

# Dental erosion due to abuse of illicit drugs and acidic carbonated beverages

Mohamed A. Bassiouny, DMD, MSc, PhD

Consumption of illicit drugs and the abusive intake of acidic carbonated beverages (particularly soda) often are associated with similar types of damage to the human dentition, the most common of which is dental erosion. The dentitions of individuals who are addicted to methamphetamines or crack cocaine can be misdiagnosed as dental caries rather than generalized dental erosion, a condition that also is associated with chronic excessive consumption of soda. Failing to identify the causative etiology could lead to a wrongful diagnosis that could in turn adversely affect treatment planning and misdirect a specified prevention protocol.

This article seeks to identify the unique clinical features of each one of these conditions, highlight the resemblances between them, and recognize the unambiguous differences in their fundamental

characteristics. Three representative cases—involving a methamphetamine user, a crack cocaine addict, and an avid consumer of diet soda—are presented. In each case, the patient has admitted to the cause of their poor oral health. The dental, oral, and paraoral manifestations of each case are documented and differentiated from one another, and the factors that contributed to the associated disease process are discussed.

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According to 1965-1996 data from the United States Department of Agriculture (USDA), when Americans—especially adolescents—reached for something to quench their thirst, their preferred drink was increasingly an acidulated carbonated beverage.<sup>1</sup> A 2005 study of school children (ages 6-12) by Blum et al confirmed this growing trend in the US population.<sup>2</sup> Today, with the seemingly endless varieties available, the consumption of soft drinks continues to be the people's choice.<sup>3,4</sup> According to the USDA's Economic Research Service data, Americans consumed an average per capita total of 46.4 gallons of soft drinks in 2003.<sup>5</sup> Between 1999 and 2002, teenagers in the US who consumed carbonated soft drinks consumed an average of 28 oz daily.<sup>6</sup> Excessive and frequent consumption of acidulated carbonated beverages has been shown to cause severe dental erosion that can ravage the human dentition in a considerably short period of time.<sup>6-11</sup>

Ironically, for many years, high sugar content was thought to be the greatest hazard of sugar-sweetened (regular) soda.<sup>12</sup> Health authorities and the general public focused on the high sugar content as a primary health concern because it contributed to weight gain, obesity, and many other serious systemic health and dental issues.<sup>13-19</sup> Such deeply rooted beliefs have led the avid consumers of soft drinks (more females than males) to switch from the regular to the diet

variety of soda to alleviate concerns of gaining weight.<sup>6,16</sup> This mindset led to an ongoing increase in the national consumption of diet soda.<sup>3</sup> Regular and diet soda differ in terms of how they are sweetened, however, their composition is otherwise almost identical, especially in terms of the acids used.<sup>20,21</sup> Citric acid, which is known to have a high potential for erosion, is the essential acidic compound found in both regular and diet soda.<sup>22,23</sup> Therefore, while consuming diet soft drinks may avoid the adverse effects of sugar consumption, the erosive action of the acidic constituents remains.

The widespread abuse of methamphetamine in the US has swept through both rural and urban communities from Hawaii eastward.<sup>24,25</sup> Likewise, in 2009, the National Institutes of Health's Community Epidemiology Work Group reported that indicators of crack cocaine use remained high or stable (or fell only slightly) in several areas of the nation.<sup>26</sup> These sympathomimetic-producing drugs have serious effects on the users' systemic health, including shortness of breath, increased respiration, cardiovascular events, hyperirritability, hyperthermia, nausea, vomiting, diarrhea, and xerostomia.<sup>27-30</sup> Dental complications associated with methamphetamine and crack cocaine abuse include "rotted, crumbling, or falling apart dentition."<sup>27,30-33</sup> This condition, commonly known as *meth mouth*, has been diagnosed as *rampant decay*.<sup>27,30-33</sup>

The dentitions of those who abuse illicit drugs and those who consume soda in excess may demonstrate similar clinical and radiographic features, which can make it difficult to identify the causative factors and diagnose the cause of the lesions that devastate the dentition. In order to facilitate accurate diagnoses, the clinically related manifestations should be differentiated. In addition, the causative relationship of their etiologies and the exacerbating factors associated with the dental hard tissue diseases must be discussed.

This report seeks to identify the unique clinical dental and oral features associated with chronic abuse of methamphetamine, crack cocaine, and diet soda. The similarities between the dental lesions/defects associated with each will be highlighted; in addition, the specific differences between them will be emphasized and the contributory factors for each discussed.

