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A scurvy case in an infant from Monte da Cegonha (Vidigueira – Portugal)

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Abstract Monte da Cegonha is a roman *villa* in Vidigueira, south of Portugal, built in the first quarter of 1st century and preserved until the 12th century. Inside a basilica from the 4th century, probably one of the first Christian Churches in Iberian Peninsula, some human burials were found. One of them, exhumed in 1986 is here reported. It concerns an almost complete and well-preserved skeleton of an infant. A series of porous abnormalities affecting the skull and the scapulae are the most striking expressions of this condition and their distribution pattern suggests scurvy. Even when differential diagnosis is done, which is here discussed, scurvy remains the most probable aetiology.

Key words Non-adult skeleton; scurvy; Christian church; Monte da Cegonha; Portugal.

Resumo Monte da Cegonha é uma *villa* romana na Vidigueira, sul de Portugal, construída no primeiro quartel do século I e preservada até ao século XII. Nela existe uma basílica Cristã datada do século IV, considerada como uma das primeiras da Península Ibérica, onde foram encontrados alguns enterramentos. Neste trabalho apresentam-se os resultados do estudo de um dos esqueletos exumados em 1986. Trata-se de um indivíduo com cerca de um ano, bastante completo e bem preservado onde foram observadas várias alterações poróticas que afectam tanto o esqueleto craniano como o pós-craniano. Após diagnóstico diferencial o escorbuto foi considerado como a etiologia mais provável para essas lesões.

Palavras-chave Esqueleto de não adulto; escorbuto; basílica Cristã; Monte da Cegonha; Portugal.

Introduction

Monte da Cegonha is a roman *villa* in Vidigueira (Southeast of Portugal) built in the first quarter of the 1st century. This roman *villa* was preserved until the 12th century.

The infant here reported was inhumed inside a basilica dated from the 4th century; probably one of the first Christian Church's built in Iberian Peninsula (Alfenim, 1992; Alfenim and Lopes, 1994). Inside the basilica, the inhumations seem to have occurred between the 4th and 6th centuries, before Concílio de Braga (Council of Braga) in 572 AD (Alfenim, 1992; Alfenim and Lopes, 1994).

The pathological case here analyzed was observed in an individual from Monte da Cegonha excavated in 1986. During this excavation 8 individuals were exhumed from an ossuary, 6 adults and 2 non-adults (Ferreira and Cunha, 2001).



Figure 1. The young child skeleton displayed in laboratory.

About the young child

The skeleton subject of this study was almost complete and in a reasonably good state of preservation (Figure 1).

The individual was inhumed in a supine position on a grave built with bricks (sepulture 1, square AII: 21) oriented South-North, with the cranium to the South, taking advantage of the West wall of the basilica (Alfenim, 1992). Graves inside the basilica had a West-East orientation. More archaeological information about the excavation is missing.

The skeleton was analyzed morphologically and morphometrically. Age at death was determined on the basis of tooth development (Ubelaker, 1989), the degree of ossification and diaphyseal length of long bones, following the methodologies recommended by Scheuer and Black (2000). The skeleton was of an infant about 1 year (± 4 months) at the time of death.

Description of the lesions

The main pathological features are porous lesions which are defined following Ortnier and Ericksen (1997), as a localized, abnormal increase of fine holes, visible without magnification, that penetrate the lamellar bone surface.

In the Monte da Cegonha infant, these lesions are mainly observed in the cranium. The postcranial skeleton was not affected except for both scapulae. This pathological condition was characterized by active porotic abnormalities distributed bilaterally in the following bones: parietal bones (Figure 2); greater wing of the sphenoids (Figure 3); temporal bones (Figure 4); orbital roof (Figure 5); right zygomatic bone (the left zygomatic bone is missing); maxilla (posterior surface and infraorbital foramen); mandible (coronoid process); supraspinous fossa of both scapulae (Figure 6).



Figure 2. Detail of right parietal showing a porous lesion with hypertrophic bone formation.



Figure 3. Abnormal porosity of greater wings (left and right) of the sphenoid.



Figure 4. Porous lesion in the left temporal bone.



Figure 6. Abnormal porosity in supraspinous fossa of left scapula.

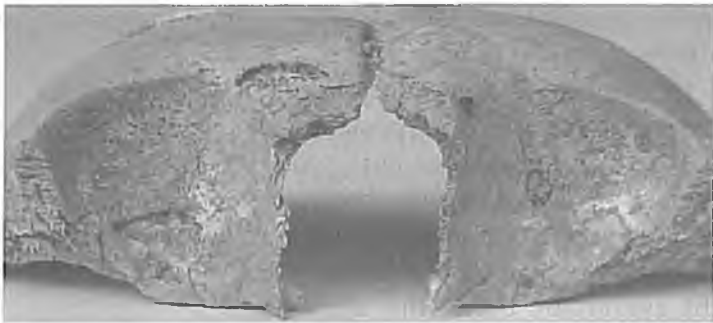


Figure 5. View of the orbital roof showing bilateral porous lesions with hypertrophic bone formation.

