Femoral bowing deformity: possible aetiologies in a 14th-19th century skeleton from Constância (Portugal)

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Abstract

In the years of 2002 and 2003, 151 skeletons (106 adults and 45 non adults) were unearthed from the ancient necropolis of São Julião Church (Constância, Portugal) dated from the 14th-19th centuries. Of the individuals analysed, one in particular, an adult female (Sk. 31) exhibited an abnormal femur morphology. Macroscopically the bone lesions were characterized by a bilateral antero-posterior thinning of the shaft and an antero-lateral bending of the proximal third of the femur diaphysis. An increased cortical thickness in the concave side of the femur shaft was revealed through conventional X-ray analysis. In addition with the described bone changes, the individual also showed small bone nodes on the inner surface of the frontal bone and an undisplaced calcaneocuboid fracture on the left calcaneus. Although femoral bowing deformity is a common manifestation in many vitamin D deficiencies (i.e. residual rickets or osteomalacia), other conditions, such as physiological bowing deformities or coxa vara, may also produce similar long bones features. The aims of the present case-study are to present the main possible etiologies for the skeletal changes observed, as well as to discuss the impact of postmortem damage in the differential diagnosis.

Keywords: paleopathology, differential diagnosis, bone plasticity, developmental anomalies, São Julião necropolis.

1. Introduction

The human skeleton is frequently perceived as a static, hard and unchanging structure (Franz-Odendaal et al., 2006; Seeman, 2007). However, this is a misleading view since bone is a living, multifunctional and physiologically dynamic tissue that is in continuous adaptation to the surrounding environment (Gross and Bain, 1993; Lieberman, 1997; Cullinane and Einhorn, 2002; Roberts et al., 2004). Accordingly, any endogenous (congenital or inherited) or exogenous (dietary deficiencies, radiation, drugs, abnormal biomechanical load) disturbances that occur during the formation and development of the skeleton, for instance, in early embryonic stages or during childhood and adolescence, may affects its normal architecture (Adler, 2000). It should be stated that bone is a highly specialized type of connective tissue (Martin, 1991; Cullinane and Einhorn, 2002; Marks and Odgren, 2002; Young et al. 2006) composed of an organic matrix (about 30%) that is strengthened by calcium and phosphate minerals (about 70%) (Young et al. 2006). Both organic and inorganic components have a well-defined function: the inorganic confers the rigidity that characterizes bone, whereas the network of collagen fibers (matrix) allows some degree of flexibility (Lieberman, 1997; Jee, 2001; Marks and Odgren, 2002; Iyo et al., 2004; Seeman, 2007; Young et al. 2006).

Under abnormal biomechanical load, two main bone tissue properties are challenged: elasticity and plasticity (McGuinnis, 1999). Elasticity can be defined as the ability of bone to strength when an extreme force is applied, returning to its original configuration after its removal (McGuinnis, 1999; Griffith et al., 2005). Nevertheless, and when the biomechanical charges exceeds the elastic limit, the bone undergoes deformation that persists, even after the mechanical force being removed (Cail et al., 1978; Frassico et al., 1997; Pearson and Lieberman, 2004; Griffith et al., 2005). This change is called plastic deformity (Cail et al., 1978). In biomechanical terms, plastic deformity can be defined as a specific adaptation

acquired during an individual's growth and development, which reflects the malleability of certain body tissues in response to environmental stimuli (Knüsel, 2000). Skeletal plasticity contributes to the intrinsic bone toughness helping in the dissipation of energy and forming "plastic zones" near incipient cracks, which act as major impediments for crack propagation (Zimmermann et al. 2011). One conspicuous manifestation of bone plasticity is bowing or bending anomalies, especially of the long bones. In severe cases, and when the elastic limit is overreached, bone may even break (Cail et al., 1978).

