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Research Article

Subadult scurvy in Andean South America: Evidence of vitamin C deficiency in the late pre-Hispanic and Colonial Lambayeque Valley, Peru

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ABSTRACT

Scurvy is a disease caused by vitamin C deficiency and is a key paleopathological indicator of subadult health and nutritional status in the past. Yet, little is known about scurvy in human remains from South America and the Peruvian Central Andes in particular. In the Lambayeque Valley Complex on the north coast of Peru, a sample of 641 archaeologically recovered subadults (A.D. 900–1750) were scored for the skeletal manifestations of vitamin C deficiency, testing the hypotheses that scurvy was common in this region and that prevalence increased following European contact. The findings reveal only five convincing cases of scurvy; overall prevalence appears extremely low, and scurvy did not become perceptibly more common following conquest. Of diagnostic interest, complex ectocranial vascular impressions were documented in two cases. Though rarely attributed to scurvy, examination suggests they formed during scorbutic episodes. Another Colonial Period subadult may demonstrate comorbidity between scurvy and rickets. This work also provides new questions for the investigation of scurvy in Andean South America.

1. Introduction

Scurvy is a metabolic disorder produced by chronic inadequate intake of vitamin C. As one of the central goals of paleopathology and bioarchaeology is to reconstruct health and nutrition in the past, scurvy ranks as a key variable in the assessment of nutritional stress and dietary adequacy in human populations. While scurvy has long been underreported in the paleopathological literature, it has received increasing perception, focus, and diagnostic rigor, particularly over the last decade. This visibility is largely due to the development of Donald Ortner's diagnostic criteria of scurvy in the skull (Ortner and Eriksen, 1997; Ortner et al., 1999, 2001) and postcranial skeleton (Ortner, 2003). Subsequent studies have shed new light on dietary insufficiency, subsistence economy, human–ecology synergisms, urbanism, and socioeconomic inequality (e.g., Melikian and Waldron, 2003; Lewis, 2004; Brickley and Ives, 2006, 2008; Mays, 2008; Waldron, 2009; Lewis, 2010; van der Merwe et al., 2010a,b; Brown and Ortner, 2011; Geber and Murphy, 2012).

While the skeletal evidence for scurvy spans thousands of years and almost the entire world, Brickley and Ives, 2008, Table A1), only one study has emerged from Andean South America. Ortner and colleagues (1999) examined 363 subadult crania from Peru curated at the Smithsonian Institution's National Museum of Natural History (NMNH). This work found probable cases in just over 10.5% of the examined crania to establish that scurvy was: (1) indeed present in the ancient coastal and highland Andes, and; (2) was evidently a common disease condition in pre-Hispanic Peru. However, most of these human remains were selectively collected from the devastated landscapes of looted cemeteries along Peru's central coast during the early 20th century expeditions of Hrdlička (1914). Key questions regarding cultural, spatial, temporal, and epidemiological variation could not be addressed as corresponding contextual data were either destroyed by looting or lost by insufficient documentation during surface collections that focused mostly on crania. Since no postcranial remains accompanied the NMNH Peruvian crania, broader assessment of lesion distributions within affected individuals was also inhibited.

Scurvy has yet to be assessed in the arid Lambayeque Valley Complex of Peru's north coast for additional contextual information regarding local Lambayeque ecology and cultural history, see the Online Supplemental Materials.This region and its five rivers provided the setting for one of the independent centers of Andean cultural development beginning around 1500 B.C. Key

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developments spanned Cupisnique, Moche, and Sicán cultures that were characterized by complex socioeconomic organization, diverse subsistence economies, and innovative technologies (Alva, 2012; Alva and Donnan, 1993; Alva Meneses, 2008; Dillehay, 2011; Donnan, 1989; Donnan, 1990a,b, 2012; Elera, 1986; Hayashida, 2006; Heyerdahl et al., 1995; Klaus et al., 2013a,b; Shimada, 1990, 1995, 2013; Shimada, 1994, 1999, 2000; Shimada et al., 2013; Tschauner, 2001; Wester, 2012). Following European conquest in the 16th century, the region became central to the Colonial viceroyalty. Emerging bioarchaeological reconstructions of late pre-Hispanic and Colonial Lambayeque have revealed multifaceted spatial and temporal patterns of health variation and a range of pathological conditions endured by local communities (Farnum, 2002; Klaus, 2012; Klaus and Tam, 2009, 2010; Klaus et al., 2009, 2010; Shimada et al., 2004; Toyne, 2011a,b). Increasing focus has been placed on reconstructing metabolic stress (Farnum, 2002; Klaus and Tam, 2009), but scurvy has remained unreported.

In this report, two basic hypotheses are addressed. Using a sample of archaeologically documented remains, it is first hypothesized that as with the earlier work of Ortner et al. (1999) on remains from Peru's central coast, scurvy was equally common in the north and will reflect evidence of a comparable prevalence. Second, it is hypothesized that scurvy became more common during the post-contact Colonial period when biological stress broadly increased (Klaus and Tam, 2009).

Table 1
Diagnostic criteria for scurvy used in this study, drawn from Ortner (2003), Ortner et al. (1999), and Brickley and Ives (2008).

