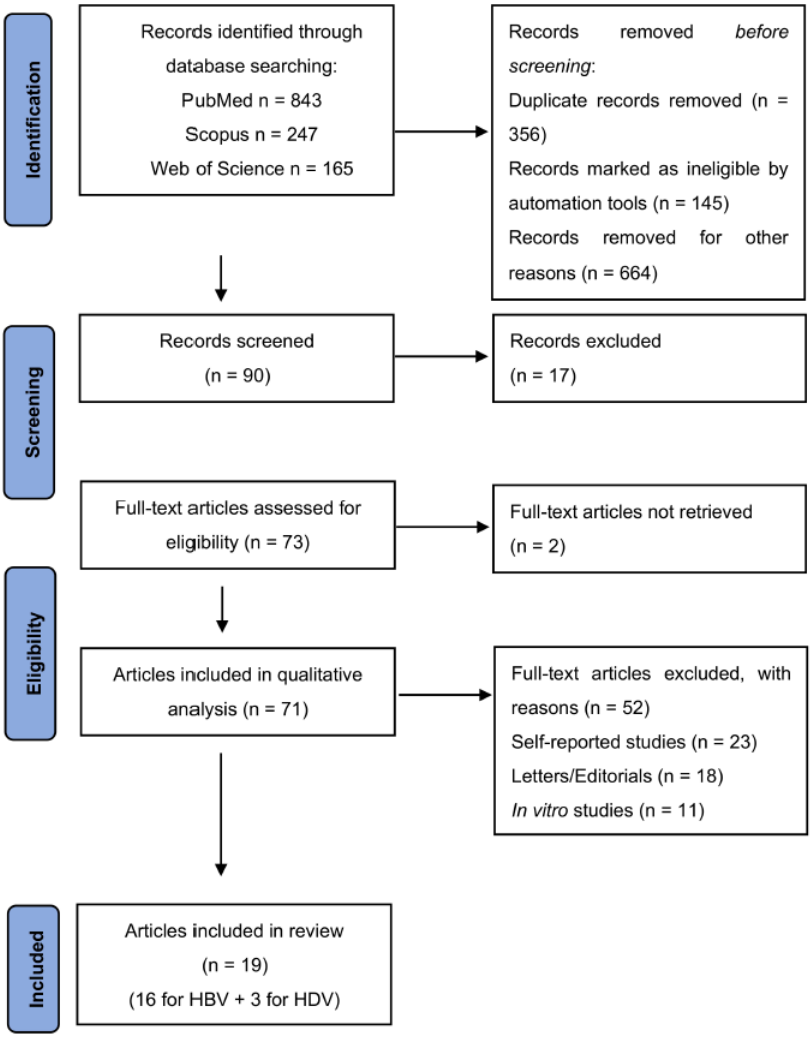


Figure 1. PRISMA flow diagram. HBV = hepatitis B virus; HDV = hepatitis D virus.

2.3. Exclusion Criteria of the Studies

Some of the generated search articles were excluded on the following premises: (1) they reported in vitro and experimental animal studies, (2) self-reported studies focusing on dental practitioners and dental students' knowledge of the transmission of the virus, and (3) letters and editorials. All other remnant studies were included for further analysis.

2.4. Information Extraction and Review Structuring

The selected papers were carefully read, and relevant information for the review was extracted. The review consisted of three parts: (1) information on HBV infection and oral implications, (2) information on HDV infection and oral implications, and (3) future perspectives and proposed development of the subject.

3. Results

3.1. HBV Infection and Oral Implications

The search for papers on HBV infection and oral implications returned 58 results, ranging from 1977 to 2022. After applying the exclusion criteria, 16 papers were finally selected for critical reading and idea synthesis. Additional papers were extracted from these papers' references list, if they were not generated by the database search.

The earliest papers on the subject were published between 1984–1985, focusing on the detection of HBV antigens (or viral particles) in the oral fluids of infected patients. The study by Polloch et al. identified HBV surface antigens (HBsAg) in the GCF samples of HBV-infected patients [30]. In 1985, Ben-Aryeh et al. performed a similar study, which concluded that HBsAg was found in 90% of the assessed GCF samples originating from HBV-infected patients [31]. The surface antigen was also found in the saliva samples of the same patients. It could be speculated that the saliva might have been contaminated with the blood of gingival origin due to gingival inflammation. However, the authors found no significant correlation between the presence of HBsAg in the saliva samples and the gingival status of the participating patients in terms of gingival inflammation or the gingival bleeding index [31]. This led the authors to suggest that the source of HBsAg presence in the saliva samples was, in fact, the GCF [28]. The virus circulates in the general bloodstream, reaches the lymphatic system and eventually into the GCF due to a difference in osmotic pressure. Then it reaches the saliva, using the GCF as a carrier [32]. This hypothesis is also endorsed by the 1984 study by Hurlem et al., who suggested that HBV-infected patients may pose a higher risk of viral transmission in the dental office by double carriers: saliva and increased gingival bleeding when dealing with gingival inflammation [33].

This hypothesis is further emphasized by a more recent study by Kamimura et al., who found a strong correlation between occult blood traces in saliva and the presence of viral HBV particles [34]. This study highlighted how HBV DNA particles were found in saliva samples, particularly of elderly patients diagnosed with periodontitis [34]. The study further speculates that this may pose an increased risk of horizontal HBV transmission in the family, where the probability of contact with infected saliva is quite elevated [34]. The risk is further enhanced if patients suffer from periodontitis, with an increased gingival bleeding index [34]. A study by Farghaly reached similar conclusions suggesting that patients with periodontitis showed a higher proportion of hepatitis exposure and a higher detectability of salivary HBsAg [35]. Additional risk factors were considered to be the rural residence of patients or a medical history of past blood transfusions. Thus, the study concluded that the presence of periodontitis, severe gingival bleeding and poor oral hygiene were associated with the risk of hepatitis and the detectability of salivary hepatitis markers [35]. Similar results were generated in the study by Sharifian et al., who considered that the most frequent risk factors for HBV infection in studied patients were positive periodontal diagnosis and family history [36].

The clinical settings of unfavorable dental and periodontal diagnosis and liver damage were assessed by Yang et al., who concluded that an increased number of absent teeth were associated with an increased risk of primary liver cancer [37]. A study by Nagao et al. also highlighted that periodontitis might be correlated with viral liver disease [38]. However, the results seem inconclusive so far, as a 2011 study by Anand et al. found that the number of dental caries and the periodontal status of patients with nonalcoholic cirrhosis did not differ significantly from that of the controls without any liver disease [39]. Nevertheless, other oral health issues, such as halitosis, have been directly linked to HBV infection and periodontitis, including a study by Hun Han et al. [40]. The authors concluded that patients with periodontitis, HBV infection and neglected tongue-brushing had the highest prevalence of volatile sulphur halitosis, suggesting that liver function should be evaluated in patients dealing with bad breath [40].

The periodontal management of patients with an HBV infection was studied by Seshima et al., who reported a case of effective, regenerative periodontal therapy [41]. The

patient suffered from HBV infection and diabetes mellitus, which can significantly impact the body's healing and regenerative capabilities [41]. However, considering the medical history of the patient, the authors reported a clinical improvement in the periodontal parameters [41]. A study by Ting et al. suggested the use of statins as an adjunctive to periodontal therapy in patients with an HBV infection [42]. This is justified by the antiviral properties of statins, as well as their antibacterial capabilities, including on important periodontal pathogens such as *Porphyromonas gingivalis* [42]. Concerning the surgical management of periodontal patients with an HBV infection, Hong et al. reported no episodes of postoperative bleeding in patients, despite a significant correlation of the international normalized ration (INR) with HBV infection diagnosis [43]. The authors suggested that it was not only INR values that should be considered when evaluating patients with liver diseases for procedures with a post-surgical bleeding risk [43].

From an immunological perspective, certain inflammation mediators were targeted in the saliva samples of patients with an HBV infection [44]. Pro-inflammatory interleukins (IL-2 and IL-4), as well as anti-inflammatory ones (IL-10), expressed significantly more elevated levels in the saliva samples of HBV infected patients than in the healthy controls, as depicted by the enzyme-linked immunosorbent assay (ELISA) used in this study [44]. The same immunological method (ELISA) was proposed in the study by Gharavi et al. as a diagnosis tool for HBV infection in samples of saliva, with good sensitivity and specificity [45].

3.2. HDV Infection and Oral Implications

The search for papers on the oral implications of HDV infections retrieved 13 papers, of which only 3 could be selected, for critical reading after applying the exclusion criteria. This low number of papers on the subject suggests a limited current understanding of the subject and should stimulate future developments of the topic.

In a 1986 article, Cottone et al. raised awareness among dental practitioners and members of the dental office team of the possibility of the transmission of the newly identified, at that time, HDV virus [46]. The authors stated that the hepatitis D virus could pose a serious threat to all members of the dental team and thus encouraged vaccination against the HBV virus, as it would also offer protection against HDV [46].

One of the main reasons why the literature on the oral implications of HDV is limited, is that the viral infection is mainly conditioned by a co-existing or pre-existing infection with HBV. Hence, the patient target group is limited only to HBV-positive persons. Even though the association of the HBV and HDV viruses is generally accepted and agreed upon, some authors have reported exceptions to this. In 2016, Weller et al. detected HDV in the salivary glands of Sjogren syndrome patients [47]. Their micro-array analysis showed that HDV was present in more than 50% of the samples originating from primary Sjogren syndrome patients [47]. The novelty of the study was the fact that the identification of HDV was independent of any HBV presence. This suggests that HDV is able to set up an independent presence without HBV, at least at the salivary gland level, and exhibits a unique tissue tropism [47]. The results of this study raise significant awareness from an oral health perspective, as Sjogren syndrome is considered to be a major trigger for dental and periodontal problems, as well as an extra-hepatic manifestation of liver diseases, including viral infections.

Currently, there is insufficient data on whether HDV particles could be carried by saliva, similar to HBV. Only one study, performed by Isaeva et al., focused on this topic but found no detection of HDV antibodies in saliva samples originating from patients with HBV and HDV infection [48]. Despite the fact that the saliva samples were positive for HBV antigens and antibodies, this was not the case for HDV, suggesting a lower concentration of these elements in the saliva than for HBV [48]. Nevertheless, the matter should be addressed by complementary research in order to increase its scientific understanding.

4. Future Perspectives

As shown by the literature review (Table 1), currently, there is sufficient data on the oral implications of HBV infection and little, or almost no insight, into these implications in HDV ones. HBV infection and oral implications cover mostly the detection of viral antigens in saliva and gingival fluid and less about the clinical, dental, or periodontal status of infected patients [49]. There is also a gap in the literature regarding the assessment of various pro-inflammatory elements in samples of gingival fluid or saliva, as this can have relevance for the characterization of low-grade inflammatory periodontal reactions and their elements in this type of patient. Regarding HDV infection, this has received little attention from the perspective of oral health implications, mainly because patients with HBV and HDV co-infection or supra-infection may be more difficult to gather for larger studies. The epidemiology of the HDV infection may vary significantly from region to region, and as HBV vaccinations continue to gain popularity, the spread of the HDV virus may also decelerate.

Table 1. Synopsis of the selected papers and their findings.

Reference	Hepatitis Virus Type	Findings
Polloch et al., 1984 [30]	HBV	HBsAg detection in gingival crevicular samples of infected patients
Hurlem et al., 1984 [33]	HBV	HBV infected patients may pose a higher risk of viral transmission in the dental office, via saliva and gingival bleeding
Ben-Aryeh et al., 1985 [31]	HBV	HBsAg detection in 90% of gingival crevicular fluid and saliva samples of infected patients
Cottone et al., 1986 [46]	HDV	HDV infection is a significant risk in the dental office, recommended HBV vaccination for members of the dental team
Farghaly et al., 1998 [35]	HBV	Patients with periodontitis showed a higher percentage of hepatitis exposure and a higher detectability of salivary HBsAg
Anand et al., 2001 [39]	HBV/HCV	Periodontal status of patients with nonalcoholic cirrhosis did not differ significantly from that of controls with no liver disease
Azatyan et al., 2001 [44]	HBV	Elevated expression of pro-inflammatory interleukins in saliva samples of HBV infected patients
Lamster et al., 2007 [32]	HBV/HCV	Gingival crevicular fluid could be an important source for traces of hepatitis viruses' presence in saliva
Hong et al., 2012 [43]	HBV	No episodes of postoperative bleeding in periodontal management of HBV infected patients, independent of INR values
Hun Han et al., 2013 [40]	HBV	Direct link between volatile sulphur halitosis, periodontitis, and HBV infection
Nagao et al., 2014 [38]	HBV/HCV	Periodontitis might be correlated with viral liver disease
Ting et al., 2015 [42]	HBV	Use of statins as an adjunctive periodontal therapy in patients with HBV infection
Seshima et al., 2016 [41]	HBV	Effective and stable results of regenerative periodontal therapy in patients with HBV infection
Weller et al., 2016 [47]	HDV	Identification of independent HDV in 50% of salivary gland samples from HDV infected patients with primary Sjogren syndrome
Yang et al., 2017 [37]	HBV	Increased number of absent teeth is associated with increased risk of primary liver cancer
Sharifian et al., 2019 [36]	HBV	Risk factors for HBV infection: positive periodontal and family history
Gharavi et al., 2020 [45]	HBV	HBV can be detected in saliva samples of infected patients by means of ELISA method
Isaeva et al., 2020 [48]	HDV	No detection of HDV antibodies in saliva samples from patients with HBV and HDV infection
Kamimura et al., 2021 [34]	HBV	Strong correlation between occult blood traces in saliva and HBV presence in saliva samples

HBV—hepatitis B virus; HCV—hepatitis C virus; HDV—hepatitis D virus; HBsAg—hepatitis B surface antigen; ELISA—enzyme linked immunosorbent assay.

