Oral Infections and the Risk of Mortality in the Iron Age

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Abstract

Medically unattended oral abscesses may spread to their surrounding area or even throught the entire body. Depending on the individual`s constitution, such processes may lead to a life-threatening situation or death. (Pre)historic case studies deliver more information about distribution and adverse effects of oral infections and the risk of mortality. In this particular case report an iron aged skeleton of a 35- to 45-year-old woman from Eulau (Naumburg/ Saale, Germany) shows multiple periapical lesions and their effects on the viscerocranium. Furthermore, alterations on the long bones can be associated with chronic inflammation processes.

Keywords: Oral infection; Periapical lesion; Iron Age

Introduction

In historic and prehistoric times oral infections played an important role in the morbidity of populations. Without medical treatment, an oral abscess spreads to the surrounding area or throughout the entire body was very high. Depending on the constitution of an individual, such a process can lead to a life-threatening situation or even death. The risk of mortality due to odontological infections has been described in prehistoric case studies (1).

Materials and methods

In the present case study, the well preserved skeleton of a 35 to 45-year-old woman shows multiple periapical lesions and further effects on the viscerocranium and postcranial skeleton. The burial site is part of an iron aged cemetery from Eulau - a site near Naumburg (Saale), Central Germany.

The age and sex diagnosis is based on classical methods. For age estimation we used the ectocranial suture closure (2), tooth wear pattern (3, 4) and the auricular surface of the ilium (5, 6). To determine the sex morphological traits of the pelvis and the skull were appraised (7, 8). In addition metric criteria like the vertical diameter of the caput humeri and caput femoris were also comprised and compared with other individuals from the same site.

For the radiographic evaluation of the periapical lesions in the upper jaw we took digital volume tomography (DVT) and panoramic radiograph (PR) images.

Results

In the upper jaw massive bilateral inflammatory changes to the teeth and alveolar bone structures are visible (Figures1a-c). On the left an ante mortem tooth loss of the second premolar (P25) caused by an inflammation process can be seen. The alveolar bone is broadened and shows a densified surface presumably caused by gangrene. Furthermore, a distinct perforation of the maxillary sinus accompanied by maxillary stomatitis is visible. Further centers of inflammation derived from the first molar (M26) which shows a gross caries lesion (caries profunda) with an open pulp. Here, the osteolytic process induced a perforation of the alveolar bone to the buccal side (Figures2a,b). In the right jaw there is also a molar (M17) with a distinct caries lesion and an open pulp. This infected pulp caused a periapical reaction and yet another opening in the maxillary sinus. It should be noted that all caries lesions had a cervical origin.

The multiple centers of inflammation are accompanied by porous alterations on the bone surface which indicates massive vascularisation of the corticalis. On the left side these structural changes range, in reduced form, from the alveolar bone to the infraorbital rim. In addition, the left infraorbital foramen is slightly enhanced and the left zygomatic process is merely thickened. Furthermore, postcranial changes can be linked – directly or indirectly – to the oral infections. This is evident especially in the lower extremities displaying periostal reactions, which can be associated with chronic inflammation processes. The right distal part of the radius shows a swelling and in addition, osteoarthritical reactions as well as osteophytes and osteolytic changes at the last two lumbar vertebrae including spondylolysis (L-5) (Figures 3a,b).

Discussion

Moisture, temperature and the absorption of food in the mouth provide ideal living conditions for germs. Injuries to the mucous membrane can offer the entrance for facultative pathogens which may induce an infection in this new environment (change from aerobic to anaerobic) may induce an infection. The extent of the reaction depends upon the immunity of the host and the virulence of the germs (1). In general, inflammations can be differentiated into specific (e.g. mycobacterium tuberculosis, treponema pallidum) and unspecific reactions. Unspecific inflammations of the oral cavity are induced by ubiquitous living, pyogenic germs like Streptococci, Staphylococci, Fusobacteria, Peptostreptococci, Porphyromonas and Bacteroides species (9). The inflammation process begins in the soft tissue and continues into the bone structure in terms of periostitis, osteitis or osteomyelitis. The major causes are transmitted inflammation processes of the gums (e.g. marginal periodontitis) or

destroyed by caries (10, 11). Via lympho- or haematogenous distribution oral infections can spread to organ systems causing e.g. mediastinitis or pericarditis (12, 13, 14). On the other hand metabolic diseases, like diabetes or nutritional deficiencies (e.g. vitamin C) have a negative influence on the progress of inflammatory processes by reducing the immune response (15, 16, 17).

In certain cases it is possible that the inflammatory changes in long bones and vertebrae are in primarily connected with oral infections. But a secondary infection of the postcranial bones cannot be ruled out because the chronic inflammation in the jaw has likely impaired the immune system. The alterations described in the upper jaw suggest a significant physical impairment of the woman's health. It is very likely that this woman died because of massive osteolytic processes in the jaw and the surrounding area of the viscerocranium it is very likely that this woman died as a result of the ongoing chronic inflammation and this was not a singular case in (pre)historic times.