Bowing of the long bones corresponds to an abnormal deviation from its longitudinal axis (Stevenson, 2006; Oestreich, 2008), which may assume the form of a gentle arc, or present a more conspicuous angulation (Stevenson, 2006). It is a dynamic phenomenon mediated by distinct factors, such as, the intrinsic properties of bone, the biomechanical stresses exerted and the bone remodelling capacity (Stevenson, 2006). It is pertinent to mention that anatomically, all long bones show a mild and variable degree of bowing (Stuart-Macadam et al., 1998; Shinohara et al., 2002; Stevenson, 2006). For example, the femur and the tibia show an anterolateral bowing, the fibula a posterior bowing, whereas the radius and ulna are medially bowed (Stevenson, 2006). Under normal circumstances, bone has the capacity to adapt to biomechanical loading through the remodelling process. This means that the functional loading of a bone will has a considerable effect on its external form and internal structure (Adler, 2000). In long-standing loading, however, bone may be incapable to repair and readjust its architecture to the external forces; as a consequence, bending deformities may form. Abnormal or pathological bowing may express as an accentuation of the normal long bone curvature, as a localized curvature, or a distinct angulation (Stevenson, 2006; Oestreich, 2008). In some cases (e.g. *tibia recurvata*), an increased cortical thickness on the concave side and a cortical narrowing on the convex side may also form (Adler, 2000). Besides prolonged

loading, other pathological condition of the congenital and metabolic spectrum may also cause bowing deformities.

In this paper, a case of bilateral femur deformity observed in a female skeleton exhumed from the ancient necropolis of Constância is presented and described. Further goals are: (1) to discuss the possible etiologies for the condition, highlighting the most probably causes, and (2) to discuss the impact of postmortem damage in the differential diagnosis.

2. Material and methods

In the years of 2002 and 2003, during the renewal of the historical centre of the village of Constância (Fig. 1), human skeletal remains were discovered in the village main square - Praça Alexandre Herculano [Alexandre Herculano square]. This discovers confirmed the location of the ancient necropolis of São Julião Church, dated from the 14th-19th centuries (Garcia, 2004). The chronology is based on the period of construction and subsequent abandonment of the São Julião Church, which has probably occurred in the first decades of the 19th century (Coelho, 1999). There are few historic information's available with regard to the exact localization of the S. Julião Church and associated necropolis. It is known that the church suffered a massive destruction in the 19th century during the French invasions. For instance, it was burned and its unique paintings stolen and/or destroyed. As a consequence, the temple was abandoned, probably in the year of 1811, and demolished. Some documents states that in the year of 1820, and after the royal authorization of D. João VI (1767-1826) a new Pelourinho (a symbolic monument that firms the village status) was built in the centre of the new Praça Alexandre Herculano (Coelho, 1999). This fact and the construction of a new cemetery in the year of 1833 has probably dictated the abandonment of the ancient necropolis (Garcia, 2004).

The archaeological and anthropological survey allowed the recovery of 151 skeletons: 106 adults and 45 non adults (Assis, 2006). The skeleton (Sk. 31) described in this paper was found in a shallow grave without evidence of coffin use (Fig. 2.1). The body was inhumed in extended supine position, following the West-East alignment. The upper limbs were flexed over the chest and the lower limbs were extended and parallel. A cooper medal was recovered over the chest (Assis, 2006).

The Sk.31 skeleton exhibited some postmortem destruction, mainly in the skull and innominate bones, axial skeleton and at the upper and lower extremities of some long bones (Fig. 2.2). These taphonomic changes were characterized by bone breakage, longitudinal cracking and cortical detachment with exposition of spongy bone, especially in those bone pieces mostly composed of trabecular bone (e.g. articular regions, vertebrae). The femora were the best preserved bones, despite some breakage and erosion of the upper and lower extremities (posterior anatomic plane) due to the contact with moisten soil. Apart from cortical erosion, no other texture and colour changes were noticed. The macroscopic evaluation of the taphonomic bone changes was conducted following the recommendations described in Buikstra and Ubelaker (1994) and White and Folkens (2005). The analysis of the preserved bone pieces revealed a middle-aged to old female individual. The sex diagnosis was based on skull and ilium morphology (Ferembach et al., 1980; Buikstra and Ubelaker, 1990; White and Folkens, 2005), and in the metrics of the long and foot bones (Wasterlain, 2000). The age-at-death estimation considered the metamorphic changes at the auricular surface of the ilium (Lovejoy et al., 1985).

