Joint Bone Spine 83 (2016) 495–499

Available online at
ScienceDirect
www.sciencedirect.com

Elsevier Masson France
EM|consulte
www.em-consulte.com/en

Review

Bone infarcts: Unsuspected gray areas?

Pierre Lafforgue[a,b,*], Sophie Trijau[b]

[a] Aix-Marseille université, faculté de médecine, 27, boulevard Jean-Moulin, 13005 Marseille, France
[b] Service de rhumatologie, hôpital Sainte-Marguerite, 270, boulevard de Sainte-Marguerite, 13009 Marseille, France

ARTICLE INFO

Article history:
Accepted 6 November 2015
Available online 25 May 2016

Keywords:
Bone infarction
Avascular necrosis
Osteonecrosis

ABSTRACT

There is agreement to label as bone infarcts avascular necrosis (AVN) occurring in the metaphyses or diaphyses of long bones, the terms AVN or osteonecrosis being used at the epiphyses. One might expect bone infarction to hold no mysteries. Oddly enough, however, scientific evidence about bone infarcts is extraordinarily scant. The prevalence of bone infarcts is unknown. The main sites of involvement are the distal femur, proximal tibia, and distal tibia. In patients without sickle cell disease or Gaucher's disease, involvement of the upper limbs and lesions confined to the diaphysis are so rare as to warrant a reappraisal of the diagnosis. Although widely viewed as a generally silent event, bone infarcts causes symptoms in half the cases. Standard radiographs are normal initially then show typical high-density lesions in the center of the marrow cavity. A periosteal reaction is common and may be the first and only radiographic change. Magnetic resonance imaging consistently shows typical features and therefore, in principle, obviates the need for other investigations. Bone infarcts are multifocal in over half the cases and, when multifocal, are usually accompanied with multiple foci of epiphyseal avascular necrosis. Thus, bone infarcts, whose prognosis is good per se (with the exception of the very low risk of malignant transformation), are usually a marker for systemic avascular necrosis. Consequently, patients with bone infarcts must be investigated both for known risk factors and for other foci of avascular necrosis, which may, in contrast, have function-threatening effects.

Bone infarcts are often mistakenly viewed as a radiological oddity that has no clinical impact. However, although the radiological diagnosis is usually considered easy, errors are common, and some of their aspects remain poorly known. Bone infarction was described as a manifestation of caisson disease before being reported in other settings in 1939 [1,2]. Very few studies have specifically addressed bone infarcts. They included fewer than 20 patients and were published many years ago, with the most recent – to the best of our knowledge – having been reported in 1990 [3–6]. Furthermore, the few relatively recent publications are anecdotal case reports [7–14] that fail to shed new light on the topic. We recently identified 109 cases in 31 patients that, when combined with our earlier case series reported in 1990, provide updated information on bone infarction.

1. Definition

The name bone infarct has not been defined in detail. Traditionally, the term "bone infarct" is reserved for the death of bone and marrow tissue due to ischemia, without infection, and located in the metaphysis and/or diaphysis of a long bone. The same process located at the epiphysis is known as "avascular necrosis" (AVN) or osteonecrosis, which is also the term generally used to designate ischemic cell death of carpal and tarsal bones. For ischemic aseptic cell death affecting flat bones, such as the pelvis, ribs, and skull, both "bone infarction" and "osteonecrosis" terms are used in the literature.

Bone infarction occurring as a chronic non-inflammatory condition is the most common situation and the focus of this article, as opposed to acute bone infarction with an inflammatory response. Chronic bone infarction affects bone marrow areas containing large numbers of adipocytes. Symptoms are minimal and the date of onset therefore usually unclear. Acute bone infarction is ischemic necrosis of hyperplastic bone marrow in patients with sickle cell disease or Gaucher's disease, which can affect any part of the skeleton and causes excruciating pain. The manifestations of acute bone infarction are identical to those of acute osteomyelitis, even

* Corresponding author. Service de rhumatologie, hôpital Sainte-Marguerite, 270, boulevard de Sainte-Marguerite, 13009 Marseille, France.
E-mail address: pierre.lafforgue@ap-hm.fr (P. Lafforgue).

http://dx.doi.org/10.1016/j.jbspin.2016.02.003
1297-319X/

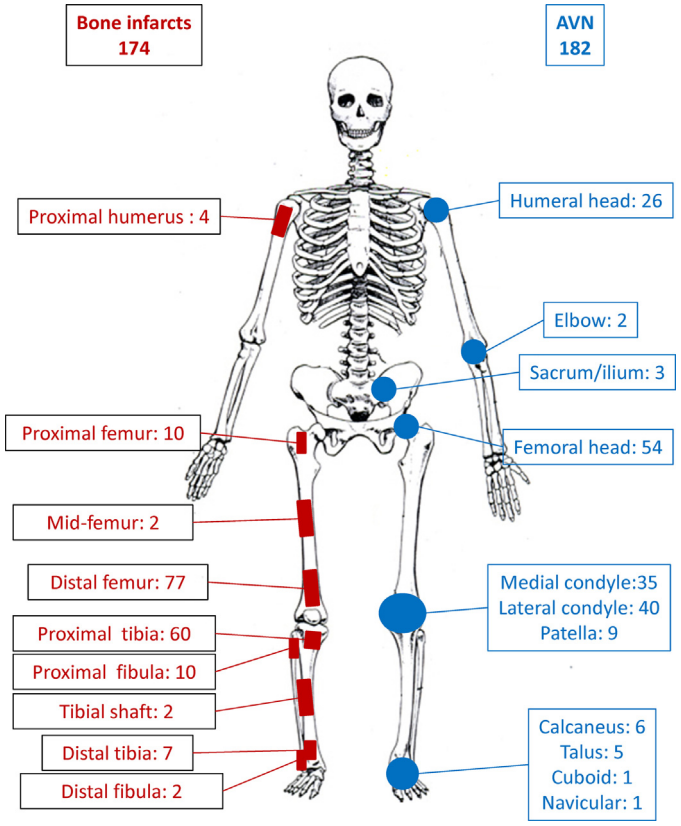

Fig. 1. Distribution of bone infarcts and other forms of avascular bone necrosis in 51 patients (adapted from [5] and additional personal observations).

regarding the laboratory and imaging findings [15–17]. Acute bone infarction contributes to the functional impairments seen in patients with sickle cell disease or Gaucher's disease.

