QUESTION 1

My patient is receiving orthodontic treatment, and her oral hygiene has taken a turn for the worse. What preventive and interceptive measures are available for improving and maintaining good oral hygiene and cariogenic control in orthodontic patients?

Background

The maintenance of effective oral hygiene is critical during orthodontic treatment. It is the responsibility of the orthodontist, the general dentist and their teams to collaborate in providing oral hygiene instruction, monitoring and motivation to all patients undergoing orthodontic treatment. It is hoped that these techniques will become habits that stay with the patients for the remainder of their lives.

White spot lesions around the surfaces of banded or bonded teeth are a common orthodontic problem, estimated to occur in 13% to 50% of patients.[1,2] Orthodontic appliances make tooth-brushing and flossing more difficult, and they increase the surface area available for accumulation of bacterial plaque and food particles. Decalcification may subsequently occur when the intraoral pH decreases.

Risk Factors

Important risk factors for enamel decalcification include the patient's oral hygiene, diet, individual salivary flow and excess or failed bonding cement around the bracket or band (**Table 1**).

Table 1 Risk factors for decalcification of enamel with orthodontic appliances

Risk factor	Description and preventive methods
Oral hygiene	• Decalcification around orthodontic appliances may occur within 4 weeks[3] (with poor oral hygiene) • Patient must have knowledge and understanding of cariogenic process and risk factors for periodontal disease • Dental team responsible for providing information to patient
Diet	• Diet high in fermentable carbohydrates presents risk • Appliances may restrict ability of tongue and saliva to eliminate food particles, leading to breakdown of complex carbohydrates in oral cavity and prolonged exposure of teeth to acid • Carbonated drinks high in sucrose, phosphoric acid, citric acid decrease intraoral pH • Volume of fluid, frequency and duration of exposure affect rate of decalcification
Individual salivary characteristics	• pH, flow rate and buffering capacity of saliva influence degree of demineralization • Greater risk of decalcification with lower salivary flow rates (e.g., labial surface of maxillary incisors) • Lower risk of decalcification with higher salivary flow rates (e.g., lingual surfaces) • Saliva important for washing away food particles and bacteria • Saliva also delivers fluoride to enamel; concentration about 20 μg/L or 0.02 ppm and total daily release of 11.4 μg[4] • Low levels of ambient fluoride may protect enamel against demineralization[5]
Excess bonding cement or failure of the bonding cement	• Risk of decalcification not increased by use of banded rather than bonded attachments • Well-cemented molar bands may protect teeth, especially if cement has true chemical or adhesive bond to enamel (e.g., glass ionomer, zinc polycarboxylate cements) • Leakage and decalcification only if cement holding band fails or band is improperly contoured around tooth anatomy • Excess bonding resin around orthodontic brackets should be removed as it may be a nidus for accumulation of plaque (because of rough, unpolished surface) and may make debonding more difficult

Methods to Reduce the Risk of Decalcification

For patients with active dental disease and those at high risk of caries, orthodontic treatment should be delayed. For patients with a history of caries, orthodontic treatment should be initiated only when they have been assessed over a period of time as having good periodontal health, low risk of caries and excellent oral hygiene habits. A period of 3 months of sustained good oral hygiene is probably sufficient to evaluate a patient's ability to conform to a stringent oral hygiene regimen.

Before treatment, a diet counselling session, including a 5-day detailed diet record, is recommended, particularly for patients with a history of caries. The cariogenic potential of foods should be explained to all patients, and patients' understanding of the concept should be verified by having them explain it in their own words. Patients should be advised to avoid sugar-containing foods and to drink water instead of high-sugar drinks.