The skeleton was examined macroscopically and through conventional radiography, using the services of the Clínica Universitária de Imagiologia from the Hospitais da Universidade de Coimbra (HUC). This analysis complemented the detailed description of the

bony lesions according to their distribution and morphology. This procedure was concluded with the differential diagnosis.

3. Palaeopathological description

In the skeleton under analysis, the more conspicuous changes were noticed in the skull, femora and in the left calcaneus.

In the skull remains, it was possible to observe the presence of small bone nodes on the endocranial surface of the frontal bone (Fig. 3.1 and 3.2). These bony appositions were located between the frontal crest and the meningeal grooves and showed a well-defined morphology. With a bilateral and symmetrical distribution, the lesions were characterized by thin depositions of new bone with a striated appearance. In the remaining skull fragments, no additional bone change or thickness abnormities were observed. The radiological analysis revealed an increase density in the frontal area affected (Fig. 3.3).

The visual inspection of the long bones revealed a marked, bilateral antero-lateral curvature of the femora, more conspicuous in the lower third of the shaft. Bilateral short head-neck proportions, more evident on the posterior side, was seen, which may configures a case of coxa vara (Fig. 4, and 5). Figure 6 clearly illustrates the femoral deformity observed in the Sk. 31 individual, when an affected bone was compared with a healthy one from another individual from the Constância necropolis. Alongside the described changes, a bilateral hyperthrophy at the site of attachment of the *linea aspera* muscles was seen (Fig. 7). A slight periosteal reaction was also noticed on the lower third of the right femur. The use of conventional X-ray revealed an increased cortical thickness in the concave side of the femoral shaft. Some bilateral linear radiopacities on the distal portion of the femur shafts were also observed (Fig. 8).

The visual examination of the foot bones showed the presence of an undisplaced fracture at the calcaneocuboid joint of the left calcaneus (Fig. 9). The lesion was characterized by an antero-superior compression with healing evidences. Degenerative joint changes were also seen on the affected joint. The X-ray analysis showed an increased radiopacity in the fracture area (Fig. 10). On the left cuboid bone, only a slight lipping was observed surrounding the joint.

4. Discussion

Bowing deformities of the long bones are a common occurrence in many congenital, developmental and metabolic conditions (Table 1). For instance, it is observed (e.g. tibia) in cases of neurofibromatosis (or von Recklinghausen's disease) in addition with other skeletal changes, such as asymmetrical bone length, cranial suture defects, kyphoscoliosis, pseudoarthrosis, ribs deformity, posterior vertebral body scalloping (Zimmerman and Kelley, 1982, Ortner, 2003, Kjellin, 2009) and massive erosion of the cortical bone, with formation of cyst-like areas of tissue demineralization (Zimmerman and Kelley, 1982; Ortner, 2003). Other conditions that may cause bowing of the long bones are fibrous dysplasia and osteogenesis imperfecta. Fibrous dysplasia causes thinning of the cortical bone with loss of tissue definition, as well as fibro-osseous formation in the medullar cavity. The presence of radiographic cyst-like areas, pathological fractures, (Russel and Chandler, 1950; Harris et al., 1962; Zimmerman and Kelley, 1982; Bianco et al, 2003; Ortner, 2003), and Shepherd's crook deformity on the proximal femur (Chen et al., 2005) are also common features of the disease. Osteogenesis imperfecta, an inherited condition caused by a deficiency in collagen type I production (Parsons, 1980; Zimmerman and Kelley, 1982; Ortner, 2003; Borghei and Tehranzadeh, 2009), acts diminishing bone elasticity and increasing the risk of fractures with exuberant callus formation (after minor trauma) (Zimmerman and Kelley, 1982). As a

consequence, severe deformities, namely kyphoscoliosis, defective growth, and shortening of the axial skeleton and long bones (Parsons, 1980; Zimmerman and Kelley, 1982; Aufderheide and Rodríguez-Martín, 1998) are produced. In the case under discussion, neurofibromatosis and fibrous dysplasia are improbable diagnoses due to its massive bone destruction, a feature not seen in the Sk.31 individual. The absence of long bones fractures and/or limb shortening also permits excluding a case of osteogenesis imperfecta. The absence of symmetric bone callus in the femora and/or other macroscopic or radiographic signs of healed fractures make a diagnosis of bilateral fracture less probable.

