Possible case of scurvy from the Roman site Viminacium (Serbia)

Šarkić N1. y Redžić S2.


2 Institute of Archeology (Belgrade, Serbia).

Corresponding author: nsarkic@gmail.com

INTRODUCCIÓN

The excavation of the necropolis of the Roman town Viminacium (Eastern Serbia) in 2015, brought to light an individual with unusual pathological changes. The skeletal remains that were found in grave G-5663, trench 478, dating from Late Antiquity can be described as well-preserved. The individual was buried in an extended supine position, with SW-NI orientation and with no burial goods (Figure 1).

Palabras clave:
Malnutrición
Enfermedad
Paleopatología
Deficiencia de Vitamina C
Retraso en el Desarrollo
Antigüedad Tardía

Keywords:
Malnutrition
Disease
Paleopathology
Vitamin C deficiency
Developmental Delay
Late Antiquity

ABSTRACT

In 2015, during the excavation of the necropolis of the Roman site Viminacium (eastern Serbia), dated to Late Antiquity, human skeletal remains of a young female, showing visible pathological changes, were found in grave G-5663, trench 478. Epiphyses of femurs, tibias and the right radius did not show any sign of fusion, although the third molar had already erupted, which could lead to the conclusion that a developmental delay occurred. Macroscopic examination of the skeleton revealed porotic changes and new bone formation that were visible on all the long bones and skull - most likely connected to scurvy (vitamin C deficiency).

RESUMEN

Durante la excavación en el año 2015 de la necrópolis del yacimiento romano de Viminacium (Este de Serbia), los restos óseos humanos de una mujer joven, mostrando cambios patológicos visibles, se encontraron en la tumba G-5663, sondeo 478. La epífisis de ambos fémures, tibias y radio derecho no mostraron ningún signo de fusión, aunque el tercer molar ya había erupcionado, lo que podría llevar a la conclusión de que se produjo un retraso en el desarrollo. El examen macroscópico del esqueleto reveló cambios poróticos y nueva formación ósea visibles en todos los huesos largos y en el cráneo, probablemente relacionados con el escorbuto (deficiencia de vitamina C).
Possible case of scurvy from Viminacium

Material and Methods

Although it is recommended to perform histological analysis when pathological changes on bones indicate that an individual might have suffered from scurvy (Schultz, 2001; Ortner, 2003; Schultz et al., 2007) there is an obvious disadvantage of using this technique, as it can cause the destruction of a sample. The other main problem is that histology can only ascertain if a new bone is present by examining cross sections, but these changes can often be visible to the naked eye (Sinnott, 2015). In the study of the individual G-5663, only macroscopic analyses were performed, as permission for destructive sampling of the study material could not have been obtained.

Human skeletal remains, found in the tomb G-5663 were cleaned and analysed in the laboratory for Physical Anthropology in the site of Viminacium. The osteological material was washed with lukewarm water and a soft brush. Reconstruction of broken fragments was done using transparent and easily removable glue “OHO”.

Preservation Index (PI) was used for the calculation of the degree of skeletal preservation, proposed by Walker et al. (1988) and modified by Safont et al. (1999). It considers the preservation of different bone groups (humeri, ulnas, radii, femurs, tibias, fibulae, scapulae, clavicles, pelvis, sacrum, mandible, splanchnocranium and neurocranium) by using the equation PI = bones preserved/bones considered x 100. According to this method, the state of preservation of the individual G-5663 can be assessed as good, with the Preservation Index = 81%. Unfortunately, none of the long bones was preserved entirely, so it was impossible to perform measurements of the postcranial skeleton.

Due to bad preservation of the coxal bone, the determination of the sex of this individual was based on cranial and mandibular morphology, using methods of Acsadi & Nemeskeri (1970); Herrmann et al. (1990); Schutkowsky (1993); Buikstra & Ubelaker (1994), and Loth & Henneberg (1996).

