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Osteomyelitis Associated With Pseudomonas Aeruginosa and Acinetobacter Baumannii Involving Hard Palate – An Unusual Case Report

Dr. Sayani Shome[1] Dr. Rudra Prasad Chatterjee[2] Dr. Jayanta Kumar Chattopadhyay[3] Prof. Dr. Sk. Abdul Mahmud[4] Dr. Sanjeet Kumar Das[5]

[1]Post-graduate trainee, Dept. of Oral and Maxillofacial Pathology,Guru Nanak Institute of Dental Sciences and Research, Kolkata

[2]Assistant Professor, Dept. of Oral and Maxillofacial Pathology, Guru Nanak Institute of Dental Sciences and Research, Kolkata

[3]Reader, Dept. of Oral and Maxillofacial Surgery, Awadh Dental College and Hospital, Jamshedpur

[4]Professor, Dept. of Oral and Maxillofacial Pathology, Guru Nanak Institute of Dental Sciences and Research, Kolkata

[5]Senior Lecturer, Dept. of Oral and Maxillofacial Pathology, Guru Nanak Institute of Dental Sciences and Research, Kolkata

Corresponding Author: Dr. Sayani Shome, Post-graduate trainee, Dept. of Oral and Maxillofacial Pathology,Guru Nanak Institute of Dental Sciences and Research, Kolkata

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Abstract

Osteomyelitis is a debilitating, necrotizing disease arising from bone and its marrow due to pathogenic microbes. Occurranace of osteomyelitis in maxilla is less common than in mandible due to sufficient vascularity that wards off microbiota associated with this disease process. Chronic osteomyelitis is almost an indispensible consequence of immunocompromised states in elderly patients such as long standing case of insulin dependent diabetes mellitus, chronic kidney disease, malignancies, individuals undergoing radiation therapy etc. Osteomyelitis involving hard palate due to virulent bacteria like *Pseudomonas aeruginosa* and *Acinetobacter baumannii* is an unique event that demands judicious clinical diagnosis and management.

Keywords: Osteomyelitis, hard palate, immunocompromised state, *Pseudomonas aeruginosa*, *Acinetobacter baumannii*

Introduction

The word “Osteomyelitis” is a conjoint derivation of two Greek words ‘osteone’ (bone) & ‘muilenos’ (bone-marrow)[1]. John Hunter in 1764 devised the term sequestra & involucrum for necrosed bone chips and newly formed bony contour respectively[2]. Later in 1852, Edouard Chassaignac from France described the clinical course of this inflammatory bone disease[2]. It is a polymicrobial chronic relapsing condition arising primarily from medullary cavity and gradually reaches periosteum via haversian system rarely involving maxilla due to profound vascular supply compared to mandible[3]. Impaired host-immune system is a strong risk-factor of osteomyelitis[3] where *Pseudomonas aeruginosa*[4] and *Acinetobacter baumannii*[5] are two affiliated causative microorganisms.

Case Report

A 48 years old male patient reported to us with chief complaints of discomfort in the upper jaw while eating

with foul smell since 1 year. He initially developed a painful gum swelling in the upper anterior jaw which was diagnosed as peri-apical abscess by local dental surgeon and the offending tooth was extracted. Later on, he was advised to undergo complete oral prophylaxis after which there was spontaneous exfoliation of other anterior teeth in the upper arch.

Patient was suffering from chronic kidney disease (CKD) along with diabetes mellitus since 15 years with uncontrolled hyperglycemia (Fasting blood sugar 134.0 mg/dl and Post-prandial blood sugar 395.0 mg/dl) and slightly raised serum creatinine level. He also had another co-morbidity that is essential hypertension, for which he takes regular antihypertensive medication. No relevant past surgical history revealed on interrogation. He had previous history of persisting deleterious oral habits of chewing khaini 3-4 times a day for more than two decades.

