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Corticosterapie-induced bone infarction: About an observation and review of the literature

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Abstract
Bone infarction is an aseptic osteonecrosis localized to the metaphyses or diaphyses of the long bones, and sometimes flat bones, excluding epiphyseal osteonecrosis and small bones of the tarsus and carpus. The usual form is little or no pain. We report our observation, the metaphyso-diaphyseal bone infarction in a 53-year-old patient with a history of taking corticosteroids in the long run following a painful ankle, discovered late on X-ray which, showed irregular opacity.

Keywords: Bone infarction, corticosteroid therapy, sickle cell disease

Introduction
Bone necrosis, whether epiphyseal or metaphyso-diaphyseal, occurs as a result of ischemia, related to an intrinsic anomaly (obstruction by thrombosis, sickle cell, gaseous or fatty microembol) and / or an extrinsic disruption of bone circulation (trauma, vasculitis). Bone marrow cell death then occurs followed by edema causing local hypertension, which promotes the formation of a vicious circle. Then the osteocytes die and at the same time a fibro-inflammatory remodeling is created and, later, a bone reconstruction [1].
It is an ischemic necrosis, definitive, of a medullary sector generally quite extensive. Essentially, these infarctions occur in territories of yellow marrow, consisting mainly of adipocytes, which is the case of the metaphyses and diaphyses of the long bones of the adult, unlike the axial skeleton which is spared [2]. We do not know the incidence of this condition in our environment but it remains a rare clinical entity in the world or even exceptional. It is therefore obvious that this case may justify a publication but if it is far from representing its prevalence.

Observation
A 53-year-old woman with no particular history, followed for spontaneous pain in the right lower limb, first occurring when walking and then becoming permanent even at rest. She had benefited from corticosteroid injections: Kenacort retard 80 mg/2 mg per week for ten weeks for right knee osteoarthritis before being replaced by kamcilone 2 mg tablets in 2015 for several months. The clinical examination found me overweight (105 kg for 1.55 m) with a calculated BMI of 43.7. Examination of the spine shows a flexible spine, Lasègue and Wassermann maneuvers were negative, absence of root signs and peripheral neurological disorders. Mobilization of the right ankle is limited in dorsiflexion. The pressure on the lower third of the right leg is painful. The knees are unharmed. The biological assessment is unremarkable, in particular the inflammatory assessment (Count of white blood cells, CRP, sedimentation rate, leukocyte formula) hemoglobin electrophoresis is normal; the lipid profile with the profile of hypercholesterolemia and the normal liver function. X-ray of the right leg shows a well-circumscribed opacity in swirls of smoke located in the lower metaphyseal-diaphyseal leading to the suspicion of a neoplastic lesion [Fig 1].
A one-piece bone resection-biopsy after bone trepanation [Fig 2] is performed to rule out sarcomatous degeneration. The histological study shows large masses of dense collagenous tissue with focal and irregular calcification. Bony trabeculae are mostly non-vital and have empty lacunae suggestive of bone infarction.

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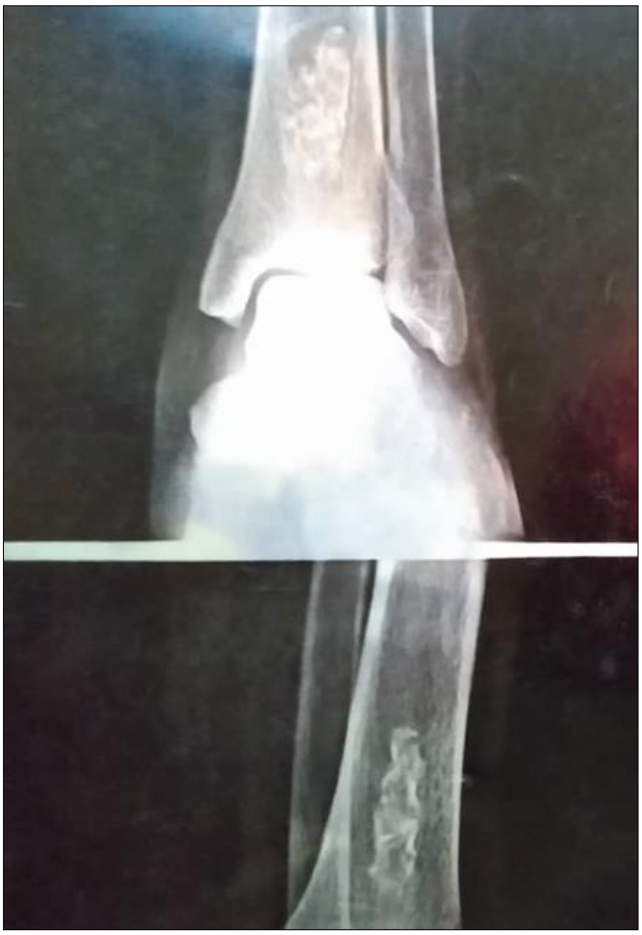

Fig 1: Standard X-ray of the leg (front and profile) showing a well-circumscribed opacity in volutes of smoke located in the lower metaphysodiaphyseal of the right leg

A one-piece bone resection-biopsy after bone trepanation [Fig 2] is performed to rule out sarcomatous degeneration. The histological study shows large masses of dense collagenous tissue with focal and irregular calcification. Bony trabeculae are mostly non-vital and have empty lacunae suggestive of bone infarction.