Methods based on epiphysis fusion and tooth eruption were used for the age estimation of this individual. We relied on Scheuer & Black (2000) methods for the timing of fusion of the epiphysis, while the methods of Smith (1991), Ubelaker (1989), Hillson (2002) and AlQahtani (2009) were used for teeth eruption methods. Dental development is less affected by environmental and physiological conditions, such as nutrition and hormone imbalance, because it is under strict genetic control (Konigsbers & Holman, 1999). It is a common agreement in the scientific community that the estimation of age, based on tooth development, approaches more closely to chronological age than the estimation based on bone fusion (Garn et al., 1959, 1965; Lewis & Garn, 1960; Cardoso et al., 2013). Therefore, the age was estimated according to tooth eruption, as this method is consider to be more reliable.

In addition to this, dental and paleopathological analyses were conducted. Descriptive methods were used for the analysis of pathological changes (Campillo 2001). The remains were examined macroscopically: visual inspection with naked eye under natural light; and microscopically: with 10X magnifying glass. Ortner and colleagues’ (Ortner & Ericksen, 1997; Ortner et al., 1999, 2001) criteria for describing and identification of the lesions were employed.

Results

Sex and age assessment

The sex of this individual was determined as female based on the lack of nuchal crest (“1” accor-
According to the method of Acsadi & Nemeskeri 1970; form and size of the mastoid process – very small and round (“2” according to the method of Acsadi & Nemeskeri 1970); minimal expression of mental eminence (“1”, according to the method of Acsadi & Nemeskeri 1970); the shape of mandible - gracile, without inversion of the gonium, which are considered to be characteristics of female individuals (Herrmann et al. 1990); and with straight posterior border of the mandibular ramus (“-1” according to the method of Loth & Henneberg 1996).

Considering the age estimation, it was noticed that there was asynchronicity between epiphyses fusion and tooth eruption. The epiphyses of both femurs, both tibias and right radius were not fused (Figure 2). The timing of fusion varies greatly in different parts of the skeleton, in response to the function of the soft tissues with which that element is associated (Cunningham et al., 2000), and it also depends on the sex of the individual, as fusion happens earlier in girls than in boys (Scheuer & Black, 2000). Each bone in the body has a predictable age range when fusion of epiphysis occurs. This fact is considered to be of great importance in forensic and legal studies. For that reason it has been studied by many researchers, such as Saunders (1992), Bass (1995), Scheuer & Black (2000), Steele & Bramblett (1988) and to name a few. According to this methodology, the age of the individual G-5663 was less than 14 years (Table 1). On the other hand, third molars were fully erupted (Figure 3 and Figure 4, black arrows), with parallel root walls, but with apices that remained open (Figure 5, red circles), which led to age estimation of 18.5-19.5 years, according to Smith (1991) and AlQahtani (2009), or > 15 < 20 according to Ubelaker (1989) and Hillson (2002).

<table>
<thead>
<tr>
<th>Fusion of long bone (Scheuer &amp; Black 2000)</th>
<th>Male (expected age)</th>
<th>Female (expected age)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Radius ep. proximal</td>
<td>14 - 17</td>
<td>11.5 - 14</td>
</tr>
<tr>
<td>Radius ep. distal</td>
<td>14 - 17</td>
<td>16 - 20</td>
</tr>
<tr>
<td>Femur ep. proximal</td>
<td>14 - 19</td>
<td>12 - 16</td>
</tr>
<tr>
<td>Femur ep. distal</td>
<td>14 - 18</td>
<td>16 - 20</td>
</tr>
<tr>
<td>Tibia ep. proximal</td>
<td>15 - 19</td>
<td>13 - 17</td>
</tr>
<tr>
<td>Tibia ep. distal</td>
<td>15 - 18</td>
<td>14 - 16</td>
</tr>
</tbody>
</table>

Figure 2: Diaphysis of right radius with pathological changes. Epiphyseal fusion did not occur.