Considering the setting, pilot studies on smaller groups of patients could be generated in order to probe the particularities of oral and periodontal health in patients with an HBV + HDV infection. The first step of this project would be to compare clinical data on the oral

and periodontal health status of HBV + HDV infected individuals, such as the number of missing teeth, periodontal diagnosis, and the type of diagnosed periodontal conditions, in terms of the severity and rate of progression, as compared to the controls. The ideal circumstance would be to include patients who do not suffer from other systemic diseases that could influence the manifestation of periodontitis (such as diabetes mellitus), but this would remain to be established by the study design and group characteristics [50]. An immunological analysis via the ELISA method would be necessary in order to measure specifically targeted pro-inflammatory mediators in GCF samples that have relevance in both periodontitis and HBV + HDV infections pathogenesis. The local and systemic effects of periodontal therapy in patients diagnosed with periodontitis and HBV + HDV infection should also be evaluated, from a clinical and immunological standpoint, in order to detect improvements in the expression of inflammatory mediators as a sign of the inflammatory reaction's modulation.

As the prevalence of HDV infection in Romania experienced recent rising trends [51] and considering the general increase in population mobility after the COVID-19 pandemic, the development of such a research project could be of significant interest and deliver valuable and high-novelty results. With the experience gained from the previous HCV infection study, we plan to apply the same principles and management of the project in this new research direction in order to improve existing knowledge and increase scientific awareness of the topic.

5. Conclusions

The existing literature offers sufficient background information on the oral implications of HBV infection in order to fundamentally support the development of a research project on the topic of HBV + HDV co-infection, where data is scarce and has significant gaps.

Author Contributions: Conceptualization, I.R. and P.S.; methodology, A.C.; validation, D.M.P. and M.V.B.; formal analysis, D.N.G.; investigation, D.N.G., F.B., F.M.N. and C.M.I.; resources, M.V.B. and A.C.; data curation, D.N.G.; writing—original draft preparation, D.N.G.; writing—review and editing, F.B., D.M.P. and A.C.; visualization, P.S.; supervision, I.R.; project administration, P.S. All authors have read and agreed to the published version of the manuscript.

Funding: This research received no external funding.

Institutional Review Board Statement: Not applicable.

Informed Consent Statement: Not applicable.

Data Availability Statement: Not applicable.

Acknowledgments: A.C. shares an equal contribution to that of the first author and thus can be considered a main author.

Conflicts of Interest: The authors declare no conflict of interest.

References

1. Nomura, Y.; Okada, A.; Hanada, N. Future Prospective of Oral Microbiome Research. *Appl. Sci.* **2022**, *12*, 55. [CrossRef]
2. Schulz, S.; Stein, J.M.; Schumacher, A.; Kupietz, D.; Yekta-Michael, S.S.; Schittenhelm, F.; Conrads, G.; Schaller, H.-G.; Reichert, S. Nonsurgical Periodontal Treatment Options and Their Impact on Subgingival Microbiota. *J. Clin. Med.* **2022**, *11*, 1187. [CrossRef] [PubMed]
3. Mombelli, A. Microbial colonization of the periodontal pocket and its significance for periodontal therapy. *Periodontology 2000* **2017**, *76*, 85–96. [CrossRef]
4. Fiorillo, L.; Cervino, G.; Laino, L.; D'Amico, C.; Mauceri, R.; Tozum, T.F.; Gaeta, M.; Cicciù, M. *Porphyromonas gingivalis*, Periodontal and Systemic Implications: A Systematic Review. *Dent. J.* **2019**, *7*, 114. [CrossRef] [PubMed]
5. Cecoro, G.; Annunziata, M.; Iuorio, M.T.; Nastri, L.; Guida, L. Periodontitis, Low-Grade Inflammation and Systemic Health: A Scoping Review. *Medicina* **2020**, *56*, 272. [CrossRef] [PubMed]
6. Slots, J. Periodontitis: Facts, fallacies and the future. *Periodontology 2000* **2017**, *75*, 7–23. [CrossRef] [PubMed]

7. Guarnieri, R.; Zanza, A.; D'Angelo, M.; Di Nardo, D.; Del Giudice, A.; Mazzoni, A.; Reda, R.; Testarelli, L. Correlation between Peri-Implant Marginal Bone Loss Progression and Peri-Implant Sulcular Fluid Levels of Metalloproteinase-8. *J. Pers. Med.* **2022**, *12*, 58. [CrossRef] [PubMed]
8. Bosshardt, D. The periodontal pocket: Pathogenesis, histopathology and consequences. *Periodontology 2000* **2017**, *76*, 43–50. [CrossRef] [PubMed]
9. Matsumoto, Y.; Zhang, B.; Kato, S. Lymphatic networks in the periodontal tissue and dental pulp as revealed by histochemical study. *Micros. Res. Tech.* **2002**, *56*, 50–59. [CrossRef] [PubMed]
10. Beck, J.; Papapanou, P.; Philips, K.; Offenbacher, S. Periodontal Medicine: 100 Years of Progress. *J. Dent. Res.* **2019**, *98*, 1053–1062. [CrossRef] [PubMed]
11. Williams, R.; Offenbacher, S. Periodontal medicine: The emergence of a new branch of periodontology. *Periodontology 2000* **2000**, *23*, 9–12. [CrossRef]
12. Battancs, E.; Gheorghita, D.; Nyiraty, S.; Lengyel, C.; Eördegh, G.; Baráth, Z.; Várkonyi, T.; Antal, M. Periodontal Disease in Diabetes Mellitus: A Case–Control Study in Smokers and Non-Smokers. *Diabetes Ther.* **2020**, *11*, 2715–2728. [CrossRef] [PubMed]
13. Antal, M.; Battancs, E.; Bocskai, M.; Braunitzer, G.; Kovács, L. An observation on the severity of periodontal disease in past cigarette smokers suffering from rheumatoid arthritis- evidence for a long-term effect of cigarette smoke exposure? *BMC Oral Health* **2018**, *18*, 82. [CrossRef] [PubMed]
14. Antal, M.; Braunitzer, G.; Mattheos, N.; Gyulai, R.; Nagy, K. Smoking as a Permissive Factor of Periodontal Disease in Psoriasis. *PLoS ONE* **2014**, *9*, e92333. [CrossRef] [PubMed]
15. Caton, J.; Armitage, G.; Berglundh, T.; Chapple, I.; Jepsen, S.; Kornman, K.; Mealey, B.; Papapanou, P.; Sanz, M.; Tonetti, M. A new classification scheme for periodontal and peri-implant diseases and conditions—Introduction and key changes from the 1999 classification. *J. Periodontol.* **2018**, *89*, S1–S8. [CrossRef] [PubMed]
16. Tu, T.; Block, J.M.; Wang, S.; Cohen, C.; Douglas, M.W. The Lived Experience of Chronic Hepatitis B: A Broader View of Its Impacts and Why We Need a Cure. *Viruses* **2020**, *12*, 515. [CrossRef] [PubMed]
17. Chang, M. Hepatitis B virus infection. *Semin. Fetal Neonatal Med.* **2007**, *12*, 160–167. [CrossRef]
18. Guvenir, M.; Arikan, A. Hepatitis B Virus: From Diagnosis to Treatment. *Pol. J. Microbiol.* **2020**, *69*, 391–399. [CrossRef] [PubMed]
19. Herrscher, C.; Roingeard, P.; Blanchard, E. Hepatitis B Virus Entry into Cells. *Cells* **2020**, *9*, 1486. [CrossRef]
20. Nicolini, L.A.; Orsi, A.; Tatarelli, P.; Viscoli, C.; Icardi, G.; Sticchi, L. A Global View to HBV Chronic Infection: Evolving Strategies for Diagnosis, Treatment and Prevention in Immunocompetent Individuals. *Int. J. Environ. Res. Public Health* **2019**, *16*, 3307. [CrossRef] [PubMed]
21. Noverati, N.; Bashir-Hamidu, R.; Halegoua-DeMarzio, D.; Hann, H.-W. Hepatitis B Virus-Associated Hepatocellular Carcinoma and Chronic Stress. *Int. J. Mol. Sci.* **2022**, *23*, 3917. [CrossRef]
22. Rizzo, G.E.M.; Cabibbo, G.; Craxì, A. Hepatitis B Virus-Associated Hepatocellular Carcinoma. *Viruses* **2022**, *14*, 986. [CrossRef]
23. Rizzetto, M.; Stroffolini, T. Forty-Five Years after the Discovery of the Hepatitis D Virus: Where Do We Stand? *Viruses* **2021**, *13*, 555. [CrossRef]
24. Rizzetto, M. Hepatitis D Virus: Introduction and Epidemiology. *Cold Spring Harb. Perspect. Med.* **2015**, *5*, a021576. [CrossRef]
25. Brillanti, S. Management of Delta Hepatitis 45 Years after the Discovery of HDV. *J. Clin. Med.* **2022**, *11*, 1587. [CrossRef]
26. Taylor, J.M. Infection by Hepatitis Delta Virus. *Viruses* **2020**, *12*, 648. [CrossRef]
27. Miao, Z.; Zhang, S.; Ou, X.; Li, S.; Ma, Z.; Wang, W.; Peppelenbosch, M.; Liu, J.; Pan, Q. Estimating the Global Prevalence, Disease Progression, and Clinical Outcome of Hepatitis Delta Virus Infection. *J. Infect. Dis.* **2019**, *221*, 1677–1687. [CrossRef]
28. Gheorghe, D.N.; Camen, A.; Popescu, D.M.; Sincar, C.; Pitru, A.; Ionele, C.M.; Nicolae, F.M.; Danilescu, C.M.; Roman, A.; Florescu, C. Periodontitis, Metabolic and Gastrointestinal Tract Diseases: Current Perspectives on Possible Pathogenic Connections. *J. Pers. Med.* **2022**, *12*, 341. [CrossRef]
29. Surlin, P.; Gheorghe, D.N.; Foia, L.; Surdu, A.; Toma, V.; Solomon, S.M.; Rogoveanu, I. Periodontal Implications of Hepatitis C Infection. In *Hepatitis C—From Infection to Cure*; IntechOpen: London, UK, 2018. [CrossRef]
30. Pollock, J.; Andors, L.; Gulumoglu, A.; Ells, P. Direct measurement of hepatitis B viral antibody and antigen markers in gingival crevicular fluid. *Oral Surg. Oral Med. Oral Pathol.* **1984**, *57*, 499–503. [CrossRef]
31. Ben-Aryeh, H.; Ur, I.; Ben-Porath, E. The relationship between antigenaemia and excretion of hepatitis B surface antigen in human whole saliva and in gingival crevicular fluid. *Arch. Oral Biol.* **1985**, *30*, 97–99. [CrossRef]
32. Lamster, I.; Ahlo, J. Analysis of Gingival Crevicular Fluid as Applied to the Diagnosis of Oral and Systemic Diseases. *Ann. N. Y. Acad. Sci.* **2007**, *1098*, 216–229. [CrossRef]
33. Hurlen, B.; Jacobsen, N.; Hurlen, P. Hepatitis B serum markers and oral health in a group of Norwegian male prisoners. *Acta Odontol. Scand.* **1984**, *42*, 53–58. [CrossRef]
34. Kamimura, H.; Watanabe, J.; Sugano, T.; Kohisa, J.; Abe, H.; Kamimura, K.; Tsuchiya, A.; Takamura, M.; Okoshi, S.; Tanabe, Y.; et al. Relationship between detection of hepatitis B virus in saliva and periodontal disease in hepatitis B virus carriers in Japan. *J. Infect. Chemother.* **2021**, *27*, 492–496. [CrossRef]
35. Farghaly, A.G.; Mansour, G.A.; Mahdy, N.H.; Yousri, A. Hepatitis B and C virus infections among patients with gingivitis and adult periodontitis: Seroprevalence and public health importance. *J. Egypt. Public Health Assoc.* **1998**, *73*, 707–735. [PubMed]
36. Sharifian, A.; Ashtari, S.; Hatami, B.; Mohebbi, S.R.; Naderi, N. Risk factors of transmission and natural history of chronic hepatitis B infection in Iranian patients. *Gastroenterol. Hepatol. Bed Bench* **2019**, *12* (Suppl. 1), S149–S155.