Probable diagnosis

The distribution pattern of the lesions as well as the characteristics of the lesion, made us believe that we are dealing with a probable case of scurvy.

Scurvy lesions tend to be bilateral and characterized by porosity and, in some cases hypertrophic bone formation (Ortner and Ericksen, 1997). While these lesions resemble those seen in anemia and infection, their distinctive anatomical location and association with muscle action such as chewing should differentiate them from other pathological conditions (Ortner and Ericksen, 1997; Ortner *et al.*, 1999; Ortner *et al.*, 2001).

The diagnosis of scurvy is based on a series of porous abnormalities and their distribution patterns, being a bilateral

porous lesion of the greater wing of the sphenoid and adjacent sites in the skull the most common expression of scurvy. Ortner and Ericksen (1997) and Ortner *et al.* (2001) consider that bilateral porous injuries in the greater wing of the sphenoid are the pathognomonic sign of scurvy. The haemorrhage related to the greater wing of the sphenoid and adjacent bones is associated with the anatomy and vascular supply of the temporal muscle (Ortner and Ericksen, 1997).

Scurvy is a condition caused by lack of vitamin C resulting in defective collagen synthesis with consequent skeletal growth retardation and haemorrhagic phenomena, which can lead to porous lesions (Ortner *et al.*, 1999). Vitamin C is important in connective tissues formation, including collagen and cement material (Ortner and Ericksen, 1997). A defect in the cement substance that binds the endothelial layer in blood vessels (Ortner and Putschar, 1985) causes increased susceptibility to haemorrhage that can result from even normal movement or muscle activity such as chewing or eye motion (Ortner and Ericksen, 1997).

The young children are particularly vulnerable to haemorrhage with subsequent inflammatory response because of their fast growth, accompanied by rapid remodeling replacement and growth of tissue create conditions in which the periosteum is less tightly attached to the underlying bone (Ortner and Ericksen, 1997; Ortner *et al.*, 1999; Ortner, 2003). So, haemorrhage can easily occur between the periosteum and bone in infants and young children, provoking inflammation, which results in bone porosity. Another symptom of scurvy is possible haemorrhage of the eye (Ortner and Ericksen, 1997), with consequent porous lesions on the orbital roof as seen in Figure 5.

Scurvy provokes painful limbs that minimize most physical activity but appetite is minimally affected (Ortner and Ericksen, 1997). The muscle activity due to eating and chewing can cause chronic haemorrhage and associated inflammatory response in those areas of the skull anatomy related to chewing (Ortner and Ericksen, 1997; Ortner *et al.*, 2001). In the case reported here, porosity is associated with bony surfaces related to chewing (greater wings of the sphenoid, temporal bones, zygomatic, mandible) (Figures 3 and 4).

The lesions found in scapulae (Figure 6) can be explained by a similar anatomical arrangement and provide additional confirmation for scurvy diagnosis (Ortner and Ericksen, 1997).

Other diseases that can produce similar lesions are rickets and anemia (Ortner and Putschar, 1985). The lesion pattern of rickets is very different than scurvy. Anemia also provokes porous lesions as does infection. Scurvy can occur in association with anemia, but anemia lesions are caused by marrow hyperplasia and scurvy lesions are the result of a superficial inflammatory response to bleeding (Ortner and Putschar, 1985; Ortner, 2003). Since there was no evidence of marrow hyperplasia in the present case, anemia diagnosis was excluded.

Rickets is a systemic disease usually caused by an inadequate intake of vitamin D and its precursors, with a great prevalence in high latitudes populations, which is not the present case. The rachitic skull shows porous bone but with external and internal subperiosteal bone deposition that can also occur on the facial bones (Ortner, 2003). Early rickets manifestations are seen in the metaphysis of long bones. In the infant skeleton here presented the long bones are not affected, and the skull lesions are not compatibles with rickets.

Conclusions

Scurvy, iron-deficiency anemia and rickets are nutritional deficiency diseases and many occur in the same case. However, the typical and distinctive pattern of scurvy was decisive to the present diagnosis. In this sense and considering the discussion above, scurvy seems the most probable aetiology.

In conclusion and regarding differential diagnosis, plus the patterned results of the palaeopathological study done on the young child from Monte da Cegonha, the most probable diagnosis points to a metabolic disease, namely, scurvy, or Möller-Barlow disease (Ortner, 2003).

This case seems to be the first one reported to the Portuguese archaeological record, which does not reveal any descriptions of cases of scurvy in children prior to this one.

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