Three representative individual cases were selected. Each case embodied the typical characteristics of the oral and paraoral manifestations of the condition precipitated by the respective etiology, which was confirmed by the patient's self-response questionnaire.

## Case report No. 1: methamphetamine

A 29-year-old man admitted to using methamphetamine for more than 3 years prior to his current dental visit. His systemic,



Fig. 1. Frontal view of the dentition associated with the methamphetamine abuser. Note the eroded teeth and occasional brown discoloration of dentin.



Fig. 2. Oral pantomograph of the methamphetamine abuser shown in Fig. 1. Note the typical sharp line of demarcation of the advancing lesions.

dental, and habitual health histories indicated that he continuously experienced dryness of the mouth; to restore hydration, he frequently sipped 2 to 3 (12 oz) cans of regular soda every day. He did not recall seeking any routine dental care during that time, except for emergency molar extraction. He came to the university dental clinic to “fix his mouth.” The patient’s systemic health history was unremarkable aside from a casual mention of occasional skin irritation and itching.

The head and neck examination revealed no abnormality except for cracked lips. The patient’s dentition revealed symmetrical destruction of the right and left sides of the dental arches (Fig. 1). The damage to the vestibular aspects of the dentition was severe and extended to the proximal and biting surfaces of multiple teeth (Fig. 1). The palatal aspects of the maxillary anterior teeth showed signs of erosive damage, demonstrated by tooth surface loss that left a smooth featureless morphology in the dentin core, suggesting the effect of the erosive process with constant grinding. Multiple molars were missing, confirming the history of emergency extractions. Some teeth had lost their coronal segments, while others showed remnants of crowns whose dentin cores were partially destroyed and discolored (brown with a dirty grayish tint). Upon exploration, these dentin cores felt cartilaginous or soft in consistency. The enamel bordering the cervically located discolored lesions on the facial aspects

appeared opaque white in color and had a chalky texture. Similar lesions were detected on the palatal aspects of the maxillary dental arch. The lesions that caused this extensive damage to all aspects of the dentition had taken a generalized pattern. The location, distribution, and clinical and radiographic characteristics of these lesions suggested dental erosion that is caused by multiple sources of acids (Fig. 2).

The associated periodontal soft tissues demonstrated signs of chronic marginal gingivitis, as demonstrated by the inflamed free margin of the gingiva that appeared cherry red, edematous, glistening, and had lost the entire stippling contour. The inflamed gingival tissues appeared swollen and hypertrophied, resulting in the formation of multiple pseudo-periodontal pockets. Several areas of the attached gingiva and the mucosal lining also exhibited white patches and signs of inflammation, and appeared edematous. These patches were more pronounced on the facial surface of the lining mucosa in the anterior/premolar segments, although they were present on the posterior segments as well. The superficial white film was removed readily by scraping the white patches with a blunt instrument, leaving a raw, red, inflamed tissue that bled easily. The presence and characteristics of these white patches suggested a *Candida* infection. Dental plaque and *materia alba* were found on most of the dentition, a sign of neglected oral hygiene. No restorative intervention

was detected, confirming that this badly disintegrated dentition had not received professional dental care. The patient had little saliva on the floor of the oral cavity or on the mucosal lining and teeth; the saliva that was present was rubbery and thick.

The radiographic images of this patient’s dentition reflected the clinical findings, with no significant bone loss except in the edentulous areas (Fig. 2). No infrabony pockets were detected. Multiple root remnants were present, as were areas of apical rarifying osteitis, evidently associated with the badly disintegrated teeth. According to the radiographs, the coronal defects of the partially destroyed teeth appeared to have distinct lines of demarcation that distinguished the involved (eroded) lesions from the nonaffected remaining tissue. These distinct lines of demarcation are the typical indication of an advancing erosive process.

### Case report No. 2: cocaine

A fairly apprehensive, fidgety, anxious 51-year-old man sought treatment for a partially edentulous, badly damaged dentition. This patient was seeking dental care because he realized that his teeth had been neglected for a long time and wanted his mouth “fixed” and the “rotted” teeth extracted. The systemic health history was unremarkable, although he experienced a weight gain of 10 lb over the past 7 years, after quitting a cocaine habit of approximately 18 years. His blood pressure was within the normal limits; however, he was



Fig. 3. Oral pantomograph of a patient who abused cocaine for 18 years. Note the decimated dentition due to dental erosion and the typical sharp line of demarcation of the advancing lesions.

suffering from visual impairment due to a cataract in the left eye. He took mild analgesics to control the occasional toothache, and penicillin or amoxicillin (when prescribed) during emergency dental visits to treat dental abscesses. No history of allergies was reported, although the patient was unsure of his reaction to sulfa drugs.

