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COVID-Associated Avascular Necrosis of the Maxilla —A Rare, New Side Effect of COVID-19

[1]Divya Sharma, Assistant Professor, Department of Oral and Maxillofacial Surgery, faculty of Dental Sciences, Manav Rachna Dental College, MRIIRS, Faridabad.

[2]Yajas Kumar, Assistant Professor, Department of Oral and Maxillofacial Surgery, faculty of Dental Sciences, Manav Rachna Dental College, MRIIRS, Faridabad

[3]Tabish Bashir, Department of Orthodontics and Dentofacial Orthopaedics, Career Post Graduate Institute of Dental Sciences and Hospital, Lucknow

[4]Priya Sharma, Department of Oral and Maxillofacial Surgery, Rishiraj College of Dental Sciences and Research Centre Bhopal

[5]Resham Irshad, Department of Orthodontics and Dentofacial Orthopaedics, Institute of Dental sciences, Bareilly

Corresponding Author: Divya Sharma, Assistant Professor, Department of Oral and Maxillofacial Surgery, faculty of Dental Sciences, Manav Rachna Dental College, MRIIRS, Faridabad.

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Type of Publication: Case Report

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Abstract

Purpose: The purpose of this paper is to highlight a fascinating, unusual case of a patient who had maxillary avascular necrosis brought on by COVID-19 infection.

Methods and Results: After the surgical management for this patient was finished, our team reviewed their medical record in the past. The patient, who was a male 72-year-old, was admitted to the Career Post Graduate Institute of Dental Sciences and Hospital, Lucknow, for surgical treatment of an infarcted maxilla that had resulted from an infection with COVID-19. Using PubMed, a review of the literature was done. The review and discussion of 25 articles.

Conclusions: Patients who have COVID-19 infection enter a hypercoagulable state, which can result in a number of problems in the head and neck area. In our case report, we describe a patient who experienced avascular necrosis of the maxilla as a result of COVID-19 infection. Due to the high prevalence of possible systemic consequences, thromboembolic prophylaxis is essential for COVID-19 patients.

Keywords: Covid-19, Necrosis, Maxilla, Infection

Introduction

Although COVID-19 infection is commonly thought of as a pulmonary disease, complications have been seen in a variety of other organs, including the heart, kidneys,

head, and neck area. The emergence of a hypercoagulable state, which can result in thromboemboli and, ultimately, cerebral and myocardial infarctions, is a systemic side effect of COVID-19. There haven't been any reports of visible occlusions on computed tomography angiography (CTA) in the head and neck area.

The purpose of this article is to provide a rare instance of bilateral maxillary necrosis due to COVID-19 infection-related blockage of the left internal maxillary artery. A CTA showing blockage of the left internal maxillary artery and branches of the pterygoid plexus verified this condition. A review of the patient's surgical care and the pathophysiology based on the literature is done. Understanding the pathophysiology of a hypercoagulable state caused by COVID-19 infection, how it affects the head and neck region, and how to prevent this complication in the perioperative phase is crucial for the head and neck surgeon.

Case Report

A 72-year-old man with a past medical history significant for persistent atrial fibrillation (status after ablation in 2009 and on daily 5 mg of warfarin), diabetes mellitus type II, coronary artery disease, and hypertension presented to an outpatient clinic in January 2021 with his main complaints of headaches, blurry vision in his left eye, loss of taste, and an upper respiratory infection. His international normalized ratio at presentation was 4.5. Due to pneumonia brought on by COVID, the patient's condition swiftly worsened, and he spent almost a month in the intensive care unit (ICU). Warfarin was kept at a therapeutic dose as part of the venous thromboembolic (VTE) prophylaxis together with sequential compression devices. By using a biopsy, a head CT without contrast, a carotid artery duplex scan, and a bilateral lower extremity venous duplex, it was possible to rule out temporal arteritis, cerebrovascular infarction, bilateral internal carotid stenosis, and deep vein thrombosis. He also started to get exposed bone in his right and left maxilla while he was in the hospital. He was given 81 mg of aspirin daily and 5 mg of apixaban twice daily after making a full recovery. He reports daily compliance with medication for all prescriptions.

After being released from the hospital, he visited a nearby dental and maxillofacial surgeon in May 2021 with complaints of exposed maxillary bone. He was then forwarded to the Department of Oral and Maxillofacial Surgery at Career Post Graduate Institute of Dental Sciences and Hospital, Lucknow; he appeared at the clinic in September 2021. Prior to the referral, no medication was started; the patient blamed the delays in care to insurance and financial concerns.