Figure 3: Mandible with sign of porotic leasion (red circle) and 3th molar fully erupted (black arrow).

Figure 4: Hard plate with micro and macro porosity and 3th molar fully erupted (black arrow).
Possible case of scurvy from Viminacium

Dental analysis

In total, 25 alveoli with 21 teeth were recovered, while other were lost post-mortem. Dental analysis indicated the presence of supragingival calculus on 6 teeth. According to the method of Brothwell (1981), the degree of calculus formation can be estimated as “slight”, or degree 1, “small amount”, according to the methodology of Buikstra & Ubelaker (1994).

Description of the lesions

Porotic lesions were noted in various parts of the skull. Ectocranial porosity spotted on the parietal bones can be characterised by an ‘orange peel’-like porosity (Figure 6). A small fragment of sphenoid bone was preserved with notable porosity (Figure 7, red circle). The lesion noticed on hard plate displays a mixture of micro and macro porosity (Figure 4). On the maxilla, abnormal porosity is noticeable on the alveolar bone, however, a large part of it was lost post-mortem (Figures 8 and 9). On the mandible, porosity was spotted on the mandibular condyle (Figure 10) and on the alveolar bone (Figure 3, red circle). Abnormal porosity is visible on the occipital bone fragment (Figure 11) and on temporal bones too (Figure 12). New bone formation can be seen on the endocranial as well (Figure 13).

On postcranial skeleton, abnormal porosity was noticeable on a rib fragment, on diaphysis of left humerus, diaphysis of left radius, proximal epiphysis of left clavicle, on distal epiphysis of left tibia, on left talus (Figure 14), on diaphysis and epiphysis distal of
Figure 9: Maxilla with dental alveoli porosity and post-mortem fracture.

Figure 10: Porosity on mandibular condyle.

Figure 11: Abnormal porosity on occipital bone fragment.

Figure 12: Abnormal porosity on temporal bones.

Figure 13: New bone formation on inner surface of skull.

Figure 14: Abnormal porosity on left talus.
right femur, on diaphysis of right radio and on right talus. Irregular plaques of new bone formation were noticed on the inner surface of the skull, distal epiphysis of left humerus (Figure 15), proximal epiphysis of left ulna, and proximal epiphysis of left femur. Also, on a fragment of left tibia, on left fibula, on diaphysis of right radius, diaphysis of right ulna, diaphysis of right tibia and on diaphysis of right fibula (Figure 16).

The overall appearance of the lesion suggests an active manifestation of periostitis, without signs of healing.

**Differential diagnosis**

The macroscopic analysis of the individual G-5663 showed that she suffered from a kind of pathological condition that was followed by new bone formation on all the preserved long bones and on the ectocranial, and porosity on the skull and postcranial skeleton. Bone porosity is a destructive process, the result of the action by osteoclasts. Ortner et al. (2001) argued that it is the pattern of porous lesions at multiple sites within a skeleton that identifies the presence of a systemic disease with, in most cases, a single underlying cause. It can occur due to several disorders including treponematosis, rickets, anaemia and scurvy, however, the distribution of porosity tends to be quite different (Ortner, 2012).

In treponematosis, porotic lesions are mostly common in the skull: frontal, parietal and facial parts which are, in the initial stage, characterized by clustered and confluent pits; becoming in later phases crater-like bone formation called caries sicca. In a postcranial skeleton most commonly affected is the tibia - ten times more often than the rest of long bones- (Ortner, 2003) that typically becoming thicker, with gummatous periostitis and a snail-track pattern of lesions. The pathological changes noticed on the individual G-5663 were spread over all long bones and their form was quite different from those typical for treponematosis. For those reasons we can discard treponematosis as a possible diagnosis.