37. Yang, B.; Petrick, J.; Abnet, C.; Graubard, B.; Murphy, G.; Weinstein, S.; Männistö, S.; Albanes, D.; McGlynn, K. Tooth loss and liver cancer incidence in a Finnish cohort. *Cancer Causes Control* **2017**, *28*, 899–904. [CrossRef]
38. Nagao, Y.; Kawahigashi, Y.; Sata, M. Association of Periodontal Diseases and Liver Fibrosis in Patients with HCV and/or HBV infection. *Hepat. Mon.* **2014**, *14*, e23264. [CrossRef]
39. Anand, A.; Pardal, P.; Sachdev, V. Dental caries and periodontal disorders in chronic liver disease. *Med. J. Armed Forces India* **2001**, *57*, 26–30. [CrossRef]
40. Han, D.; Lee, S.; Lee, J.; Kim, Y.; Kim, J. Association between viral hepatitis B infection and halitosis. *Acta Odontol. Scand.* **2013**, *72*, 274–282. [CrossRef]
41. Seshima, F.; Nishina, M.; Namba, T.; Saito, A. Periodontal Regenerative Therapy in Patient with Chronic Periodontitis and Type 2 Diabetes Mellitus: A Case Report. *Bull. Tokyo Dent. Coll.* **2016**, *57*, 97–104. [CrossRef]
42. Ting, M.; Whitaker, E.; Albandar, J. Systematic review of the in vitro effects of statins on oral and perioral microorganisms. *Eur. J. Oral. Sci.* **2015**, *124*, 4–10. [CrossRef] [PubMed]
43. Hong, C.; Scobey, M.; Napenas, J.; Brennan, M.; Lockhart, P. Dental postoperative bleeding complications in patients with suspected and documented liver disease. *Oral Dis.* **2012**, *18*, 661–666. [CrossRef] [PubMed]
44. Azatyan, V.; Yessayan, L.; Shmavonyan, M.; Melik-Andreasyan, G.; Perikhanyan, A.; Porkshenyan, K. Evaluation of IL-2, IL-10, IL-4 and γ-interferon levels in the oral fluids of patients with hepatitis C, B and HIV. *J. Infect. Dev. Ctries.* **2019**, *13*, 069S–074S. [CrossRef] [PubMed]
45. Gharavi, M.; Esmaeili, M. Evaluation of HBs-Ag and anti-HBc levels in serum and saliva of patients with hepatitis B. *Med. J. Islam. Repub. Iran* **2020**, *34*, 101. [CrossRef]
46. Cottone, J. Delta hepatitis: Another concern for dentistry. *J. Am. Dent. Assoc.* **1986**, *112*, 47–49. [CrossRef]
47. Weller, M.; Gardener, M.; Bogus, Z.; Smith, M.; Astorri, E.; Michael, D.; Michael, D.; Zheng, C.; Burbelo, P.; Lai, Z.; et al. Hepatitis Delta Virus Detected in Salivary Glands of Sjögren's Syndrome Patients and Recapitulates a Sjögren's Syndrome-Like Phenotype in Vivo. *Pathog. Immun.* **2016**, *1*, 12. [CrossRef]
48. Isaeva, O.; Ilchenko, L.; Kichatova, V.; Potemkin, I.; Amon, E.; Saryglar, A.; Al-Sharabi, S.; Kyuregyan, K.; Mikhailov, M. Detection of markers of hepatitis b and d virus infection in biological media and dried blood spots. *Klin. Lab. Diagn.* **2020**, *65*, 95–99. [CrossRef]
49. Mahboobi, N.; Porter, S.; Karayiannis, P.; Alavian, S. Oral fluid and hepatitis A, B and C: A literature review. *J. Oral Pathol. Med.* **2011**, *41*, 505–516. [CrossRef]
50. Lim, G.; Janu, U.; Chiou, L.-L.; Gandhi, K.K.; Palomo, L.; John, V. Periodontal Health and Systemic Conditions. *Dent. J.* **2020**, *8*, 130. [CrossRef]
51. Gheorghe, L.; Csiki, I.; Iacob, S.; Gheorghe, C.; Trifan, A.; Grigorescu, M.; Motoc, A.; Suceveanu, A.; Curescu, M.; Caruntu, F.; et al. Hepatitis Delta Virus Infection in Romania: Prevalence and Risk Factors. *J. Gastrointest. Liver Dis.* **2015**, *24*, 413–421. [CrossRef]

Oral Diseases Associated with Nonalcoholic Fatty Liver Disease in the United States

Journal of Dental Research
2019, Vol. 98(11) 1219–1226

DOI: 10.1177/0022034519866442
journals.sagepub.com/home/jdr

J.A. Weintraub[1], G. Lopez Mitnik[2], and B.A. Dye[2]

Abstract

The US prevalence of nonalcoholic fatty liver disease (NAFLD) is 30.6% and increasing. NAFLD shares some risk factors with periodontitis and dental caries. We explored the association between NAFLD and several oral conditions among US adults, using data from the cross-sectional, nationally representative National Health and Nutrition Examination Survey (NHANES), 1988 to 1994. NAFLD was assessed with ultrasonography (USON), the screening gold standard not available in the more recent NHANES, and the noninvasive Fibrosis Score (FS), Fatty Liver Index (FLI), and US Fatty Liver Index (US-FLI) as other screening alternatives. There were 5,421 eligible dentate adults aged 21 to 74 y with complete relevant data, with transferrin levels ≤50%, without hepatitis B or C, who were not heavy drinkers. Multivariable models were developed to examine the independent effects of moderate-severe periodontitis, untreated dental caries, caries experience, and tooth loss (<20 teeth) on NAFLD while controlling for clinical, biological, and sociodemographic factors. Weighted estimates for odds ratios (ORs) and 95% CIs were calculated with logistic regression. Between 17% and 24% of adults had NAFLD depending on the classification criteria. In adjusted models, as compared with those with better oral health, adults with <20 teeth were more likely to have NAFLD depending on the measure (USON: OR = 1.50, 95% CI = 1.11 to 2.02; FS: OR = 4.36, 95% CI = 3.47 to 5.49; FLI: OR = 1.99, 95% CI = 1.52 to 2.59; US-FLI: OR = 2.32, 95% CI = 1.79 to 3.01). People with moderate-severe periodontitis were more likely to have NAFLD (USON: OR = 1.54, 95% CI = 1.06 to 2.24; FS: OR = 3.10, 95% CI = 2.31 to 4.17; FLI: OR = 1.61, 95% CI = 1.13 to 2.28; US-FLI: OR = 2.21, 95% CI = 1.64 to 2.98). People with any untreated caries were more likely to have NAFLD (USON: OR = 1.51, 95% CI = 1.20 to 1.90; FLI: OR = 1.80, 95% CI = 1.33 to 2.44). NAFLD was associated with tooth loss, periodontitis, and, for some NAFLD measures, untreated dental caries but not overall caries experience after controlling for several key sociodemographic and behavioral factors. Results suggest that further evaluation is needed to better understand this health–oral health interrelationship and potential opportunities for medical-dental integration.

Keywords: oral health, periodontitis, tooth loss, dental caries, NHANES, cross-sectional studies

Introduction

Nonalcoholic fatty liver disease (NAFLD) is the excessive lipid accumulation in the liver not due to excess alcohol consumption. NAFLD is the most common chronic liver disease in the United States; adult prevalence from 2011 to 2012 was estimated to be 30.6% and increasing (Ruhl and Everhart 2015). About 24% of the population worldwide is potentially affected (Younossi et al. 2016). The prevalence is higher among those with type 2 diabetes, obesity, and hyperlipidemia (Younossi et al. 2016; Le et al. 2017). People with NAFLD can progress to nonalcoholic steatohepatitis (NASH) and NASH-related liver cirrhosis.

NAFLD is a risk factor and determinant of metabolic syndrome (Yki-Järvinen 2014; Lonardo et al. 2015). The disease also induces and enhances insulin resistance and increases the risk of type 2 diabetes (Yki-Järvinen 2014; Lonardo et al. 2015). Consumption of simple sugars (glucose and fructose) is a factor leading to NAFLD and dental caries. Chronic fructose exposure (e.g., high-fructose corn syrup) can lead to inflammation, liver fat accumulation, NAFLD, and metabolic syndrome (Lustig 2014; Yki-Järvinen 2014; Lonardo et al. 2015; Softic et al. 2016). A study with a murine model showed that injecting mice with a *Streptococcus mutans* strain, a causative factor in dental caries, and feeding them a high-fat diet could induce or expedite liver fibrosis and NASH (Naka et al. 2014; Naka et al. 2018).

Several hypotheses link NAFLD and periodontitis, through periodontal pathogens, inflammatory mediators, and oxidative stress (Han et al. 2016). *Porphyromonas gingivalis* was found in higher frequencies in patients with NAFLD than in non-NAFLD controls (Yoneda et al. 2012). In the population-based

[1]Adams School of Dentistry, University North Carolina at Chapel Hill, Chapel Hill, NC, USA
[2]National Institute of Dental and Craniofacial Research, National Institutes of Health, Bethesda, MD, USA

A supplemental appendix to this article is available online.

Corresponding Author:
J.A. Weintraub, Adams School of Dentistry, University North Carolina at Chapel Hill, 4508 Koury Oral Health Sciences Building, Chapel Hill, NC 27599-7450, USA.
Email: Jane_Weintraub@unc.edu

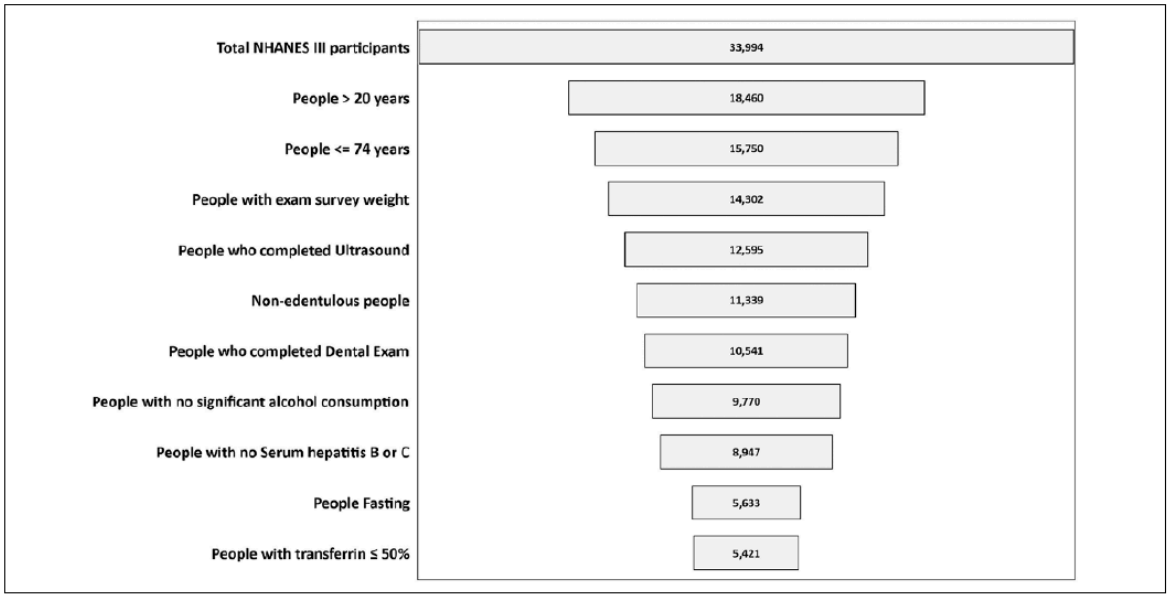


Figure 1. Number of individuals meeting eligibility criteria for analytic sample. NHANES III, National Health and Nutrition Examination Survey (1988 to 1994).

cohort Study of Health in Pomerania, NAFLD incidence increased in participants with prior elevated periodontal clinical attachment loss (Akinkugbe et al. 2017). Other investigators found a significant relationship between liver steatosis and periodontitis, especially among people with advanced rather than mild liver disease (Alazawi et al. 2017).

NAFLD and NASH are diagnosed in several ways. A liver biopsy is the most definitive, but it is invasive and costly, can have complications, and is not used for screening purposes. Ultrasonography (USON) has become the standard noninvasive diagnostic method, but this imaging technique is not feasible or available for most population-based field studies. Other approaches have been developed through a combination of demographic and clinical and/or laboratory variables, such as liver enzymes, to construct an algorithm for low-cost screening purposes.

We had 2 aims for this secondary data analysis: first, to explore the association between NAFLD and the prevalence of periodontitis, dental caries, and permanent tooth loss among a nationally representative sample of US adults, controlling for selected potential confounders; second, to determine if the same NAFLD associations with oral health status with USON are found with other noninvasive screening methods of categorizing NAFLD.

Methods

Study Population

Data from the cross-sectional National Health and Nutrition Examination Survey, 1988 to 1994 (NHANES III), were analyzed because USON information is only available for these years. In our study, we included dentate persons aged 21 to 74 y with USON rated as "confident" or "absolute" based on NHANES III criteria (Centers for Disease Control and Prevention 2011). Participants were excluded if they had an incomplete dental examination, had not been fasting (required for some laboratory measurements), or had other etiologies for liver disease (e.g., classified with "significant alcohol consumption" [Chalasani et al. 2012], or had hepatitis B/C). People who had transferrin >50% were not part of the study sample, because excessive iron from hereditary hemochromatosis can lead to liver dysfunction and fibrosis (Cheng et al. 2009). See Figure 1 for selection details. Survey participants provided written informed consent, and additional details regarding NHANES III are located elsewhere (Centers for Disease Control and Prevention 2017b).

Sociodemographic Variables

Demographic characteristics, such as age, sex, education, race/ethnicity, and marital status, were obtained from the NHANES III household interview. Categorizations of these variables are shown in Table 1.

Poverty status categories were determined by comparing the ratio of family income with the US Department of Health and Human Services' federal poverty guidelines (Dye et al. 2017).

Clinical and Behavioral Variables

Based on the average of 3 measures, hypertension was diagnosed if systolic blood pressure was ≥130 mm Hg or diastolic blood pressure was ≥85 mm Hg. A person was considered

Table 1. Distribution of Demographic, Behavioral, Clinical, and Oral Health Characteristics in Analytic Sample: NHANES III (1988 to 1994).

