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CASE REPORT
Cosmetic

Case Report: Peri-oral Vascular Compromise Due to Calcium Hydroxyapatite

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Summary: The increasing popularity of nonsurgical cosmetic treatments with the use of hyaluronic acid and calcium hydroxyapatite shows how effective and versatile these treatments can be. However, this is very likely to increase the number of complications that occur. Treatment protocols exist for vascular complications due to injections with hyaluronic acids. However, protocols for such events due to injections with calcium hydroxylapatite are less defined. The author suggests an exemplified protocol for the basis of managing such life-changing complications of impending necrosis after injections with calcium hydroxyapatite. *(Plast Reconstr Surg Glob Open 2022;10:e4193; doi: 10.1097/GOX.0000000000004193; Published online 18 May 2022.)*

A 23-year-old woman presented for a cosmetic consultation regarding her unbalanced facial profile. Clinical examination revealed she had class II malocclusion, which was further exaggerated after her recent lip augmentation. Both surgical and nonsurgical options were discussed. She opted for nonsurgical intervention. Injections with hyaluronic acid and calcium hydroxyapatite were reviewed, and it was decided by an experienced injector to proceed with the latter. A total of 1.5 ml (CAHA, Radiesse with lidocaine, Merz Aesthetics) was injected across four injections (two in midline, and one either side of midline) using a serial puncture technique and a 27G needle in the supraperiosteal plane. There were no signs of vascular compromise at the time of treatment (ie, blanching, pain, or skin discoloration). However, 12 hours later, the patient complained of severe pain, paraesthesia, and paleness along the distribution of the ascending mental and submental arteries (Fig. 1). In addition, she also complained of pain during swallowing. Immediate face-to-face assessment revealed extensive livedo reticularis with capillary refill time delayed to 6 seconds in the mid-lower lip.

Aspirin 300 mg was given immediately. Using a 25G, 50-cm cannula with an entry point away from site of livedo reticularis, 600 units of hyaluronidase, and 5-ml saline were injected into the superficial subcutaneous plane. The hyaluronidase was repeated every 45 minutes for a total of four rounds until there was clinical evidence of reperfusion. A course of oral steroids (prednisolone) was initiated at 60 mg, with a tapering regimen to reduce the risks of steroid dependence and steroid withdrawal symptoms. In an effort to dilate the arterial blood supply, sildenafil was added at a dose of 50 mg per day for 3 days. In addition, low-molecular-weight heparin was administered subcutaneously for 7 days given the extensive nature of complication. For prophylactic infection control, the patient was subsequently placed on doxycycline 200 mg initially and then a maintenance dose of 100 mg daily, and acyclovir 200 mg was given four times a day for 7 days. The patient was reviewed regularly and received two sessions of hyperbaric oxygen. After 7 days, there were signs of dried debris/superficial necrotic tissue (Fig. 2). The wound was conservatively debrided to promote wound healing, and appropriate wound care advice was given to the patient (Fig. 3).

DISCUSSION

Vascular compromise is a function of embolization or external compression of the vasculature. Hyaluronidase is an enzyme which can be used to dissolve hyaluronic acid based dermal fillers. However, when the material injected in calcium hydroxyapatite, poly-L-lactic acid, then very little can be mitigated. Although vascular complications can be avoided with sound anatomical knowledge, clinicians must also learn to recognize the

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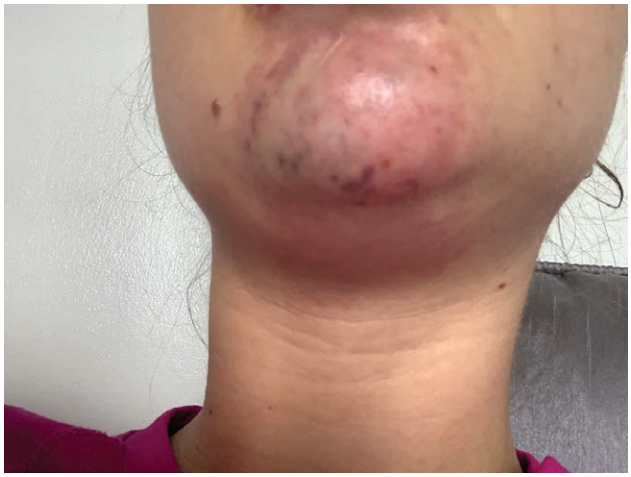

Fig. 1. Patient photograph taken 12 hours after filler injection. Skin discoloration and extensive demarcation of the ischemic area are visible in the chin and neck.

presenting features of such complications.[1] The chin region is considered a safer area to inject with dermal fillers, as very few cases of vascular complications have been reported in the literature. However, the author believes the literature is too optimistic in the reported prevalence of cases involving impending necrosis following dermal filler injections, although it is a statistically rare complication.[2]

The typical initial presentation of a vascular compromised scenario is pain out of proportion to what can be expected, although delayed presentations are not uncommon. This may be due to the addition of lidocaine within the filler or due to the filler being trapped at a bifurcation point, which is then dislodged distally.[3,4] Blanching and pallor also tend to present immediately followed by livedo reticularis (mottled skin), which may occur within minutes or hours. Natural sequelae over 1–7 days include blisters, bullae, and pustules with eventual necrosis. Sometimes these features can be difficult to diagnose, given they may resemble herpetic outbreaks.[5]