Conclusion

It is abundantly described in literature that local infections are able to cause osteomyelitis or osteoarthritis via haematogenous or lymphogenous spread (18, 19). But oral infections and inflammatory changes of postcranial bones are frequently examined separated from each other. Relations between these two are rarely established. However, recent studies indicate that oral infections are able to spread to the entire body and manifest themselves in form of acute or chronic inflammatory processes. The progress of clinical manifestation depends on the immune defence of the person affected as well as the virulence of the germs (12, 13, 14). So, in pre-antibiotic times oral infection may have been ascribed an important role. It is apparent, that the consequences of oral infections are still insufficiently investigated and that there is need for further studies.

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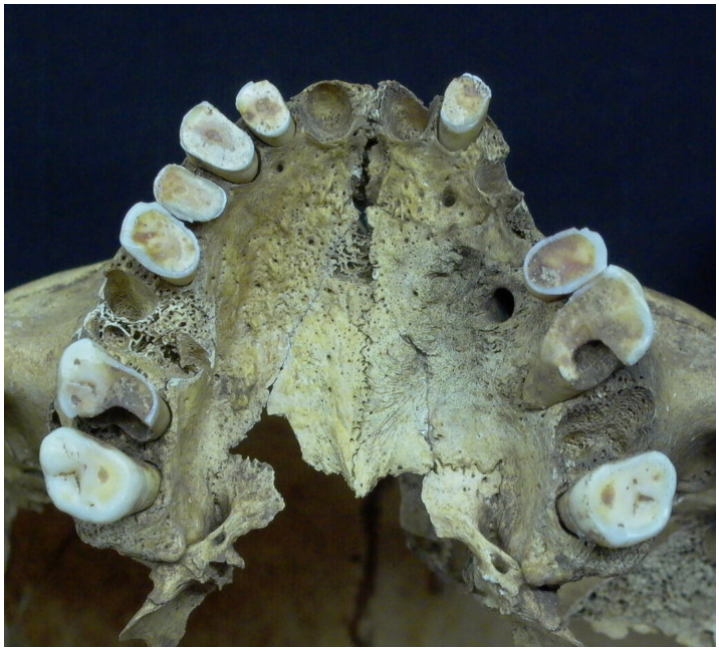

Figure 1a Left side of the upper jaw: an ante mortem tooth loss of the second premolar (P25). Furthermore, a distinct perforation in the maxillary sinus.

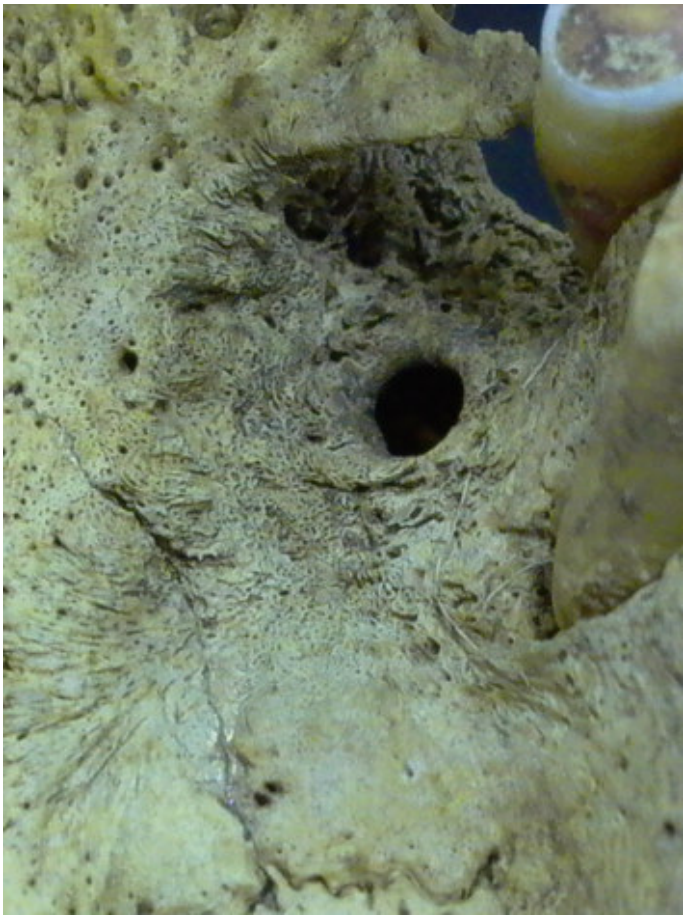

Figure 1b Left upper jaw: the alveolar bone is broadened and shows a densified surface, presumably caused by gangrene.