	% (SE) or Mean ± SE				
	All (*N* = 5,421)	USON Positive (*n* = 1,030, 17%)	FS > −1.455 (*n* = 1,337, 24%)	FLI > 30 (*n* = 1,089, 24%)	US-FLI > 30 (*n* = 794, 22%)
Demographic characteristics					
Age, y					
21 to 39	56.56 (1.25)	12.44 (1.20)	6.67 (0.75)	17.85 (1.44)	12.89 (1.44)
40 to 59	31.68 (1.13)	21.93 (1.80)	36.16 (1.49)	30.30 (2.26)	29.08 (2.07)
60 to 74	11.76 (0.75)	23.84 (2.20)	72.98 (2.29)	32.18 (1.85)	39.66 (2.73)
Sex					
Female	52.14 (0.99)	20.56 (1.72)	22.49 (1.17)	29.41 (2.11)	28.38 (1.78)
Male	47.86 (0.99)	13.32 (1.04)	25.68 (1.37)	18.11 (1.03)	16.52 (1.54)
Race/ethnicity					
Non-Hispanic White	75.41 (1.36)	16.44 (1.12)	25.08 (1.02)	23.39 (1.63)	21.28 (1.66)
Non-Hispanic Black	11.19 (0.75)	12.17 (1.24)	26.86 (1.90)	26.40 (1.44)	13.59 (1.25)
Mexican American	6.17 (0.56)	24.63 (2.25)	15.10 (1.16)	28.02 (1.59)	41.60 (2.77)
Other race[a]	7.23 (1.01)	20.86 (4.22)	18.12 (3.02)	18.21 (2.78)	26.64 (4.74)
Education					
Less than high school	18.54 (1.19)	21.48 (2.11)	30.99 (2.03)	31.19 (1.70)	30.34 (2.14)
High school/GED or equivalent	34.26 (0.96)	17.75 (1.69)	26.37 (1.85)	25.02 (1.99)	26.19 (2.03)
More than high school	47.2 (1.36)	14.25 (1.43)	19.82 (1.34)	19.52 (1.43)	16.09 (1.63)
Marital status: married	64.88 (1.09)	19.18 (1.31)	25.89 (1.10)	26.06 (1.74)	24.61 (1.68)
Poverty status, FPG					
<100%	11.31 (0.86)	16.41 (1.88)	22.69 (2.30)	26.43 (2.50)	23.49 (2.55)
100% to 199%	20.13 (1.24)	17.69 (2.14)	24.45 (2.43)	26.62 (1.94)	26.72 (2.36)
≥200%	68.56 (1.50)	16.80 (1.25)	23.88 (1.25)	22.31 (1.78)	20.38 (2.01)
Behavioral and clinical characteristics					
Hypertension	27.45 (1.13)	27.50 (2.22)	44.29 (2.30)	41.93 (1.94)	38.33 (2.19)
Diagnosed diabetes	15.86 (0.85)	34.96 (2.63)	73.72 (2.09)	50.54 (3.22)	62.09 (3.11)
Abnormal HDL	40.11 (1.41)	24.30 (1.96)	29.57 (1.73)	40.09 (2.37)	36.13 (2.62)
Elevated triglycerides	25.39 (1.32)	34.19 (2.32)	33.08 (2.43)	56.52 (2.89)	49.47 (2.96)
Elevated fasting glucose	24.10 (0.98)	30.66 (2.32)	58.18 (1.90)	43.65 (2.90)	49.63 (2.85)
Elevated AST	3.72 (0.48)	42.19 (4.66)	27.02 (3.63)	48.81 (3.45)	50.78 (4.96)
Elevated ALT	5.18 (0.56)	46.05 (4.65)	17.82 (3.07)	50.92 (4.81)	65.82 (6.04)
AST/ALT	1.40 ± 0.03	1.13 ± 0.03	1.62 ± 0.06	1.12 ± 0.03	1.07 ± 0.03
Platelet count	268.70 ± 2.70	267.78 ± 5.90	232.79 ± 2.74	266.63 ± 3.24	262 ± 3.86
Serum albumin, g/dL	4.19 ± 0.02	4.18 ± 0.03	4.02 ± 0.02	4.10 ± 0.02	4.09 ± 0.02
Smoker					
Current	26.49 (1.03)	15.45 (1.84)	18.64 (1.84)	23.52 (2.25)	18.25 (2.33)
Former	23.48 (0.81)	21.86 (2.06)	34.53 (1.44)	32.41 (3.23)	35.46 (3.14)
Never	50.03 (1.18)	15.11 (1.04)	22.09 (1.57)	19.35 (1.20)	17.02 (1.24)
BMI					
<25	45.37 (1.21)	6.83 (0.85)	13.14 (1.53)	0.42 (0.09)	2.47 (0.53)
≥25 to <30	33.43 (0.90)	16.21 (1.29)	25.87 (1.70)	19.27 (1.63)	21.46 (2.05)
≥30	21.21 (1.03)	39.00 (2.55)	43.93 (2.91)	78.21 (2.05)	63.83 (2.69)
Central obesity[b]	32.51 (1.06)	32.54 (1.95)	43.70 (2.08)	57.54 (2.04)	49.34 (2.34)
Oral health characteristics					
Caries experience	94.54 (0.62)	16.87 (1.08)	24.45 (0.89)	23.37 (1.40)	22.07 (1.50)
Untreated dental caries	27.38 (1.24)	20.45 (1.52)	27.42 (2.05)	32.90 (2.25)	28.32 (2.29)
DMFT	11.96 ± 0.18	13.21 ± 0.39	16.01 ± 0.26	13.24 ± 0.31	14.32 ± 0.36
DFT	8.75 ± 0.18	8.62 ± 0.30	9.61 ± 0.29	8.38 ± 0.21	8.85 ± 0.27
DMFS	36.96 ± 0.75	43.86 ± 1.86	57.41 ± 1.34	44.69 ± 1.6	50.30 ± 1.78
DFS	21.48 ± 0.58	21.88 ± 1.16	26.76 ± 0.94	21.38 ± 0.69	24.08 ± 1.12
DS	1.66 ± 0.11	2.18 ± 0.31	1.74 ± 0.18	2.34 ± 0.26	2.01 ± 0.24
DS/DFS	13.03 (0.79)	14.77 (1.61)	13.49 (1.45)	18.34 (1.32)	16.20 (1.31)
No. of permanent teeth	24.36 ± 0.11	22.96 ± 0.31	21.30 ± 0.34	22.77 ± 0.32	22.22 ± 0.28
<20 permanent teeth	12.85 (0.66)	26.06 (2.68)	54.41 (2.41)	39.75 (3.28)	41.08 (3.21)
Gingival recession, mm	0.21 ± 0.01	0.33 ± 0.04	0.46 ± 0.03	0.31 ± 0.04	0.37 ± 0.03
Pocket depth, mm	1.45 ± 0.02	1.54 ± 0.03	1.49 ± 0.03	1.52 ± 0.03	1.51 ± 0.03
Loss of attachment, mm	1.00 ± 0.03	1.23 ± 0.07	1.51 ± 0.05	1.24 ± 0.06	1.35 ± 0.06
Periodontal disease	12.26 (1.01)	25.08 (2.39)	38.63 (2.62)	37.28 (2.77)	34.42 (3.01)
Moderate or severe periodontitis	8.82 (0.52)	27.32 (4.29)	45.88 (2.92)	36.42 (3.99)	39.85 (3.13)

ALT, alanine aminotransferase; AST, aspartate aminotransferase; BMI, body mass index; DFS, decayed and filled surfaces; DFT, decayed and filled teeth; DMFS, decayed, missing and filled surfaces; DMFT, decayed, missing and filled teeth; DS, decayed surfaces; FLI, Fatty Liver Index; FPG, federal poverty guideline; FS, Fibrosis Score; GED, General Education Diploma; HDL, high-density lipoprotein; NHANES, National Health and Nutrition Examination Survey; US-FLI, US Fatty Liver Index; USON, ultrasonography.
Column % in "All" column; row % in others.
[a]Including multiracial.
[b]Waist circumference.

diabetic if diagnosed as diabetic, if she or he was taking insulin or medication for diabetes, if the fasting plasma glucose was >126 mg/dL, or if the nonfasting plasma glucose (first or second venipuncture) was >200 mg/dL. Abnormal HDL (high-density lipoprotein) cholesterol was <40 mg/dL for men or <50 mg/dL for women. Serum triglycerides level was considered elevated if ≥150 mg/dL. AST (aspartate aminotransferase) >37 U/L for men or >31 U/L for women was treated as elevated AST; similarly, ALT (alanine aminotransferase) >40 U/L for men or >31 U/L for women was considered elevated. Cigarette smoking was categorized as current, never, and former. Body mass index (BMI; kg/m^2) was grouped into 3 categories: normal, overweight, and obese (Centers for Disease Control and Prevention 2017a), and central obesity was defined as waist circumference >102 cm for men and >88 cm for women.

NAFLD Definitions

NAFLD was assessed with 4 criteria: USON and 3 algorithms with noninvasive measures (Appendix Table 1).

The gold standard was based on the USON examination. NAFLD was deemed present if the hepatic steatosis assessment was moderate-severe. The technical and clinical methods are documented elsewhere (Westat Inc. 1988).

The Fibrosis Score (FS; Angulo et al. 2007) was calculated with an algorithm that includes 6 variables (age, BMI, hyperglycemia as a measure of diabetes, platelet count, the liver enzymes AST/ALT ratio, and albumin). A value < –1.455 is considered the absence of significant fibrosis and >0.676 the presence of significant fibrosis. The cut point ≥ –1.455 was used in this analysis to categorize intermediate and advanced fibrosis. The FS is designed to detect advanced fibrosis and has an indeterminate area between cut points.

The Fatty Liver Index (FLI) was developed in Italy as a simple way to use routine clinical and laboratory measures to identify patients at greater risk for fatty liver for further evaluation and lifestyle counseling (Bedogni et al. 2006). The algorithm uses 4 variables: BMI, waist circumference, triglycerides, and gamma-glutamyl-transferase. The FLI varies between 0 and 100, and the cut point of <30 is used to rule out fatty liver and ≥60 to rule it in (Bedogni et al. 2006). We used the <30 cut point in this analysis.

The US Fatty Liver Index (US-FLI; Ruhl and Everhart 2015) was developed for better prediction than the FLI in a multiethnic population such as the United States. It incorporates race/ethnicity and age, which are not included in the FLI, and a different set of biomarkers. A score ≥30 is considered NAFLD for the US-FLI.

Oral Health Variables

Dental caries experience was defined as ≥1 decayed or filled permanent tooth surfaces and untreated dental caries as having any decayed permanent tooth surfaces. Permanent tooth loss experience, excluding third molars, was categorized as <20 teeth versus ≥20 teeth, as this number of teeth has been considered necessary for a functional occlusion (Gotfredsen and Walls 2007). The prevalence of moderate periodontitis was defined as ≥2 teeth with loss of attachment ≥4 mm at interproximal sites or ≥2 teeth with pocket depth ≥5 mm at interproximal sites. Severe periodontitis was defined as ≥2 teeth with loss of attachment ≥6 mm at interproximal sites and ≥1 teeth with pocket depth ≥5 mm at interproximal sites. For this study, moderate and severe were combined. Details of the dental caries and periodontal measurements can be found elsewhere (Drury et al. 1996; Page and Eke 2007).

Statistical Analysis

Descriptive analyses were conducted to calculate estimates and standard errors with Taylor series linearization. Indicator variables were used for coding hypertension, diabetes, and other elevated markers; most laboratory measures were analyzed as continuous variables. Unadjusted and adjusted multivariable logistic regression models were developed to examine the relationship among oral health variables (moderate-severe periodontitis, untreated caries, caries experience, and tooth loss) while controlling for sociodemographic factors, with the NAFLD condition as the outcome. Odds ratios (ORs) and 95% CIs were calculated with logistic regression.

Our modeling approach began with developing unadjusted models, followed by including all the covariates in multivariable models (full models). Last, we used a backward selection procedure to produce final models containing only significant covariates ($P \leq 0.05$) associated with the 4 NAFLD measures. Receiver operating characteristic (ROC) curves were calculated to the 3 NAFLD algorithms to the USON findings. The ROC curve graphs the true-positive (sensitivity) and false-positive (1 – specificity) rates on the *y*- and *x*-axes, respectively, with area under the curve calculated. All the analyses were conducted in SAS 9.4 software with the Survey Procedures (SAS Institute Inc.) and STATA 15.1 (StataCorp) incorporating survey design and sample weight variables as appropriate.

Results

Population Characteristics

The characteristics of the population studied (N = 5,421) are shown in Table 1. Just over half (57%) were 21 to 39 y old; 32%, 40 to 59 y; and 12%, 60 to 74 y. About half (52%) were women. The majority (75%) were non-Hispanic White; 11%, non-Hispanic Black; 6%, Mexican American; and 7%, "other." The majority (81%) had at least a high school education. Sixty-eight percent were ≥200% of federal poverty guidelines. About a fourth of the population was diagnosed with hypertension, elevated triglycerides, and elevated fasting glucose and was a current smoker. Among participants, 16% had diabetes, 21% were considered obese; and 33% had excessive waist circumference.

