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Case Report
Clinical Pathology

Avascular necrosis of the midface secondary to disseminated intravascular coagulation

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Abstract. To the authors' knowledge, avascular necrosis of the midface secondary to disseminated intravascular coagulation has yet to be described following a hypoxic syncopal episode secondary to 'heat stroke'. A slow, progressive loss of anterior maxillary bone and the collapse of the nasal dorsum in a healthy young man with no other known medical co-morbidities led to the diagnosis. Following debridement, a staged reconstruction of the maxilla–nasal complex was successfully performed.

Key words: avascular necrosis; midface; disseminated intravascular coagulation.

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Avascular necrosis of the midface is a rare condition due to the rich vascular supply from bilateral branches of the external carotid artery.[1] On mobilization of the maxilla during the Le Fort I down-fracture, the hard and soft tissues remain well-perfused despite detachment of circumvestibular vessels and, on occasion, loss of one or both of the contributions of the greater palatine arteries. The reliable perfusion of the maxilla during this procedure is well documented.[2] The more commonly reported causes of osteonecrosis of the midface include orthognathic surgery, trauma, infection, bisphosphonates, and radiation.[2–4]

Disseminated intravascular coagulation (DIC) is also a rare condition that is characterized by the widespread pathological activation of the coagulation cascade, and can lead to ischaemic necrosis of tissues and, more seriously, 'end-organ' failure. DIC is commonly associated with major trauma, head injuries, infection, and obstetric complications.[5]

A unique case of avascular necrosis of the midface as a complication of DIC is reported. This followed an episode of unconsciousness and severe dehydration as a result of 'heat stroke'. The clinical presentation of this rare event and the approach to reconstruction of the patient's maxillary and nasal complex are described.

Case report

A 29-year-old male bricklayer, with no known co-morbidities, suffered an episode of 'heat stroke' while returning home on foot from a building site. He was found lying unconscious on the roadside with twitching limbs, and an initial Glasgow Coma Scale score of 3/15 was recorded. After transfer to a nearby tertiary hospital, a clinical, biochemical, and radiographic assessment indicated that he had suffered an anoxic brain injury with severe cognitive deficits. He was immediately intubated, ventilated, and transferred to the intensive care unit (ICU) and a tracheostomy was performed to protect the airway.

Subsequently, he developed multi-organ failure and required renal dialysis. Further biochemical studies confirmed a diagnosis of DIC with a poor prognosis. However, after 2 months, his condition improved and he was transferred to a rehabilitation centre

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in his home state to manage his neurological deficits, which included dysarthria, impulsive behaviour, poor memory, and a gait disturbance. Brain magnetic resonance imaging (MRI) demonstrated marked bi-frontal, cerebral, and cerebellar atrophy.

After 6 months of rehabilitation therapy, his attending physicians noted that his nasal dorsum appeared to be slowly collapsing and that his maxillary anterior teeth were 'intruding'. Presuming an infective cause, he was referred to the maxillofacial surgery unit for assessment. Clinically, a saddle nose deformity was evident with obstruction of his nasal airways, greater on the left side (Fig. 1A). His maxillary anterior teeth, previously in occlusion, were noted to be mobile and had displaced superiorly (Fig. 1B). A 3D computed tomography (CT) scan demonstrated lysis of the anterior portion of the maxilla (Fig. 1C) causing collapse of the cartilaginous nasal septum. It was concluded that a process of bony necrosis was occurring, and although he remained afebrile and asymptomatic, subsequent blood cultures confirmed the presence of *Candida albicans, Aspergillus fumigatus aspergillosis* and beta-haemolytic *Staphylococcus aureus*, but without any markers of an acute infection. An area of occipital alopecia, inconsistent with male pattern baldness, was also observed and presumed to be secondary to scalp pressure and circulatory compromise while the patient was unconscious in the ICU. A diagnosis of avascular necrosis of the midface secondary to DIC was made.

A treatment plan was then devised that commenced with extraction of the compromised maxillary teeth and debridement of the necrotic maxilla and associated structures. This was to be followed by a staged reconstruction of the maxilla–nasal complex.

In the first procedure, under antibiotic cover, all maxillary teeth from second premolar to second premolar were extracted together with debridement of the associated non-vital bone. No purulence was present and an attempt at closure of a large anterior oronasal fistula was undertaken. The wound broke down in the early postoperative phase and was obturated with a temporary maxillary prosthesis.

Six months later, the maxillary defect was reconstructed with a deep circumflex iliac artery bone flap. The donor fragment was osteotomized into three segments to shape a new maxillary complex, and access to the recipient facial vessels was achieved via an incision parallel to the right nasolabial fold. The pedicle was orientated posteriorly on the graft to facilitate the anastomoses.

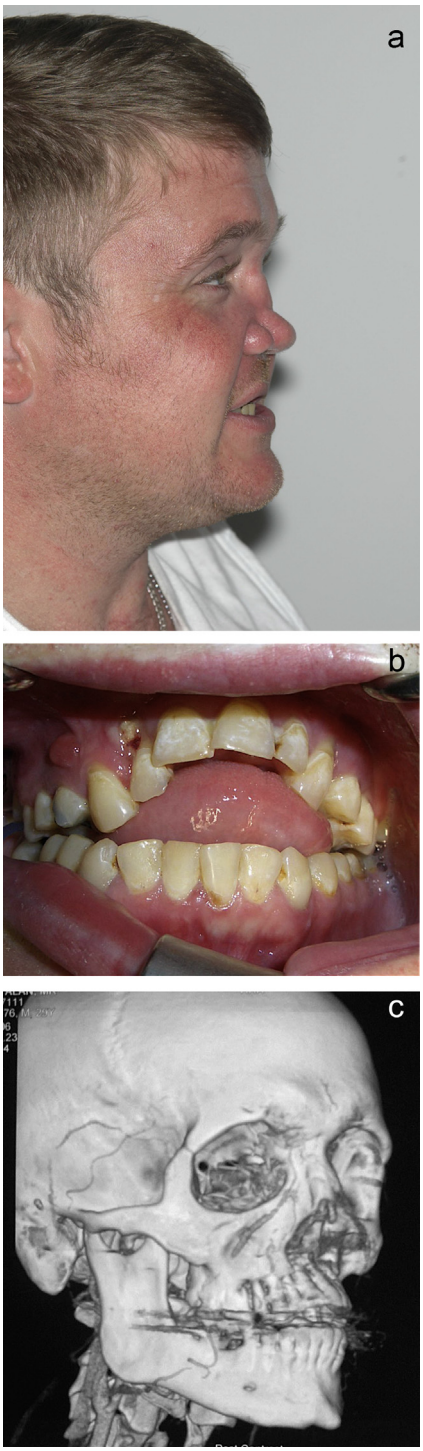

Fig. 1. (A) Lateral facial view demonstrating a 'saddle nose' deformity. (B) Anterior open bite due to osteonecrosis of the anterior maxilla. (C) Three-dimensional CT scan showing bony lysis around the anterior teeth.

After a further 5 months, a costochondral graft was harvested from the seventh rib to reconstruct the nasal deformity. The graft was shaped to restore the dorsal contours by creating a peg on the cephalic end to insert into a bony tunnel prepared in the naso-frontal region midline as far as the antero-inferior wall of the frontal sinus. This enabled the graft to be cantilevered to elevate the nasal tip. The caudal end was contoured to enable the collapsed lower lateral cartilages to be raised by suturing to the costal cartilage. A third procedure was performed 2 months later