Severe Delayed Hemorrhage After Cosmetic Rhinoplasty in the Setting of Maxillary Vascular Malformation

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Abstract: Late epistaxis after rhinoplasty is a rare but potentially life-threatening occurrence. This case report concerns a 20-year-old woman who had severe epistaxis 5 and 10 days after a closed rhinoplasty with internal osteotomies and who ultimately required transcatheter arterial embolization for definitive control of the hemorrhagic source. Arterial hypervascularity, with signs of arteriovenous malformation, of the midface at the level of the piriform aperture and maxilla was seen on angiography.

Key Words: rhinoplasty, hemorrhage, epistaxis, osteotomies, angiography, transcatheter arterial embolization

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Immediate postoperative bleeding secondary to bony, mucosal, or soft tissue trauma is the most common complication of rhinoplasty.[1] Measures to help prevent or decrease postoperative epistaxis include the discontinuation of anticoagulant drugs; nasal injection with local anesthetic with epinephrine; the use of topical oxymetazoline, phenylephrine, or cocaine; and performance of surgery in reverse Trendelenburg position at a 15-degree angle.[1–3] Most often, immediate postoperative bleeding resolves with simple measures such as head elevation, gentle digital pressure, and application of topical vasocontrictive agents.[1,4] Significant hemorrhage beyond this point is rare.

However, the infrequent occurrence of epistaxis several days after rhinoplasty can be very difficult to manage and is a cause of significant concern to the surgeon and distress to the patient. We present the case of a 20-year-old female patient who had severe delayed epistaxis after a closed rhinoplasty, which was precipitated by an underlying bilateral maxillary vascular malformation.

CASE REPORT

A 20-year-old healthy woman underwent an elective closed rhinoplasty under general anesthesia to correct her dorsal hump deformity. She did not have symptoms of nasal obstruction and had no personal or family history of excessive bleeding or bruising.

Initially, the nose was meticulously infiltrated with 1% lidocaine with epinephrine. Specifically, the injection was carried out in the submucosal perichondrial plane of the septum, the columella, the entire length of the dorsum, and the subperichondrial plane of the lateral cartilage bilaterally. Cocaine-soaked pledges were then inserted in each nostril. After adequate vasoconstrictive time, the pledges were removed, and through an intercartilaginous incision, the dorsal nasal bone was rasped and the dorsal cartilaginous septum sharply reduced and contoured. Bilateral internal lateral osteotomies were carried out in a low-to-low fashion to close the open-roof deformity. The incisions were primarily closed with chromic sutures and an external splint was applied. The patient did well, showed no signs of hemorrhage, and was discharged to home after a brief routine stay in our recovery room.

Subsequently, the patient presented to our office with significant bilateral epistaxis on postoperative day 5. The splint was removed and the bleeding controlled after placement of bilateral Rhino Rockets (Shippert Medical Technologies Corporation, Centennial, CO). She was then admitted to the hospital for observation where she stayed for 3 days without evidence of rebleeding.

On postoperative day 10, the patient presented to the emergency department with another episode of uncontrollable severe epistaxis and was taken urgently to the operating room for exploration. Intraoperatively, no bleeding points were identified, the mucosal lining was intact, and the source of bleeding appeared to be posterior in the nasopharynx (right worse than the left). After the intranasal application of several topical hemostatic agents, the nostrils were packed with pledges, which temporized the hemorrhage, and the patient was transferred intubated to the surgical intensive care unit. She was hemodynamically stable and did not require blood transfusion.

The following morning, an interventional neuroradiologist proceeded with a craniofacial angiogram via a right transfemoral approach and embolization of the facial and sphenopalatine arteries bilaterally (Fig. 1). Moreover, there was angiographic evidence of bilateral hypervascularity and early shunting, without clear nidus, in the midface at the level of the frontal process of the maxilla and the piriform aperture (Fig. 2). The embolization was successful and the patient did not have further episodes of epistaxis. She was extubated the following

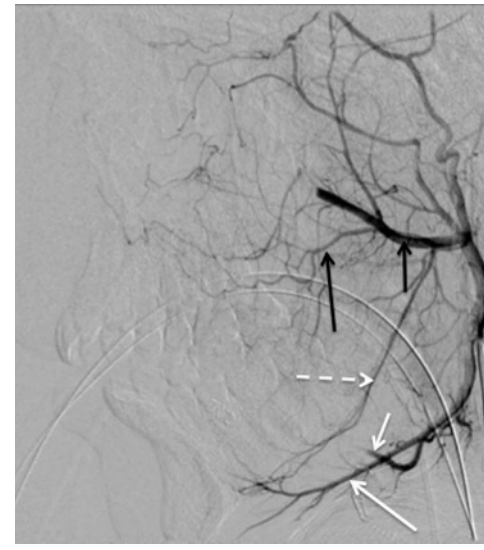

FIGURE 1. Catheter angiography of the right external carotid artery demonstrating embolization of the right facial artery, on lateral view. The facial artery stump (short white arrow) is seen adjacent to the lingual artery (long white arrow). The internal maxillary (short black arrow), transverse facial (long black arrow), and inferior alveolar (dashed white arrow) arteries are seen.