Twenty-seven percent had untreated dental caries, and 13% had <20 permanent teeth. The mean numbers of permanent

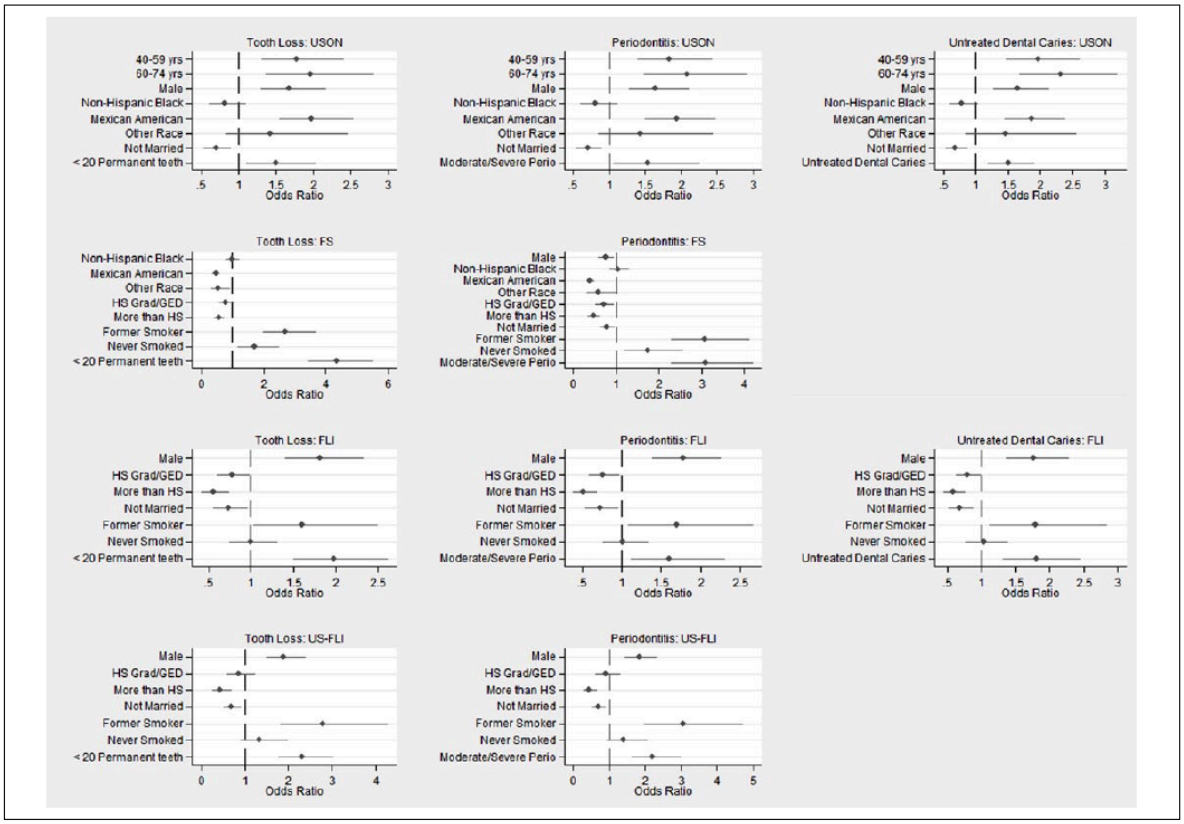


Figure 2. Odds ratios and 95% CIs for variables in final logistic regression models for tooth loss, periodontitis, and untreated dental caries with the use of different nonalcoholic fatty liver disease measures. FLI, Fatty Liver Index; FS, Fibrosis Score; GED, General Education Diploma; HS, high school; US-FLI, US Fatty Liver Index; USON, ultrasonography.

teeth and decayed and filled teeth were 24.36 and 8.75, respectively. The prevalence of moderate or severe periodontal disease was 9%. The USON prevalence of NAFLD was 17% but was higher when assessed by the algorithms, ranging from 22% (US-FLI) to 24% (FLI and FS).

Relationships between NAFLD and Each Oral Health Measure

Figure 2 shows the ORs and 95% CIs for the oral health measures and covariates in the final regression models for the NAFLD assessments, graphed as forest plots. The significant ORs for the models are summarized in Table 2. In the United States, NAFLD as determined by USON was associated with untreated dental caries (OR = 1.51, 95% CI = 1.20 to 1.90), moderate or severe periodontitis (OR = 1.54, 95% CI = 1.06 to 2.24), and having <20 teeth (OR = 1.50, 95% CI = 1.11 to 2.02) but not overall caries experience. The findings were similar per the FLI, with slightly higher ORs and wider CIs. When NAFLD was determined with the US-FLI and FS, there were significant relationships with moderate or severe periodontitis (OR = 2.21, 95% CI = 1.64 to 2.98, and OR = 3.10, 95% CI = 2.31 to 4.17, respectively) and with <20 teeth present (OR = 2.32, 95% CI = 1.79 to 3.01, and OR = 4.36, 95% CI = 3.47 to 5.49, respectively) but not with dental caries (caries experience or untreated caries).

Relationships between Demographic Covariates and NAFLD Measures

The Appendix Tables show the progression from unadjusted models to full models, including all covariates and then removal of insignificant covariates to produce the final models. For USON (Appendix Table 2), smoking was associated with NAFLD in 4 of 5 unadjusted models, but the association disappeared in those full model groups, remaining insignificant in final models. Being non-Hispanic Black was also associated with NAFLD but in the opposite direction as smoking. Both followed similar patterns: significant in unadjusted models, not significant in multivariate models. Generally, being older, male, Mexican American, and unmarried was associated with NAFLD (USON) in unadjusted models and remained

Table 2. NAFLD Final Models and Oral Health Indicators.

	Oral Health Status								
	<20 Permanent Teeth			Prevalence of Moderate or Severe Periodontitis			Prevalence of Untreated Caries (≥1 DS)		
NAFLD Assessments	OR	LCI	UCI	OR	LCI	UCI	OR	LCI	UCI
USON	1.50	1.11	2.02	1.54	1.06	2.24	1.51	1.20	1.90
FS	4.36	3.47	5.49	3.10	2.31	4.17	—	—	—
FLI	1.99	1.52	2.59	1.61	1.13	2.28	1.80	1.33	2.44
US-FLI	2.32	1.79	3.01	2.21	1.64	2.98	—	—	—

Dashes (—) indicate not statistically significant at $P < 0.05$.
DS, decayed surfaces; FLI, Fatty Liver Index; FS, Fibrosis Score; LCI, lower 95% CI; NAFLD, nonalcoholic fatty liver disease; OR, odds ratio; UCI, upper 95% CI; US-FLI, US Fatty Liver Index; USON, ultrasonography.

Table 3. ROC Analysis Comparing Ultrasonography Assessment with Other NAFLD Assessments.

Criteria	AUROC	SE	95% CI	Sensitivity, %	Specificity, %	Correctly Classified, %	PPV, %	NPV, %
FS	0.57	0.01	0.56 to 0.59	39.18	76.12	69.12	27.75	84.25
FLI	0.69	0.01	0.68 to 0.72	59.68	79.81	75.94	41.32	89.26
US-FLI	0.72	0.01	0.71 to 0.75	64.31	81.55	78.18	45.84	90.39

AUROC, area under the receiver operating characteristic; FLI, Fatty Liver Index; FS, Fibrosis Score; NAFLD, nonalcoholic fatty liver disease; NPV, negative predictive value; PPV, positive predictive value; ROC, receiver operating characteristic; US-FLI, US Fatty Liver Index.

significant in the final multivariate models for all 4 groups. For 3 algorithm-derived NAFLD screening tools, low education, unmarried status, and smoking remained significant in the final models; however, being Mexican American remained significant in the final FS model, whereas being male remained associated with NAFLD in the FLI and US-FLI final models.

Relationship among NAFLD Measures

ROC curves are shown in the Appendix Figure. The area under the ROC curve (Table 3) was least for the FS (0.57), greater for the FLI (0.69), and greatest for the US-FLI (0.72). The FLI and US-FLI areas under the curve were significantly different from that of the FS. Thus, if USON is not available, the US-FLI provides the best NAFLD diagnostic estimate of these 3 other noninvasive assessments in the US population.

Discussion

We found significant associations between moderate-severe periodontitis and tooth loss with all 4 NAFLD assessments, though only 2 assessments were associated with untreated dental caries. More important, the best performing of the 3 screening alternatives to USON, the US-FLI, was unable to show an association with untreated dental caries. This raises an important question for public health researchers: the selection of the screening tool and how that may affect any potential predictive explanatory variables being assessed—in this case, oral health variables. Our findings suggest that choosing the best screening assessment (US-FLI) would result in an inability to explore a potential relationship between NAFLD and dental caries, when we know that an association exists with the standard USON. Choosing a slightly less predictive screening assessment (e.g., FLI) would enhance the researcher's ability to ascertain an association between NAFLD and dental caries. There may be more misclassification with the algorithmic screening tools than with ultrasound, as more variability is introduced with each risk marker.

The analytic sample was limited to those 21 to 74 y old with complete data for key explanatory variables. The greatest number of people were excluded because an USON assessment was not available. A comparison of this age group in the analytic sample with those excluded indicated no substantial differences by sex, but the mean age of the analytic sample was older, and there were some differences by race/ethnicity, with more non-Hispanic Blacks and Mexican Americans in the sample. The US-FLI is the only NAFLD assessment that includes race/ethnicity in the algorithm. This factor may explain the better fit of this measure.

In a systematic review, there was a significant association between NAFLD and clinical microbial periodontal parameters in 11 of 12 studies (Alakhali et al. 2018). Qiao and colleagues (2018) found a positive association between NAFLD and self-reported categories of tooth loss in males but not females. In our study, there was a greater odds of tooth loss positively associated with NAFLD in men than women with 3 of the 4 NAFLD measures.

Other investigators have noted the higher fatty liver prevalence among men as compared with women and among Mexican Americans as compared with non-Hispanic Whites, with a lower prevalence among non-Hispanic Blacks as compared with non-Hispanic Whites in the United States (Ruhl and

Everhart 2015). However, the prevalence among Hispanics varies with country of origin (Fleischman et al. 2014). Risk can also vary by disease severity—for example, male sex and Mexican American ethnicity have been identified as risk factors for NAFLD but not advanced fibrosis (Le et al. 2017).

Although USON is a well-accepted technique, the sensitivity and specificity can vary depending on the operator, the presence of obesity, and the extent of steatosis (Ballestri et al. 2017). Because USON is limited in its ability to detect inflammation (Li et al. 2018), there are newer diagnostic imaging tests available, such as transient elastography, magnetic resonance elastography, resonance-based fat quantitative techniques, and ultrasound elastography, though each has limitations, including cost and clinical availability (Li et al. 2018; Younossi et al. 2018). Comparisons of diagnostic performance of some of the different noninvasive NAFLD methods have been made (Shah et al. 2009; McPherson et al. 2010; Aykut et al. 2014; Cui et al. 2015) with results varying depending on the stage of the disease process (Aykut et al. 2014). Another panel of biomarkers, the Fibrosis-4 score (Sterling et al. 2006), had better areas under the ROC as compared with other noninvasive fibrosis panels but was not significantly different from the FS (Shah et al. 2009). More important, the noninvasive tests generally have a higher negative predictive value than positive predictive value and are better at screening to exclude people with advanced fibrosis among those with NAFLD (McPherson et al. 2010). Since effective therapeutic treatment is lacking, general screening for people without other NAFLD risk factors, such as type 2 diabetes or metabolic syndrome, is not recommended (Chalasani et al. 2012; European Association for the Study of the Liver et al. 2016).

The associations found have implications for health care, as oral conditions may be part of the constellation of either risk factors or consequences of NAFLD, or both. Many people do not know if they have NAFLD. As dentists gain access to medical records with the demographic and laboratory information needed for input into algorithmic NAFLD screening tools, they will have the potential means to evaluate NAFLD risk factors and refer patients for medical assessment as necessary. Conversely, our findings suggest that primary care providers whose patients have NAFLD may want to advise them to see their dentist and hygienist because of NAFLD associations with poor oral health status.

Strengths and Limitations

Our study has several strengths. NHANES III is a large, nationally representative study. People are selected randomly, not on the basis of any preexisting disease or condition, from a variety of geographic areas. Many clinical and biological variables were available, including NAFLD and clinically determined oral health measures, a rare combination. The dental caries data are high quality, since the examination is comprehensive, and dental examiners are trained and calibrated. There was masking in the sense that no examiners knew who had NAFLD.

The gold standard used was USON; thus, "fibrosis" was not confirmed by liver biopsy. We used 3 widely reported assessment algorithms for NAFLD, which are noninvasive screening tools with differing diagnostic utility. We explored how using these screening tools would affect associations between NAFLD and oral conditions when imaging or other more expensive methods were not available for epidemiologic studies, and we found some differences regarding caries. We used older data since USON measurements were not obtained in the more recent NHANES.

Children and adults aged ≥75 y did not receive USON, which limits the generalizability of our analysis with respect to our youngest and oldest populations. The oral conditions studied and NAFLD prevalence all increase with age (Dye et al. 2007; Chalasani et al. 2012). NHANES III used only a partial mouth design for periodontal assessments. We know that this substantially underestimates the prevalence of periodontal disease (Eke et al. 2010). Although NAFLD and oral diseases share common risk factors, it was not the primary aim of this study to tease out specific predictors or pathways to better explain causality or directionality. Variables known to be associated with periodontal disease, such as diabetes, were included in ≥1 algorithms and thus could not be used as independent variables. Many potential explanatory factors were not included, such as cariogenic or periodontal pathogens or sugar consumption, because information was not available, limited, or not useful.

Conclusion

In this study based on a large, representative US population, NAFLD was significantly associated with tooth loss, moderate to severe periodontitis, and, for some NAFLD measures, untreated caries, after adjusting for several key sociodemographic factors. The US-FLI provided information in greater agreement with the USON measure than the FS or FLI. Our findings emphasize the importance of understanding the connections between diseases in the oral cavity and other organ systems.

Given that NAFLD prevalence is increasing, studies with contemporary data are needed to better understand its relationship to oral health. Results suggest another oral-systemic link and potential opportunities for medical-dental integration.

Author Contributions

J.A. Weintraub, B.A. Dye, contributed to conception, design, data analysis, and interpretation, drafted and critically revised the manuscript; G. Lopez Mitnik, contributed to design, data analysis, and interpretation, drafted and critically revised the manuscript. All authors gave final approval and agree to be accountable for all aspects of the work.

Acknowledgments

This research was partially supported by the National Institute of Dental and Craniofacial Research, and 2 of the authors were paid

a salary by the National Institutes of Health. The authors declare no potential conflicts of interest with respect to the authorship and/or publication of this article.

