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Concurrent Mandibular Pyogenic Granuloma and Acute Osteomyelitis Following SARS-CoV-2 Infection: A Case Report

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ABSTRACT

This report details a complex oral manifestation of SARS-CoV-2 infection in a 50-year-old female recovering from moderate COVID-19. The patient presented with severe mandibular pain, which upon comprehensive clinical, radiographic, and histopathological evaluation was diagnosed as concurrent advanced pyogenic granuloma and acute osteomyelitis with sequestration in the same jaw quadrant, accompanied by severe bilateral facial paresthesia. The diagnostic process was protracted, with osteomyelitis only confirmed following a subsequent presentation with exposed bone. Management necessitated sequential surgical debridement, targeted antibiotic therapy, and neuropathic pain control with Gabapentin, culminating in eventual resolution. This singular co-occurrence of a reactive vascular lesion and an invasive bony infection suggests a unified pathophysiological model wherein SARS-CoV-2-induced endotheliopathy, cytokine dysregulation, and immune dysfunction collectively establish a permissive local environment for both aberrant angiogenic proliferation and bacterial invasion of bone. The case underscores the imperative for heightened clinical suspicion of complex, co-existing maxillofacial pathologies in post-COVID patients and illuminates the potential therapeutic dilemmas inherent in managing such intertwined inflammatory and infectious sequelae.

Keywords: COVID-19; Osteomyelitis; Pyogenic granuloma; Oral manifestations.

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Introduction

While COVID-19 first announced itself as a severe respiratory illness, we have since learned that the SARS-CoV-2 virus casts a much wider net, affecting numerous systems throughout the body [1]. The disease is notoriously unpredictable, striking people differently; some feel nothing, while others face life-threatening organ failure [2]. One of the more personal and visible sites of these unexpected effects is the mouth. Doctors and researchers have cataloged a surprising variety of oral sores, ulcers, and taste disturbances in COVID-19 patients, though pinning them directly on the virus remains a complex challenge [3]. It's believed these oral issues erupt from a perfect storm of factors: the virus itself potentially injuring delicate mouth tissues, a weakened immune system allowing secondary infections, and the body's own intense inflammatory response running out of control [4,5].

In this landscape, we see conditions such as pyogenic granuloma, a typically harmless, overgrown bit of tissue often triggered by local irritation or trauma [6]. Its presence in COVID-19 patients suggests that the virus's inflammatory chaos may be a new trigger. On the more severe end lies osteomyelitis, a serious and painful bone infection that is typically a sequel to a dental infection or injury [7]. Finding these two conditions-one a reactive soft tissue growth, the other a deep-seated bone infection-arising together in the same jaw of a COVID-19 patient is remarkably rare. This unusual pairing forces us to consider a troubling question: could the unique biological turmoil caused by SARS-CoV-2 be creating a perfect environment for such disparate pathologies to co-exist? This report documents that very occurrence, seeking to understand the hidden connections.

Case Description

A 50-year-old female was referred to Shahid Beheshti Hospital in Kashan, Iran, with chief complaints of fever, headache, fatigue, and lethargy of seven days' duration. Following written informed consent, the patient underwent diagnostic evaluations, including a chest CT scan and an SARS-CoV-2 polymerase chain reaction (PCR) test, which confirmed a diagnosis of COVID-19. Subsequently, she was hospitalized for 14 days to manage her systemic condition. Approximately 1.5 days before her scheduled discharge, the patient developed sudden, severe jaw pain. The medical team made a provisional diagnosis of a secondary bacterial or fungal infection empirically. She was initiated on a broad-spectrum antimicrobial regimen consisting of Cefixime (400 mg q12hr), Metronidazole (400 mg q6hr), an anti-fungal Nystatin oral suspension (20 drops q6hr), and a 0.2% Chlorhexidine mouthwash (BID). She was discharged 36 hours after this treatment began. Two days post-discharge, the patient returned for a follow-up appointment with worsening symptoms. Her oral condition had deteriorated significantly, presenting with a severe oral lesion, generalized class III tooth mobility, and a recurrent fever. The previous antibiotic regimen and Acetaminophen (1 tablet q6hr) were re-prescribed to manage the fever, and she was urgently referred to an oral medicine specialist for further investigation.

