



Case Report

Posttraumatic chronic cranial osteomyelitis due to a superficial wound - A clinical and neuroradiological case report

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ABSTRACT

Background: Osteomyelitis is a progressive infection of bone and bone marrow by microorganisms, resulting in inflammatory destruction of bone, bone necrosis, and new bone formation. Skull involvement is a rare occurrence which mainly affects children with chronic inflammatory diseases of paranasal sinusitis, or malignant otitis. In adults, cranial vault osteomyelitis can occur after cranial surgery or head trauma.

Case Description: We describe an unusual case of chronic cranial osteomyelitis occurred 3 months following a mild traumatic brain injury. The causative mechanisms along with the diagnostic modalities are discussed.

Conclusion: Focal cranial vault osteomyelitis, in the absence of severe trauma, can be challenging to diagnose. Imaging findings and patient history should be carefully investigated to make a correct diagnosis.

Keywords: Cranial vault osteomyelitis, head injury complication skull osteomyelitis, posttraumatic osteomyelitis, posttraumatic skull osteomyelitis

INTRODUCTION

Osteomyelitis is an inflammatory process accompanied by bone destruction caused by an infecting microorganism. The decline of fulminant osteomyelitis of the skull from a routine event to a rare occurrence has largely paralleled the emergence of potent antibiotics. Today, osteomyelitis of the skull usually presents as a chronic process that occasionally complicates craniotomies and scalp injuries.^[8] Despite the relative infrequency of this entity, it is essential to be skilled in the recognition and treatment of this potentially dangerous condition. New diagnostic modalities, the introduction of broad-spectrum antibiotics, have led to a decrease in the rate of treatment failure in cranial osteomyelitis. Early recognition of initial nonspecific

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symptoms is key to diagnosing and managing this treatable but life-threatening condition. However, early recognition is not possible in all the cases and antibiotic treatment cannot be started properly. Here, we describe a unique case of cranial osteomyelitis which occurred 3 months following a traumatic scalp wound.

CASE REPORT

We report a case of 45-year-old medical nun, involved in a car accident while being in a humanitarian mission in Senegal. She presented with a superficial skin wound in a cranial vault at the right parietal level that, after prompt disinfection, was treated with 2 days antibiotic regimen (two caps of amoxicillin 1 g). 3 months later, she came back to Italy and started to complain of a slight persistent headache with a sense of oppression, apparently not related to the previous head trauma. At admission, no fever, no soft tissue swelling neither skin infection was present. The previous superficial wound, reported in anamnesis, was completely healed. Neurological examination was normal.

Laboratory tests showed: hemoglobin 13.3 g/dL, total leukocyte count of 9000/uL, neutrophils 67%, lymphocytes 24% with erythrocyte sedimentation rate of 22 mm/h, C-reactive protein (CRP) 0.5 mg/dL, and procalcitonin <0.05 ng/mL. Blood cultures tests were negative for bacteria, although IgG and IgM were positive for *Mycoplasma pneumoniae*.

The patient underwent brain magnetic resonance imaging (MRI) with the following protocol: axial and coronal FSE T2-weighted images; 3D-fluid-attenuated inversion recovery, diffusion-weighted imaging, and multiplanar T1-weighted images, before and after Gadolinium-DTPA (Gadovist 0.1 mg/kg) administration.

On T2-weighted images, MRI examination showed a high signal diploic area in the right parietal bone, with an extension from the outer cortical bone to the inner table. After gadolinium administration, an intense and homogeneous enhancement of the diploic lesion was depicted. The pathological process involved the galea and the underneath dura mater. A slight contrast-enhancement inside the surrounding diploae, next to the main lesion, was present [Figure 1].

For a better evaluation of the bone involvement, the patient underwent brain computed tomography (CT) which showed a circumscribed bone lytic defect with jagged edges. The osteolytic area was better depicted by volume rendering technique reconstruction that showed a “hole-like” image, without debris within the parietal bone. Slightly suffusion in the near soft tissues was also present [Figure 2].