For more than 30 years, this patient also was a heavy smoker (2 packs of cigarettes a day), but he claimed to have quit smoking a year before seeking treatment. He also admitted to drinking beer concurrent with his addiction to crack cocaine. The patient claimed to have stopped the crack cocaine habit at 44 years of age, when he realized his health was deteriorating. The patient received neither medical nor dental health supervision during his 18-year dependency on cocaine and the subsequent recovery period; as a result, the patient cited numerous dental-related episodes that resulted in the extraction of several teeth on an emergency basis. For several years, he suffered from bleeding gums, bad taste and odor in his mouth, loose teeth, abscessed teeth, and frequent toothaches. He also reported nasal and sinus problems that manifested by congestion, stuffiness, discharge, and occasional pain. The patient reported no history of accident to his teeth or jaws, nor did he complain of temporomandibular joint disorders, masticatory muscle disorders, or changes in bite or alignment.

This patient appeared stocky, slightly overweight, with no apparent general health deficits apart from displaying anxious behavior. There were no signs of alterations in the appearance of the facial skin or lips, and no palpable lymph nodes, enlarged salivary, or thyroid glands. The intraoral examination disclosed a grossly devastated dentition. Most of the posterior teeth were missing, while the remaining few had residual roots; some were loosely attached to the gingiva and in an advanced mobile state. All of the remaining anterior teeth were affected by carious-like lesions that invaded both the cervical and the proximal aspects. These lesions were present throughout the remaining dentition; they had devoured most of the coronal parts of the teeth and extended to the roots, which were barely visible above the gum line.

Despite the massive destruction of the remaining dentition and multiple pulp involvements, this patient only had a single restoration, a clear sign of total oral health negligence. The presence of massive plaque formation, calculus deposits, and materia alba was clearly evident. The marginal gingiva was inflamed and edematous; it bled upon touch and had receded at several sites. Deepening gingival pockets in several sites displayed the signs of attachment loss. The dorsal surface of the tongue was coated and bilateral mandibular tori were present.

The patient had lost his vertical support and consequently suffered collapsed occlusion. His oral pantomograph confirmed his dental and periodontal status, but showed normal bone density and architecture in both arches (Fig. 3). The maxillary sinuses appeared to be enlarged and exhibited some degree of radiopacity. The mandibular borders of the sinus draped over the premolar-molar areas to a level close to the alveolar bony crests, where the edentulous areas displayed ridge resorption. A generalized loss of alveolar bone height was noted (Fig. 3).

The damaged teeth and the remaining roots that were destroyed beyond pulp involvement showed sharp lines of demarcation that separated the affected tissue from the healthy tooth structures. This type of demarcation line is a typical sign of an advancing erosion lesion, unlike the diffused line associated with progressing caries. Areas of apical rarifying osteitis were displayed below several roots and teeth; suggesting the presence of chronic pathosis. The 2 mandibular condyles and rami appeared normal. Because none of the remaining teeth were salvageable, total clearance of the remaining badly damaged teeth and roots was deemed the most realistic and practical treatment approach. Maxillary and mandibular treatment prostheses were recommended, to be followed by complete dentures after the healing phase and the adaptation period. The patient accepted the treatment protocol and consented to the start of the process, but his attendance for scheduled follow-up dental appointments was erratic.

### Case report No. 3: diet soda

The patient was a single mother in her early thirties who lived on a limited income; her health history was noncontributory. She reported no complaint of altered salivary secretion or dry mouth; however, she was a mouth breather. Presumably because of the financial constraints involved in raising 2 children on her own, she did not seek any dental health services for an extended period of time (more than 2 decades). She reported consuming 2 liters of diet soda daily for the past 3 to 5 years; she chose diet soda because she feared gaining weight by consuming regular sodas and expressed awareness of the relationship between refined sugar and tooth decay.



Fig. 4. Frontal view of the patient's dentition associated with consumption of diet soda. Note the eroded dentition and the generalized dark yellowish discoloration of dentin.



Fig. 5. Oral pantomograph of the patient in Fig. 3, demonstrating varying degrees of damage to the dentition. Note the tissue loss on the left side compared to the right side and in the maxillary arch compared to the mandibular arch.