Anatomical site	Criteria
Cranial vault	Abnormal regions of porosity <1 mm in diameter penetrating cortical bone; woven bone deposition; impressions of vascular rami or networks
Greater wing of the sphenoid bone	Abnormal regions of porosity <1 mm in diameter penetrating cortical bone
Orbital plate	Abnormal regions of porosity <1 mm in diameter penetrating cortical bone; new bone deposition
Temporal bone	Abnormal regions of porosity <1 mm in diameter penetrating cortical bone
Zygomatic bone, internal and external surfaces	Abnormal regions of porosity <1 mm in diameter penetrating cortical bone; new bone deposition
Anterior maxilla	Abnormal regions of porosity <1 mm in diameter penetrating cortical bone; woven bone deposition
Infraorbital foramen	New bone deposition
Hard palate	New bone deposition
Coronoid process of the mandible, medial surface	New bone deposition
Long bone diaphyses	New bone deposition
Long bone metaphyses	Metaphysial fractures, cortical thinning, deposition of new bone
Supra- and infraspinous fossa of the scapula	Abnormal regions of porosity <1 mm in diameter penetrating cortical bone; new bone deposition
Ribs	Fractures adjacent to oestochondral junction; flaring rib ends

2. Pathogenesis and lesion characteristics

Humans and other great apes share a mutation of the gene that produces L-gluno-γ-lactone oxidase, and we fail to produce this final enzyme crucial to the synthesis of ascorbic acid, or vitamin C (Nishikimi and Udenfriend, 1976, 1977; Stuart-Macadam, 1989; Brown and Ortner, 2011; Weinstein et al., 2001). Obtaining at least 10 mg/day of dietary vitamin C is required. Vitamin C accomplishes hydroxylation of proline and lysine into hydroxyproline and hydroxylysine, which are necessary to form collagen fibril polypeptide precursors (Hodges, 1980). Insufficient dietary intake of vitamin C leads to defective Type 1 collagen formation, which in turn promotes production of defective osteoid, fragile blood vessels prone to rupture, and periosteal membranes with a propensity to tear. Depressed immune function, compromised blood formation, and suboptimal metabolism of iron and folate are additional corollaries (Tamura et al., 2000; Weinstein et al., 2001; Akikusa et al., 2003; Lewis, 2007).

Hemorrhage is a hallmark of scurvy. Outside the circulatory system, the body treats blood as an inflammatory agent targeted for removal. The inflammatory reaction in response to hemorrhage in subadults can affect both the cranial and postcranial skeleton. The vascular response in the cranium may stimulate an incursion of osteoclasts into existing cortical bone that creates channels for newly formed capillaries (usually less than 1 mm in diameter [Ortner et al., 1999; Kozłowski and Witas, 2012]), thus providing pathways for white cells to remove extravasated blood. Yet, such new vessels are themselves likely to be structurally compromised and deficient in collagen, thus exacerbating hemorrhage and inflammation in a feedback loop (Brown and Ortner, 2011, 198). Should hemorrhage elevate the periosteum, new, hypertrophic bone will form hematomas; osteoblasts migrate via chemotaxis to hematomas, which then begin to organize into connective tissue (Ragsdale and Lehmer, 2012).

Common skeletal sites manifesting subadult scurvy include the superior eye orbits, ecto- and endocranial regions of the cranial vault, alveolar bone, the hard palate, and the posterior maxilla and mandible (Table 1). Ortner and colleagues (1999, 2001) argue abnormal bilateral porosity of the greater wing of the sphenoid bone is virtually pathognomonic for scurvy, produced by chronic bleeding of ruptured connective or vascular tissue owing to minor trauma or normal muscular functions such as chewing (Ortner, 2012). Endocranially, scurvy can produce epidural bleeding as arteries in the dura rupture and leak into surrounding tissue space such that hematoma separates the dura and periosteum from the bone and tears bridging vessels between the arachnoid and dura layers of the meninges (Kumar et al., 2009; also Lewis, 2004).

In the infra-cranial skeleton, movement of the muscles of the rotator cuff is implicated in the formation of porous lesions and new bone deposition in the supra- and infraspinatus fossa of the scapula. Osteochrondral junctions of ribs and long bone metaphyses may fracture. New bone $\leq$1 cm thick may be deposited on affected regions of long bone diaphyses. The most massive subperiosteal hematomas are observed on the weight-bearing long bones of the lower limb, especially in children old enough to be walking (Ortner, 2003, 384). Sharpey's fibers, which attach periosteum to bone, are shorter and less numerous in children and have less resistance to tearing and bleeding (Caffey, 1978; Lewis, 2007). Brown and Ortner (2011) and Geber and Murphy (2012) recently identified the ilium and the foramen rotundum of the sphenoid bone as potential sites of scorbutic inflammation.

A common view holds that scurvy manifests in bone following reintroduction of vitamin C into the diet following an episode of deprivation (Brickley and Ives, 2008). In essence, skeletal signs of this disorder may represent signs of recovery, implying that lesion formation is akin to a Cartesian switch that is flipped upon the conclusion of a scorbutic episode. However, multiple animal, clinical, and experimental studies (i.e., Dalldorf, 1929; Hamm and Elliot, 1938; Brailsford, 1952; Hodges et al., 1971) abundantly contribute to a progressive model of skeletal lesion formation in response to insufficient vitamin C. Pathophysiologic and cellular functional perspectives thus seem to involve a spectrum of responses, first spanning a period of progressive drawdown of bioavailable vitamin C that promotes poor collagen formation, bleeding, and inflammatory response. Only small amounts of vitamin C are needed for

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