The extraoral facial profile of the patient was apparently normal [Fig.1]. Intraoral examination revealed presence of blackish, necrotic bare palatal bone which is covered by redundant folds of firm palatal mucosa on the posterior aspect with empty sockets on the anterior region [Fig.2]. The palate was completely mobile. Radiographic images were advised to delineate involvement of the structures in close proximity. Orthopantomogram (OPG) revealed complete destruction of palate at the level of alveolar process [Fig.3]. Computed tomographic (CT) imaging of head and neck region revealed the presence of bilateral apparently normal maxillary antrum and normal nasal septum in midline with mild soft tissue thickening in the posterior palate along with destruction of the palatal process anteriorly [Fig.4]. Chest poster-anterior radiograph was also found to be normal with respect to anatomic structures. [Fig.5]

Depending upon all the clinical presentations and medical co-morbidities, we provisionally diagnose the case as Mucormycosis. Differential diagnosis was chronic osteomyelitis involving palate.

All the routine hematological investigations along with liver function test, kidney function tests and serology for HIV 1, HIV 2 and HBV, HCV have been performed. Fasting blood sugar and post-prandial blood sugar were excessively high with uncontrolled glycosylated hemoglobin (HbA1c %). All other parameters were within normal limit and serology was negative for aforementioned components. Patient was referred back to consultant endocrinologist and swabs from the anterior dento-alveolar region and posterior palatal region were taken by us in two separate sterile glass tubes for microbial culture [Fig.6]. Culture for fungal etiology was found to be negative and it was turned out to be the causal effect of *Pseudomonas aeruginosa* (confluent growth) on the anterior dento-alveolar region and *Acinetobacter baumannii* (scanty growth) from the posterior palatal region, after overnight incubation.

Fibreoptic antrostomy was performed to rule out invasion within the nasal cavity and maxillary antrum after written consent had been received from the patient. He was advised to undergo complete removal of necrotic hard palate and radial forearm flap coverage to wrap the bare area, under general anaesthesia and the procedure was performed under extreme supervision. Sequestered bone fragments from the gross specimen [Fig.7] were processed and evaluated histopathologically by hematoxyline and eosin stain. Histopathological features [Fig.8a and Fig.8b] revealed necrotic osseous tissue almost devoid of entrapped osteocytes and infiltrated with non-specific chronic inflammatory cells. Our patient was on concurrent antimicrobial therapy which was parenteral Piperacillin

and Tazobactam combination (1.5 gm twice daily for 7 days intravenously).

Discussion

Association of mixed (aerobic and anaerobic) microbial flora[6] in osteomyelitis along with fungi and virus[3] has been established where inflammatory exudates jeopardize the vascular supply in the confined anatomical area[3] and also directly invades endothelium of the capillaries[1] leading to avascular necrosis of the bony compartment. Though involvement of cranio-facial skeleton in this disease process is quite unusual in industrialized countries[7], hematogenous spread of local infection instigates pathogenesis of jaw osteomyelitis[8]. Individuals with poor immunosurveillance and immunocompromised systemic conditions are habitat for the ubiquitous microorganism like *Pseudomonas aeruginosa*[9]. *Pseudomonas aeruginosa* is a gram-negative, non-sporing, non-capsulated, aerobic motile melanin-like pigment producing bacillus[5]. It is considered as an opportunistic pathogen due to it's rare encounter in a healthy individual[5]. A. B. Mori in her documented study among the health workers in an oncology hospital in Goiania, established striking prevalence of *Pseudomonas sp.* in their saliva, where it was a mixed colony of *P. aeruginosa*, *P. stutzeri* and *P. fluorescens*[9]. Osteomyelitis of microbial origin are prudent in immunocompromised patients[3] like in diabetes mellitus and allied health hazards and one of the early reports had been published by British physician, Dr. Benjamin Brodie in 1832[10]. Association of osteomyelitis with long standing diabetes mellitus is prudent and supports our case too. Depending upon the history given by the patient and clinical course of the disease we provisionally diagnose the case as of fungal etiology, precisely mucormycosis keeping in mind the differential diagnosis of chronic osteomyelitis. Chronic osteomyelitis demands immediate intervention by surgical methods and of course by administering oral or parenteral antibiotics as the case may be. Long standing diabetic state coupled with odontogenic infection had terminated into osteomyelitis in palate in our case. As the serum creatinine level of our patient was raised, assumption of chronic kidney disease (CKD) is also inferred here. CKD patients are susceptible to develop infection of urinary tract, peritoneum and bacteremia by *Pseudomonas aeruginosa*[11]. The course of infection by *P. aeruginosa* is unostenstatious in most of the time but it can lead to vascular invasion resulting in arterial obliteration and thrombosis pertaining to necrosis of the tissue[12]. P. aeruginosa is able to produce bluish green phenazine pigment known as pyocyanin, greenish yellow pigment known as fluorescin and pyorubin (red) in pigment enhancing media[5]. Endarteritis along with melanin-like pigment production give rise to blackish necrotic appearance of the affected tissue[12]. This statement reinforces gross clinical appearance of the lesion over hard palate in this case. The involved part of the palate was completely necrotic and blackish corroborating infarction due to less vascularity and regional tissue hypoxia. *Acinetobacter baumannii* was another causative organism isolated in our case, which is responsible to cause nosocomial infections, osteomyelitis, wound infections in immunocompromised patients[5]. It is gram-negative, glucose non-fermenter bacilli which are habitat of soil, water and so called as saprophytes, often is found to reside in moist human skin as non-pathogenic strain[5]. It follows classical microbial pathogenesis in human being that implicates interactions of capsular lipopolysaccharides with Toll-like receptor 4 (TLR 4)[13]. Role of *Acinetobacter sp.* in osteomyelitis is evident in our case which is according to the previous literature. Hard palate, being an indelible part of head and neck anatomy renders direct communication with the base of the skull. Deadly

