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Review

Tibial hyperostosis: A diagnostic approach

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ABSTRACT

Tibial hyperostosis may be encountered in musculoskeletal imaging, incidentally or during the investigation of a leg pain. Hyperostosis involves the exuberant production of osseous tissue and results in cortical, periosteal and/or endosteal thickening of the bone. As a long bone with thick cortices, the tibia has a significant probability of being affected by ubiquitous bone diseases. As a tubular long bone, the tibia is likely to be involved in extensive infectious conditions such as osteomyelitis. As a bone of the lower limb, the tibia undergoes high stresses and may be affected by decrease in bone strength or repetitive submaximal stress. The tibia is also particularly involved in some bone sclerosing dysplasias and Paget's disease. In this work, we aim at highlighting the main conditions leading to tibial hyperostosis and try to provide key elements to narrow down the several diagnostic possibilities. Osteoid osteomas, fatigue or insufficiency fractures, infectious conditions, vascular lesions, sclerosing bone dysplasias and Paget's disease represent the main challenging diagnoses to discuss.

1. Introduction

Tibial hyperostosis may be encountered incidentally or during the investigation of a leg pain. Considering the wide range of diagnostic possibilities, this condition is not uncommon, especially in institutions that focus on musculoskeletal imaging. Hyperostosis results in the exuberant production of osseous tissue which is incorporated to the paleocortex with deformation of the outer bone contour and/or narrowing of the medullary canal. It may be responsible for a focal or diffuse enlargement of the bone. As a long bone with thick cortices, the tibia has a significant probability of being affected by ubiquitous conditions such as osteoid osteoma and cortical bone diseases such as Paget's disease and sclerosing bone dysplasias. As a tubular long bone, the tibia is frequently involved in infectious conditions such as osteomyelitis. As a bone of the lower limb, the tibia is exposed to the consequences of high mechanical stresses. Considering this wide range of diagnostic possibilities, tibial hyperostosis has to be properly investigated. The clinical and radiological patterns of tibial hyperostosis are not univoque but some elements are key features for the diagnostic approach.

In this review we will discuss the essential conditions leading to tibial hyperostosis. We will also give clues to properly carry out additional imaging examinations in order to narrow down the several diagnostic possibilities.

2. Conditions associated with tibial hyperostosis (Table 1)

The term "rare disease" means that less than 200,000 people are affected in the US population according to the Office of Rare Diseases (ORD) of the National Institutes of Health (NIH).

2.1. Osteoid osteoma

Osteoid osteomas account for 12% of benign skeletal neoplasms [1]. These benign bone tumours are composed of osteoid, highly-vascular connective tissue stroma and bone at various stages of maturity [2]. The shaft of long bones is preferentially involved, especially in the lower limb (femur and tibia in most cases). Osteoid osteomas are mainly observed in children or young adults between 7 and 25 years old with a male predominance [3]. Clinically, osteoid osteomas are typically responsible for inflammatory pain promptly relieved by the administration of salicylates or non-steroid anti-inflammatory drugs. There is no biological inflammation. Osteoid osteoma is a unilateral condition responsible for a focal hyperostosis. Osteoid osteomas are possibly sub-periosteal, intracortical, endosteal, in the cancellous bone and exceptionally in the medullary canal. In tubular bones, it has been hypothesized

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Table 1
Conditions associated with tibial hyperostosis according to clinical, biological and imaging findings.

				Vascular conditions			Bone sclerosing dysplasia			
	Osteoid osteoma	Infectious conditions	Fractures	Vascular malformation	Cortical hemangioma	Paget	Melorheostosis	Camurati-engelmann	Ribbing	Endosteal hyperostosis
Age	Child–adults	Child–adults	Adults	Child–adults	Child–adults	Elderly	Adults	Child–adults	Adults	Child–adults
Pain	Inflammatory	Inflammatory	Mechanical and inflammatory	Recurrent pain ↑ with exercise	Recurrent pain ↑ with exercise	None	Variable	Recurrent	Recurrent	Variable
Biology findings	None	Inflammation	None or calcium/25OH vitamin D deficiency	None	None	↑ Serum bone ALP ↑ urinary excretion of OH-proline	None	None	None	None
Uni/bilateral	Unilateral	Unilateral	Unilateral	Unilateral	Unilateral	Unilateral[a]	Unilateral	Bilateral symmetrical[b]	Unilateral or asymmetrical	Bilateral symmetrical[b]
Focal/extensive	Focal	Extensive may be focal (Brodie)	Focal may extend along cortices	Focal or extensive	Focal	Extensive	Extensive, sclerotomal distribution	Extensive	Extensive	Extensive
Additionnal imaging features	Lacuna = arterial central enhancement (MRI)	Cavity (Brodie) with rim enhancement (MRI) – cortical irregular thickening – circumferential periostitis-sequestra	Fracture line horizontal-longitudinal	Mass of vessel-phleboliths	Lobulated trabeculated-"soap bubbles" aspect (CT)	Involvement of anterior tibial tubercle-cortical thickening/coarsening-bone architecture disorganization	Aspect of "melting wax flowing down the side of a candle"	Fusiform enlargement of diaphysis	Fusiform enlargement of diaphysis	Narrowing of medullary canal

ALP = alkaline phosphatase.
[a] Possibly bilateral.
[b] Possibly asymmetrical.