Disturbances in the bone metabolism and cell functioning may also induce limb deformities. For example, lateral and anterior bowing of the long bones is a common observation in later stages of the Paget disease, often combined with increased thickness of the skull bones - "cotton wood" appearance, vertebral compression fractures, cortical thickness and coxa vara (Parson, 1980; Zimmerman and Kelley, 1982; Ortner, 2003; Brickley and Ives, 2008; Mays, 2008). Despite the presence of femoral bowing and coxa vara, none of the aforementioned features of Paget disease were seen in the Sk. 31 individual. Furthermore, the X-ray analysis did not revealed the cortical thickness and the medullar narrowing commonly seen in this condition. The bone formations observed in inner surface of the Sk.31 skull are also distinct from those of Pagetic origin. A possible case of *hyperostosis frontalis interna* (e.g. Barber et al., 1997; Belcastro et al., 2006) seems to be the most probably etiology for the lesions observed.

The lack of traits of bone tissue fragility associated with osteoporosis (i.e. marked thinning of cortical bone, biconcave or wedged vertebrae deformity and pathological fractures in the femoral neck and at the distal end of radii - Parson, 1980; Zimmerman and Kelley, 1982; Ortner, 2003; Mays, 2008) led to the exclusion of this metabolic condition from the differential diagnosis.

One mechanical consequence of vitamin D deficiency is bending deformities on the long bones. Vitamin D deficiency is considered the primary cause of rickets, since it plays a major role in calcium absorption (Zimmerman and Kelley, 1982; Ortner, 2003; Pettifor, 2003; Mays *et al.*, 2007; Brickley and Ives, 2008; Mays *et al.*, 2009). Other conditions that may produce similar effects are chronic renal tubular failure, lower rate of calcium in the diet and chronic intestinal disorders (Zimmerman and Kelley, 1982; Ortner, 2003; Mays *et al.*, 2009). An inadequacy of calcium produces an accumulation of normal osteoid and consequently, a failure or a delay in the endochondral mineralization (Parson, 1980; Ortner and Mays, 1998; Cheema et al., 2003; Pettifor, 2003; Brickley and Ives, 2008; Mays, 2008; Mays *et al.*, 2009). In long-standing rickets, biomechanical forces acting upon weakened, poorly mineralized bones will produce bending deformities, mainly in femora and tibiae (Stuart-Macadam, 1988; Ortner and Mays, 1998; Ortner, 2003; Pettifor, 2003; Brickley and Ives, 2008; Mays, 2008), that persist into adulthood, a condition termed residual rickets (RR) (Ortner, 2003; Mays, 2008; Waldron, 2009). These bowing deformities may be followed by thickening of the concave face of long bones, medio-lateral widening of proximal femora, bone shortening, coxa vara and angulation of the knees (Brickley and Ives, 2008). Additional skeletal features include kyphosis or scoliosis, lateral narrowing of the pelvis, abnormal shape of ilia, and anterior bending of sacrum (Brickley *et al.*, 2005; Brickley and Ives, 2008). According with Brickley et al. (2010), long bones bending in adults constitute one of the most important evidences of vitamin D deficiencies occurred in infancy. Actually, most cases of RR reported in the literature (e.g. Lunardini et al., 2005; Haduch et al., 2009; Brickley et al., 2010; Kacki et al., 2011) exhibit bone deformities, affecting, normally, most bones from the lower limb. A marked degree of bowing (lateral or medial) is often described for tibiae (e.g. Lunardini et al., 2005). Apart from the bilateral femoral bowing and coxa vara, Sk. 31 does not present any other skeletal deformity compatible with RR. As mentioned previously, the tibia and fibula of

the Sk.31 individual, as well as the remaining long bones, presented a normal morphology. Furthermore, no metric differences in the length and diameters of Sk. 31 femora where found when compared with the mean values obtained among the females sample of Constância (Table 2). The presence of transverse radiolucent band on the distal metaphysis seems to suggest, however, that this individual may have suffered of some type of physiological stress during her growth and development. In the literature, the presence of arrested growth lines (also called Harris's lines) is normally interpreted as an evidence of recovery from acute or chronic stress episodes, such as malnutrition or severe illness (Burgener et al., 2006; Pinhasi, 2008), and signifies renewed or even increased growth following periods of inhibited bone growth (Steinbock, 1976).