outcome of untreated osteomyelitis in hard palate can further lead to involvement of skull base with significant mortality rate of approximately up to 10% to 20%[14]. The histopathologic features of bone fragments in our case is also consistent with the light microscopic features in osteomyelitis which shows necrotic bones along with loss of osteocytes from the lacunae and necrosis of the marrow space with infiltration of chronic inflammatory cells[1]. The first line antipseudomonal β-lactam is the combination of Piperacillin and Tazobactam that has a tremendous effect to reduce mortality has been reported previously and one of the successful studies had been conducted by Lodise T P *et al.* among patients with *Pseudomonal* infection in Albany Medical Centre, New York, USA[15]. In our case, intravenous Piperacillin-Tazobactam combination was administered as anti-microbial adjunct to surgical correction to the anatomic defect in hard palate and this reinforces former articles.

Conclusion

Hard palate is a component of maxilla and palatal bone. Osteomyelitis involving maxilla is a rare entity due to presence of significant amount of cancellous bone which provides enormous blood supply. If it gets involved otherwise, any other co-morbidities like diabetes, CKD etc that often magnify the course of this complex disease. Subsequently the presence of *P. aeruginosa* and *Acinetobacter sp.* in the osteomyelitis of palate and maxilla is documented thus necessitating culture sensitivity and proper management.

