CASE REPORT

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PSEUDOANEURYSM OF THE PROXIMAL FACIAL ARTERY PRESENTING AS OROPHARYNGEAL HEMORRHAGE

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Abstract: *Background.* Penetrating trauma to the neck traversing zones II and III may cause considerable damage to soft tissues and neurovascular structures. Delayed sequelae of vascular injuries, such as pseudoaneurysm (PA), may present weeks to months after the initial injury.

Methods. We report an unusual case of a traumatic PA of the proximal facial artery that ruptured into the oropharynx.

Results. A 30-year-old man presented with oropharyngeal hemorrhage one month after a gunshot wound to the neck. Angiography revealed a PA of the proximal facial artery, which was treated with embolization. The arterial injury leading to the pseudoaneurysm had not been detected by arteriography at the time.

Conclusions. PAs are rare complications of penetrating neck trauma. To our knowledge, this is only the second report of PA involving the proximal facial artery, and the first of a facial PA rupture into the pharynx. © 2001 John Wiley & Sons, Inc. *Head Neck* **23:** 259–263, 2001.

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Pseudoaneurysms (PAs) are contained collections of turbulent blood flow that are in direct communication with an artery and are distinguished from true aneurysms, which involve all three layers of the vessel wall.[1,2] They typically arise from partial arterial wall injuries, which result in hematoma initially contained by the surrounding soft tissues. The ensuing thrombus becomes a PA as it liquefies and acquires a pulsatile arterial flow contained only by a fibrous capsule.[3–7] This can then expand under the strain of arterial pressure and can rupture.[7–9] PAs are delayed phenomena, often presenting days, months, or even years after the initial trauma.[2,6,7,8,10] The initial injury to the affected artery may be undiagnosed at the time of initial trauma, despite surgical exploration or arteriography.[11–14]

PAs of the extracranial arterial system (ECAS) are extremely uncommon.[15] Most of the few reported cases originated from the distal facial[4–8,16,17] or the superficial temporal arteries.[8,18] These vessels are most vulnerable due to their relatively superficial course along the mandible and zygoma, respectively.[8,19,20] Even more

rare are PAs of deeper vessels, such as the common carotid,[21,22] external carotid,[9,10,20,23] internal carotid,[1,11,22,24,25] internal maxillary,[19] lingual,[26] and proximal facial artery.[3]

We present an unusual case of a PA of the proximal facial artery presenting as oropharyngeal hemorrhage, one month after a through-and-through gunshot wound to the neck traversing zones II and III. Vessel injury in this region went unrecognized at the time of initial bilateral arteriography and endovascular treatment of extensive vascular injury on the contralateral side. To our knowledge, this is only the second report of a PA of the proximal facial artery in the world literature. The physician should be aware of this entity as a possible delayed sequela of penetrating neck injury.

CASE REPORT

A 30-year-old man presented to an outside hospital after a gunshot wound to the neck. The entrance wound was anterior to the left mastoid tip (zone III) and the exit wound was in the midportion of the right mandibular body (zone II). Subsequently, the patient developed an expanding hematoma in the right neck. Arteriography revealed active hemorrhage from the proximal right lingual and facial arteries (Fig. 1A). This was completely controlled with embolization in the angiography suite, using both polyvinyl alcohol particles and microcoils. Arteriography of the contralateral (left) ECAS (Fig. 1B) revealed no direct evidence of vessel injury, but flow of contrast was noted to be sluggish, consistent with spasm. A partial fracture was noted in the right mandibular body but did not require treatment. He subsequently underwent tracheotomy, and direct laryngoscopy revealed mucosal wounds on the superior left oropharyngeal wall, right tongue base, and inferior right oropharyngeal wall. No other procedures were performed. He was decannulated and was discharged 5 days after injury after uneventful recovery.

Five weeks later he awoke with massive hematemesis. He reported that the previous evening he had felt a vague tearing sensation on the