Patients should be taught effective flossing and tooth-brushing techniques with their braces in place, and should then be asked to demonstrate their ability to perform these techniques. They should understand the importance of brushing after meals and of timing themselves while they brush, to ensure an appropriate duration of brushing. An electric or ultrasonic toothbrush can be recommended to increase motivation and to improve oral hygiene, as these tools have been associated with lower plaque scores around brackets.[6]

Once orthodontic treatment has been initiated, the general dentist and the orthodontist should work as a team, continuously motivating the patient, encouraging effective oral hygiene practices and proper nutrition and diet, and assessing gingival health and caries risk at each appointment. If evidence of poor oral hygiene and decalcification appear during treatment, and these problems continue despite efforts to improve, the orthodontic wires may be removed. This may increase the total treatment time, but it makes oral hygiene procedures simpler and offers the patient a chance to improve. If poor oral hygiene persists, the orthodontic attachments should be removed and treatment discontinued, to prevent further decalcification until effective oral hygiene practices, good gingival health, a positive attitude and improved dental IQ are demonstrated over time.

A Cochrane review has recommended that orthodontic patients who are at risk of caries should use a 0.05% sodium fluoride rinse daily during treatment, in addition to fluoridated toothpaste.[7] Fluoride ions prevent plaque activity and adhesion by blocking enzyme systems, reducing enamel demineralization and promoting remineralization. Unfortunately, however, the evidence for the use of fluoride-releasing bonding materials, such as glass ionomer cement, in reducing the incidence and severity of white spot lesions in vivo was weak.[7]

The use of fluoride varnish at sites that are highly susceptible to caries may be advantageous, as it contains a high concentration of fluoride, hardens on contact with enamel, adheres to enamel for a prolonged period and is well tolerated.[8] Fluoride-containing resins and elastomers cause an initial high burst of fluoride, which in turn causes the formation of calcium fluoride and helps to remineralize and protect etched enamel. Over the following days and weeks, the fluoride release tapers down, with small amounts being released over extended periods. This causes the formation of fluorapatite on enamel at the bracket base, where it is most needed. This process exemplifies the advantage of sustained, low-dose fluoride release. In addition, it has been shown that these materials can imbibe fluoride (thus becoming recharged), such that the fluoride can then be re-released.[9-11] However, fluoridated elastomers have been found to enlarge when placed intraorally, which would increase the surface area to which plaque could adhere.[9,10]

If visible white spot lesions are detected when the braces are removed, they should not be treated immediately with concentrated fluoride agents. This is because subsurface soft lesions take longer to remineralize then outer lesions. The presence of excess fluoride would cause a precipitate of calcium phosphate to form on the enamel surface (creating an unsightly white opaque appearance), which would in turn block the surface pores and limit remineralization of the inner surface. This may arrest the lesion but prevent its normal repair, leading to unesthetic, opaque white spot lesions on the surface of the enamel. White spot lesions observed after debonding should therefore be treated with 2 to 3 months of good oral hygiene (the fluoride within saliva permitting a more controlled degree of remineralization), followed by professional application of topical fluoride during regular 6-month recall appointments at the dentist's office.

Conclusion

Before bonding and banding orthodontic appliances, it is the responsibility of the general dentist and the orthodontist to assess the patient's risk of caries, to provide comprehensive oral hygiene instructions and possibly even to review the patient's diet. The risk assessment should be repeated at intervals during treatment, and continual motivation and reminders of oral hygiene practices should be given, to prevent the development of white spot lesions.

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QUESTION 2

How should a dentist evaluate severe acute pain involving the teeth and other orofacial areas, and how can the dentist determine whether the patient needs conventional dental interventions?

Background

Dentists are routinely asked to diagnose and treat pain of presumed dental origin, sometimes at the patient's request but also upon referral by a physician. In the majority of cases, orofacial pain is of odontogenic origin, and it usually resolves after appropriate, routine dental interventions. However, some patients never experience relief of pain in their teeth or adjacent areas, even though clinical and radiographic examinations indicate that the therapy was successful.

In one case, a 36-year-old man presented to the dental office with a chief complaint of spontaneous and severe shooting pain in the left maxillary teeth. Upon questioning, he also reported additional pain, which he described as "feeling as though a knife is boring through my left eye." His left eye had suddenly become red, with tearing and swelling; the eyelid had also become very heavy and drooping. The differential diagnosis for this presentation should include a nondental condition of neurovascular origin known as trigeminal autonomic cephalalgia.