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Legends Figure

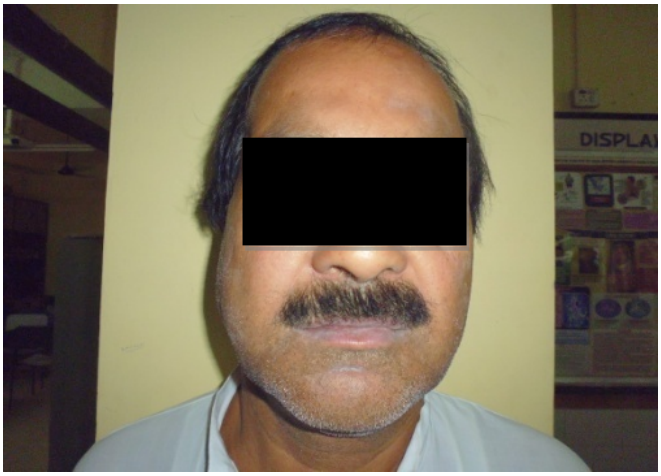

Fig.1: showing apperantly normal extra-oral profile

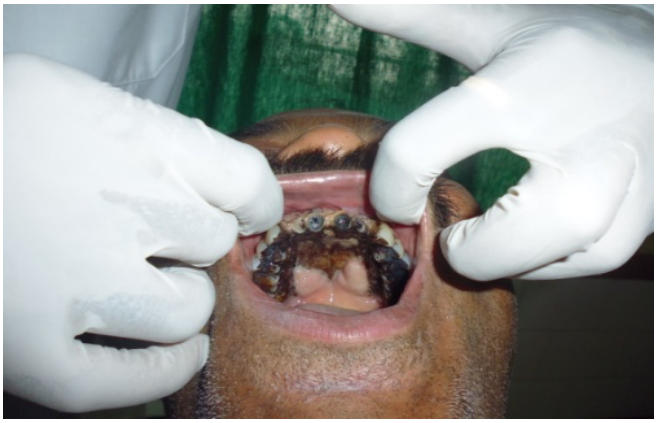

Fig.2 : showing denuded, bare palatal bone with remaining few teeth

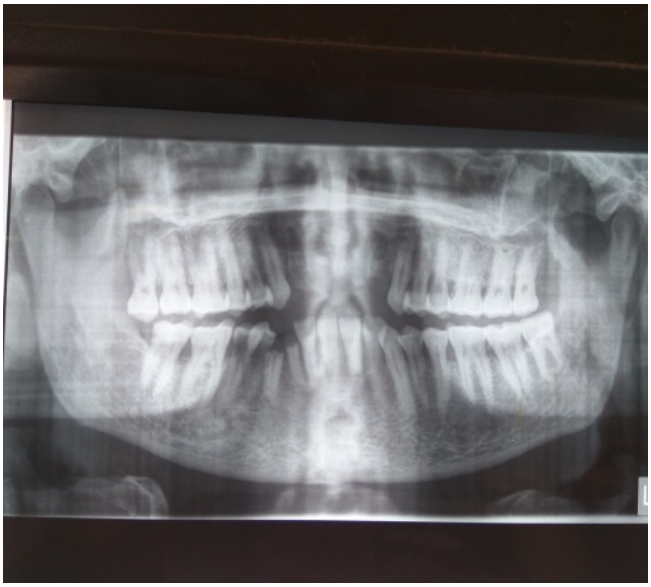

Fig.3 : OPG revealed complete destruction of palate at the level of alveolar bone

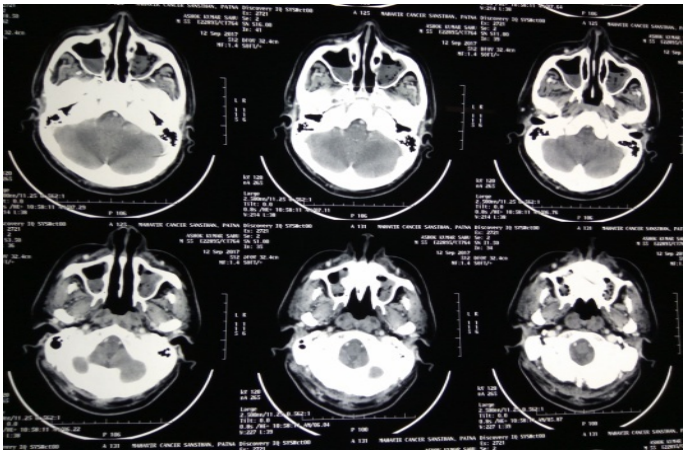

Fig.4: CT Scan revealed mild soft tissue thickening in posterior palate and destruction of anterior palate. Antrum was devoid of any pathology