ORCID iD

J.A. Weintraub https://orcid.org/0000-0001-7178-2028

References

Akinkugbe AA, Barritt AS, Cai J, Offenbacher S, Thyagarajan B, Khambaty T, Singer R, Kallwitz E, Heiss G, Slade GD. 2017. Periodontitis and non-alcoholic fatty liver disease, a population-based cohort investigation in the Study of Health in Pomerania. J Clin Periodontol. 44(11):1077–1087.

Alakhali MS, Al-Maweri SA, Al-Shamiri HM, Al-Haddad K, Halboub E. 2018. The potential association between periodontitis and non-alcoholic fatty liver disease: a systematic review. Clin Oral Investig. 22(9):2965–2974.

Alazawi W, Bernabe E, Tai D, Janicki T, Kemos P, Samsuddin S, Syn WK, Gillam D, Turner W. 2017. Periodontitis is associated with significant hepatic fibrosis in patients with non-alcoholic fatty liver disease. PLoS One. 12(12):e0185902.

Angulo P, Hui JM, Marchesini G, Bugianesi E, George J, Farrell GC, Enders F, Saksena S, Burt AD, Bida JP, et al. 2007. The NAFLD fibrosis score: a noninvasive system that identifies liver fibrosis in patients with NAFLD. Hepatology. 45(4):846–854.

Aykut UE, Akyuz U, Yesil A, Eren F, Gerin F, Ergelen R, Celikel CA, Yilmaz Y. 2014. A comparison of FibroMeter NAFLD Score, NAFLD fibrosis score, and transient elastography as noninvasive diagnostic tools for hepatic fibrosis in patients with biopsy-proven non-alcoholic fatty liver disease. Scand J Gastroenterol. 49(11):1343–1348.

Ballestri S, Nascimbeni F, Baldelli E, Marrazzo A, Romagnoli D, Targher G, Lonardo A. 2017. Ultrasonographic fatty liver indicator detects mild steatosis and correlates with metabolic/histological parameters in various liver diseases. Metab Clin Exp. 72:57–65.

Bedogni G, Bellentani S, Miglioli L, Masutti F, Passalacqua M, Castiglione A, Tiribelli C. 2006. The fatty liver index: a simple and accurate predictor of hepatic steatosis in the general population. BMC Gastroenterol. 6:33.

Centers for Disease Control and Prevention. 2011. National Health and Nutrition Examination Survey, 1988–1994. Data documentation, codebook, and frequencies. Hepatic/gallbladder ultrasound and hepatic steatosis (HGUHS); [accessed 2018 Aug 22]. https://wwwn.cdc.gov/nchs/data/nhanes3/34a/HGUHS.htm#GUPCHSC.

Centers for Disease Control and Prevention. 2017a. About adult BMI. Atlanta, GA; Centers for Disease Control and Prevention; [accessed 2018 Oct 3]. https://www.cdc.gov/healthyweight/assessing/bmi/adult_bmi/index.html.

Centers for Disease Control and Prevention. 2017b. National Health and Nutrition Examination Survey, 1988–1994. Questionnaires, datasets, and related documentation; [accessed 18 Oct 2018]. https://wwwn.cdc.gov/nchs/nhanes/nhanes3/default.aspx.

Chalasani N, Younossi Z, Lavine JE, Diehl AM, Brunt EM, Cusi K, Charlton M, Sanyal AJ. 2012. The diagnosis and management of non-alcoholic fatty liver disease: practice guideline by the American Association for the Study of Liver Diseases, American College of Gastroenterology, and the American Gastroenterological Association. Hepatology. 55(6):2005–2023.

Cheng R, Barton JC, Morrison ED, Phatak PD, Krawitt EL, Gordon SC, Kowdley KV. 2009. Differences in hepatic phenotype between hemochromatosis patients with HFE C282Y homozygosity and other HFE genotypes. J Clin Gastroenterol. 43(6):569–573.

Cui J, Ang B, Haufe W, Hernandez C, Verna EC, Sirlin CB, Loomba R. 2015. Comparative diagnostic accuracy of magnetic resonance elastography vs. eight clinical prediction rules for non-invasive diagnosis of advanced fibrosis in biopsy-proven non-alcoholic fatty liver disease: a prospective study. Aliment Pharmacol Ther. 41(12):1271–1280.

Drury TF, Winn DM, Snowden CB, Kingman A, Kleinman DV, Lewis B. 1996. An overview of the oral health component of the 1988–1991 National Health and Nutrition Examination Survey (NHANES III–Phase 1). J Dent Res. 75 Spec No.:620–630.

Dye BA, Mitnik GL, Iafolla TJ, Vargas CM. 2017. Trends in dental caries in children and adolescents according to poverty status in the United States from 1999 through 2004 and from 2011 through 2014. J Am Dent Assoc. 148(8):550–565.e7.

Dye BA, Tan S, Smith V, Lewis BG, Barker LK, Thornton-Evans G, Eke PI, Beltran-Aguilar ED, Horowitz AM, Li CH. 2007. Trends in oral health status: United States, 1988–1994 and 1999–2004. National Center for Health Statistics. Vital Health Stat. 11(248):1–92.

Eke PI, Thornton-Evans GO, Wei L, Borgnakke WS, Dye BA. 2010. Accuracy of NHANES periodontal examination protocols. J Dent Res. 89(11):1208–1213.

European Association for the Study of the Liver, European Association for the Study of Diabetes, European Association for the Study of Obesity. 2016. EASL-EASD-EASO clinical practice guidelines for the management of non-alcoholic fatty liver disease. Obes Facts. 9(2):65–90.

Fleischman MW, Budoff M, Zeb I, Li D, Foster T. 2014. NAFLD prevalence differs among Hispanic subgroups: the Multi-ethnic Study of Atherosclerosis. World J Gastroenterol. 20(17):4987–4993.

Gotfredsen K, Walls AWG. 2007. What dentition assures oral function? Clin Oral Implants Res. 18 Suppl 3:34–45.

Han P, Sun D, Yang J. 2016. Interaction between periodontitis and liver diseases. Biomed Rep. 5(3):267–276.

Le MH, Devaki P, Ha NB, Jun DW, Te HS, Cheung RC, Nguyen MH. 2017. Prevalence of non-alcoholic fatty liver disease and risk factors for advanced fibrosis and mortality in the United States. PLoS One. 12(3):e0173499.

Li Q, Dhyani M, Grajo JR, Sirlin C, Samir AE. 2018. Current status of imaging in nonalcoholic fatty liver disease. World J Hepatol. 10(8):530–542.

Lonardo A, Ballestri S, Marchesini G, Angulo P, Loria P. 2015. Nonalcoholic fatty liver disease: a precursor of the metabolic syndrome. Dig Liver Dis. 47(3):181–190.

Lustig RH. 2014. Fat chance: beating the odds against sugar, processed food, obesity and disease. New York (NY): PLUME, Penguin Group.

McPherson S, Stewart SF, Henderson E, Burt AD, Day CP. 2010. Simple non-invasive fibrosis scoring systems can reliably exclude advanced fibrosis in patients with non-alcoholic fatty liver disease. Gut. 59(9):1265–1269.

Naka S, Nomura R, Takashima Y, Okawa R, Ooshima T, Nakano K. 2014. A specific *Streptococcus mutans* strain aggravates non-alcoholic fatty liver disease. Oral Dis. 20(7):700–706.

Naka S, Wato K, Hatakeyama R, Okawa R, Nomura R, Nakano K. 2018. Longitudinal comparison of *Streptococcus mutans*–induced aggravation of non-alcoholic steatohepatitis in mice. J Oral Microbiol. 10(1):1428005.

Page RC, Eke PI. 2007. Case definitions for use in population-based surveillance of periodontitis. J Periodontol. 78(7 Suppl):1387–1339.

Qiao F, Fu K, Zhang Q, Liu L, Meng G, Wu H, Xia Y, Bao X, Gu Y, Shi H, et al. 2018. The association between missing teeth and non-alcoholic fatty liver disease in adults. J Clin Periodontol. 45(8):941–951.

Ruhl CE, Everhart JE. 2015. Fatty liver indices in the multiethnic United States National Health and Nutrition Examination Survey. Aliment Pharmacol Ther. 41(1):65–76.

Shah AG, Lydecker A, Murray K, Tetri BN, Contos MJ, Sanyal AJ; Nash Clinical Research Network. 2009. Comparison of noninvasive markers of fibrosis in patients with nonalcoholic fatty liver disease. Clin Gastroenterol Hepatol. 7(10):1104–1112.

Softic S, Cohen DE, Kahn CR. 2016. Role of dietary fructose and hepatic de novo lipogenesis in fatty liver disease. Dig Dis Sci. 61(5):1282–1293.

Sterling RK, Lissen E, Clumeck N, Sola R, Correa MC, Montaner J, Sulkowski MS, Torriani FJ, Dieterich DT, Thomas DL, et al; APRICOT Clinical Investigators. 2006. Development of a simple noninvasive index to predict significant fibrosis in patients with HIV/HCV coinfection. Hepatology. 43(6):1317–1325.

Westat Inc. 1988. National Health and Nutrition Examination Survey. Gallbladder ultrasonography procedure manual; [accessed 2018 Oct 18]. https://wwwn.cdc.gov/nchs/data/nhanes3/manuals/gallblad.pdf.

Yki-Järvinen H. 2014. Non-alcoholic fatty liver disease as a cause and a consequence of metabolic syndrome. Lancet Diabetes Endocrinol. 2(11):901–910.

Yoneda M, Naka S, Nakano K, Wada K, Endo H, Mawatari H, Imajo K, Nomura R, Hokamura K, Ono M, et al. 2012. Involvement of a periodontal pathogen, *Porphyromonas gingivalis* on the pathogenesis of non-alcoholic fatty liver disease. BMC Gastroenterol. 12:16.

Younossi ZM, Koenig AB, Abdelatif D, Fazel Y, Henry L, Wymer M. 2016. Global epidemiology of nonalcoholic fatty liver disease: meta-analytic assessment of prevalence, incidence, and outcomes. Hepatology. 64(1):73–84.

Younossi ZM, Loomba R, Anstee QM, Rinella ME, Bugianesi E, Marchesini G, Neuschwander-Tetri BA, Serfaty L, Negro F, Caldwell SH, et al. 2018. Diagnostic modalities for non-alcoholic fatty liver disease (NAFLD), non-alcoholic steatohepatitis (NASH) and associated fibrosis. Hepatology. 68(1):349–360.

Original Paper

Clinical Study on Abfraction Lesions in Occlusal Dysfunction

ADI RUSU OLARU[1], MIHAI RAUL POPESCU[1], LUCIAN PAUL DRAGOMIR[1], ANNE-MARIE RAUTEN[2]

[1]Department of Occlusology and Fixed Prosthetics, Faculty of Dental Medicine, University of Medicine and Pharmacy of Craiova, Romania

[2]Department of Orthodontics, Faculty of Dental Medicine, University of Medicine and Pharmacy of Craiova, Romania

ABSTRACT: The abfraction theory states that under the action of the occlusal forces non-axially transmitted, the flexion of the tooth occurs in the cervical area, which initially leads to the appearance of cracks in the enamel and dentin, followed by the destruction of the dental structure. These lesions allow bacterial plaque retention, lead to dental hypersensitivity and can affect the vitality of the dental pulp. Thus, the study included 102 participants, of both sexes, 54% representing the male gender (55 subjects) and 46% the female gender (47 subjects), aged between 20 and 80, from the urban area 76% (77 subjects) and rural 24% (25 subjects), who came to the Dental Medicine office, between August 2018 and August 2019, representing 57.3%, of the total number of patients treated during the aforementioned period. They have been described the acid and abrasive processes involved in the generation of these lesions,and special attention was paid to the role of mechanical stress occurring at the occlusal level, due to the transmission of forces outside the dental axis.

KEYWORDS: *Cervical lesions, abfraction, dental abrasion, occlusal trauma.*

Introduction

The abfraction represents the pathological loss of the dental tissue structure caused by para-axial biomechanical forces, resulting in the tooth enamel, dentine and tooth pulp being distanced from the place of force application [1].

Specialized studies [2,3,4] indicate, as etiological factors for abfraction also: abrasion, erosion, corrosion or combinations thereof.

Abfraction represents a type of cervical non-carious lesion, charaterized by the loss of dental tissues with various clinical aspects.

Cervical wear can be accompanied by dental abrassion and erossion due to interaction of chemical, biological and behaviour factors.

The clinical aspect can vary based on the type and severity of the involved etiologic factors.

Special attention was paid to mechanical stress at the occlusal level due to the action of the forces transmitted outside of the dental axis.

Abfraction lessions can appear accompanied by pathological wear of the tooth as a consequence of the interaction of chemical, biological and behaviour factors [5].

The location of the abfraction lesions according to some authors [6] is mainly in the cervical area of the teeth, as this region is the most vulnerable part of the tooth. In this area the enamel has a lower quality with a lower protein and mineral content than the other dental areas.

Grippo suggested bruxism as a main cause of these lesions and described five categories of abfraction injuries: cracks, horizontal streaks, lesions in the form of a plate located at the enamel level, crescent-shaped lesions, depression on the tip of the cusp at the premolars and molars.

Depending on the damage to the dental tissue, the abfraction lesions are grouped into three types:

I. lesions present only at the enamel level;

II. enamel and dentin lesions;

III. lessions that have progressed to the level of the dental pulp.

Due to the fact that the destruction of the hard dental structures (enamel, dentine, cement) is progressive, the soft tissues of the tooth (dental pulp) can also be affected with the appearance of dental hypersensitivity.

Objectives

The purpose of this study was to analyze and highlight the different clinical aspects of the abfraction lesions, based on certain criteria of division of the studied group.