Taken together the neuroradiological findings along with the history of traumatic head injury, a circumscribed osteomyelitis lesion was strongly suspected. A biopsy for microbiological testing and histopathological studies was proposed, but the patient refused to give consent.

Considering that no consensus exists regarding the duration of antibiotic treatment for osteomyelitis,^[7] the patient was treated

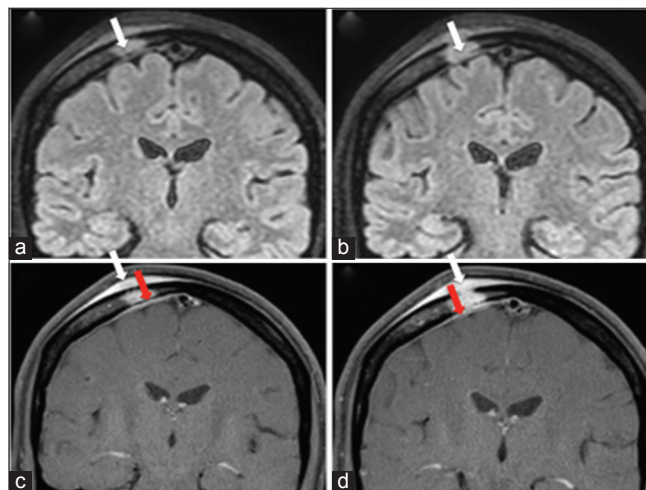


Figure 1: Magnetic resonance imaging (MRI) examination at clinical onset. (a and b) Coronal fluid-attenuated inversion recovery. A focal high signal intensity at the soft subgaleal extracranial structures in the right parietal bone is well depicted (white arrow). (c and d) Coronal Dixon T1-weighted images after Gadolinium-DTPA administration. The diploic lesion shows intense and homogeneous contrast-enhancement. Fat suppression technique well demonstrates the involvement of the galea (white arrows) and the subjacent dura mater (red arrows). A slight contrast-enhancement inside the surrounding diploae, next to the main lesion, was present.

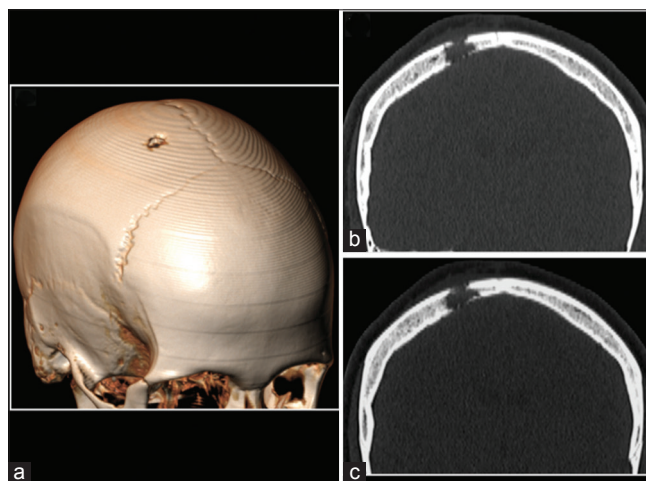


Figure 2: Computed tomography (CT) scan examination. (a) Volume rendering technique reconstruction; (b and c) Coronal multiplanar reconstruction with bone algorithm. An osteolytic area, with jagged edges, at right parietal bone is well depicted by CT scan.

with a broad-spectrum antimicrobial therapy for 4–6 weeks. In particular, ampicillin-sulbactam 3 g IV q6h was given without clinical sequels. At the end of the therapy, a follow-up MRI was performed which showed a progressive reduction of the lesion. At 1-year follow-up, the neuroradiological investigations showed complete regression of the lesion. In particular, postcontrast T1-weighted MRI images demonstrated complete regression

