

Prevention and therapy of acid-induced dental tissue loss (erosions)

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Erosions (Fig. 1) arise due to the chronic action of acids of non-bacterial genesis on plaque-free tooth surfaces provided there is undersaturation with regard to tooth mineral. The critical pH value in the saliva is 4-4.5 (Stephan 1966, Larsen and Nyvad 1999).



Fig. 1: Generalized severe acid-induced tooth structure losses with exposure of the dentin in a thirty-year old male who had enjoyed soft drinks for years 3-5 times daily.

Acids can act on the tooth structure either exogenously, for example on consumption of acidic foods and drinks, or endogenously due to gastric acid. Depending upon the individual predisposition, acid-induced losses of tooth structure become clinically evident only on frequent chronic action over a longer period.

Primary prevention should consist generally of appropriate information about causes and avoidance of erosive tooth damage within the scope of the established prevention strategies and individual counselling. Further measures related to the population depend upon the prevalence of erosive losses of tooth structure and should therefore be discussed specifically for the country in question. Secondary prevention comprises above all the early and differential diagnostically correct detection of the early stages of erosions within the scope of screening examinations and individually coordinated causal measures.

For better understanding of prevention and therapy strategies, the pathogenesis and

ultrastructure of erosions are described briefly below.

In erosive demineralization there is centripetal loss of substance in the enamel, which is manifested as clinically visible surface defect in the case of continuous exposure to acids. A partially demineralized zone with reduced microhardness is found on the eroded enamel surface (Lussi et al. 1995), which corresponds ultrastructurally more or less to a classical etching pattern (Meurman and Frank 1991) (Fig. 2).

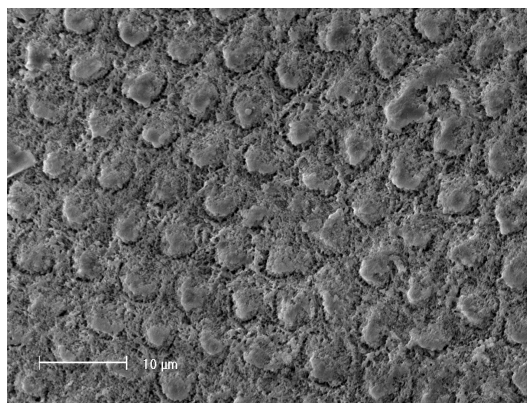


Fig. 2: Typical etching pattern in the enamel after erosion with citric acid.

Thus the ultrastructure of enamel erosion differs fundamentally from initial enamel caries, in which the zone of the greatest demineralization lies under a pseudo-intact surface layer (Thylstrup and Fejerskov 1994). In the dentin after short-time action of acids, there is firstly loss of mineral in the region of the peritubular dentin and with longer action time enlargement of the dentin tubules with demineralization of the intertubular dentin (Noack 1989, Meurman et al. 1991). In this case the organic matrix is exposed. Both in laboratory experiments (*in vitro*) and in samples which were worn in the mouth (*in situ*), this organic covering layer is displayed regularly (Fig. 3) but the role that it plays clinically is so far unclear. In contrast to caries, which as a rule always requires invasive therapy as from a certain stage, acid-induced tooth structure defects come to a standstill independently of their extent, if sufficient causal or symptomatic measures are adopted. As a rule no restorative treatment is then necessary, unless there are aesthetic or functional impairments.

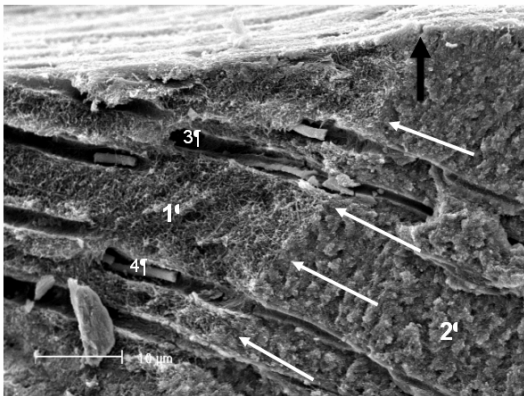


Fig. 3: Dentin erosion after action of hydrochloric acid (cross-section). On the right of the black arrow the sample surface was protected by resin. In the area of acid action (on the left of the black arrow) the demineralized organic dentin matrix (1) can be seen clearly in sharp delineation to the unchanged dentin (2) (white arrows); (3) dentinal tubule, (4) odontoblast process.