In rickets, porosity is generally apparent in subchondral surfaces of the growth plate where small and circular areas (like pores) of a subchondral osteoid are not mineralized (Ortner & Mays, 1998). However, rickets is a systemic disease of early childhood, which has the highest peak of prevalence between six months and two years (Ortner, 2003). Normally, in an adult skeleton, changes such as porosity and roughening of a bone will be lost (Brickley et al., 2010). Deformity, which occurs during growth in individuals with rickets, such as bowing of long bones, especially those of lower extremities, can persist and be recognisable in an adult skeleton (Brickley et al., 2010). As we have already mentioned, the individual that is the object of our study is an adult, so porosity noticed on the skull and long bones is not likely to be a consequence of rickets. Also, bowing of the long bones was not noticed. For those reasons, a diagnosis of rickets can be rejected.
In some cases of anaemia, the porosity of the skull vault and/or orbital roof can be apparent (Miquel-Feucht et al., 1999; Walker et al., 2009). Changes in less severe cases are expressed in a form of small porotic lesions, while in advanced stages the affected area has a cribrotic appearance. In the case of the individual G-5663, the orbital roofs were not preserved so we could not assure if those changes were produced, while porotic changes were spotted on the skull. Nevertheless, these kinds of changes can be assigned for many different infections and metabolic diseases. Furthermore, the changes that were described on the long bones are not typical for anaemia. It was however noted by Stuart-Macadam (1989) that scurvy often occurs with anaemia, so a possibility of co-morbidity should not be ruled out.

Scurvy, a metabolic disease caused by a deficiency in dietary vitamin C can stimulate porous lesions of the cortex and porous hypertrophic bone formation. The most characteristic sign of scurvy is the porous lesion of the greater wing of the sphenoid bone (Ortner & Ericksen, 1997; Ortner et al., 1999, 2001). Porotic changes on the skull can also appear on the maxilla, the coronoid process and alveolar of the mandible, on the orbital roof, the hard plate, the parietal and the occipital bone (Ortner & Ericksen, 1997; Ortner et al., 1999; Ortner, 2003; Brickley et al., 2010). The localization appears to occur at sites where supporting muscles attach. In a postcranial skeleton periosteal new bone reaction and abnormal porosity can be spotted on all long bones, feet and hands in individuals affected by this disease. Some authors also report the presence of ossified haematomas in long bones, especially in the tibia and femur (Maat, 2004, Van der Merwe et al., 2010) but it seems that those changes are not always accompanying scurvy. In historical and medical records antemortem tooth loss, which usually starts with upper incisive, was often associated with scurvy (Fain, 2005; Olmedo et al., 2006; Velandia et al., 2008). Unfortunately, in the case of the individual G-5663, this part of the alveolar bone of maxilla was damaged post-mortem, so we cannot assure whether some of the teeth were lost antemortem, but the porosity of alveoli can be noted.

It could be difficult to distinguish porotic changes caused by trauma, infectious or metabolic disease on any isolated bone, with those produced in the early stages of scurvy. However, as we can see in Table 2, the individual G-5663 exhibits almost the entire changes characteristic for scurvy, except ossified haematomas where the presence of this on the bones in individuals with scurvy was recorded by only a small number of authors; and changes in the roof orbits and the vertebrae, which were not preserved.

### Table 2. Skeletal elements commonly affected by scurvy

<table>
<thead>
<tr>
<th>Orbits</th>
<th>UO</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maxila</td>
<td>P</td>
</tr>
<tr>
<td>Mandibular ramus</td>
<td>P</td>
</tr>
<tr>
<td>Alveolar bone</td>
<td>P</td>
</tr>
<tr>
<td>Hard plate</td>
<td>P</td>
</tr>
<tr>
<td>Sphenoid bone</td>
<td>P</td>
</tr>
<tr>
<td>Ectocranial</td>
<td>P</td>
</tr>
<tr>
<td>Endocranial</td>
<td>P</td>
</tr>
<tr>
<td>Vertebrae</td>
<td>UO</td>
</tr>
<tr>
<td>Long bones</td>
<td>P</td>
</tr>
<tr>
<td>Ossified haematomas</td>
<td>A</td>
</tr>
</tbody>
</table>