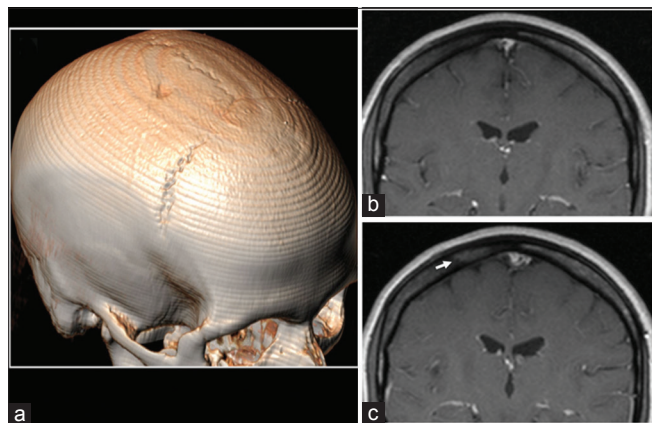


Figure 3: Computed tomography (CT) and magnetic resonance imaging (MRI) at 1-year follow-up showing a complete regression of the lesion. A, CT scan showing a minimal bone irregularity; (b and c) postcontrast T1 WI-weighted MRI images showing a complete regression of the lesion with a normal inner and outer cortical tables. Only a slightly contrast-enhancement of the parietal diploe was observed at the pathological area (white arrow).

of the lesion with a normal inner and outer cortical tables and minimal bone irregularity on CT examination. Only slightly contrast-enhancement of the parietal diploe was depicted at the pathological area [Figure 3].

DISCUSSION

Osteomyelitis is a bone marrow infection related to different pathogenetic mechanisms potentially affecting any anatomic district.^[10] Cranial vault osteomyelitis is a rare, life-threatening condition that requires early diagnosis and treatment before it can affect the central nervous system.^[7]

Osteomyelitis mainly affects a child with chronic inflammatory disease of paranasal sinuses.^[20] Furthermore, it can occur in adult patients as a complication of otitis.^[3] In adults, cranial vault osteomyelitis can occur after neurosurgical intervention or traumatic brain injury (TBI).^[1,7]

Hematogenous spreading of microorganism is the most frequent route of bone contamination, while after TBI bone infection is related to direct bony infection.^[10,12]

According to the literature, based on the site of the infection and the pathogenesis, cranial osteomyelitis can be divided into two main categories. Accordingly, it can be distinguished as sino-rhino-otogenic (SRO) and non-SRO (NSRO).^[12]

NSROs, an object of our interest, are divided into iatrogenic, posttraumatic, and hematologic.

In adults, posttraumatic skull osteomyelitis generally occurs as a complication of minor or major head injuries associated with cranial fractures, mainly determined by direct bacteria penetration after skull injuries.^[6,13] Cases of acute osteomyelitis complicating

closed or exposed fracture have been largely reported.^[18] and *Staphylococcus aureus* was the main microorganism responsible for the infection. However, cranial osteomyelitis, especially in chronic forms, are not always bacterial being other causative pathogens, such as mycobacteria, fungi, and parasites responsible for the infection. Our clinical case had a chronic and not acute form of cranial osteomyelitis. In this regard, few reports have been already published.

Only one report, published, in particular, in 1998, it was described as a case of infected wound complicated by frontal osteomyelitis.^[14] In 2001, Rodríguez-Hernández *et al.* described a case of *Aspergillus fumigatus* cranial infection secondary to accidental cranial traumatism that occurred in an immunocompetent patient.^[17] Another case of chronic nonhealing ulcers of the scalp after trauma in 11-year-old boy was reported.^[9] Finally, a scalp ulcer secondary to chronic osteomyelitis discovered >50 years after the causal trauma was reported.^[19]