2. Epidemiology

The prevalence of bone infarcts is unknown. Anecdotal case reports frequently describe bone infarcts as rare or very rare, and only about 25 cases have been reported over the last 25 years [7–14]. Obviously, few cases will be deemed worthy of publication, and the literature therefore provides no information on prevalence. Over 26 years, we have seen 51 patients with bone infarctions[1]. This number is small compared to that of patients with femoral head AVN seen over the same period. However, it undoubtedly constitutes an underestimation, since the cases were identified manually, as the ICD-9 has no specific code for bone infarcts. The cases are distributed equally between males and females, and most of the patients are 25 to 50 years of age [5–14].

The most common sites of involvement are the metaphyses or metaphyseal-diaphyseal regions of the knee (distal femur, proximal tibia, and proximal fibula), which accounted in our experience for 85% of all bone infarcts (Fig. 1). The proximal femur is the next most common site. The upper limb is only very rarely involved, with the main location being the proximal humeral metaphysis. Bone infarcts confined to the diaphysis are also exceedingly rare. Therefore, considerable circumspection is in order when considering a possible diagnosis of bone infarct affecting the upper limb or confined to the diaphysis.

[1] Personal unpublished case series, including ref. [5].

Bone infarction is usually multifocal. This characteristic was pointed out in the very first publications, then confirmed in most of the reported cases. Our 51 patients had 174 bone infarcts in all and only 14 patients had a single focus. The same 51 patients also had 189 foci of AVN located in the epiphyses of long bones or small tarsal bones. This multifocal distribution of the lesions highlights the systemic nature of the necrotic bone disease.

3. Diagnosis

Bone infarcts are classically identified in one of two ways: either fortuitously when imaging studies are performed to investigate another disease, such as knee osteoarthritis; or non-fortuitously during an imaging workup for epiphyseal AVN. However, although many bone infarcts are asymptomatic, others cause bone pain. In our experience, the infarct was the only detectable cause of pain in 45% of cases. The corresponding proportion among published cases is difficult to determine. Thus, the widespread belief that bone infarcts are asymptomatic is in large part unfounded.

The diagnosis relies only on imaging studies and, among these, on radiography and magnetic resonance imaging (MRI). The findings on standard radiographs depend on the time since the infarction [1,3,5,18,19] (Fig. 2). Initially, for an unknown but probably prolonged period, the radiographs may remain normal, with the diagnosis being established only by MRI. The early radiographic changes are non-specific and may erroneously cause concern when they consist of ill-defined areas of lucency or sclerotic areas in the center of the medullary canal [3,6,19]. A periosteal reaction

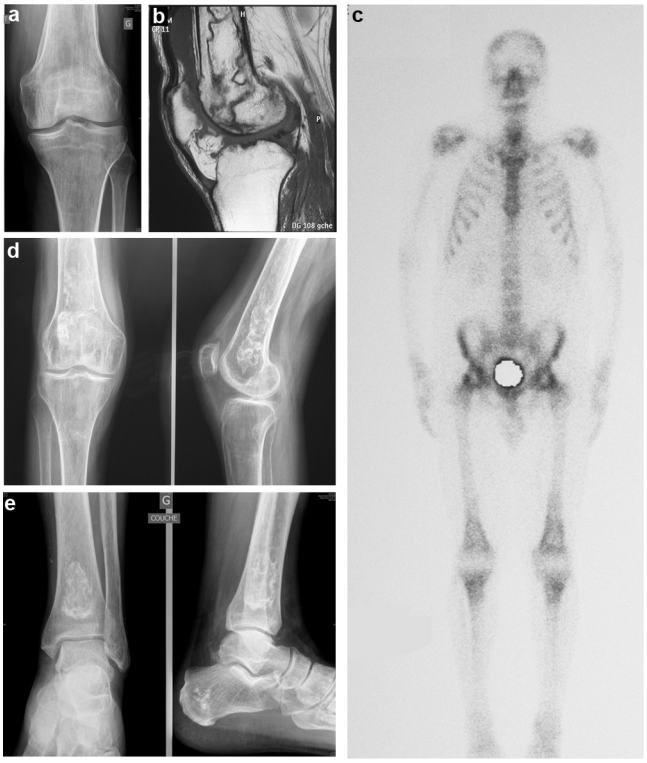

Fig. 2. Radiographic features. a: a periosteal reaction may be the only radiographic finding; b: magnetic resonance imaging confirming the bone infarct in the same patient as in (a); c: radionuclide bone scan showing symmetrically increased uptake in the distal femoral and proximal tibial metaphyses and a bull's eye sign at both hips in the same patient as in (a); d: typical appearance with a calcified lesion in the center of the bone marrow sparing the cortical bone and extending into the femoral metaphysis and diaphysis. Note the minimal periosteal reaction over the medial aspect of the metaphysis; e: typical appearance of a distal tibial infarct in a patient with multiple foci of glucocorticoid-induced avascular bone necrosis. (Note the calcifications related to avascular necrosis of the talus).