This patient reported that she recognized her deteriorating dentition with teeth breaking down partially or entirely, although this was a painless process. This report was confirmed by the patient's oral examination (Fig. 4). Aside from endodontic treatment of teeth No. 8 and 10 and an old amalgam restoration in tooth No. 3, no other signs of restorative intervention were observed. The entire dentition was in a catastrophic state, the posterior segments were damaged more severely than the anterior segments, while the mandibular arch was affected more than the maxillary.

The majority of the left maxillary arch dentition and both mandibular quadrants were drastically destroyed. The damaged sections of the dental arches had residual roots that were submerged below the level of the gingival margin or further, at the alveolar bone level. The visible cervical segments of these roots were soft upon exploration and a majority of their apices were surrounded by rarifying osteitis. The occlusal outlines of the root stumps disclosed semi-lunar borders that exhibited sharp demarcation lines on the radiographs; these lines were evident in other defects that affected the coronal segments of the remaining teeth (Fig. 5). The decalcified chalky white enamel surfaces were a telltale sign of dental erosion, as were the softened dentin cores, the partial or total

coronal loss of teeth in the entire dentition and the progression and generalized distribution of the lesions.

This erosive activity appeared to be related to an extrinsic dietary etiology. The lack of brown discoloration of dentin commonly associated with the consumption of sugar-sweetened acidic beverages points directly to excessive consumption of diet soda as the cause of dental erosion in this case.<sup>20</sup> The specific locations and severity of these erosion lesions disclosed the patient's pattern of acidic beverage consumption. This pattern was confirmed by discussing the patient's habits. She indicated sipping the beverage directly from a can or a bottle and holding the fluid in her mouth before swallowing. She also mentioned that when doing so, she habitually leaned on her left side against the arm of the sofa while watching television. The massive bilateral destruction of the mandibular and the left side of the maxillary dentition resulted in a collapsed bite.

The free gingiva was severely inflamed without significant deepening of the periodontal pockets. It appeared red and swollen and bled spontaneously upon touch. Dental plaque, materia alba, and calculus deposits accumulated in the retention areas of the badly eroded coronal segments of the dentition. The mouth breathing further aggravated the patient's

dental and periodontal health by making the dry anterior segments of the dental arches more vulnerable to acidic challenge and bacterial plaque assaults.

A few of the severely eroded teeth also displayed brown discoloration that is not commonly associated with diet beverage consumption, suggesting that the patient consumed junk food containing carbohydrates along with the diet soda. Upon revisiting this issue with the patient, she confirmed consuming potato chips. The entire dentition of this patient, apart from the maxillary right posterior segment (with moderate damage on the lingual aspect), appeared both clinically and radiographically to be in a total state of destruction (Fig. 4 and 5). This case posed poor prognosis and nullified any attempt for rehabilitation.

## Discussion

The decimation of dentition associated with methamphetamine and crack cocaine abuse could masquerade as aggressive destructive lesions of the hard dental tissues.<sup>20,30</sup> These lesions are strikingly similar to the advanced dentition destruction associated with excessive consumption of diet soda. The etiology of these lesions can remain unknown if the patient does not disclose drug abuse in his or her habitual health histories, which can make ascertaining the specific causative etiology

a challenge. Furthermore, the untoward effects on the dentition and oral health induced by these culprit substances obscures the origins of these lesions, which can hinder the discovery of the true underlying condition and lead to a misdiagnosis. As a result, it is almost impossible to implement a proper treatment plan with a positive prognosis, and any preventive measure taken could often miss the intended target.

The 3 cases reported here had several common denominators. All 3 were adults who lived in an urban community with fluoridated public water and all came from similar socio-economic backgrounds. The first 2 cases were individuals addicted to drugs (methamphetamine and crack cocaine), while the third case involved an avid consumer of diet soda. The lesions that affected the dentitions of these patients had almost identical features.

### **Lesion characteristics and identification**

Clinical and radiographic findings revealed some unique characteristics of the lesions that have exhibited a generalized pattern throughout the dentition. Some lesions were limited to the coronal segments of the involved teeth, while others displayed extensive damage to more than one aspect (or to the entire coronal surface of the teeth). Due to the invasiveness of these lesions, several dental units had lost their coronal structure, leaving remaining roots at the gum level. The exposed surfaces of these roots displayed a smooth cartilaginous consistency with uniform thickness. Excavation uncovered a hard sublayer of dentin. Multiple roots were severely shortened by the damage from both ends and were attached only to the gingival tissues. Radiographic images of these roots reflected a consistent pattern of radiolucent defects at the cervical end, separated from the intact healthy tissue beneath by a sharp line of demarcation. Root resorption, condensation osteitis, and/or bony rarefaction were sporadically evident at the apical regions; in addition, a considerable number of teeth (more posterior than anterior) were lost.

