FEMORAL BOWING DEFORMITY: POSSIBLE AETIOLOGIES IN A 14TH-19TH CENTURY SKELETON FROM CONSTÂNCIA (PORTUGAL)

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Introduction

Bowing or deformity of the long bones corresponds to an abnormal deviation from its longitudinal axis. This alteration may assume the form of a gentle arc, or of a more conspicuous angulation. Bone bowing is a dynamic phenomenon overlapped by distinct factors, such as, the intrinsic properties of bone, the biomechanical stresses exerted and its remodelling capacity.

Morphologically, long bones show a mild degree of bowing - physiological bowing, that usually resolves itself during normal growth and development.2,9,5. Pathological bowing may appear as an accentuation of the normal long bone curvature, as a localized curvature, or a distinct angulation5. Several congenital, traumatic or metabolic conditions can be pointed out as possible aetiologies.9

The necropolis

 Constância is a small village in the centre of Portugal, and located between two important Portuguese rivers, the Tejo and the Zêzere.

- Archaeological excavation: ancient necropoles of Constância
- Total sample: 161 skeletons (165 adults and 46 subadults)
- Chronology: 14th-19th centuries A.D.

Sk.31: biological and funerary profile

Old Female (>50 years old)5
- Post mortem damage of the axial skeleton and pelvis
  - Shallow grave without evidence of coffin use
  - Extended supine with flexed arms and straight legs
  - Aligned on a West-East axis

Paleopathological description

Skull

1. Highly fragmented
2. Presence of small nodes in the inner portion of the frontal bone – hyperostosis frontalis interna (HFI)7,3,9
3. Increased radiopacity in the affected area

Femurs

1. Bilateral femoral bowing: anterolateral curvature
2. a. Cortical thickening in the femoral shaft, more pronounced in the convex face; b. Bilateral Harris lines8

3. Morphological differences between an affected (on the left) and a normal (on the right) femur from a Constância’s female skeleton. Increased curvature of the lower third of the affected diaphysis. Normal morphometric values (table 1)

4. Pronounced femoral neck angle: presence of coxa valga10
5. Periosteal reaction in the lower third of the right femur
6. Hypertrophy at the site of attachment of the linea aspera muscles

Long bones bowing (PBD)

Bowed femur: anteversion of the diaphysis > 15 °

Bowed left femur

Left calcaneus

1. Undisplaced intra-articular calcaneal fracture at the calcaneocuboid joint
2. a. Antero superior compression with healing evidences;
b. Degenerative joint lesions
3. Fracture line. Increased radiopacity

Discussion

A range of pathological conditions might have cause Sk.31 femoral bowing (table 2). Paget’s disease and osteoporosis seems unlikely, since none of the most distinctly features are present7,8,12. Neurofibromatosis and fibrous dysplasia are improbable due to its massive bone destruction9,12. The absence of long bones fractures and/or limb shortening, rules out a case of osteogenesis imperfecta and bilateral trauma9. PBD occurs in children and most commonly affects the forearm. The adult form is rare and the fibula is the most affected bone2. Nevertheless, this hypothesis must be considered in the present diagnosis. With the exception of the femoral bowing, all other bony changes due to active rickets are absent. In prolonged rickets, biomechanical forces can produce limb deformities that persist into adulthood - residual rickets9.

Hypertrophy of the linea aspera can be associated with this extensive muscle involvement. The presence of Harris lines may also indicate some degree of physiological stress during growth16. However, it is almost impossible to establish a direct relationship between rickets and Harris lines. Even so, coxa valga10, in its acquired form, can be attributed to rickets or osteomalacia. Looser’s zones are considered “pathognomonic” of osteomalacia11. Due to post mortem damage, none of the specific locations (e.g. scapula body, pubic ramus) were recovered. Forced dorsiflexion is pointed as the main cause of the undisplaced calcaneal fracture. Since only long bone bending deformities are present it is difficult to differentiate between residual rickets and osteomalacia. Consequently, we must consider this two conditions as possible aetiologies.

Table 2. Differential diagnosis for Sk. 31 femoral deformities2,7,8,10,17,12

<table>
<thead>
<tr>
<th>Metabolic conditions</th>
<th>Paget’s disease</th>
<th>Sk.31</th>
<th>Osteomalacia</th>
<th>Sk.31</th>
</tr>
</thead>
<tbody>
<tr>
<td>Osteoporosis circumscripta (early stages)</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>“V-shaped” deformities</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>No</td>
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<tr>
<td>Cranial vault thickness (later stages)</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Thickening and lamination of long bone cortex</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Long bones bowing</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Cortical bone thinning</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Biconvex or wedge vertebra</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Looser’s zones of radiopacity</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Long bones bowing</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Miscellaneous conditions</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
</tbody>
</table>

Hypertrophy of the linea aspera can be associated with this extensive muscle involvement. The presence of Harris lines may also indicate some degree of physiological stress during growth16. However, it is almost impossible to establish a direct relationship between rickets and Harris lines. Even so, coxa valga10, in its acquired form, can be attributed to rickets or osteomalacia. Looser’s zones are considered “pathognomonic” of osteomalacia11. Due to post mortem damage, none of the specific locations (e.g. scapula body, pubic ramus) were recovered. Forced dorsiflexion is pointed as the main cause of the undisplaced calcaneal fracture. Since only long bone bending deformities are present it is difficult to differentiate between residual rickets and osteomalacia. Consequently, we must consider this two conditions as possible aetiologies.

Table 1. Comparative morphometric analysis with Constância’s female standards (femur)11

<table>
<thead>
<tr>
<th>Femur Measurements (cm)</th>
<th>Bowed left femur</th>
<th>Constância’s female mean values (n=10)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maximum length</td>
<td>418</td>
<td>418.5</td>
</tr>
<tr>
<td>Physical length</td>
<td>415</td>
<td>414.2</td>
</tr>
<tr>
<td>Transverse diameter (middle of the diaphysis)</td>
<td>25</td>
<td>25.56</td>
</tr>
<tr>
<td>Sagittal diameter (middle of the diaphysis)</td>
<td>27.5</td>
<td>27.68</td>
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</table>