The basis of appropriate therapy for cranial osteomyelitis is a precise diagnosis. Treatment of this infectious disease cannot be standardized due to the variable clinical presentations, locations, severities, etiologies, and the lack of data from randomized controlled trials. The spectrum of treatment goes from antibiotic therapy alone to combination with simple incision and drainage to invasive surgery. However, most diagnoses are delayed needing a combination of surgical debridement, correction of the primary source of infection and long-term use of antimicrobial agents. Patients with extracranial involvement or with acute localized osteomyelitis tend to be managed more conservatively than those with an intracranial extension or with chronic destructive forms. In these cases, the infected foci should be cleaned out thoroughly as early as possible, and the skin flap or myocutaneous flap should be to repair the wounds; thus, the good results can be achieved. There are various standard skull base approaches available for the removal of involved bones. These techniques require special care for the type of procedure and the appropriate timing of surgery. Surgical procedures may involve debridement of necrotic tissue, bone biopsy and culture, handling of the dead space after surgery, and when required, and bone stabilization.^[2] Prompt, appropriate treatment leads to normalization of neurological deficits, early resolution of associated symptoms, and improvements on radiographic imaging.

The treatment for cranial osteomyelitis includes a course of culture-guided long-term intravenous broad-spectrum antibiotic therapy which can take several months before a complete resolution is achieved.^[5] For bacterial or fungal infection, 3–6 months of culture-directed antimicrobial therapy remains a general protocol.^[5] For bacterial infection, antimicrobial therapies include aminoglycoside and a β -lactamase antibiotic, a third-generation cephalosporin, or an oral quinolone ciprofloxacin.^[16] For fungal infection, the use of high-dose amphotericin B is recommended.^[16] In cases of tissue hypoperfusion and hypoxia, hyperbaric oxygen therapy is a useful adjunct to antibiotics and surgical therapies

and plays a key role in the management of chronic, refractory infection.^[16]

To the best of our knowledge, our case is the first reporting late osteomyelitis after a soft tissue trauma becoming symptomatic following a complete wound healing.

In our case, the mechanism underlying the bony infection can be related to the microorganism penetration from the skin with initial infection of the galea. Subsequently, extended from the galea to the diploe bone through the Haversian canal.^[12]

In these cases, physical examination, and patient history may provide information to guide the diagnosis. In this scenario, neuroradiological investigations have an important role for the diagnosis of osteomyelitis, since laboratory testing (i.e., erythrocyte sedimentation rate, CRP levels, white blood cell, and procalcitonin) could not be helpful. In bone tissue diseases, CT and MRI are able to identify the site and the extension of the infection, showing benign or malignant features.^[15] Acute osteomyelitis is characterized by an osteolytic area, with defined edges first involving the diploe; subsequently, the infection extends into the cortical bone, through the inner and outer tables. CT scan is the most sensitive imaging method to evaluate bone erosion and periosteal remodeling although it is less capable to identify the initial intertrabecular stages and the intracranial extension of the infection. MRI has a high sensitivity for the diagnosis of osteomyelitis foci since from the early stage of disease when the edema is the only pathological finding. Moreover, MRI allows the involvement of the surrounding soft tissues and the intracranial structures to be evaluated. It should be considered, however, that osteomyelitis can mimic other bone disease, such as primary or secondary bone tumors or Langerhans cell histiocytosis.^[4]

Bone marrow edema can be detected in the early stage of disease (1–2 days after the onset of infection), and an early diagnosis prevents osteomyelitis complication such as bone and soft tissues abscess, chronic osteomyelitis, osteonecrosis, and cranial structures involvement with subdural or brain abscess.^[11]

Focal cranial vault osteomyelitis, in the absence of severe trauma, is challenging to diagnose. Imaging findings and patient history can help in making a correct diagnosis. In uncertain cases, labeled leukocyte scintigraphy or bone biopsy can confirm the diagnosis.^[10]

Finally, serial MRI examinations will provide information about the infection progression and its response to antibiotic therapy.

CONCLUSION

Osteomyelitis is a well-known inflammatory process accompanied by bone destruction. Early recognition of initial nonspecific symptoms is key to diagnosing and managing this treatable but life-threatening condition. Our case demonstrates that cranial osteomyelitis can occur even months later following a mild TBI.

Accordingly, imaging findings and patient history should be carefully investigated to make a correct diagnosis.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient has given her consent for her images and other clinical information to be reported in the journal. The patient understand that her name and initial will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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