Many of afore-mentioned lesions of RR are also common to osteomalacia, a condition that may results from vitamin D deficiency, but also from poor nutrition, lack of sunshine exposition or successive pregnancies coupled with lactation (Zimmerman and Kelley, 1982; Chadha et al., 2001; Ortner, 2003; Kamath et al., 2005). There are several pathological features that should be considered in a diagnosis of osteomalacia, such as buckling of the scapular body and pubic ramus, narrowing of the thorax, ribs curvature and sternum angulation, vertebral body compression, also known as "codfish vertebra", scoliosis and kyphosis, diffuse osteopenia and Looser-Milkman's zones of radiolucency associated with bilateral and symmetrical pseudofractures (Parson, 1980; Sittampalam and Rosenberg, 2001; Ortner, 2003; Kamath et al., 2005; Brickley and Ives, 2008; Waldron, 2009). For some authors (e.g. Brickley et al., 2005; Brickley and Ives, 2008; Waldron, 2009), Looser's zones are considered "pathognomonic" of osteomalacia. In a large-scale systematic study of archaeological cases of osteomalacia (post-medieval England), Ives and Brickley (2014) reported a set of lesions that should be considered in the study of adult vitamin D deficiency osteomalacia, especially in fragmented and poorly preserved individuals. These pathological

features include pseudofractures on the inferior border of the scapula spinous process and lateral border, coupled with unhealed or healing rib pseudofractures. Fractures on the iliac crest and pubic ramus and unhealed fractures on the distal ulna and clavicle, albeit less frequent, may also be found (Ives and Brickley, 2014). New diagnosing findings were advanced by the author's such as the presence of pseudofractures affecting the coracoid process of the scapula, the medial ilium, the proximal femur (neck and shaft), and the transverse process of the thoracic vertebrae. Periosteal new bone formation, often exhibiting a spiculated appearance near the fracture edges was also described (Ives and Brickley, 2014). Although the postmortem damage of some key anatomic areas (e.g. scapula body), no evidence of pseudofractures or fractures (unhealed and healing) compatible with those described for osteomalacia were recorded in the skeletal remains of the Sk. 31 individual. The only traumatic lesion was observed on the calcaneocuboid joint of the left calcaneus. According with Daftary and co-authors (2005), the calcaneus is the tarsal bone more affected by intra- and extraarticular fractures, accounting for about 2% of all fractures. The fractures that affect the calcaneocuboid articulation are normally of the extraarticular type and involve the anterior process (Paley, 1994; Daftary et al., 2005). These fractures may be caused by avulsion (small lesions with eventual bone displacement) or compressive (impaction of the calcaneocuboid joint with formation of a compression fracture at the anterior process) forces (Paley, 1994). The traumatic lesion observed in the left calcaneus of the Sk. 31 female individual was probably caused by a compression force that generated an undisplaced fracture. It should be stated that anterior process fractures of the calcaneus are uncommon (Daftary et al., 2005). Etiologically, it may be produced by forced abduction of the forefoot with a fixed calcaneus (Daftary et al., 2005), or exaggerated dorsiflexion (Judd and Kim, 2002; Daftary et al., 2005).

Further conditions that should be considered in the present differential diagnosis are coxa vara and plastic bowing deformity (PBD).

Coxa vara refers to a decreased inclination of the angle formed by the neck and shaft of the femur (Stevenson and Hall, 2006), which places the greater trochanter prominently above the level of the femoral head (Barnes, 2012). Coxa vara may be unilateral or bilateral and may manifests solely, as part of a multiple malformation syndrome (e.g., skeletal dysplasia), in association with metabolic (e.g., rickets, osteomalacia), endocrine (e.g., hypoparathyroidism) and genetic (e.g., Gaucher disease, osteogenesis imperfecta) conditions, or following infection and trauma (e.g., fracture of the femoral neck, injury of the femoral head) episodes (Castriota-Scanderbeg and Dallapiccola, 2005; Hudgins and Vaux, 2006). It may also occur as a consequence of a faulty intrauterine position (Castriota-Scanderbeg and Dallapiccola, 2005). An overlap between coxa vara and slipped capital femoral epiphysis often exists; that is, coxa vara may predispose to the development of femoral head slippage and vice-versa (Castriota-Scanderbeg and Dallapiccola, 2005). In spite of the postmortem damage of the great trochanter of the Sk. 31 femora, it seems that it was placed at the same level or slightly above of the femoral head, which may reinforces a diagnosis of coxa vara. Moreover, in the literature (e.g. Barnes, 2012), coxa vara is frequently described in association with shortened or bowed femur.