A maxillofacial CT without contrast revealed osteolytic changes of the hard palate, supe rior alveolar ridge, left maxilla, zygoma, zygomatic arch, left ethmoid air cells (lamina papyracea), pterygoid plates, and pterygoid palatine fissure (Figs 2*A- D*). A neck CTA demonstrated an abrupt cutoff of a small branch of the left internal maxillary artery located adjacent to the medial aspect of the left mandible, representing occlusion (Fig 3). Additionally, the left pterygoid plexus was nonfilling, indicating further occlusion of these branches. An edentulous maxillary ridge was seen during a clinical examination, with exposed, infarcted bone on the labio- buccal surface, reaching from the left posterior maxilla anteriorly to the left canine region as well as from the keratinized gingiva mid-height of the alveolus to the depth of the vestibule. On the labial surface of the right maxilla, in the premolar area, there was also a single, 8-mm section of the infarcted bone (Fig. 1). The bone had a little pus

infection. The patient reported hypoesthesia in the left cranial nerve V-2's distribution; cranial nerve VII was generally intact on both sides.

The patient was brought to the operating room, where the maxilla was debrided (Figs. 4A,B). Few Pseudomonas aeruginosa, few Klebsiella pneumoniae (with resistance to ampicillin), few Candida tropicalis, and few Enterococcus species were found in the cultures that were acquired. Osteonecrosis of the maxilla was confirmed by a biopsy of the material. Achieved was primary closure (Fig. 5). Amoxicillin/clavulanic acid 875 mg/125 mg twice daily were prescribed for the patient for 10 days. The patient continued to take 5 mg of apixaban every 12 hours as prescribed. After a surgical procedure, the patient is still being monitored by the Career Post Graduate Institute of Dental Sciences and Hospital, Lucknow surgical team.

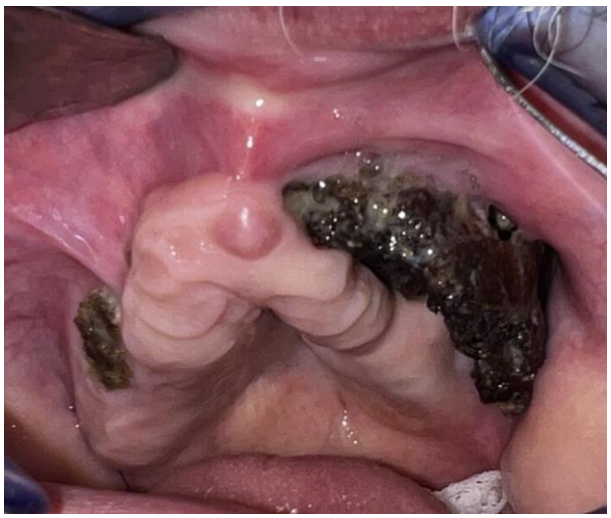

Figure 1: Clinical photo from the patient's initial presentation OMFS clinic. There is necrotic, exposed bone on the labio-buccal surfaces of the left and right maxillary quadrants with suppuration.

Discussion

In the maxillofacial region, COVID-19 problems are starting to emerge. It has been noticed that COVID-19 patients are more likely to get thrombo-emboli and fungal infections, such as mucormycosis, as a result of their treatment. These consequences have a multifactorial etiology that includes adverse drug reactions, endothelial cell destruction, patient immobility, and other aspects of critical disease.1,2 Both of these complications were of concern to this patient.

The CTA and the exam outcomes that excluded giant cell temporal arteritis, deep vein thrombosis, and cerebrovasular accident support the authors' theory that the patient had numerous thromboemboli. The patient may experience vision problems and maxillary necrosis as a result of the thromboemboli. Numerous biopsies and cultures were carried out on the patient with a high suspicion for invasive fungal species; no fungal contamination or colonisation was discovered. Another possibility is that the anticoagulation that was provided was insufficient to treat the hypercoagulable condition brought on by COVID-19 infection.

