

Case Report: Skeletal Fluorosis - An Uncommon Cause of Bone Densification

S. Naimi^{1*}, S. Faiz¹, Y. Bouktib¹, A. Elhejjami¹, B. Boutakioute¹, M. Ouali Idrissi¹, N. Cherif Idrissi El Ganouni¹

¹Radiology Department, Arrazi Hospital, Mohammed VI University Hospital, Marrakech, Morocco

DOI: [10.36347/sasjm.2024.v10i03.003](https://doi.org/10.36347/sasjm.2024.v10i03.003)

| Received: 28.12.2023 | Accepted: 04.02.2024 | Published: 04.03.2024

*Corresponding author: S. Naimi

Radiology Department, Arrazi Hospital, Mohammed VI University Hospital, Marrakech, Morocco

Abstract

Case Report

Skeletal fluorosis is an uncommon toxic bone condition marked by the excessive buildup of fluoride in the skeletal structure. The disease manifests as an endemic issue in certain regions of the world and arises from prolonged ingestion or, rarely, inhalation of elevated levels of fluoride. Key imaging features include bilateral patchy bony sclerosis, enthesopathy, ligament, and periosteal bony proliferation, as well as ossification of spinal ligaments. Management of fluorosis generally focuses on symptom treatment.

Keywords: Bone consolidation, Imaging, fluorosis.

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INTRODUCTION

Fluorosis is a widespread disease, chiefly causing dental and skeletal manifestations due to the consumption of elevated levels of fluoride, with water being the primary source. In North African nations, it is frequently linked to poisoning from contaminated water sources. The seriousness of skeletal fluorosis is evident in the emergence of skeletal deformities and neurological issues. Management of fluorosis typically revolves around symptom control.

CASE

A 58-year-old patient, being treated for fluorosis, presents with bilateral hearing loss. Our patient lived in a region of Morocco where the problem of fluorosis is endemic. A temporal bone CT scan is performed as part of the etiological assessment. On the sections passing through the skull base, a diffuse condensation of the bones at the base is observed. The purpose of this article is to demonstrate a rare cause of diffuse bone consolidation, which is skeletal fluorosis.

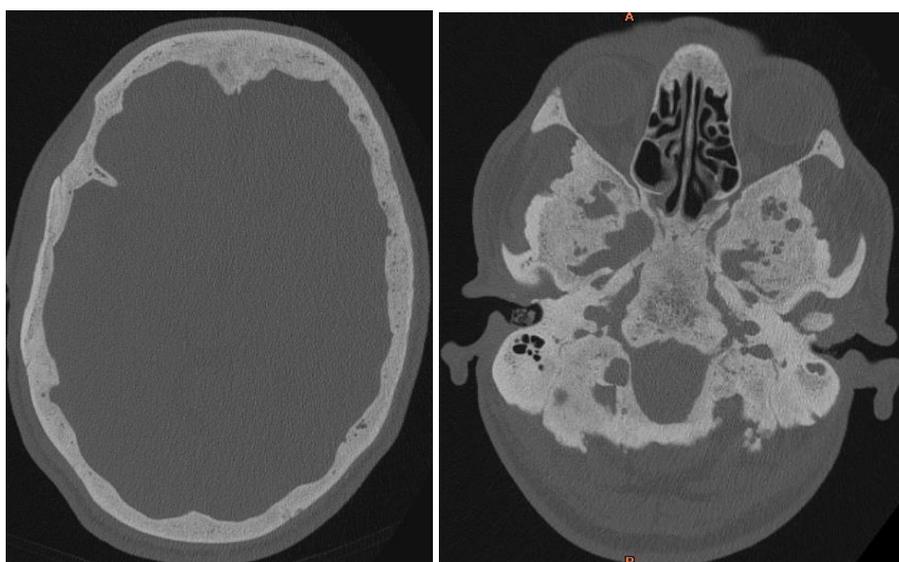


Figure 1: Axial section in bone window: diffuse bone condensation of the diploe and the bones at the base of the skull