Other condition that may eventually explain the femoral deformity observed in Sk. 31 is PBD. Plastic bowing deformity is frequent in children and causes an exaggeration of the normal age-related bone angulation (Cheema, 2003). Varus angulation is a common observation in neonates and infants after birth; however it resolves gradually during growth and development (Shinohara et al., 2002; Stevenson, 2006; Chafetz et al., 2008), especially when the child starts to walk (normally by 18-24 months of age). The persistence of this angulation after the age of two is considered abnormal and termed as physiological or

developmental bowing. In the lower extremity, this condition is more frequent in overweigh child and in those that starts walking at an early age (Cheema, 2003). In contrast, bowing of the forearm bones seems to develop as a result of compressive or longitudinal forces produced during a fall onto an outstretched hand (Stuart-Macadam et al., 1998; Oestreich, 2008).

From the above-mentioned differential diagnosis, coxa vara and PBD, solely and/or combined, are the most probable explanations for the femoral deformity observed in the Sk. 31 individual. Inferring if those conditions were inherited or acquired during development is, however, difficult due to the lack of other skeletal evidences. It should be mentioned that the postmortem damage observed in the skull and innominate bones and in the axial skeleton has impacted the differential diagnosis and the overall interpretation. During burial, several biological, chemical and physical agents play a determining role in the preservation of human remains (Mays, 1998; White and Folkens, 2005; Stodder, 2008; Turner-Walker, 2008). These taphonomic and diagenetic agents (for a review see, e.g.: Nielsen-Marsh et al., 2000; Hedges, 2002; Collins et al., 2002; Turner-Walker, 2008) are normally responsible for the differential preservation of bone elements (Mays, 1998; Pinhasi and Bourbou, 2008; Jackes, 2011), with substantial impact on the paleopathological record (Stodder, 2008). The church of São Julião and associated necropolis were located in the vicinity of the Tagus River, in an area that is affected by seasonal floods. The presence of ground-water in the burial environment is considered a powerful destructive agent (Hedges and Millard, 1995). Therefore, one may suggest that the environmental conditions of the ancient necropolis of Constância may have contributed to the postmortem damage observed.

Independently of the underlying etiology, one may assume that this femoral bowing deformity was probably disabling during walking. For example, coxa vara is described as causing stiffness, pain and limited abduction and internal rotation (Barnes, 2012). Waddling gait is also reported as a common complication of bowed legs (Castriota-Scanderbeg and

Dallapiccola, 2005). If so, these biomechanical constraints may eventually explain the conspicuous hypertrophy of the linea aspera, which is the site of attachment of the adductors and *vastus* muscles (White and Folkens, 2005), and probably had contributed to the compression fracture observed on the left foot.

5. Final remarks

Distinct pathologies may impact the normal skeletal morphology. In paleopathology, an accurate differential diagnosis depends not only from the type and "specificity" of the bone changes observed, but also from the preservation of the skeletal remains. This paper described a case of bilateral femur deformity observed in a female skeleton exhumed from the ancient necropolis of the Constância's village. While some conditions were "easily" discarded from the differential diagnosis due to the absence of severe macroscopic and radiologic bone changes (e.g., neurofibromatosis, osteogenesis imperfecta, fibrous dysplasia, Paget's disease and osteoporosis), others, such as residual rickets and osteomalacia, were more difficult to exclude. Nevertheless, the absence of pseudofractures and/or other skeletal deformities makes a diagnosis of vitamin D deficiency less probable. Of the overall possibilities, coxa vara, probably associated with plastic bowing deformity, emerged as the most plausible etiologies. This case-study has also emphasized the negative impact that a postmortem damage has in the differential diagnosis and in the study of past human conditions.

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