Lack of fusion of the epiphysis, noted on the individual G-5663, could be another argument in the diagnosis of scurvy. Normally, in female individuals, at the time when the third molar fully erupts, most of the epiphyses should be partly or fully fused. The fact that this did not occur could be in correlation with the pathological condition present in this individual. This could be explained by crepitus that has been frequently noted in cases of scurvy in young adults and children and it can lead to delayed fusion of long bones and possible discrepancies in osteological ageing of those individuals (Sinnott, 2015).

All the above mentioned arguments can lead us to the conclusion that diagnosis of scurvy would be the most plausible one.
**Discussion**

Scurvy is a metabolic disorder caused by a lack of ascorbic acid, vitamin C, in the diet. In the case of humans, some of the non-human primates and guinea pigs, it cannot be produced by the body itself (Maat, 2004). Vitamin C is available in most of the fresh fruits and vegetables, so appearance of scurvy is usually connected with natural or social disasters, such as long-term droughts or besieged places, and specific life conditions, like life of sailors on transoceanic sailing ships (Miladinović-Radmilović & Vulović, 2015). However, there are, in fact, various factors or lifestyle issues that might increase the risk of scurvy, such as food allergy diets, eating disorders (anorexia nervosa or bulimia), and certain cancers (Mayland et al., 2005). Cooking food can also be one of the factors, as it reduces vitamin C content by 20% to 40% (Hirschmann & Raugi, 1999).

Vitamin C is required for the hydroxylation of proline to hydroxyproline, an important amino acid in collagen (Maat, 2004). Collagen is the main protein component of the connective tissue, including bone (Stuart-Macadam, 1989; Akikusa et al., 2003). Defective collagen will result in fragile capillaries and weakened bone tissue, which can result in capillary bleedings, bone infinctions and absence of repair activities (Maat, 2004). Clinical consequences of scurvy include bleeding gums, tooth loss, pain in the extremities, vertigo, faintness, excessive sweating, hemorrhagic spots in the eyes, xerosis, hyperkeratosis, bent and coiled body hairs, and impaired wound healing (Hodges et al., 1969; Hirschmann & Raugi, 1999). Treatment with vitamin C results in rapid, often dramatic, improvement (Hirschmann & Raugi, 1999), but without a treatment the disease can have a fatal outcome (Van der Merwe et al., 2010).

As mentioned before, scurvy shares some common features with some types of anaemia, such as porosity on skull and roof orbits, so we also considered the possibility of co-morbidity. In fact, experiments performed on a guinea pig show that progressive anaemia is one of the symptoms of dietary deficiency in vitamin C (Mettier & Chew, 1932). As all of the lesions were still active at the time of death, without signs of healing, we can presume that scurvy - or a combination of scurvy and anaemia - resulted in a lethal outcome for this young girl.

**Conclusion**

A young female individual, discovered in the necropolis dating from the Late Roman Period, presented pathological signs that can be related to deficit of vitamin C. This can be associated with malnutrition - low intake of nutrients, or other conditions that provoke a chronic loss of nutrients (diarrhoea, for example). As vitamin C is available in most of fresh fruits and vegetables, its deficiency is usually connected with some kinds of specific life conditions; such as periods of famine, war or long sea-voyage. In the case of this individual the historical period in which she lived – Late Antiquity – was very turbulent. The decay of the...
Roman Empire, invasions of “barbarians”, and a siege of the city could lead to food shortage and possible diet disturbance.

Although the individual was already an adult (base on dental age estimation), none of the recovered epiphyses were fused. This kind of a developmental delay can be associated with scurvy, but is rarely mentioned in paleopathological literature. It is important to be aware of this characteristic in order to avoid mistakes in age estimations when there is reasonable suspicion that the object of a study suffered from scurvy.

References