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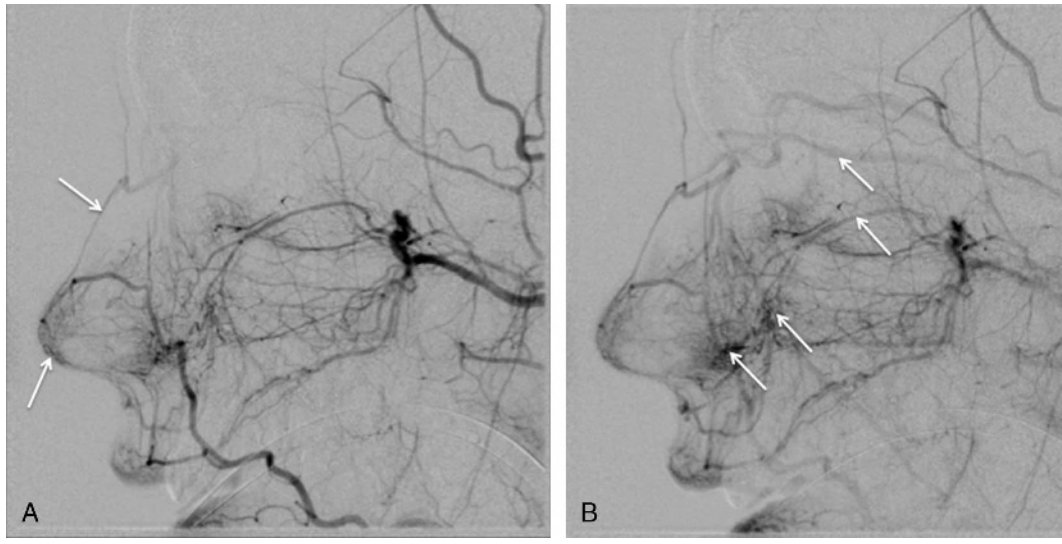

FIGURE 2. A, Selective angiogram showing early venous filling of the plexus of the nasal tip and the dorsum (arrows). B, Significant venous engorgement and early filling (arrows), indicative of abnormal shunting, are seen at the level of the left anterior maxilla and piriform aperture. These findings represent hypervascularity and are suggestive of arteriovenous malformation, corresponding to the areas of internal lateral osteotomies.

day and was discharged home 3 days later. On outpatient follow-up, the patient remained well and asymptomatic.

DISCUSSION

Severe bleeding after rhinoplasty occurs in less than 1% of patients.[4] Partial arterial injury secondary to the osteotomy with late formation of a false pseudoaneurysm has been reported.[5] To our knowledge, this is the first report of severe epistaxis in the background of vascular anomaly at the osteotomy sites.

The etiology of the delayed nature of the hemorrhage is not fully understood. We suspect that the postoperative edema and vasoconstrictive drugs promoted early hemostasis. In the next few days, as the edema subsided, the patient became more active and higher craniofacial intravascular pressures ensued, causing late epistaxis.

Significant epistaxis after maxillofacial surgery cannot be underestimated because it can be life threatening and warrants prompt evaluation of the patient. The first step is to transfer the patient to the hospital to assess the need for resuscitation and if indicated to establish a secure airway according to the advanced trauma life support protocol.[6,7] Next, anterior and posterior nasal packing, or balloon tamponade, should be attempted, although the success of these measures is unpredictable and the rates are as low as 29%.[6,7]

Intractable epistaxis secondary to vascular malformations is typically treated with transcatheter arterial embolization (TAE) with very high success rates.[8–10] However, postoperative epistaxis without knowledge of a vascular anomaly warrants operative exploration of the nasal cavity before TAE. An attempt to identify and control potential bleeding points intraoperatively is worthwhile and allows for better packing and the use of hemostatic agents. It has been shown that the risk of recurrent bleeding decreases significantly—approximately 4 times—once hemostasis using electrocoagulation is achieved.[11]

In cases where intraoperative exploration fails to provide definitive control of the postoperative hemorrhage, TAE is the procedure of choice. The arteries most commonly embolized after maxillofacial surgery are the internal maxillary and facial.[6] The success rate of TAE is as high as 96% and the complication rates are approximately 3%, which include skin and mucosal necrosis, cranial nerve injuries, and cerebrovascular accidents.[7] An attempt to perform surgical arterial ligation is not recommended in the setting of postoperative epistaxis unless absolutely necessary because it is technically challenging and less precise or efficacious than TAE.

CONCLUSIONS

Severe intractable posterior epistaxis after rhinoplasty can present late. Although very rare, an underlying maxillary vascular anomaly in the setting of osteotomies may exacerbate the hemorrhage and should be suspected. Prompt intervention is crucial, starting with ensuring a secure and definitive airway. The surgeon should have a low threshold proceeding with intraoperative exploration followed by TAE when operative treatment measures fail.

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