The Recognition of Primate Phylogeny and Osseous Affliction: When Primates are Lionized

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Editorial

Skulls contain the preponderance of criteria/characteristics used identification of skeletons. This is especially true for primates of both human and non-human lineages. When that shape is altered, reliable identification is less assured. So, too, is it problematic to identify the source of skull pathology, unless it's severe. Even that can be controversial. One approach has been referred to as phylogenetic bracketing. It is predicated on previous observations of recognized disorders in con-specifics, or closely or even remotely related organisms, not necessarily of the same family or even order.

This assumes that the disorder of interest manifests in a similar manner across phylogenetic lines. While this is clearly the case for the spondyloarthropathy, crystalline and osteoarthritis forms of arthritis [1], the trans-phylogenetically reproducibility of findings in other disorders has not been confidentially accepted.

Yaws is the classic example. Mark Skinner first recognized classic modifications in a post-cranial chimpanzee skeleton [2], but his diagnosis was not accepted until DNA studies subsequently identified the infection in Pan (chimpanzees). Its skeletal imprint was then recognized as a population phenomenon in a unique sample, the Cotton-Powell collection. The affliction in this sample is at variance with what has been noted other chimpanzee collections (i.e., those in the Cleveland Museum of Natural History, US National Museum of Natural History, Harvard MCV and Central African Museum in Treuven), an as yet unexplained variation either in exposure or susceptibility to the vector of yaws, Treponema pertenue [2-4].

An extraordinary skeletal alteration is that of skull (predominantly facial bones) referred to as leontiasis ossea, because [5,6] perceived resemblance to lion skulls. I personally do not see that resemblance to this proliferative process, but find the in vivo appearance more like the proliferative changes found in lepromatous leprosy. The term was once used for the soft tissue changes of that disease. Virchow, however, actually was relating the appearance of the skull changes to elephantiasis molluscum, a progressive hyperplasia of connective tissue, according to Arrhansson’s translation of Virchow’s pathology of tumors [7].

Most of the skulls in which leontiasis ossea has been recognized have been curated as isolated specimens, without accompanying post-cranial skeletons (e.g., Royal College of Surgeons RCS 711V and RCS 711U). Diffuse periosteal reaction expands facial bones, blurring suture lines, narrowing the nasal opening and obliterating the maxillary and sphenoid sinuses. The etiology of the facial bone changes has been difficult to confidentially etern from such a limited perspective.

Leontiasis ossea presents as massive thickening of facial bones [8,9], to be distinguished from osteomatoses [10], a benign neoplasm, which presents as projecting masses often on pedicles. Paget's disease was considered [11,12], but does not blur sinuses and is associated with increased tooth separation (Table 1).

<table>
<thead>
<tr>
<th>Disorder</th>
<th>Distribution</th>
<th>Periosteal reaction</th>
<th>Suture blurring</th>
<th>Nasal opening</th>
<th>Sinus obliteration</th>
<th>Tooth separation</th>
<th>Exophytic bone mass</th>
</tr>
</thead>
<tbody>
<tr>
<td>Leontiasis ossea</td>
<td>Facial</td>
<td>+</td>
<td>+</td>
<td>Reduced</td>
<td>+</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Paget’s disease</td>
<td>Cranial</td>
<td>-</td>
<td>-</td>
<td>Reduced</td>
<td>-</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>Fibrous dysplasia</td>
<td>Maxilla and mandible</td>
<td>-</td>
<td>-</td>
<td>Not affected</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Primary hypertrophic osteoarthropathy</td>
<td>Soft tissue only</td>
<td>+</td>
<td>-</td>
<td>Not affected</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Yaws/groundou</td>
<td>Nasal</td>
<td>+</td>
<td>-</td>
<td>Increased</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Renal osteodystrophy</td>
<td>Facial</td>
<td>+</td>
<td>+</td>
<td>Reduced</td>
<td>+</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Craniofacial osteopathy</td>
<td>Isolated</td>
<td>Porous</td>
<td>-</td>
<td>Not affected</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Osteoma</td>
<td>Orbit and mandible</td>
<td>-</td>
<td>-</td>
<td>Not affected</td>
<td>-</td>
<td>-</td>
<td>+</td>
</tr>
</tbody>
</table>

Table 1: Differential distribution and character of leontiasis ossea and other proliferative skull pathologies.
Plenk and Gardner [10] suggested trauma or infection, but presented no details to support such a hypothesis. Even groundou (the term used for the peri-nasal bone destruction in yaws) has been considered as etiologic for leontiasis ossea, because of the diffuse osteitis of yaws [8]. Fibrous dysplasia is a trabecular disorder of disorganized bone enlargement [11,13,14]. Primary hypertrophic osteoarthropathy (pachydermoperiostosis), which has been found in lemurs [15], does cause facial coarsening, but not the facial bone expansion seen in leontiasis ossea. Secondary hypertrophic osteoarthropathy has not been associated with such facial alterations [16,17]. Cranio-mandibular osteopathy has been used to describe isolated facial bone periostitis in dogs [18-23]. Also referred to as Westie’s disease or lion’s jaw, it presents as isolated occipital, mandibular and/or temporal bone coral-like masses. However, it is much more limited in skull than leontiasis ossea.

Pighead disease in horses is a diet-related disorder producing skull enlargement with massive periostitis [24]. A more generally applied term in other mammals is “bighead” [25-28]. It is related to ingestion of low calcium, high phosphate diets. This diet produces changes very similar to those of renal osteodystrophy. That disorder is characterized by increased blood phosphate and decreased calcium as a result of renal failure. This leads to increased bone resorption and formation, with secondary hyperparathyroidism [29]. This disorder obliterates maxillary sinuses, causes mandibular sclerosis and facial bone changes [24,30]. The combination of renal insufficiency and hyperparathyroidism is referred to as renal osteodystrophy [31,32].

Examination of specimens in which post-cranial skeletons are preserved provides the pertinent insight. Such individuals with leontiasis ossea have the softened, remodeled (warped) bone with widened metaphyses so characteristic of vitamin D deficiency (rickets or osteomalacia) as well as the subperiostal resorption and osteitis fibrosa cystica of hyperparathyroidism [1,31]. Thus, the diagnosis of renal osteodystrophy appears to explain all manifestation [33,34]. Indeed, classic skull lesions of leontiasis ossea are found in Egyptian baboon scaphophagi where osteomalacia is so common.

While presence of skull pathology complicates recognition of phylogenetic attribution (especially when only defleshed skeletons are available for evaluation), the very character of that alteration has permitted new insights to the nature of what had been a disease of mystery. Fifty percent of animals in the London Zoo of the 1900’s had vitamin D deficiency [1], alerting human and veterinary physicians to opportunities for intervention. Attention to calcium and phosphate balance, vitamin D intake and dermal sun-related conversion to its active form are essential components of ongoing efforts to improve the lives and health of primates.

References