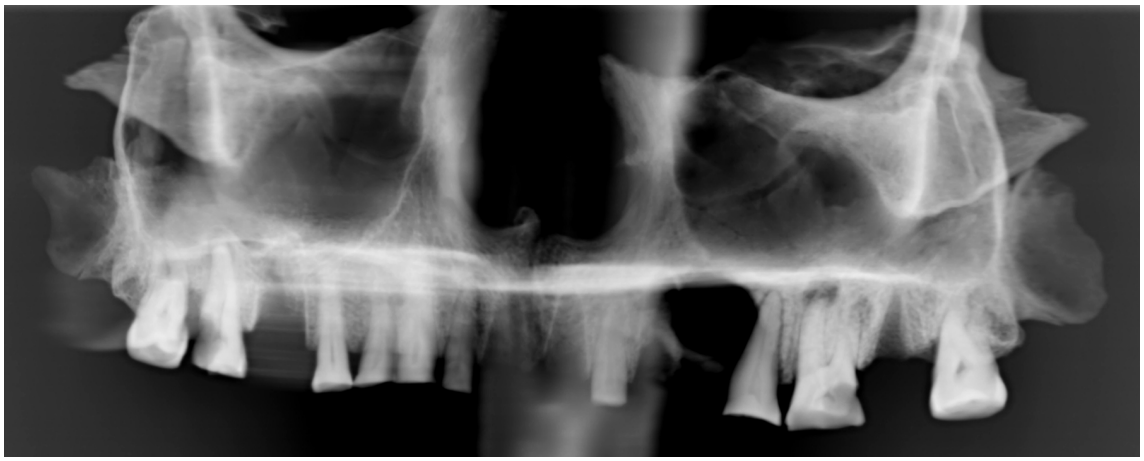

Figure 1c Panoramic radiograph image of the upper jaw: on both sides (M26, M17) periapical reactions are visble.

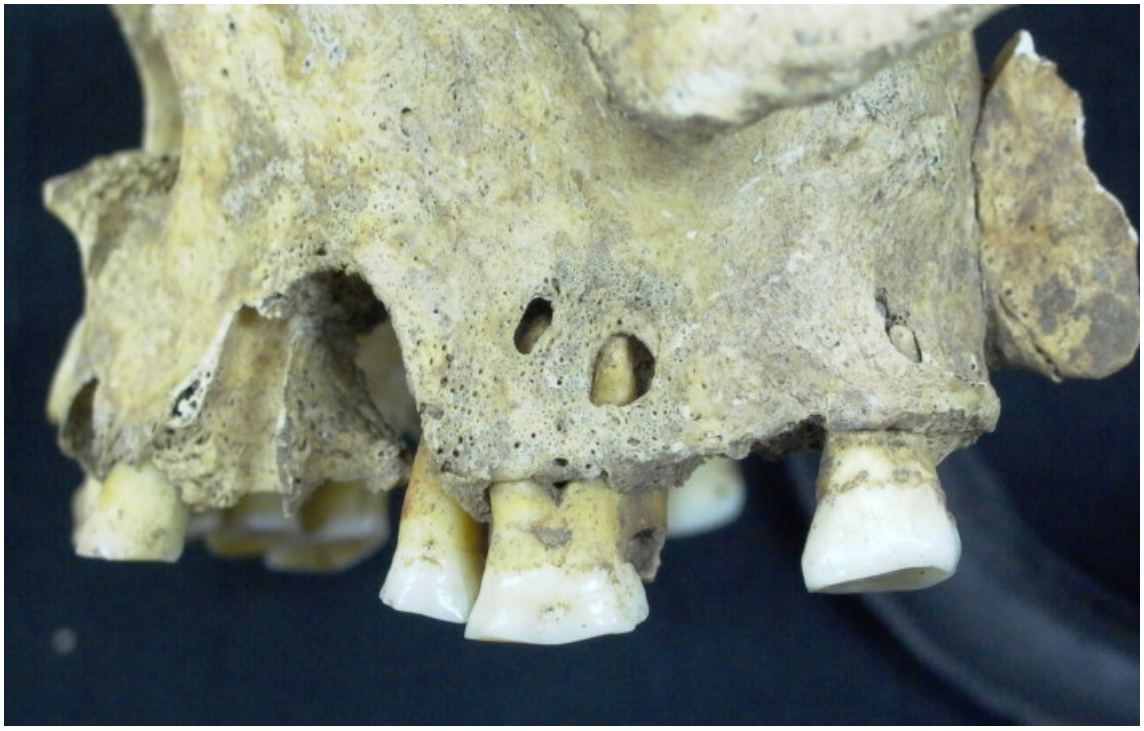

Figure 2a Left upper jaw: caries lesion and open pulp of the first molar (M26). Periapical reaction caused a perforation of the alveolar bone to the buccal side.