Some of the coronal lesions that began at the cervical third of the facial aspects appear to have progressed both superficially (in lateral and vertical directions to

the adjacent surfaces) and axially, invading the stroma of the teeth. The topographic invasion of these lesions was more extensive than the axial progression. Several lingual aspects of the remaining teeth were affected by these lesions. Enamel lesions displayed chalky white surfaces with occasional intensified opacity, forming halos at the cervical regions. These halos appeared in several cervical segments and were mistaken for immature calculus deposits. These enamel surfaces were easily scratchable; their margins were brittle and crumbled wherever underlying dentin was softened. The exposed dentin appeared either slightly yellowish, dirty grayish, or discolored with varied hues of brown. The dentin surface of some lesions presented a cartilaginous consistency that flaked easily upon excavation, while others had a texture ranging from soft to mushy. The lesions that affected the entire dentition appeared on the facial aspects of maxillary and mandibular anterior teeth, the facial aspects of mandibular molars, and the lingual aspects of maxillary incisors and molars, sites located far from the major salivary gland openings. The remaining lingual and facial aspects of the patients' dentitions (as well as the biting surfaces) were affected secondarily by the progression of the lesions. It was also noted that the mandibular arch (particularly the biting surfaces) showed more destruction than the maxillary/mandibular molars or the incisors.

The generalized clinical features of the destructive lesions that affected the dentitions of the 3 cases embodied the typical manifestations of dental erosion lesions.<sup>6,20</sup> It appears that these erosion lesions developed by challenging acids from exogenous sources, either alone or in combination with those from intrinsic sources.<sup>34</sup> The lesions of the patient in case report No. 3 appeared to be more invasive in sites that experience continued contact with gravitated acidic diet soda by holding of the beverage in the mouth for prolonged durations before swallowing. The 2 patients whose dentitions were deprived of salivary protective functions due to their preferred consumption of beverages containing acidic fluids were similar; their dentitions were exacerbated by the acidic nature of the drugs as well as the compromised salivary flow, a complication of their drug abuse.

### **Differentiation between erosion and carious lesions**

Among those who indulge in excessive intake of acidic beverages, their reluctance to wash, dilute, or neutralize the challenging acid and/or brush teeth regularly allows for ongoing decalcification of the enamel surface. The eroded superficial enamel layer becomes porous and soft; in addition, it loses its optical translucency and turns opaque white in color. This softened enamel layer becomes permeable and allows the acid to contact the subsurface layer, leading to its decalcification and porosity. Concomitant assault of acids increases thickness of the decalcified enamel layer and thus increases its optical opacity. Variations in terms of enamel decalcification and the degree of opacity may appear within the affected dentition; these variations correspond to the duration of exposure to the culprit acid and the absence of the salivary neutralizing capability. This combination of long exposure to acids and reduced saliva explains the generalized opacity of enamel through the entire dentition that is due to excessive intake of acidic fluids, in addition to the contributing factors related to the cases involving drug addiction. The erosion process' axial progression into dentin hastens the destruction of the tooth core. The decalcified softened dentin may retain its integrity and surface smoothness; it appears yellowish or displays darker hues of brown (Fig. 1). In general, the erosion lesions observed in the 3 cases had similar clinical and radiographic features. They displayed subtle differences in terms of dentin discoloration; the dentin affected by diet soda had a yellowish color, while the dentin affected by the consumption of regular soda and/or beer (as seen in case reports No. 1 and 2) contained shades of brown.<sup>6,20</sup>

The discoloration of the eroded dentin is the result of a biochemical reaction between the acid and sugar present in the medium.<sup>35,36</sup> These 2 ingredients act synchronously to create the intense brown discoloration in the organic rich dentin matrix; this discoloration intensifies as the lesion penetrates deeper into dentin and as the sugar concentration in the acidic beverage increases. The browning phenomenon was explained as a *Maillard reaction* or a glycosylation process that causes non-enzymatic browning in the dentin; as a result, it can be mistaken for dental caries.<sup>35,36</sup>