Material and Method

The study included 102 participants, of both sexes, 54% representing the male gender (55 subjects) and 46% the female gender (47 subjects), aged between 20 and 80, from the urban area 76% (77 subjects) and rural 24%

10.12865/CHSJ.45.04.07

(25 subjects), who came to the Dental Medicine office, between August 2018 and August 2019, representing 57.3%, of the total number of patients treated (Table 1).

The analysis of the different clinical aspects of the abfraction lesions was based on the use of certain criteria of division, namely: oral cavity hygiene, type of tooth affected, age, sex, environment of origin, degree of damage of dental structures in occlusal trauma, distribution of the dental elements affected by cervical lesions.

After obtaining the informed consent of the patients, the condition of the dentition, the oral hygiene, the number of teeth and their mobility, the oral symptoms of parafunction were evaluated and analyzed.

After the clinical and paraclinical examination, the observation charts were drawn up, photographies were taken and the patients were being diagnosed with changes of hard dental structure located in the cervical area.

According to the objectives pursued, the criteria based on which the patients were included in this study were the following.

The criteria for inclusion in the study: age over 20 years old; the presence of structural changes of the teeth at cervical level; the possibility to come to the dental medicine office as many times as necessary; the health state to allow the dental treatments to be carried out.

The exclusion criteria: under 18 years old; presence of psychiatric disorders; lack of availability for repeated visits to the dental medicine office.

We proceeded to question the patients in order to obtain data on how they perform the oral cavity hygiene and to verify its accomplishment with the help of bacterial plaque detectors.

The patients, part of the study, have been given a questionnaire to obtain data about their brushing technique, frequency, duration and intensity of teeth brushing.

The aim was to establish how these, associated with the parafunctions, contribute to the production of abfraction injuries.

Table 1. Questionnaire regarding oral cavity hygiene and parafunctions.

NO.	QUESTIONS	ANSWER
1	How many times per day do you perform dental brushing?	
2	How long does one dental brushing session last?	
3	What type of toothbrush do you use? (e.g. with whitening effect)	
4	Do you use abrasive toothpaste?	
5	What brushing technique do you use? (Describe how you brush your teeth)	
6	Do you use secondary means of oral cavity hygiene? (e.g. dental floss, mouthwash)	
7	Do you crack your jaws during your day or night?	

The way of hygiene of the oral cavities and the verification of its accomplishment was carried out with the help of bacterial plaque detectors.

These were in the form of sponge impregnated in solution dye, used by pressing on the faces of the teeth near the incisal edge or face occlusal, diffusing through capillary close to the gingival margin.

After the coloring of the bacterial plate, the dental surfaces with plaque were noted and plate index O'Leary was established as a percentage, dividing the number of surfaces by plate at the total number of surfaces examinated by 100.

Interpretation of values: IP<10-15% dental hygiene is good; 10-15%<IP<20-25% dental hygiene is satisfactory; 20-25%<IP<40% dental hygiene is deficient; IP >40% dental hygiene is a problem for the patient.

The oral and dental health status was evaluated and analyzed, the number of teeth affected by cervical lesions was determined by clinical examination on all surfaces, and the depth of the cervical defect was measured using the periodontal probe.

Wear surfaces generated by the parafunctions were detected.

Each patient was instructed to perform the radiological-orthopantomogram examination.

The study was approved by the University Ethics and Deontology and Scientific Commission of the University of Medicine and Pharmacy of Craiova (no.70/15.06.2018).

Results

The abfraction lesions detected showed different forms, affecting the premolars in a greater number compared to the other teeth.

Following the anamnesis, regarding the dental hygiene, it was established: 52% (53 subjects) performed the dental brushing twice a day, 37% (38 subjects) perform oral hygiene several times a day, and 11% (11 subjects) do not perform oral cavity brushing (Figure 1).

135 cervical lesions were detected, each patient presenting with 1.2 or multiple lesions at different stages of progression.

There was a higher presence of the abfraction lesions in the premolars 77% (104), in the molars 5% (7), and 18% (24) were combined canine, premolar, molar lesions.

16% (17 subjects) stated the presence of nocturnal bruxism, and in terms of depth, 46% (62) of the lesions were present only at the enamel level, 38% (51) of the lesions showed simultaneous enamel-dentine damage, and 16% (22) of the lesions showed signs of pulpal damage.

The oral hygiene was evaluated as a possible risk factor in the production of abfraction lesions and the distribution of the patients studied as follows: 69% (70 subjects) manage to remove, but not entirely the bacterial plaque using an incorrect brushing technique presenting a satisfactory hygiene, 20% (21 subjects) had an adequate brushing technique and a good hygiene, 11% (11 subjects) had poor hygiene because they did not perform oral cavity hygiene.

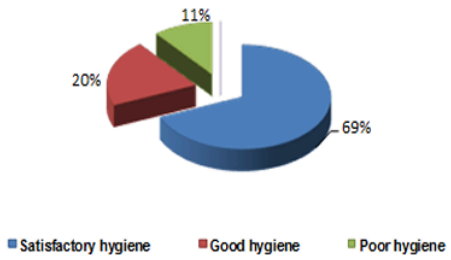


Figure 1. Subject distribution according to the oral cavity hygiene.

In the case of patients with poor hygiene, it was found the presence of untreated caries lesions, which are in various stages of progression, without experiencing pain, the presence in the treatment being imposed by the impairment of the aesthetic function.

Regarding the frequency order of dental brushing, 52% (53) of the subjects perform the dental brushing twice a day, 37% (38 subjects) several times a day and 11% (11 subjects) do not perform the daily dental brushing (Figure 2).

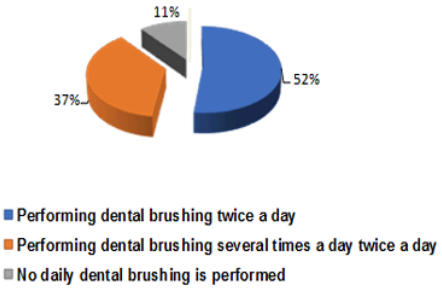


Figure 2. Frequency of dental brushing.

Regarding the duration of the dental brushing, 52% (47 subjects) of the ones that perform the brushing allocate 3-5 minutes for it and 48% (44 subjects) perform the dental brushing in less than 2 minutes (Figure 3).

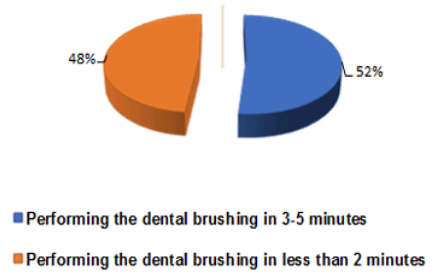


Figure 3. Time allocated to dental brushing.

The force acting on the teeth during brushing is high and the preference for toothbrushes with hard bristles was 78% (71 subjects) and 22% (20 subjects), prefers soft bristles toothbrushes and act gently during dental brushing (of the 91 who performed the daily brushing), noting the occurrence of cervical lesions with predominance in the males compared with the females (Figure 4).

10.12865/CHSJ.45.04.07

Figure 4. The acting forces on the teeth by means of the toothbrush.

The preference of some patients in the use of hard bristles toothbrushes associated with improper brushing techniques has resulted in cervical lesions.

The participants in this study presented abfraction lesions located on 1, 2, or more teeth, being in different stages of progression, having a total of 135 lesions.

Regarding the type of tooth affected, the study undertaken by us showed that: there was a significant damage due to the presence of abfraction lesions in large number at the level of premolars-77% (104 lesions) of the investigated cases; at the molar level such lesions were detected in a much smaller percentage-5% (7 lesions); and 18% (24) of the subjects had combined canine, premolar, and molar lesions (Figure 5).

There was no significant difference of the abfraction lesions present in the right or left quadrant.

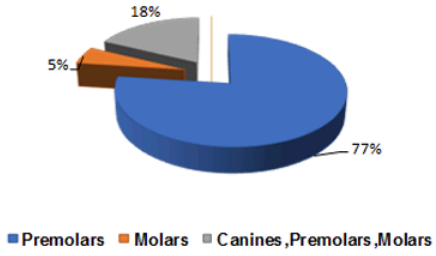


Figure 5. Distribution of the abfraction lesions according to the type of tooth affected.

The patient with abfraction lesions in the lower premolars, presenting complications due to the pain caused by inflammation of the dental pulp, so that the patient resorted to avoiding the oral cavity hygiene.

The analysis of the distributions of the abfraction lesions in the two sexes, revealed the greater presence in the male, 58% of the total of the abfraction lesions detected (78 lesions), compared with the female, 42% (57 lesions) (Figure 6).

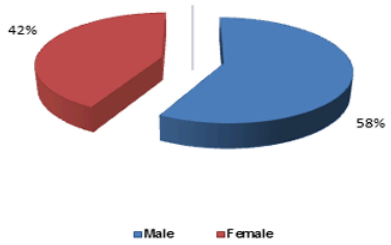


Figure 6. Distribution of abfraction lesions according to patients' gender.

Referring to the distribution of abfraction lesions according to the age of the subjects in the studied group, it was found that the majority of patients with premolar lesions were between 30 and 65 years old, the percentage of molar damage was between 32 and 72 years, and regarding combined canine, premolar, molar lesions between 25 and 72 years old.

Table 2. Distribution of abfraction lesions according to age and type of tooth affected.

Age of the subjects	Type of affected tooth	Number of lesions
30-65 years old	PREMOLARS	104
32-72 years old	MOLARS	7
25-72 years old	CANINES, PREMOLARS, MOLARS	24

The investigation of the existence of parafunctions and the transmission of occlusal forces outside the dental axis showed that 16.66% (17 subjects) presented nocturnal bruxism and 8.82% (9 subjects) presented dental abnormalities without establishing orthodontic treatment (Figure 7).

The presence of abfraction lesions was found in patients with satisfactory oral hygiene, who had dento-maxillary abnormalities.

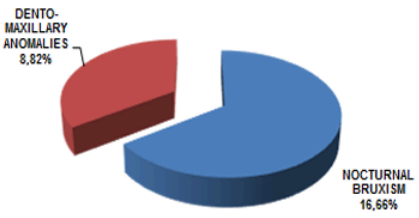


Figure 7. Investigation of the existence of parafunctions in patients with abfraction lesions.

The occlusal forces generated in the patients with bruxism led to the occurrence of the abfraction lesions and the wear facets caught in different stages of evolution, accentuated by aging.

In patients who had untreated dento-maxillary abnormalities and implicitly the transmission of non-axial occlusal forces, cervical lesions were detected cervical lesions in various stages of progression unrelated to the oral cavity hygiene, the patients presenting a satisfactory hygiene.

The examination of the degree of damage of the dental structures revealed that: the percentage of the damage only at the enamel level was 46% (62 lesions); 38% (51 lesions) of the detected lesions progressed with dentine damage, and in 16% (22 lesions) there were signs of dental pulp damage, such as the hypersensitivity described in response to an osmotic, tactile, chemical stimulus (Figure 8).

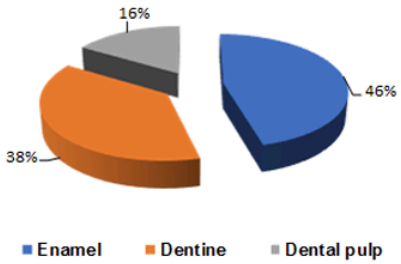


Figure 8. Analysis of the distribution of the abfraction lesions according to the degree of damage of the dental structures.

Frequently encountered were the cervical lesions with enamel and dentine damage without experiencing pain on the part of the patients, and only the aesthetic function was affected.

The action of the occlusal trauma caused by the parafunctions on the teeth for a long time resulted in the dental pulp being affected with the appearance of acute phenomena, such as the periodontal abscess with secondary genio-suborbital cellulitis (Figure 9).

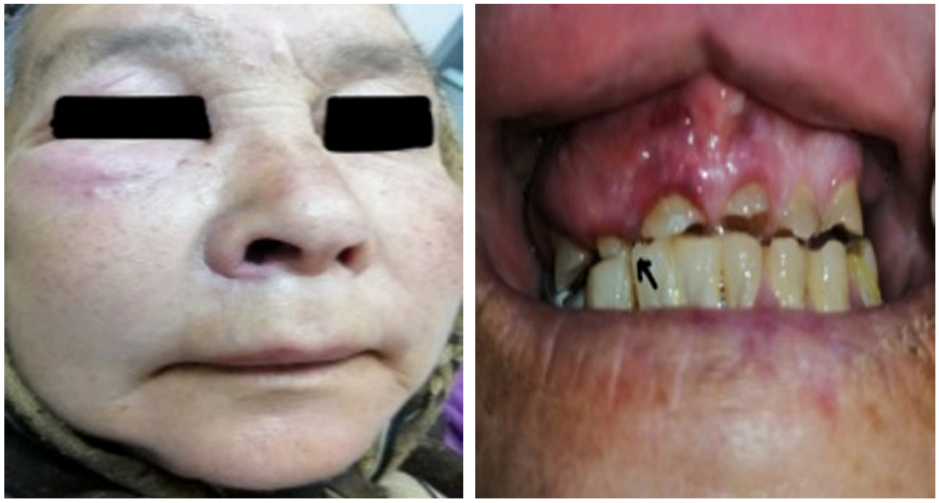


Figure 9. A 68-year-old female patient, affirmative nocturnal bruxism, with the presence of wear facets affecting the dental pulp.

10.12865/CHSJ.45.04.07

Discussions

There are theories [7,8] that justify how stress forces occurring at the cervical level lead to the occurrence of abfraction lesions due to the poor quality of the tooth enamel at this level that does not withstand the concentrated traction forces near the cervical region.

Lee and Eakle [9] proposed as etiology for abfraction a combination of: occlusal stress, the presence of parafunctions, abrasion and erosion.