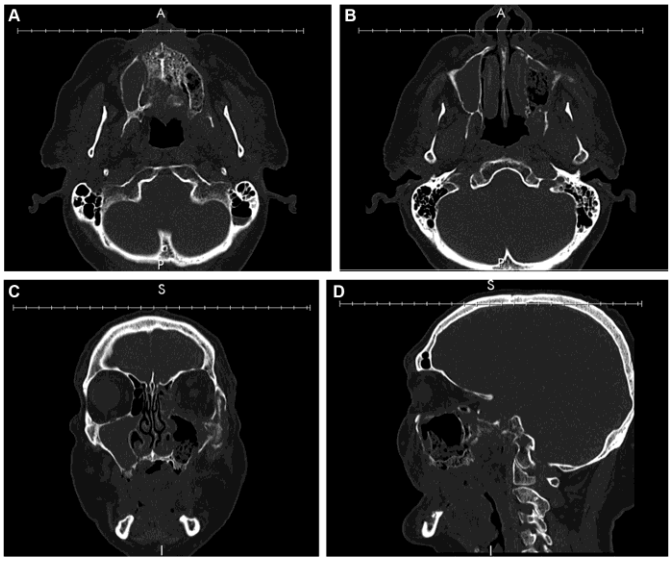

Figure 2: A, Axial view. There are osteolytic changes seen on the hard palate, superior alveolar ridge, and left maxilla. B, Axial view. There are osteolytic changes

seen on the left maxilla, zygoma, and left ethmoid air cells. C, Coronal view. There are osteolytic changes seen on the hard palate, superior alveolar ridge, left maxilla, zygoma, zygomatic arch, and left ethmoid air cells. D, Sagittal view. There are osteolytic changes seen on the hard palate, superior alveolar ridge, left maxilla, and left ethmoid air cells.

Just a few cases of necrosis or jaw osteomyelitis following COVID-19 infection have been documented. In a series of 4 individuals who had COVID-19, one case series described osteomyelitis or maxillary necrosis.[3] In a different case report, maxillary necrosis caused by COVID-19 infection was described in a single patient.4 Other oral cavity signs have also been noted, such as blisters, desquamative gingivitis, petechiae, reddish spots on the palate, and oral ulcers.3 The internal maxillary artery has not been thrombosed in any cases that have been recorded. A hypercoagulable state may be caused by COVID-19 infection, increasing the risk of both arterial and venous thrombosis, according to accumulating research. In the literature, a high prevalence of thrombotic problems has been reported. Numerous reports of arterial thrombosis have been made. In one sizable investigation, 16% of individuals with COVID-19 who were hospitalised experienced thrombotic events. Ischemic stroke (1,6%), systemic thromboembolism (1%), and myocardial infarction (8.9%) were all arterial events.5 Currently, our understanding of the pathogenesis of this prothrombotic state is insufficient. Different mechanisms have been proposed, and a wide range of factors could be involved. This syndrome is now known as "COVID-associated coagulopathy" as a result.[6]

Thrombosis and coronary artery disease associated with COVID-19 are influenced by endothelial damage and malfunction (endotheliopathy). After COVID-19 infection, endothelial inflammation has been seen in autopsy studies. Von Willebrand factor (vWF), angiopoietin 2, P-selectin, and thrombomodulin levels have significantly increased, and vWF and thrombomodulin have been shown to be significantly correlated with mortality.8–12 COVID-19 may either directly or indirectly (through the ACE2 receptor) cause prothrombotic/proinflammatory substances to be exocytosed from endothelial cells, which causes microvascular thrombosis and inflammation.[9] In COVID-19, the complement system has been hyperactivated, and elevated circulating markers are associated with severe illness. Autopsies have revealed thrombotic microvas-cular damage with significant complement protein deposition.[13] The complement system can be directly activated by COVID-19 or indirectly by endotheliopathy, which also stimulates the complement system.[14] Studies of the Middle East respiratory syndrome coronavirus in C3-deficient mice or those with blockages in the distal complement system support this by demonstrating a reduced incidence of systemic pathology.[15,16] Endothelial dysfunction and damage may be facilitated by terminal complement cascade components through a variety of mechanisms.[14] Currently, completion system targets are being researched.

Laboratory testing has supported COVID-19's hypercoagulable condition. The critically unwell have been found to have higher D-dimer, fibrinogen, factor VIII, and vWF levels while having lower anti-thrombin levels.[8,17] Additionally, the results of thromboelastography are in line with a hypercoagulable state due to shorter reaction and clot formation times,

higher maximum amplitudes, and reduced clot lysis at 30 minutes (LY30).[8] COVID-19 appears to create a profoundly proinflammatory condition, amplifying prothrombotic factors, despite the fact that critical illness is known to cause a hypercoagulable state.[18] Additionally, patients with COVID-19 pneumonia who are critically ill have plasma hyperviscosity.[19]