Intraoral examination revealed pus excretion and multiple, scattered, lobulated exophytic masses (Figure 1). Given the clinical presentation of purulent discharge alongside proliferative soft-tissue lesions, the primary differential diagnoses included pyogenic granuloma and acute osteomyelitis. Histopathological analysis of the biopsied soft tissue confirmed a diagnosis of advanced pyogenic granuloma (Figure 2). Consequently, surgical intervention was performed, including excision of the granulomatous tissue, thorough debridement, and extraction of the severely mobile teeth. A significant accompanying symptom from the onset of the oral condition was severe bilateral facial paresthesia, more pronounced on the left side, affecting the lower lip and mental region. Zinc supplementation was initiated, and the paresthesia was monitored using the Visual Analog Scale (VAS). Over three weeks of follow-up, the paresthesia improved remarkably (VAS reduced from 10 to 3). However, a recurrence of the pyogenic granuloma was noted, for which intraregional injections of Triamcinolone Acetonide were administered.

In the fourth week, the patient reported a resurgence of localized pain in the region of the right mandibular second molar, accompanied by exposed bone, pus excretion, and intensified paresthesia. These findings raised a strong suspicion of acute osteomyelitis. An orthopantomogram (OPG) revealed sequestration (Figure 3). After that, a CBCT was taken, and we observed vertical and horizontal bone loss and multiple periapical lesions (Figure 4). The area was surgically explored, and a 1.5 cm bony sequestrum was excised (Figure 5). Subsequent thorough debridement was performed until viable, bleeding bone was reached, followed by irrigation and closure. Histopathological examination of the excised bone fragment confirmed the

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diagnosis of acute osteomyelitis (Figure 2). Following the definitive diagnosis, a targeted antibacterial therapy with Cefixime (400 mg q12hr) was initiated. The patient was placed on a one-month follow-up schedule for three consecutive months. Mucosal healing progressed satisfactorily. Due to persistent paresthesia, Gabapentin (300 mg q24hr) was prescribed. The VAS score was monitored weekly; it decreased from 5 to 3 in the first week and reached zero by the fourth week. The Gabapentin dose was maintained for one additional month for consolidation. After two months, complete mucosal repair was achieved, and the patient was referred for prosthetic rehabilitation with a partial denture to address the edentulous area.

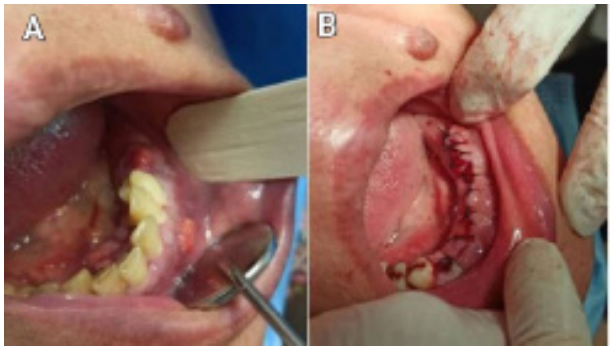

Figure 1. A) Intraoral view revealing multiple, scattered, lobulated exophytic masses B) Intraoral view after excisional biopsyreview.

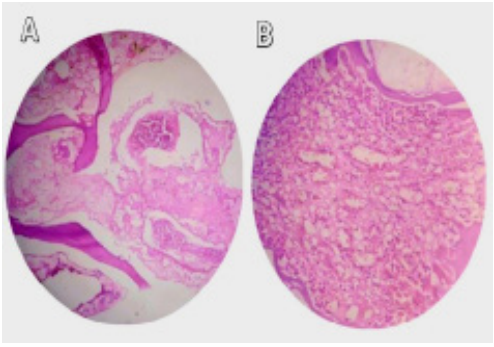

Figure 2. A) Osteomyelitis microscopic view (H&Ex100) B) Pyogenic Granuloma microscopic view (H&Ex100).

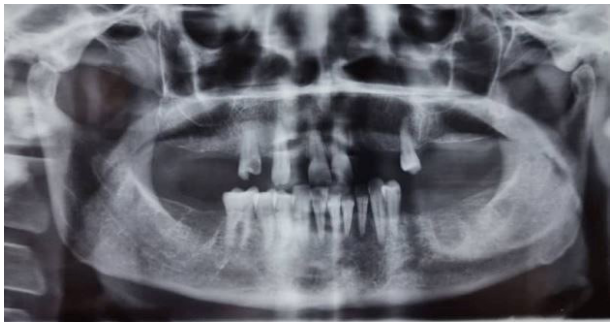

Figure 3. Radiograph in Panoramic view, yellow arrows revealing the presence of sequestration.