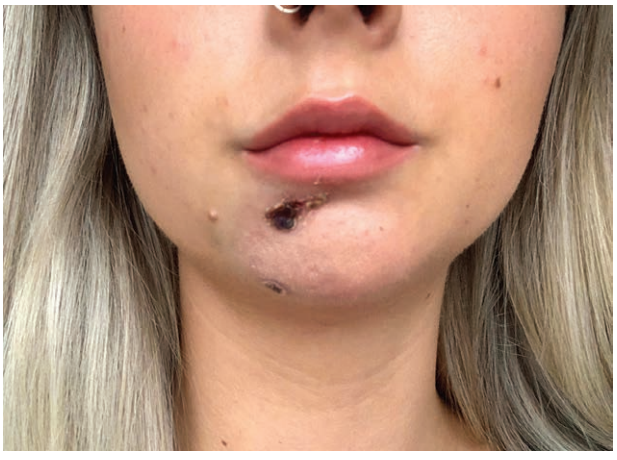

Fig. 2. Seven days post-hyaluronidase treatment. Skin changes (including pustules and crusting visible) showing signs of small necrotic areas of skin.

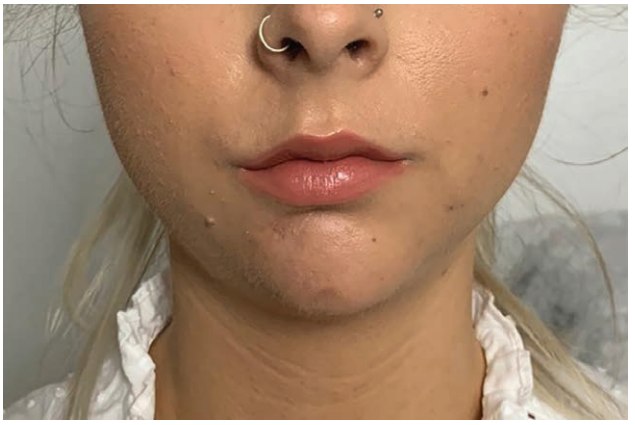

Fig. 3. Three weeks post-hyaluronidase treatment with good wound care and regular follow-ups. Superficial debridement of necrotic tissue aided the healing process.

The mainstay of management is rapid reperfusion of the affected areas. (**See table, Supplemental Digital Content 1,** which shows the key strategies for managing vascular complications due to CAHA. **http://links.lww.com/PRSGO/B973**.) Warm compress and vigorous massage promote vasodilation and may propagate the filler distally into smaller arterioles.[6] Hyaluronidase should be used despite the filler being resilient to the enzyme. The benefits of hyaluronidase include its antiinflammatory properties and hydrolyzing endogenous hyaluronic acid, which in turn will theoretically reduce localized pressure.[6,7] The author is a huge advocate for using a cannula to administer hyaluronidase rather than using the conventional method of "flooding" the area with a needle, as this may mask crucial visual signs with the risk of significant bruising. This should be repeated every 45–60 minutes until reperfusion is improving. High-dose aspirin (or clopidogrel) should also be part of the initial management plan because it will limit platelet aggregation, thus reducing the risk of embolus formation.[8] This should be followed by a maintenance dose of 75 mg for 5–7 days. Alternatively, subcutaneous injection with low-molecular-weight heparin can be used.[6] To promote vasodilation and increase blood flow, systemic drugs such as sildenafil and tadalafil can be used.[9] Although some authors are advocates for using topical nitroglycerin, its benefit remains controversial due to potential shunting of blood. A tapering course of oral corticosteroids may be considered when there is tissue edema and inflammation. The presence of necrotic tissue in a wound will inhibit optimum wound healing and should be carefully superficially debrided in a conservative manner. In vitro analysis has shown limited reversibility of vascular compromiseDwith calcium hydroxyapatite, although larger studies are needed to provide further insight.[10] Due to the mainstay of dermal filler injections being of hyaluronic acid composition, it is not routine to store sodium thiosulfate. This has now become an integral part of our emergency kit at our clinic, where we manage various types of aesthetic complications. (**See table, Supplemental Digital Content 1,** which shows the key strategies for managing vascular complications due to CAHA, **http://links.lww.com/PRSGO/B973**.)

The submental artery is the largest cervical branch of the facial artery, with an average diameter of 1.69 mm.[11] It ascends upward, running over the mylohyoid muscle, passes over the mandible, and terminates into superficial and deep branches. The presence of the submental artery and its communications with the inferior labial artery and mental artery could be the basis for a more extensive territory involvement during inadvertent vascular injection. The presenting feature of pain on swallowing is likely to be due to partial occlusion in the (superficial and deep) arterial branches of the submental artery, which supply the digastric, mylohyoid, and platysma muscles.

CONCLUSIONS

This case report describes the successful management of impending skin necrosis resulting from the submental artery after injections with CAHA filler. Prompt diagnosis and management of a vascular compromised situation is paramount to prevent undesirable cosmetic outcomes such as scars.

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