The causal therapy of acid-induced tooth structure defects starts with the identification of the acid source. This includes an anamnestic consultation which includes questions regarding exogenous and endogenous acid exposure. In addition an open nutrition protocol (Lussi 1996) can give further indications in many cases with regard to quantity, nature and frequency of acid action due to food. In the case of exogenous acid action, the causal therapy consists in changing the eating habits, which frequently does not necessarily require major changes in behaviour. For example, apart from reducing the frequency of consumption, less erosive drinks can be consumed. Both *in vitro* and *in situ* studies have shown that solely the addition of calcium can reduce considerably the erosive potential of drinks (Hughes et al. 1999a, Hughes et al. 1999b). Fruit can be consumed together with milk products. In the case of endogenous acid action, medical treatment can be indicated (e.g. in reflux diseases), but frequently causal therapy is difficult. For example, eating disorders with chronic vomiting can exist for years despite therapeutic efforts. In these cases symptomatic measures are necessary just as in unclarified exposure to acids.

The purpose of symptomatic measures is to modify the tooth surface so that the erosive demineralization and thus the loss of microhardness is reduced.

Substances that lead to acid resistant mineral precipitations in or on the tooth surface or that form permanent coatings are suitable for this. The application of dentin adhesives has been discussed as non-mineral coating (Azzopardi et al. 2004, Sundaram et al. 2007). This measure is suitable as an acute measure. Since it can be expected that these coatings are abraded at least in the medium term, their protective effect may be limited in time.

Mineral precipitates can be expected generally from oversaturated calcium/phosphate solutions such as saliva. Therefore it is frequently recommended not to clean the teeth directly after the action of acids but to wait for the "remineralization" of the tooth surfaces. Such recommendations were derived from laboratory studies with saturated/oversaturated calcium/phosphate solutions. However, *in situ* experiments have been able to prove only slight effects of waiting times (Jaeggi and Lussi 1999, Ganss et al. 2007). But there is a good explanation for these apparently contradictory findings.

In vitro the precipitation of calcium and phosphate from saturated solutions onto etched enamel depends upon different factors (Amjad et al. 1981), but crystal growth can be proven easily. However, intraorally the oversaturation of the saliva is maintained by proteins such as statherin, proline-rich proteins or histidine-rich polypeptides, so that normally no precipitation of calcium/phosphate salts takes place on clean tooth surfaces. Mineralization processes can occur only if diffusion of these proteins is obstructed, as for example by the pseudo-intact surface layer of initial caries (remineralization of the initial caries) or by plaque (formation of calculus). In the case of eroded enamel, under oral conditions neither a relevant increase of the microhardness has been proven (Collys et al. 1991, Collys et al. 1993) nor could the precipitation of mineral be shown (Garberoglio and Cozzani 1979, Allin et al. 1985). These findings also correspond to the results of *in situ* studies, that have shown only a slight effect of waiting times between erosive demineralization and brushing (Jaeggi and Lussi 1999, Attin et al. 2001, Ganss et al. 2007). A change of the oral hygiene habits,

apart from insufficient plaque control, is therefore expedient only in the case of traumatic oral hygiene techniques or extreme effects of acid.

However, mineral precipitates can be generated by the local application of fluorides. According to the form of administration, more or less pronounced covering similar to CaF_2 layers arise, which however are relatively easily soluble in acids. Contradictory estimates are published in the literature about the effectiveness of these measures (Wiegand and Attin 2003), some authors cast doubt on the sense of fluoridation measures for therapy of erosions (ten Cate et al. 2003). Therefore fluoridation recommendations that lead to the most pronounced possible precipitates are generally given. Precipitates similar to CaF_2 are the thicker the more acidic and concentrated the fluoride products are, and the longer the action time is (Saxegaard and Rølla, 1988). Therefore acid preparations should be used as frequently as possible as gels with high fluoride concentration and/or as mouth wash solutions in addition to a fluoride toothpaste (Schmidt et al. 2003, Ganß 2005, Wiegand and Attin 2003). In fact intensive fluoridation can be very effective at least under in situ conditions even with longer and frequent effects of acid. Intensive fluoridation has also proven to be clearly more effective than waiting times for reducing abrasion by brushing (Ganss et al. 2007).

