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Review

Dental erosion in archaeological human remains: A critical review of literature and proposal of a differential diagnosis protocol

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ABSTRACT

Objective: Although studies of dental wear on archaeological human remains have largely focused on mechanical wear (attrition and abrasion) in the past, chemical wear (erosion) is being increasingly identified as a separate form of wear. This paper aims to review the current state of research and to develop a protocol that may be universally used by biorchaeologists to specifically identify dental erosion.

Design and results: A critical review of literature has been done in order to highlight the issues related to diagnosis of dental erosion in archaeological human remains. The bodies of work based on the analysis of both modern and archaeological dentitions raise their separate problems. In addition to a need to re-evaluate symptoms of dental erosion, notably dentin 'cupping', it is apparent that no specific protocol is adapted from medical to archaeological sciences. Authors rather rely on tooth wear indices and photographs of modern clinical cases for diagnosis. Furthermore, the diagenetic chemical alternation has rarely been considered as a bias.

Conclusions: Here we suggest a three-step protocol: the primary method is the microscopic identification of dental erosion by SEM, followed by the exclusion of taphonomic aetiology on surrounding bone and soil pH analysis. Archaeologists should also explore possible causative agents of wear using archaeological and historic knowledge about the population being analyzed.

1. Introduction

In modern clinical literature, dental erosion, together with mechanical wear, are considered as the main factors affecting the dental crown in non-pathological conditions. Ganss (2008) defines dental erosion as the chronic exposure of the teeth to acids under the condition that the oral fluids are undersaturated with respect to tooth mineral. Its origin is non-bacterial and the effects are irreversible. Prevalence in modern populations is extremely variable, ranging from 2% to 100%, and depending on factors such as age (Bartlett et al., 2011; Jager, 2015; Linnett & Seow, 2001; Mahoney & Kilpatrick, 2003; Pontefract, 2002; Ritter, Grippo, Coleman, & Morgan, 2009; Spijker et al., 2009), sex (Al-Dlaigan, Shaw, & Smith, 2001; Bardsley, Taylor, & Milosevic, 2004), pathologies of the dental crown (Kazoullis, Seow, Holcombe, Newman, & Ford, 2007; Linnett, Seow, Connor, & Shepherd, 2002), calculus formation (Westergaard, Moe, Pallesen, & Holmen, 1993), temperature (West, Hughes, & Addy, 2000), and socio-economic status (Bardsley et al., 2004; Kazoullis et al., 2007). Although estimated to be universal in certain populations today (Fares et al., 2009), erosion has seldom been studied on archaeological teeth.

The aim of this paper is to assess critically the available research on dental erosion in archaeological human populations. In light of the dietary and lifestyle background of past populations, modern methods and knowledge of taphonomical processes, a differential diagnosis protocol will be proposed.

2. Dental erosion: the modern clinical evidence

The process of chemical dissolution of dental tissue is well understood (Bartlett, 2005; Lussi & Jaeggi, 2006; Lussi, Schlüter, Rakhmatullina, & Ganss, 2011), and is not to be confused with the mechanical forces (attrition and abrasion), which have a distinct aetiology (Grippo & Simring, 1995). Human dental enamel is a dense, highly mineralised crystalline tissue composed mainly of hydroxyapatite. Dentine is similar in molecular content, but differs in structure and composition (Hillson, 2005). Its higher organic content (approximatively 20% by weight in mature dentin, as opposed to 1% in enamel) and tubular structure render it more vulnerable to erosion than enamel (de Dios Teruel, Alcolea, Hernández, & Ruiz, 2015). However, the erosive potential and level of implication of certain factors are disputed (Ganss, 2008; Hannig et al., 2004; Linkosalo & Markkanen, 1985; Lussi & Jaeggi, 2006; Zero, 1996). This is largely due to the

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frequently observed coexistence of erosion with mechanical tooth wear (Johansson, Omar, Carlsson, & Johansson, 2012; Linkosalo & Markkanen, 1985; Lussi, Schaffner, Hotz, & Suter, 1991; Lussi, Hellwig, Zero, & Jaeggi, 2006; Mandel, 2005; Mannerberg, 1961; de Carvalho Sales-Peres et al., 2014).