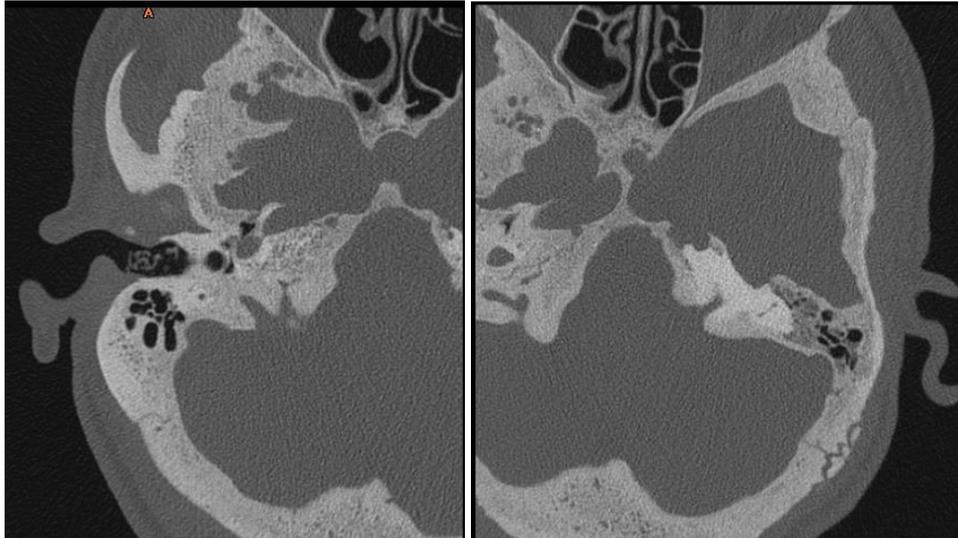


Figure 2: Axial section in bone window of the right and left petrous ridges showing diffuse condensation of the petrous apices with an impact on the left internal auditory canal

DISCUSSION

Skeletal fluorosis (SF) is a chronic metabolic bone disorder resulting from prolonged ingestion, or rarely inhalation, of fluoride ions in regions where naturally elevated fluoride levels are present. This is an important public health problem in several countries including India, where it was initially documented in the 1930s. The geographic fluoride belt stretches from Turkey to China and Japan, passing through Iraq, Iran, and Afghanistan. The prevalence of skeletal fluorosis (SF) may be on the rise, coinciding with the pollution of surface waters and the increased drilling of water wells. Indeed, the impact of fluoride on bone depends on the amount ingested and the length of exposure.

SF can arise from the consumption of water rich in fluoride, surpassing the World Health Organization (WHO) limit of 1.5 mg/l. Dental fluorosis, or mottling of teeth, occurs due to the excessive ingestion of fluoride during the formation of teeth. It can also occur due to prolonged inhalation of fluoridated gases emanating from volcanic sources, industrial waste, or the combustion of coal fires. In such cases, it may be classified as an occupational disease.

We also offer a review of non-endemic skeletal fluorosis (SF), which is not linked to medicinal or industrial exposure, as documented in the American and European literature. We emphasize the sources of fluoride, their impact on calcium metabolism, and the bone pathophysiology associated with this condition. SF is frequently asymptomatic and is serendipitously detected through radiological examination. Symptoms include bone and joint pain, deformities, fractures, and developmental dental abnormalities.

Radiological examination is considered the most effective method for the early diagnosis of fluorosis, particularly in the asymptomatic stage, which

may persist for 10–30 years before overt clinical symptoms emerge.

Characteristic imaging features include osteosclerosis, osteophytosis, and ligamentous calcifications, most notably observed in the pelvis and spine.

X-rays may reveal a solid periostitis affecting multiple bones, indicating a benign and slow-growing process. Osteopenia, though less common, may also manifest in fluorosis, particularly in younger patients at an earlier stage.

Computed tomography can display generalized trabecular heterogeneity with an overall elevation in bone density, hypertrophic spurring at bone margins, regions of relative radiolucency, and a coarse trabecular pattern. It may also present changes in the spinal canal, indentations within the epidural space, and calcified ligaments.

Magnetic resonance imaging (MRI) can be beneficial when there is a suspicion of fatigue fractures or neurological complications. High signal intensity areas in the spinal cord on T2-weighted images at the site of compression confirm compressive myelopathy. Osteosclerosis exhibits low signal intensity on both T1- and T2-weighted images.

Bone scintigraphy with technetium demonstrates diffuse uptake throughout the axial and peripheral skeleton, signifying notable bone turnover. Other differential diagnoses can be responsible for diffuse bone condensation, with the most significant ones being Paget's disease, renal osteodystrophy, myelofibrosis, osteoblastic metastasis and ankylosing spondylitis.

Skeletal fluorosis (SF) can result in substantial and debilitating deformities, such as kyphosis, limited spinal and chest movement, and deformities of extraspinal joints, particularly the hips, increasing the susceptibility to hip osteoarthritis. Moreover, several studies have reported a high incidence of genu valgum deformity.

Neurological complications may occur in 10% of patients with SF. These are primarily due to mechanical compression of the spinal cord and nerve roots resulting from osteophytosis, a significant reduction in the anteroposterior diameter of the spinal canal and intervertebral foramina, a sclerosed vertebral column, and ossified ligaments. At present, there is no specific treatment available for fluorosis, except for discontinuing the causative etiological factor. The management of SF typically revolves around alleviating symptoms through treatments such as analgesics and non-steroidal anti-inflammatory drugs as needed.

CONCLUSION

Skeletal fluorosis is a rare cause of bone sclerosis. Imaging plays a crucial role in confirming the diagnosis alongside endemic, clinical, and biological contexts. No treatment for fluorosis can currently be

proposed, except for discontinuing the underlying causative factor.

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