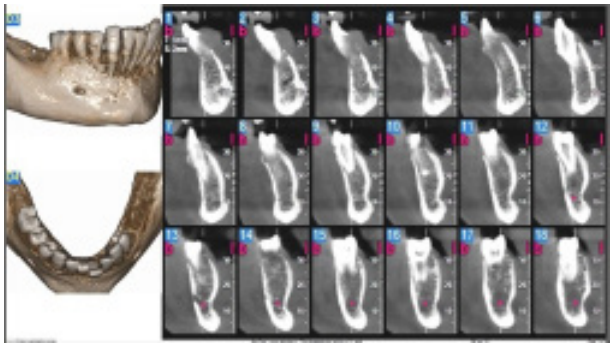

Figure 4. Cone beam computed tomography showing vertical and horizontal bone loss.

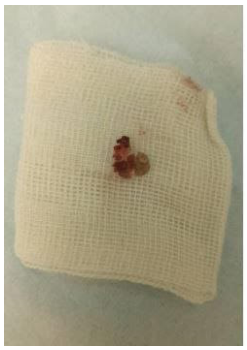

Figure 5. Excised bony sequestrum.

Discussion

This report presents a singular case of concurrent mandibular pyogenic granuloma and acute osteomyelitis in a patient recovering from COVID-19. The co-occurrence of these two distinct pathologies in the jaw represents a rare and clinically challenging sequela of SARS-CoV-2 infection, suggesting a complex interplay between the virus and the oral microenvironment that extends beyond common oral manifestations such as ulcerations or dysgeusia [3,4]. The pathogenesis of this dual presentation can be theorized to stem from the profound systemic effects of SARS-CoV-2. The virus gains cellular entry via the Angiotensin-Converting Enzyme 2 (ACE2) receptor, which is abundantly expressed on oral mucosal epithelial cells and salivary glands [8,9].

This direct viral presence may initiate local tissue injury. However, a more significant driver is likely the systemic "cytokine storm" characterized by a surge of pro-inflammatory cytokines such as IL-6 and TNF-α [10]. This hyperinflammatory state is coupled with well-documented COVID-19-induced endotheliopathy and vasculitis, which disrupt vascular integrity and promote a prothrombotic state [11,12]. In the context of pyogenic granuloma, a reactive, hyperplastic lesion fundamentally driven by angiogenic proliferation, this environment of vascular injury and elevated inflamma-

tory mediators may serve as a potent trigger [6]. The upregulation of vascular endothelial growth factor, a key mediator in pyogenic granuloma pathogenesis [13], could be significantly amplified by the COVID-19-related cytokine cascade, facilitating the development of the exuberant granulation tissue observed in our patient. Simultaneously, the same pathophysiological milieu creates a fertile ground for osteomyelitis. The initial viral and cytokine-mediated damage to the oral mucosa and microvasculature likely compromises the local immune barrier [14]. This immunocompromised state, potentially exacerbated by the transient hypoxia common in COVID-19, which can impair neutrophil function [1], would allow commensal or opportunistic oral bacteria to invade the underlying bone [16]. The ensuing infection triggers a robust inflammatory response within the confined medullary bone space, leading to ischemia, necrosis, and eventual sequestration, as was confirmed in our case [7,17].

The diagnostic challenge in this case underscores the complexity of COVID-19's oral manifestations; the initial soft-tissue biopsy correctly identified the PG, but it was only upon the subsequent presentation of exposed bone and sequestration that the underlying osteomyelitis was definitively diagnosed and confirmed histologically. The patient's severe and persistent facial paresthesia adds another layer of complexity. While it can be a known complication of both osteomyelitis and surgical trauma [18], the intensity and bilateral nature in this case suggest a possible neuropathic component linked to COVID-19. The virus's associated endotheliopathy and hypercoagulability can lead to microthrombi and ischemic injury in the vasa nervorum, the small vessels supplying peripheral nerves [12]. The successful management of this symptom with Gabapentin further supports a neuropathic etiology.

This case highlights critical clinical implications. Firstly, oral complaints in COVID-19 patients should not be dismissed as minor and may signal severe underlying pathologies, such as osteomyelitis. Secondly, the initial use of corticosteroids for the recurrent pyogenic granuloma, while standard practice, may have inadvertently suppressed the local immune response, potentially facilitating the progression of the latent osteomyelitis [19]. This illustrates the delicate therapeutic balance required in managing post-COVID inflammatory conditions. Finally, the protracted and relapsing course of this case emphasizes the necessity for long-term, multidisciplinary follow-up for patients who develop significant oral complications from SARS-CoV-2 infection.

Conclusion

In conclusion, this report demonstrates that SARS-CoV-2 infection can create a "perfect storm" of vascular injury, immune dysregulation, and hyperinflammation in the jaw, predisposing patients to the simultaneous development of reactive soft-tissue lesions and destructive bony infections. Clinicians should maintain a high index of suspicion for such co-occurring pathologies to ensure timely and comprehensive management.

Conflict of Interest

There is no conflict of interest to declare.

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