2.1. Aetiology of dental erosion

In the studies of dental erosion, several groups of aetiological factors are commonly distinguished. The consumption of acid-rich foods and beverages is the best understood and main contributing factor (Lussi et al., 2011). These products include citrus and other fruit juices (Allan, 1967; Eccles & Jenkins, 1974; Hunter, West, Hughes, Newcombe, & Addy, 2000; Meurman et al., 1990), carbonated cola-type beverages (Gedalia et al., 1991; von Fraunhofer & Rogers, 2004), herbal teas (Angmar-Månsson and Oliveby, 1980; Brunton & Hussain, 2001; Phelan & Rees, 2003), wine (Mandel, 2005; Meurman & Vesterinen, 2000; Willershausen, Callaway, Azrak, Kloß, & Schulz-Dobrick, 2009), vinegar (Järvinen, Rytomaa, & Heinonen, 1991; Linkosalo & Markkanen, 1985; Lussi, Jaeggi, & Schärer, 1993), and beer (Bartlett et al., 2011; Gandara and Truelove, 1999; Shaw & Smith, 1998).

As an addition to these extrinsic chemical factors, it is considered that intrinsic ones are also highly correlated to dental erosion (Allan, 1969; Ranjitkar, Kaidonis, & Smales, 2011; Scheutzel, 1996). These include intra-organic substances affecting the pH of the oral cavity, such as chyme (or gastric fluid), which may be a highly erosive factor when frequently projected in the mouth through the oesophagus during vomiting or regurgitating (Rolands, 2015). These symptoms are strongly associated with known behaviors and medical conditions, such as pregnancy, elevated alcohol consumption, bulimia nervosa, food poisoning, drug side-effects, and others (Järvinen, Meurman, Hyvärinen, Rytömaa, & Murtomaa, 1988; Lussi et al., 1991; Robb, Smith, & Geidrys-Leeper, 1995; Smith & Robb, 1989; White, Hayes, & Benjamin, 1978). The effects of additional conditions such as asthma have been looked into by Al-Dlaigan, Shaw, and Smith (2002), but its relation with erosion remains unclear.

The time of exposure, frequency and location of these erosive products in the mouth are mostly the result of behavioral choices. Certain diets include many acidic dietary products, and therefore individuals with frequent consumption are more at risk (Linkosalo & Markkanen, 1985; Zero, 1996). Some researchers have attempted to correlate erosion frequencies with socio-economic status (SES), comparing wear patterns of children from different economic groups, although results are conflicting and require further investigation (Al-Dlaigan et al., 2001; Bardsley et al., 2004; Milosevic, Young, & Lennon, 1994). Frequent and strenuous exercise, such as with professional athletes, has also been suggested as a behavior leading to higher risk of dental erosion. This is an example of the interactive role of causative agents: increased fluid loss during exercise, and therefore decreased salivary flow, may lead to higher possibility of acid-rich sport drink consumption and gastrointestinal reflux (Clark et al., 1989; Milosevic, 1997; Sirimaharaj, Messer, & Morgan, 2002; Young, 1995). Occupational causes have also been observed, mainly those related to workers exposed to inorganic acids (factory workers, professional swimmers) (Tuominen, Tuominen, Ranta, & Ranta, 1989; Tuominen & Tuominen, 1990; Tuominen et al., 1991), and professional wine tasters (Gray, Ferguson, & Wall, 1998). In the latter group, factors in elevating risk of erosion may not only include a more frequent consumption of acidic wines, but also an longer contact period caused by specific drinking methods – 'swishing' and gargling (Brand, Tjoe Fat, & Veerman, 2009; Johansson, Lingström, Imfeld, & Birkhed, 2004; Mulic, Tveit, Hove, & Skaare, 2011).