Recognizing the distinctive characteristics of these 2 comparable types of brown lesions and understanding their genesis is essential for identifying and diagnosing the respective etiologies. When making a differential diagnosis of erosion lesions versus carious lesions, several parameters must be considered, including the distribution of the lesions within the dentition, aspects of the tooth involved, the disparity of the lesions' presence on the facial and lingual surfaces, the specific sites in the dentition and their location relative to major salivary gland openings, the extent and size of the lesions on the surface of the tooth versus their depth (as detected clinically, radiographically, or post-excitation). Radiographic images are invaluable diagnostic tools for differentiation since they disclose the lesions' progression into tooth structures and also show the typical sharp demarcation line between the affected and healthy dentin. Systemic health, dietary, and habitual histories could reveal pertinent information that will help to distinguish between the 2 lesions.

### Role of illicit drugs

The long-term abuse of illicit drugs played significant roles (both directly and indirectly) in the development and severity of the dental erosion lesions detected in the first 2 cases. Considering the complexity of the biological factors that contributed to the demise of the 2 individual addicts' dentitions, the damage endured was similar to the known pharmacological effects and duration of action for these illicit drugs and the patient's dietary habits. Unlike the single source of acid in the third case, several sources of acids contributed to the development of erosion lesions in the first 2 cases. First, the ingredients used in preparing methamphetamine can include extremely corrosive materials such as battery acid, lantern fuel, antifreeze, hydrochloric acid, drain cleaner, and lye.<sup>37</sup> Similarly, crack cocaine is highly acidic in nature. The pharmacological action of these sympathomimetic-producing drugs (methamphetamine and crack cocaine) impact the abuser's physiological, and psychological well-being, both systemically as well as orally—particularly through the factors whose collective action adversely affects salivary flow, secretion and functions.<sup>27,30,31,37-41</sup> Once they enter the bloodstream, these drugs interfere with the

brain's dopamine re-absorption process, causing euphoria (in addition to other side effects) within a few minutes.

Methamphetamine's pharmacological effects and the severity of its negative impact may exceed those of crack cocaine due to the duration of effect (12 hours for methamphetamine compared with only 1 hour for crack cocaine).<sup>31</sup> Sympathomimetic effects of methamphetamine and crack cocaine include vasoconstriction of blood vessels; decreased salivary flow and reduction of salivary constituents. In addition, chronic methamphetamine or crack cocaine use induces mood swings, bipolar disorder, and depression—conditions that are implicated in reduced salivary flow. The psychological stresses commonly experienced during abstinence or recovery from methamphetamine or crack cocaine abuse, as well as most of the therapeutic medications used for the treatment of withdrawal symptoms, are known to reduce salivary flow.<sup>29-33,37</sup> Gastrointestinal disturbances (including hyperacidity, gastroesophageal reflux, nausea and vomiting) propel gastric acid into the oral cavity, causing further erosive damage. Neuropsychiatric complications (including anxiety and hyperthermia) may cause dryness of the mouth. The occurrence of one or more of the aforementioned events leads to decreased salivary output known as *xerostomia*, which reduces both salivary components and the redundant salivary protective functions that defend against acids and microbial disturbances in the oral environment.<sup>37-41</sup>

To compound matters, people using these drugs tend to drink acidic beverages such as soda (regular variety as a source of calories) or beer to quench their thirst; such consumption also contributes to the dramatic results of dental erosion. Hyperirritability, a common indication among illicit drug users, leads to grinding and/or clenching of teeth.<sup>40</sup> Combined with the presence of erosion-causing acids and the lack of neutralizing action due to reduced salivary flow, grinding/clenching promotes loss of softened decalcified tissues from the biting surfaces of the teeth and the formation of wear facets. Consumption of high caloric junk food and the total negligence of oral hygiene add to the cumulative adverse effects on the dental and periodontal health, as seen in the present cases.<sup>27,31,37</sup>

### Summary

Although the goal of avoiding weight gain by drinking diet soda is questionable, the danger of the erosive potential of this beverage may not be avoidable, as demonstrated in the present case involving an avid consumer. Dental erosion lesions associated with diet soda could demonstrate similar clinical features and characteristics of destruction in the hard dental tissues as those observed in patients who abuse methamphetamine and crack cocaine. The only difference is the degree of dentin lesion discoloration, which is related to the sugar/acid interaction in the medium. These lesions could mimic dental caries and must be differentiated from one another.

### Author information

Dr. Bassiouny is a professor, Department of Restorative Dentistry, School of Dentistry, Temple University in Philadelphia, Pennsylvania.

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