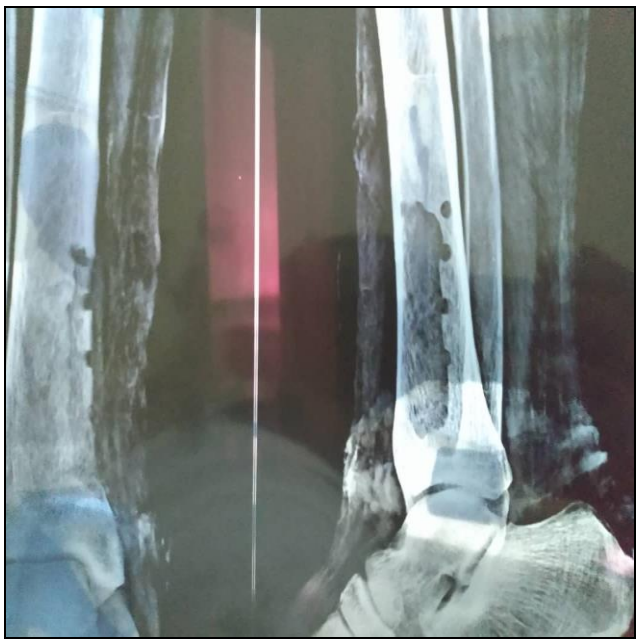

Fig 2: Standard X-ray of the leg (front and profile) showing bone defect and drill holes

Discussion

Bone necrosis, whether epiphyseal or metaphyseal-diaphyseal, occurs following ischemia, linked to an intrinsic anomaly (obstruction by thrombosis, sickling, gaseous or fatty micro-embolism, etc.) and/or a disturbance extrinsic of the bone circulation (trauma, vasculitis). Bone marrow cell death then occurs followed by edema causing local hypertension, which promotes the formation of a vicious circle. The osteocytes then die and at the same time fibro-inflammatory changes occur and, later, bone reconstruction [1, 3]. The physiopathology opposes two categories of osteomedullary infarction. The "usual", chronic and cold form, by far the most frequent, occurs with little or no symptoms. It is the equivalent to the metaphyses and diaphyses of the epiphyseal ONA, of which they share the risk factors and with which the infarctions and the acute, hot form. [4, 5]. It constitutes a rare pathology throughout the world according to published articles and in a recent study carried out in our community on bone and joint infections in sickle cell patients in Lubumbashi, Manix *et al.* [6] adds to Almeida [7] and Fatima-Zahrae Bennis [8] had found that bone damage represented 70.87% and came in third place after respiratory and otolaryngological damage; bone pathology is very common in sickle cell patients and is dominated by two major entities: Infarction and infection, infarction being much more frequent (about a ratio of 50/1) [6]. This thesis is affirmed by many authors, that the main orthopedic manifestations in sickle cell patients are osteonecrosis, osteomyelitis, septic arthritis and bone infarction [8]. These bone infarcts always occur in a particular pathogenic context and have been studied above all in barotrauma, cooking disease, cytosteatosis, cytosteatosis, Gaucher's disease [9, 10].

In our patient, the diagnosis was only made following intense, rebellious and disabling ankle pain related to a bone infarction in the absence of any history of sickle cell disease. In the literature, these lesions are often asymptomatic, discovered incidentally or during imaging performed in front of an epiphyseal ONA. Therefore, it is necessary to systematically seek an associated cause before attributing the symptomatology to the infarction. However, pain not explained by another lesion may reveal a bone infarction. Their clinical, biological and imaging presentation is then very similar to that of acute osteomyelitis, a difficult differential diagnosis [5]. The radiological images only appear at the stage of the reaction of the healthy tissue circumscribing the necrosis. Bone scintigraphy is telling much earlier and shows, in the initial phase of osteonecrosis of the etc. femoral for example, a zone of hypo fixation testifying to the circulatory anomaly and the death of the osteocytes. MRI very quickly established itself as a means of more and earlier detection of necrosis, because it highlights very well the bone marrow of the spongy bone which, in the normal subject, gives a high intensity signal [1]. In our observation, the X-ray showed a well-circumscribed opacity in wisps of smoke located in the lower metaphyseal-diaphyseal of the right leg.

Conclusion

Our observation highlights a case of corticosteroid-induced bone infarction discovered late on the basis of radiography following a sore ankle. These infarcts are often multiple, sometimes associated with epiphyseal osteonecrosis with which they share the well-known risk factors. The prognosis of bone infarctions is generally excellent, but the rare possibility of bacterial super infection or malignant transformation into sarcoma should be known. There is a need to raise awareness in our community about the danger posed by the phenomenon of self-medication.

Conflicts of interest
The authors declare no conflict of interest.

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Author contributions
Dimitri Kanyanda Nafatalewa consulted and wrote the article, Jeff Bukasa Misenga and Moise Tshiband Mosh a Bilond initiated the article and operated on the patient, Augustin Kibonge Mukakala participated in collecting patient information and arranged and translated the article in english, Manix Ilunga Banza, Vincent de Paul Kaoma Cabala, Prince Muteba Katambwa, Trésor Kibangula Kasanga, Gauthier Kibabu Wanga and Igor Mujinga wa Mujinga took part in the bibliographical research and in writing the manuscript.

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