The morphology and actions of the mouth is the final category of contributing factors. The role of physical actions in the oral cavity during food and beverage consumption, such as sipping, sucking, swallowing, and tongue movements, has been demonstrated by in vitro studies. During these experiments, the elimination of these physical factors while exposing the dental crown to extrinsic acids has resulted in a radically different, centripetal tissue loss pattern than the ones typically observed during life (Ganss, 2008). Acidic liquids are more likely to resist swallowing in remote areas of the mouth such as fissures on the occlusal surface of the posterior dentition and interproximal tooth surfaces. The secretion of saliva is a considerable contributor in protecting dental tissue against erosion, serving to neutralize the pH in the oral cavity through dilution of acids during swallowing. Moreover, it is oversaturated in elements that can aid in re-hardening dental tissue following early stages of erosion. The buffering capacity of this biofilm depends on many aspects such as secretion quantity, organic and inorganic content (Buzalaf, Hannas, & Kato, 2012; Featherstone & Lussi, 2006).

2.2. Problems in diagnosis

Some issues have arisen in identifying and diagnosing dental erosion in modern studies. Since, as Lussi et al. (2011) warn, the factors at play once the acid is introduced into the mouth are multifactorial, interactive, and their categorization conventional, they must be regarded as a whole. Some studies have attempted to identify a dominating cause whereas others could easily have been overlooked (Ganss, 2008). There also seem to be disparities between authors in the level of certainty regarding the erosive potential of some extrinsic factors (Ganss, 2008). This might be due in part to the sharp increase in prevalence over the last decades following a transition to a much more acid-rich diet (Jager, 2015). An urgent need for frequent diagnosis could have overshadowed that of a revision of causes and symptoms. Some significant concerns have nonetheless been raised.

Firstly, Zero (1996) points out the lack of systematic research about the effect of certain dietary products on dental erosion. Few studies demonstrate the erosive effect of some acidic products, such as honey (Grobler, Du Toit, and Basson, 1994), cider and beer (Gandara & Truelove, 1999; Nogueira, Souza, & Nicolau, 2000), and even certain dental hygiene products (Rytömaa, Meurman, Franssila, & Torkko, 1988). Many factors other than the pH of a product contribute to the solubility of apatite in an acidic environment, such as Ca, K and F concentration (Larsen & Nyvad, 1999). The contribution of these elements in reducing solubility rate has on occasion been overlooked and may have led to overambitious conclusions.

This issue has been tackled by Lussi et al. (2011) with the case of yoghurt. The authors have determined that if the Ca, K and F concentration of a product is higher than that of plaque fluid, it will negate the erosive effect of this product, which supports the frequent conclusions of clinical studies on the minimal or absent role of yoghurt in erosion (Al-Dlaigan et al., 2001; Mathew, Casamassimo, & Hayes, 2002; Sirimaharaj et al., 2002). In spite of this, yoghurt is still listed in questionnaires for clinical studies as in the older literature it has been said to be a contributing factor in erosion (Lussi et al., 1991). Therefore, more in vitro studies should be conducted to isolate high-risk dietary products. This would allow researchers to remove bias caused by biological and behavioral factors, which is a major cause of disparities in results of clinical studies and case reports (Ganss, 2008).

Complications in diagnosis are added as we consider that the first stage of erosion is characterised by the partial demineralisation of enamel surface, making it more susceptible to mechanical wear on the occlusal facets (Lewis & Smith, 1973). Tissue loss is therefore almost inevitably the result of attrition (wear resulting of the contact of upper and lower dentition) and abrasion (wear resulting of the contact of teeth with exogenous substances) as well. The mechanical action of the tongue (Abrahamsen, 2005; Gregg, Mace, West, & Addy, 2004) may be also considered, as it may prevent contact of acids with teeth in certain areas. For example, when regurgitation occurs the anterior mandibular dentition is protected, whilst the lingual surface of the maxillary dentition is the most vulnerable area.