Patients with trigeminal autonomic cephalalgia and other nondental causes of orofacial pain represent a significant challenge for dentists, who must be aware of and recognize orofacial conditions that may mimic odontogenic pain, so as to avoid performing unnecessary and inappropriate dental procedures.

Classification

The trigeminal autonomic cephalalgias are a group of headaches characterized by unilateral head and/or face pain with accompanying autonomic features.[1,2] According to the International Classification of Headache Disorders, the trigeminal autonomic cephalalgias include the following conditions: episodic or chronic cluster headache, episodic or chronic paroxysmal hemicrania, and short-lasting unilateral neuralgiform headache attacks with conjunctival injection and tearing (SUNCT).[2] Cluster headache and SUNCT are more common in males, whereas paroxysmal hemicrania is more common in females. Both cluster headache and paroxysmal hemicrania have a similar age of onset of 20 to 35 years; SUNCT occurs in older patients (onset between 35 and 65 years of age).

Clinical Characteristics

Cluster Headache

Cluster headache is characterized by severe, primarily unilateral pain generally located in the orbital and/or temporal regions with accompanying ipsilateral autonomic changes.[1,2] The pain may spread to the maxilla, nostril, gingiva, palate, jaws, teeth and neck.[3,4] As a result, it may be difficult to identify the source of the pain. The pain is excruciating, and patients often describe it as constant, boring and burning. The vast majority (about 93%) of patients with cluster headaches report restlessness, agitation and pacing; they may also report head-banging behaviour when pain is present.[5] Cluster headache attacks may occur from 1 to 8 times per day, with each episode beginning abruptly and lasting between 15 and 180 minutes.

Paroxysmal Hemicrania

Paroxysmal hemicrania is characterized by severe, short-lasting, unilateral pain attacks localized to the orbital region and/or temporal sites, accompanied by one or more autonomic features.[1,2] The pain may involve the orofacial and frontal regions, the neck and the occiput.[2,3,6,7] The pain is excruciating and is often described at peak intensity as boring or stabbing.[1] Most paroxysmal hemicrania attacks are spontaneous; however, triggers such as glyceryl trinitrate, alcoholic drinks and mechanical rotation or manipulation of the head and/or neck may precipitate attacks.[8] The attacks are typically 2–30 minutes in length, with between 1 and 40 attacks per day.

Short-lasting Unilateral Neuralgiform Headache Attacks with Conjunctival Injection and Tearing

SUNCT is characterized by strictly unilateral, intense pain attacks localized to the orbital, supraorbital, temporal and frontal areas; these headaches have cranial and facial autonomic features. The pain may involve areas of the head and neck, ear, nose, cheek, palate and throat,[2,9,10] and about one-third of patients report pain that is localized

Table 1 Medical conditions with symptoms mimicking those of various trigeminal autonomic cephalalgia[a]

Cluster headache	Paroxysmal hemicrania	SUNCT
Other primary headaches		
Hypnic headache Hemicrania continua SUNA Primary stabbing headache Primary cough headache Migraine with or without aura	Hypnic headache Hemicrania continua SUNA Primary stabbing headache Primary cough headache Primary exertional headache Primary headache associated with sexual activity	Primary stabbing headache
Vascular disorders		
Carotid artery dissection or aneurysm Vertebral artery dissection or aneurysm Giant cell (temporal) arteritis	Middle cerebral artery infarct Collagen vascular disease Parietal arteriovenous malformation	Cerebellopontine arteriovenous malformation Cavernous hemangioma
Tumours		
Pituitary adenoma Nasopharyngeal carcinoma Sphenoidal meningioma	Pancoast tumour Pituitary microadenoma Macroprolactinoma	Tumour of posterior fossa Pituitary lesions
Dental		
Pulpal pain Periodontal pain TMD	Pulpal pain Periodontal pain TMD	Pulpal pain Periodontal pain TMD
Trigeminal neuralgia	Trigeminal neuralgia	Trigeminal neuralgia
Maxillary sinusitis		
Head and neck trauma		