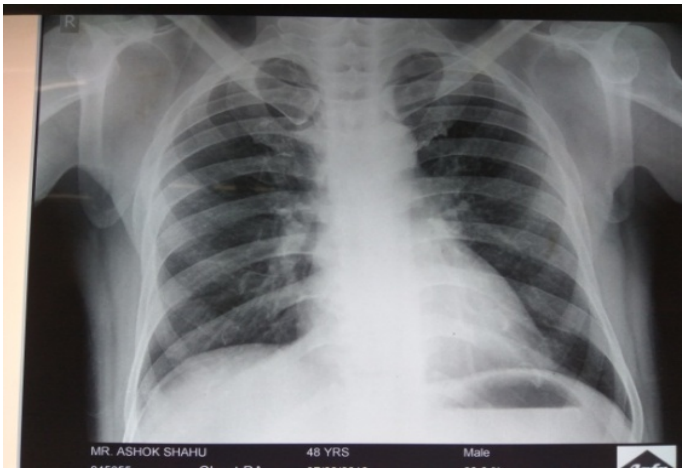

Fig. 5: shows normal PA Chest X-Ray

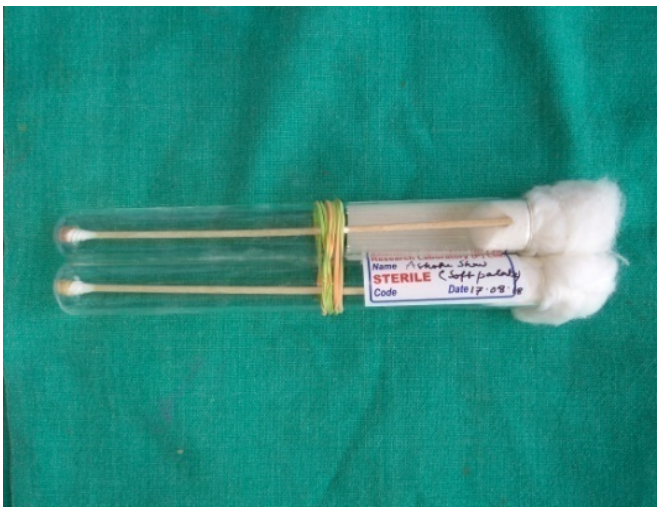

Fig. 6 showed swabs taken for microbial culture

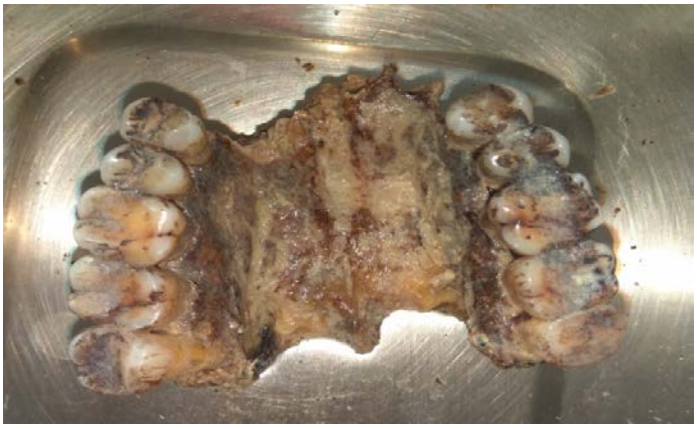

Fig.7 shows gross-specimen of the necrosed hard palate and dento-alveolar process

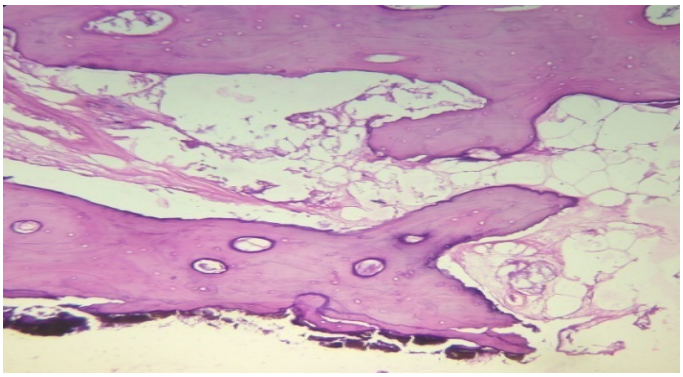

Fig.8a

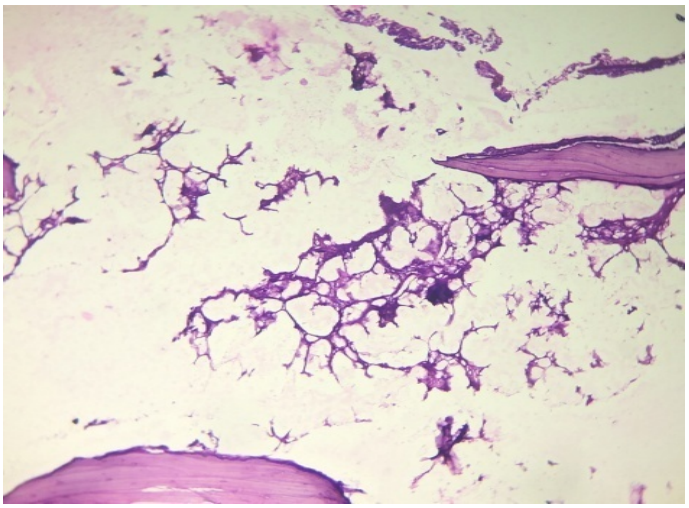

Fig.8b

Fig.8a and Fig.8b showed sections stained with Hematoxyline and Eosin revealing presence of necrotic sequestered bone, obliteration of marrow space along with infiltration of chronic inflammatory cells