According to these authors, the location of the abfraction lesions is determined by the direction of the lateral forces acting on a tooth during mastication and parafunctions when three types of stress factors appear: compression, shearing and traction, due to the elasticity of the dentin and the reduced thickness of the HUNTER-SCHREGER band in the cervical area leading to the occurrence of abfraction lesions by a pathogenic mechanism that advances rapidly when the paraaxial occlusal forces act repeatedly.

Other studies [10] on the relation of occlusal forces, which act in bruxism, with abrasion, show that the teeth can flex in the cervical region due to occlusal stress, but they cannot cite the causes of this injury without the presence of an abrasive or erosive component.

Other authors [11] reported a direct correlation between occlusal wear facets and progression of cervical lesions.

It has also been hypothesized [12,13,14] that excessive occlusal forces are the causal factor in the occurrence of abfraction and gingival recession, but not supported by adequate evidence.

Optical coherence tomography revealed the relationship between the incidence of occlusal wear and the demineralization of the cervical region of the tooth. It has also been found that in the early stages, the demineralization of the dentine leads to lesions at the parcel level, and the occlusal stress contributes to the progression of these lesions [15].

From our study it appears that the most affected by the abfraction lesions were the premolars (77.45%), which confirms the findings of other studies [16] that speak of the lower capacity of the premolars compared with the canines, to counteract the forces acting non-axially and are prone to loss of dental tissue in the cervical region.

According to other authors [17,18] occlusal forces generated at the time of exercising oral functions and parafunctions and premature contacts lead to cervical lesions. As a result, there are authors [19,20] who assert that clinical and radiographic indicators can be used to establish the presumptive diagnosis of occlusive trauma, and it is difficult to determine whether the wear facets are caused by functional contacts or parafunctional habits, such as bruxism.

Previous studies [21] have shown that the toothbrush does not cause abfraction damage alone, but together with the toothpaste, confirmed in our study by the presence of abfraction lesions in patients with good oral hygiene who have performed dental brushing several times per day.

There are studies [22,23] that have shown that there are patients who did not perform the dental brushing, also encountered in our study, but showed lesions at the cervical level, indicating that brushing is not an element of triggering these lesions but can intensify this process.

Another clinical study [24] found that the progression of cervical lesions was associated with occlusal forces, without the contribution of dietary habits, dental hygiene, wear facets or parafunctional habits.

There are studies [25] that have associated abfraction and occlusal wear with bruxism, other authors [26] with occlusal pathology, supporting the presence of several etiological factors and suggesting that dental wear is related to the anatomy of the tooth, the distribution of forces, the development of caries lesions, occlusion and parafunctions.

Other authors [27,28] found that the abfraction lesions changed with aging, influencing the prevalence and severity of these lesions, reporting a direct correlation between the occlusal wear facets and the progression of the cervical lesions.

The study carried out by us revealed the presence of the abfraction lesions in various stages of progression in the elderly but also in the young people, who presented dento-maxillary anomalies with the transmission of the paraxial occlusal forces.

Other studies [29] related to occlusal wear have associated the dehydration of the patient with the reduction of salivary protection against dental erosion, which is of greater importance in the etiology of abfractionn than bruxism.

The associations between occlusal pathology and cervical lesions are primarily the result of dental erosion in contrast to the abfraction hypothesis, which emphasizes that harmful forces that cause wear on the occlusal surfaces

and are transmitted at the cervical level do not cause abfraction damage without the presence of biocorrosion [30].

Von et al. [31] evaluated in 2004 the histological changes that occur in the periodontal structures during the use of non-axial forces, discovering the presence of areas of hyalinization and necrotic tissue, and in 2009 [32] they discovered hyalinization as an undesirable side effect in the dental orthodontics displacements.

Vier et al. [33] evaluated the effects of occlusal trauma on the dental pulp, finding that hyalinization is an irreversible response if excessive forces act for a long time.

There are authors [34] who have sought evidence to establish a causal relationship between occlusion and abfraction lesions, and other studies [35,36,37] indicate associations between non-carious lesions, bruxism and occlusal factors such as wear facets, premature contacts, but does not confirm the cause of the abfraction.

According to these studies [38,39,40], there is a correlation between bruxism and cervical lesions, but the relationship between excessive occlusal forces and the evolution of abfraction lesions is still uncertain.

There are bioengineering studies which have explored the association of occlusal stress with cervical wear, using photoelastic methods, without confirming a positive relation with abfraction lesions [41,42].

It has been argued that an occlusal trauma situated far from cervical lesions, cannot be considered as a single cause of abfractions [43].

A number of theories have been issued [44] on the etiology of these lesions, but the real causes underlying their production are still unclear, which is reflected in the specialty literature that describes acid and abrasive processes as etiological factors of these lesions, but pay special attention to occlusal stress due to forces transmitted outside the axis of the teeth.

Conclusions

The abfraction lesions in combination with other factors progress through the loss of protection of the natural tissues and evolve into the soft tissue of the tooth, the dental pulp.

Early and correct identification of the main mechanism of production of cervical lesions will allow the adoption of preventive or therapeutic measures increasing the chances of success in the management of these lesions.

The simultaneous occurrence of wear and the presence of the bacterial plaque may be an indicator in the progression of abfraction injuries.

The abfraction lesions need to be restored to prevent their further progression, exposure of the dental pulp, coronary tooth fracture and to improve the aesthetics.

Conflict of interests

None to declare.

References

1. Sarode GS, Sarode SC. Abfraction: A review-J Oral Maxillofacial Pathology, 2013,17(2):222-227.
2. Grippo JO. Abfractions: a new classification of hard tissue lesions of teeth. J Esthet Dent,1991, 3(1):14-19.
3. Grippo JO. Noncarious cervical lesions:the decision to ignore or restore. J Esthet Dent, 1992, 4(1):155-164.
4. American Academy of Periodontology. Glossary of periodontal terms, American Academy of Periodontology, 2001, Chicago.
5. Nascimento M, Dilbone D, Pereira P, Duarte W, Geraldeli S, Delgado A. Abfraction lesions: etiology, diagnosis, and treatment options. J Clinical, Cosmetic and Investigational Dentistry, 2016, 3(8):79-87.
6. Walter C, Kress E,Gotz H,Taylor H,Willershausen I,Zampelis A.The anatomy of non-carious cervical lesions, 2014,18(1):139-146.
7. McCoy G. On the longevity of teeth. J Oral Implantol, 1983, 11(2):248-267.
8. Walter C, Kress E, Gotz H, Taylor H, Willershausen I, Zampelis A. The anatomy of non-carious cervical lesions, 2014, 18(1):139-146.
9. Lee WC, Eakle WS. Possible role of tensile stress in the etiology of cervical erosive lesions of teeth. J Prosthet Dent, 1984, 52(3):374-380.
10. Grippo JO, Simring M. Dental ''erosion'' revisited. J Am Dent Assoc, 1995,126(5):619-630.
11. Grippo JO, Simring M, Schreiner S. Attrition, abrasion, corrosion and abfraction revisited: a new perspectiveon tooth surface lesions. J Am Dent Assoc, 2004, 135(8):1109-1118.
12. Stillman PR. The management of pyorrhea. Dent Cosm, 1917, 59(4):405-414.
13. Stillman PR. What is traumatic occlusion and how can it be diagnosed and corrected. J Am Dent Assoc, 1925, 12(11):1330-1338.
14. Rodier P. Clinical research on the etiopathology of gingival recession. J Periodontology.1990, 9(3):227-234.
15. Wada I, Shimada Y, Ikeda M, Sadr A, Nakashima S, Tagami J, Sumi Y. Clinical assessment of non carious cervical lesion using swept-source optical coherence tomography. J Biophotonics, 2014, 7(7):506-513.
16. Borcic J, Anic I, Urek MM, Ferreri S. The prevalence of non-carious lesions in permanent dentition. J Oral Rehabil, 2004, 31(2):117-123.
17. Kornfeld B. Preliminary report of clinical observations of cervical erosions, a suggested analysis of the cause and the treatment for its relief. Dent Items Interest 1982, 54:905-909.

10.12865/CHSJ.45.04.07

18. Chen KK, Miyake K, Terashita M. Cervical strains induced by occlusal loading. J Dent Res, 1999, 78:474.
19. Pihlstrom BL, Anderson KA, Aepli D, Schaffer EM. Association between Signs of trauma from occlusion and periodontitis. J Periodontol,1986, 57(1):1-6.
20. Jin LJ, Cao CF. Clinical diagnosis of trauma from occlusion and its relation with severity of periodontitis, 1992, 19(2):92-97.
21. Abrahamsen TC. The worn dentition-pathognomonic patterns of abrasion and erosion. Int Dent J, 2005, (4 Suppl 1):268-76.
22. Faye B, Kane AW, Sarr M, Lo C, Ritter AV, Grippo JO. Noncarious cervical lesions among a non-toothbrushing population with Hansen's disease (leprosy) initial findings. Quintessence Int, 2006, 37(8):613-619.
23. Ritter AV, Grippo JO, Coleman TA, Morgan ME. Prevalence of carious and non-carious cervical lesions in archaeological populations from North America and Europe. J Esthet Restor Dent, 2009, 21(5):324-334.
24. Sawlani K, Lawson NC, Burgess JO, Lemons JE, Kinderknecht KE, Givan DA, Ramp L. Factors influencing the progression of noncarious cervical lesions: a 5-year prospective clinical evaluation. J Prosthet Dent, 2016, 115(5):571-577.
25. Xonga FA, Sognnaes RF, Wolcott RB. Efficacy of fluoride varnishe in the prophylaxis of dental erosion. Magazyn Stomat, 1997, 39(1):49-52.
26. Bergstrom J, Eliasson S. Cervical abrasion in relation to toothbrushing and periodontal health. J Dent Res,1988, 96(5):405-411.
27. Smith WA, Marchan S, Rafeek RN. The prevalence and severity of non-carious cervical lesions in a group of patients attending a university hospital in Trinidad. J Oral Rehabil, 2008, 35(2):128-134.
28. Brandini DA, Trevisan CL, Panzarini SR, Pedrini D. Clinical evaluation of association between noncarious cervical lesions and occlusal forces. J Prosthet Dent, 2012, 108(5):298-303.
29. Young WG, Khan F. Sites of dental erosion are saliva-dependent. J Oral Rehabil, 2002, 29(1):35-43.
30. Bartlett DW, Shah P. Acritical review of non-carious cervical (wear) lesions and the role of abfraction, erosion, and abrasion. J Dent Res, 2006, 85(4):306-312.
31. Von Bohl M, Maltha J, Von den Hoff H, Kuijpers-Jagtman AM. Changes in the periodontal ligament after experimental tooth movement using high and low continuous forces in beagle dogs. Angle orthod, 2004, 74(1):16-25.
32. Von Bohl M. Am hyalinization during orthodontic tooth movement. J Orthod, 2009, 31(1):30-36.
33. Vier-Pelisser FV, Figueiredo MA, Cherubini K, Braga Filho A, Figueiredo JA. The effect of head-fractioned teletherapy on pulp tissue. Int Endod J, 2007, 40:859-865.
34. Telles D, Pegoraro LF, Pereira JC. Incidence of noncarious cervical lesions and their relation to the presence of wear facets. J Esthet Restor Dent, 2006, 18:178-183.
35. Horning GM, Cohen ME, Neils TA. Bucal alveolar exostoses: prevalence, characteristics and evidence for buttressing bone formation. J Periodontol, 2000, 71(6):1032-1042.
36. Piotrowski BT, Gillette WB, Hancock EB. Examining the prevalence and characteristics of abfractionlike cervical lesions in a population of U.S.veterans. J Am Dent Assoc, 2001, 132(12):1694-1701.
37. Pegoraro LF, Scolaro JM, Conti PC, Telles D, Pegoraro TA. Noncarious cervical lesions in adults: prevalence and occlusal aspects. J Am Dent Assoc, 2005, 136:1694-1700.
38. Ommerborn MA, Scheider C, Giraki M, Schafer R, Handschel J. In vivo evaluation of noncarious cervical lesions in sleep bruxism subjects,J Prosthet Dent.2007;98:150-158.
39. Tsiggos N, Tortopidis D, Hatzikyriakos A, Menexes G. Association between self-reported bruxism activity and occurrence of dental attrition, abfraction and occlusal pits on natural teeth. J Prosthet Dent, 2008, 100:41-46.
40. Fan J, Caton JG. Occlusal trauma and excessive occlusal forces: Narrative review, case definitions, and diagnostic considerations. J Clin Periodontol, 2018, 40:59-66.
41. Rees JS, Hammadeh M, Jagger DC. Abfraction lesion formation in maxillary incisors, canines and premolars: a finite element study. Eur J Oral Sci, 2003, 111(2):149-154.
42. Wood ID, Kassir ASA, Brunton PA. Effect of lateral excursive movements on the progression of abfraction lesions. Oper Dent, 2009, 34(3):273-279.
43. Levrini L, Di Benedetto G, Raspanti M. Dental wear: a scanning electron microscope study. Biomed Res Int, 2014, 2014:340425.
44. Pereira AFV, Poiate IA, Poiate E Jr, Miranda WG Jr. Abfraction lesions reviewed: current concepts.RGO, 2008, 56(3):321-326.

Corresponding Authors: Lucian Paul Dragomir, Department of Occlusology and Fixed Prosthetics, University of Medicine and Pharmacy of Craiova, 2 Petru Rareş Street, 200349 Craiova, Romania; e-mail: dragomirlucianpaul@yahoo.com
Mihai Raul Popescu, Department of Occlusology and Fixed Prosthetics, University of Medicine and Pharmacy of Craiova, 2 Petru Rareş Street, 200349 Craiova, Romania; e-mail: popescumihairaul@yahoo.com