However, such a therapeutic approach means that patients should fluoride frequently and possibly with different forms of preparations such as mouth rinse and gel, which means a considerable organizational and financial effort. Such recommendations are therefore only limitedly suitable for longer therapeutic application and are not suitable for preventive measures.

So far the fluoride compounds of sodium fluoride, amine fluoride or sodium monofluorophosphate known from cariology and contained generally most frequently in oral hygiene products have been examined. However, more recent studies show that the effectiveness of fluorides in the context of erosions is determined essentially by the

nature of the fluoride compound. The different effectiveness of different fluoride compounds becomes especially clear if preparations of the same pH value and same concentration are compared with one another (Schlueter et al. 2007, Ganss et al. 2008). Thus it can be shown that erosive mineral losses can be prevented practically at least under mild conditions by stannous fluoride or amine fluoride/stannous fluoride solutions, whereas sodium fluoride or amine fluoride/sodium fluoride solutions appear to be significantly less effective. A new result so far is that even a stannous chloride solution without fluoride shows effectiveness that lies in the order of magnitude of a sodium fluoride solution. Electron optical examinations have shown that after application of solutions containing tin, apparently relatively acid-resistant precipitates are formed (Ganss et al. 2008), whereby quite generally fluoride compounds with polyvalent metal ions come into view as potential erosion inhibitors. Titanium and, as already mentioned, tin must be emphasized specially in this connection (Ganss et al. 2006, Hove et al. 2007). Titanium fluoride has been examined so far in the form of experimental preparations. After treatment with titanium fluoride glaze-like deposits that are resistant to mechanical and chemical influences are formed and they can even withstand treatment with concentrated hydrochloric acid (Büyükyılmaz et al. 1997). The reaction mechanisms that lead to the formation of such deposits are not clarified, but reactions between the titanium ion and the organic constituents of the tooth structure or the oxygen available on the tooth surface as well as the formation of stable titanium dioxide are discussed. So far the good effectiveness of titanium fluoride has been shown primarily for concentrated and very acid solutions that are not suitable for domestic use. However, more practicable forms of preparation appear to develop no better effect than, for example, solutions containing stannous fluoride (Fig. 4). It can be stated in conclusion that in the symptomatic therapy with fluorides of erosions apparently the fluoride compound is significant. At present acidic preparations containing stannous fluoride appear to be the most effective erosion inhibitors.

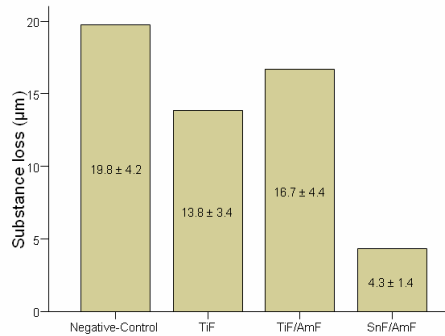


Fig. 4: Substance loss in the enamel after cyclic demineralization and remineralization *in vitro*. The samples were eroded over 7 days for 6x2 minutes daily with 1% citric acid solution and treated for 2x2 minutes daily with the test solutions in three test groups (TiF: titanium fluoride solution, TiF/AmF: titanium fluoride-amine fluoride solution, SnF/AmF: stannous fluoride-amine fluoride solution) after the first and last erosion. There was only erosion in the negative control group.

The therapy of acid-induced tooth structure losses can be summarized as follows:

Clarify the diagnosis of “erosion” carefully (differential diagnosis attrition, abrasion or wedge-shaped defect!), if confirmed:

- Anamnesis for identification of the acid exposure
- Causal measures according to the nature of the acid action (change of nutrition, internal medical therapy, psychotherapy)

If causal measures are not possible or not sufficient, or supporting:

- Symptomatic measures with an effective product (containing stannous fluoride)
- Change oral hygiene measures only in the case of traumatic oral hygiene technique or extreme acid exposure

For assessment of the success of therapy

- Photos or good situation models at intervals of 6-12 months

Restorative measures only with very pronounced losses of substance, functional disorders or clear aesthetic impairments, previously bring erosions as far as possible to a standstill.

For reference list please contact the author.