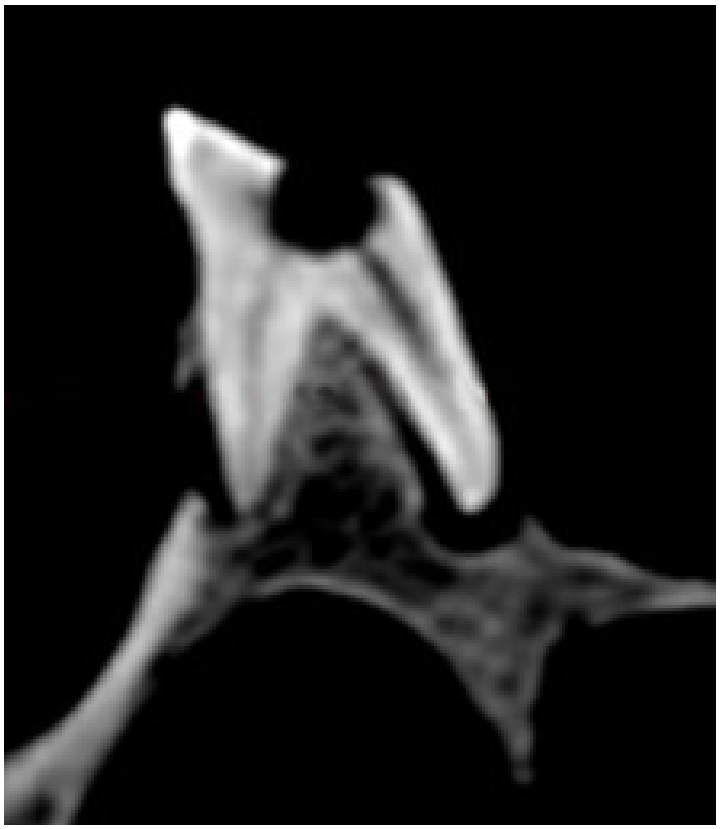

Figure 2b DVT image of the left first molar (M26): periapical reactions with openings to the palatinal and buccal side of the alveolar bone.

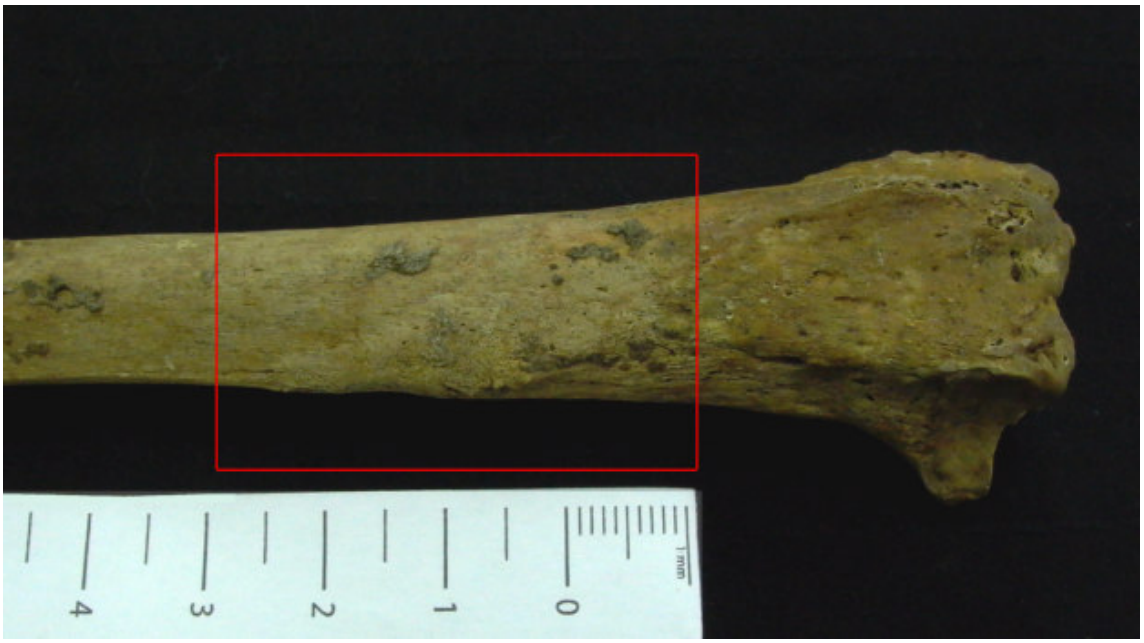

Figure 3a Swelling at the right distal part of the radius. This alteration is not caused by a fracture but is the result of a healed inflammatory process.

Figure 3b Spinal osteophytosis and osteolytic changes at the last lumbar vertebra (L-5) including spondylolisis.

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