IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861.Volume 22, Issue 3 Ser.13 (March. 2023), PP 10-11
www.iosrjournals.org

Palate osteomyelitis- "Issues untouched"

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Date of Submission: 08-03-2023 Date of Acceptance: 21-03-2023

Dear Sir,

I read with great interest the article published in your esteemed journal- "Dr. Anubhav Das Adhikari, et. al. "Osteomyelitis of the palate: A case report of a rare case." IOSR Journal of Dental and Medical Sciences (IOSR-JDMS), 22(3), 2023, pp. 57-59". However, author has missed on various aspects of this useful information. I feel, it is pertinent for the readers to update their knowledge on certain issues, which authors have missed inadvertently to give justice to the readers of this esteemed journal.

Osteomyelitis typically appears during the 5th and 6th decades of life. Osteomyelitis of maxilla was originally described by Rees in 1847 [1]. Osteomyelitis of the jaws was relatively common before the era of antibiotic therapy. Today osteomyelitis of facial bones is a rare condition. Maxillary osteomyelitis is rare compared to mandible osteomyelitis because extensive blood supply & strut like bone of the maxilla make it less prone to chronic infection [2, 3]. Macbeth in 1952 classified the etiology of osteomyelitis of maxilla into traumatic, rhinogenic and odontogenic [4].

Lew and Waldvoger [5] classify osteomyelitis into suppurative and non-suppurative, depending on its infectious character or hematogenous origin.

Osteomyelitis may develop due to contiguous infection, trauma, surgery and overuse of broad spectrum antibiotics or certain systemic conditions like diabetes, malnutrition, malignancy, radiation or other immunocompromised states [6]. Agranulocytosis, leukaemia, anaemia, nutritional deficiencies, syphilis, cancer, chemotherapy and auto-immune diseases, as well as habits of alcohol or tobacco consumption should also be ruled out [7].

Fungal osteomyelitis usually occurs in immunocompromised individuals but it can affect immunocompetent individuals also. In maxillofacial region, *Candida* induced osteomyelitis is seen very rarely. The extensive vascularity of the maxilla compared to the mandible makes it a less preferred site of involvement [8]. So, tissues need sent for histopathology, KOH mount, aerobic bacterial and fungal culture and sensitivity on the same day. Additional growth of Group A β hemolytic *Streptococci* and *Enterococcus* species is to be obtained.

It is important to note that presence of sulfur granules in the tissue aspirate, which can indicate the likelihood of Actinomycoses.

A biopsy taken from the margins may reveal chronic inflammation and vasculitis. Tuberculosis workup is not done here. Also, Serology for Hepatitis B, Hepatitis C, Hepatitis E and HIV status is not mentioned including her blood sugar levels, Haemoglobin, Erythrocyte Sedimentation Rate (ESR) and autoimmune workup (ANA, c-ANCA, and p-ANCA) with C3 and C4 levels.

Plain Radiograph of paranasal sinuses (Water's view) have not been given here. These days Computed tomography (CT) of the maxillofacial is a gold standard and has not been shown here or performed by the author.

It is pertinent to note that the general treatment of osteomyelitis usually involves surgical debridement of necrotic material and biopsies for the culture of affected tissue/bone (assuming the patient is an appropriate candidate) followed by appropriate antimicrobial therapy. Adjunctive nasal endoscopy is a non invasive approach which helps in achieving complete clearance of the necrotic remnants from the cavity.

While in hospital, in addition to bone debridement, the patient is with IV antibiotics and antifungal agents and is usually discharged with oral medication for a total of six weeks of therapy.

It is worthwhile to point out that Lentrodt et al [9] mentioned that hyperbaric oxygen therapy is an adjuvant in osteomyelitis therapy since it elicits tissue hyper-oxygenation effect, antimicrobial activity, fibroblast proliferation, neo-vascularisation, bone matrix formation increase, mineralization increase, as well as osteoblastic function improvement. He observed a protocol of 60 to 90 minute daily sessions with 100% oxygen at 2.2 and 2.4 ATM pressure during at least 15 days.

Moreover, author failed to describe the follow up, as the literature clearly mentions that the patient should be kept under strict observation for at least 6 months after stopping the treatment and in case of clinical suspicion of residual/recurrent disease; patient should be subjected to radiology and/or histopathology of the suspicious area to prevent any relapse. Besides, it is documented that the patient should be followed with weekly complete blood count (CBC), comprehensive metabolic panel (CMP), erythrocyte sedimentation rate (ESR), and C-reactive protein (CRP) to monitor for resolution during the therapy.

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