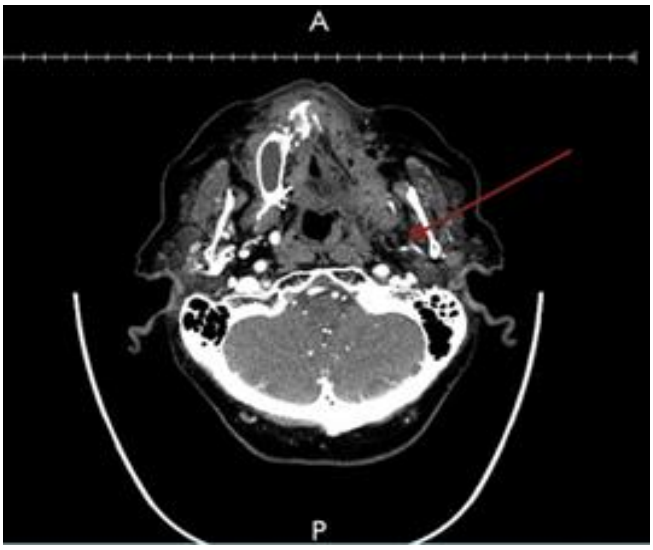

Figure 3: Neck CTA demonstrating occlusion of a branch of the left maxillary artery. Abbreviation: CTA, computed tomography angiography

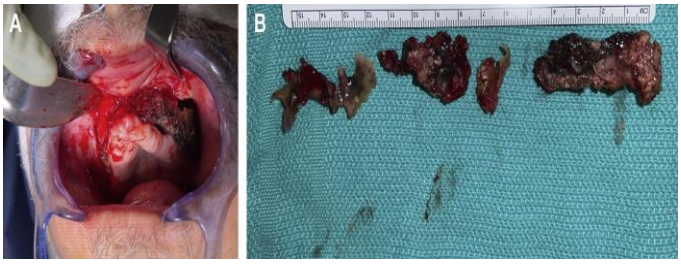

Figure 4: A, Surgical debridement of the right and left maxillary necrotic bone. B, Necrotic bone debrided from the right and left maxilla.

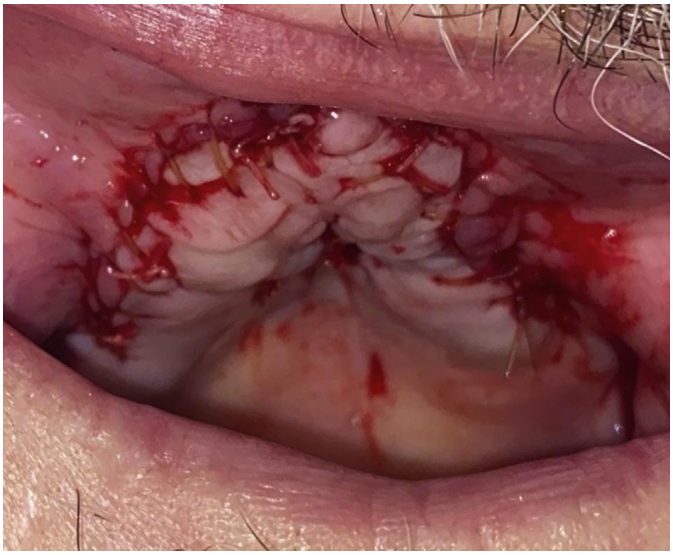

Figure 5: Primary closure of the maxilla after debridement

Bilateral maxillary necrosis may result from thromboembolism of the internal maxillary artery related to a hypercoagulable state brought on by COVID-19 infection. Even with preventive dosages of anticoagulant treatment, COVID-19 infection and immobility can enhance the odds of hospitalised patients developing complications. This complication, which was confirmed by CTA and a maxilla biopsy, is described in this article. Giant cell arteritis and other probable ophthalmologic consequences (necrosis) were ruled out. Multiple factors, including an increase in D-dimer, fibrinogen, factor VIII, and WF and a decrease in antithrombin, contribute to the pathophysiology of COVID-induced hypercoagulability. Other cellular modifications, such as those to enzymatic proteins and oxidants, neutrophil extracellular traps, and may also be involved. The critical patient's immobility and these alterations are compounded. The patient should be addressed surgically if necrosis of the maxilla or any other bone structure develops. A biopsy and cultures ought to be performed to rule out superimposed infections and confirm the diagnosis. To avoid potential complications, the managing team and surgeon should review the NIH recommendations for VTE prophylaxis.

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