Adapted with permission from Balasubramaniam and others.[12]
[a] In addition to the specific conditions listed in this table, each of the trigeminal autonomic cephalalgias may be mistaken for the other 2 types.
SUNCT = short-lasting unilateral neuralgiform headache attacks with conjunctival injection and tearing, SUNA = short-lasting unilateral neuralgiform headache attacks with cranial autonomic symptoms, TMD = temporomandibular disorder.

to the maxillary branch of the trigeminal nerve.[11] The majority of SUNCT attacks occur spontaneously or following an innocuous trigger similar to those reported for trigeminal neuralgia.[9] A SUNCT attack begins abruptly, with maximum intensity within 2 to 3 seconds, and persists a mean of 49 seconds (range 2 to 600 seconds).[10] In spite of neuralgia-like triggers of SUNCT, there are no refractory periods, as may be seen in trigeminal neuralgia.[10] Attack frequency varies from less than once daily to more than 60 attacks hourly; severe attacks may last for days.

Diagnostic Features

The key to diagnosis is the medical history, as reported by the patient. Decisive features include the rapidity of onset and the location of the pain; the quality, duration and temporal patterns of the headache episodes; the presence of triggering factors; and autonomic features.[2,5] Consideration of a differential diagnosis is required before a working diagnosis of trigeminal autonomic cephalalgia can be made, as many other conditions can mimic these headaches, including orofacial and dental pain with other causes (**Table 1**).[12]

Implications for Dentists

A thorough history taking and a comprehensive clinical examination must be conducted. If these are not performed, the dentist may attempt a variety of unnecessary and inappropriate dental interventions to treat the pain, to the patient's

detriment. Patients with trigeminal autonomic cephalalgia may report pain that arises in the midface region, which may be misinterpreted as pain originating from the teeth, jaws or temporomandibular joints.[3,4] In particular, many patients with cluster headache are seen by a dentist before receiving the correct diagnosis, and they often undergo inappropriate dental procedures that are both invasive and irreversible.[13]

Given the short duration of attacks, the frequency of recurrence, and the excruciating intensity and pulsatile quality of the pain associated with paroxysmal hemicrania, there is a potential that this condition will be misdiagnosed as dental pulpitis,[7] which may result in unwarranted dental interventions.[6] Paroxysmal hemicrania may also be misdiagnosed as a temporomandibular disorder, because the pain presents in the temporal, maxillary and ear regions, with tenderness of the ipsilateral masticatory muscles.[6,7]

In some cases, patients with SUNCT have reported pain that radiates to the adjacent teeth, which has led to dental treatments such as extraction, occlusal splints and incorrect pharmacologic therapy.[14] Trigeminal neuralgia may also be confused with paroxysmal headache or SUNCT, because all 3 of these disorders share common features, such as the excruciating intensity, intermittent temporal pattern, unilateral distribution and lancinating nature of the pain, as well as the frequency of attacks.

Conclusion

The trigeminal autonomic cephalalgias are painful and disabling primary headaches. A patient with one of these conditions may visit a dentist before any other practitioner, or the patient may be referred to a dentist by a physician because of the site and localization of the pain. It is therefore incumbent upon dentists to understand and recognize the characteristics of trigeminal autonomic cephalalgias, to avoid incorrect diagnoses and also to avoid unnecessary and inappropriate traditional dental treatments. After orofacial and dental causes of pain have been ruled out, the dentist should refer any patient with symptoms of a complex headache to the appropriate pain practitioner for appropriate diagnosis and management.

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The "Point of Care" section answers everyday clinical questions by providing practical information that aims to be useful at the point of patient care. The responses reflect the opinions of the contributors and do not purport to set forth standards of care or clinical practice guidelines. Readers are encouraged to do more reading on the topics covered. If you would like to contribute to this section, please contact editor-in-chief Dr. John O'Keefe at jokeefe@ cda-adc.ca.