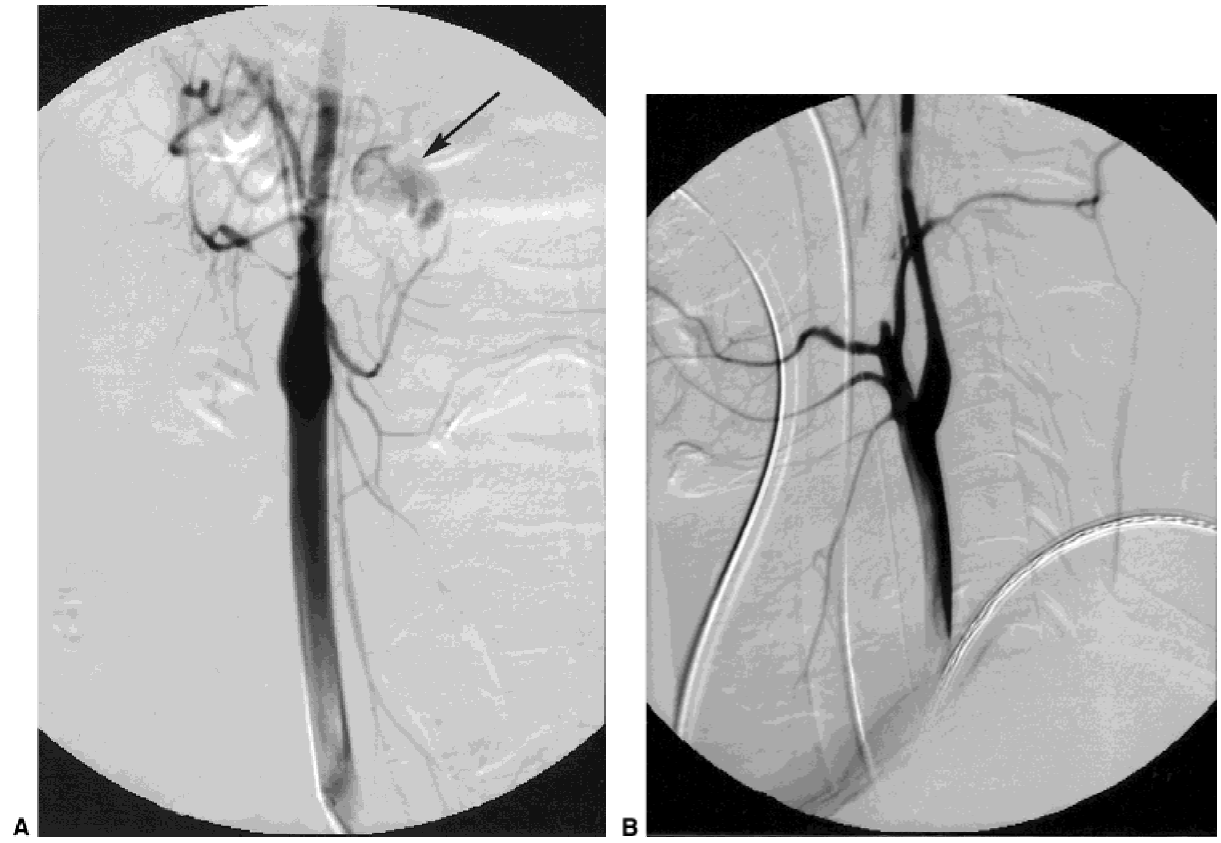

FIGURE 1. Carotid Arteriography on the day of injury. **(A),** right carotid, A-P view. Active hemorrhage from the proximal right lingual and facial arteries (arrow). **(B),** left carotid tree, lateral view. No extravasation of contrast is seen, but slow filling was noted in the lingual and facial arteries, consistent with spasm.

left side during a yawn. He went to a nearby hospital where he had 350 ml of additional hematemesis. He was subsequently transferred to our institution for further evaluation. Examination of the oral cavity and oropharynx revealed left posterior tonsillar pillar fullness. Flexible nasopharyngoscopy demonstrated a 1 ×2 cm pulsatile submucosal mass involving the left lateral pharyngeal wall, tongue base, and posterior tonsillar pillar, with fresh thrombus on its surface but no active bleeding. Arteriography showed a 1.5 ×2 cm PA arising from the proximal left facial artery at the expected origin of the ascending palatine artery (Fig. 2A). This was treated in the angiography suite with an endovascular trapping technique, consisting of microcoil emboli placed first distal to the aneurysm, then within the PA itself, and finally across the proximal facial artery. Flow ceased completely in the arterial segment and in the PA (Fig. 2B). Arteriography on the right revealed the previously placed coil emboli with no PAs or other abnormality (not shown). He had no further hemorrhage after embolization and was discharged home the following day. No further episodes of oral cavity or oropharyngeal bleeding were noted at follow-up one and six months later.

DISCUSSION

PAs are uncommon complications of arterial injury, and can result from blunt, penetrating, or surgical trauma.[2,14] They are generally felt to arise from a partial or tangential laceration of a feeding artery, which results in a local hematoma expanding into the surrounding tissues. The hematoma expands until the resistance of the surrounding soft tissues equals the systolic arterial pressure. A thrombus, which remains in continuity with the feeding vessel, is formed and then gradually liquefies. Organization of the thrombus results in a fibrous capsule lined by a pseudointimal layer. If continuity with the artery is reestablished, pulsatile, turbulent, and circular blood flow returns to the cavity, and the mass can expand once again under the strain of systolic blood pressure. This can lead to dissection into adjacent tissues and rupture of the capsule.[5–7,10,19] PAs typically present as expanding, pulsatile masses, often with an audible bruit heard almost exclusively during systole.[7] These must be distinguished from arteriovenous fistula (AVF), which can result from partial injury to both an artery and a nearby vein. AVFs present with a more continuous "machinery" bruit and often a palpable thrill.

PAs of the ECAS are rare. In a series of over 8000 aneurysms, McCollum and colleagues reported only 21 PAs of the ECAS, and of these, 19 occurred after carotid artery surgery.[15] The rarity of PAs is thought to be secondary to the small size of most of the vessels, which makes complete transection much more likely than partial lacera-

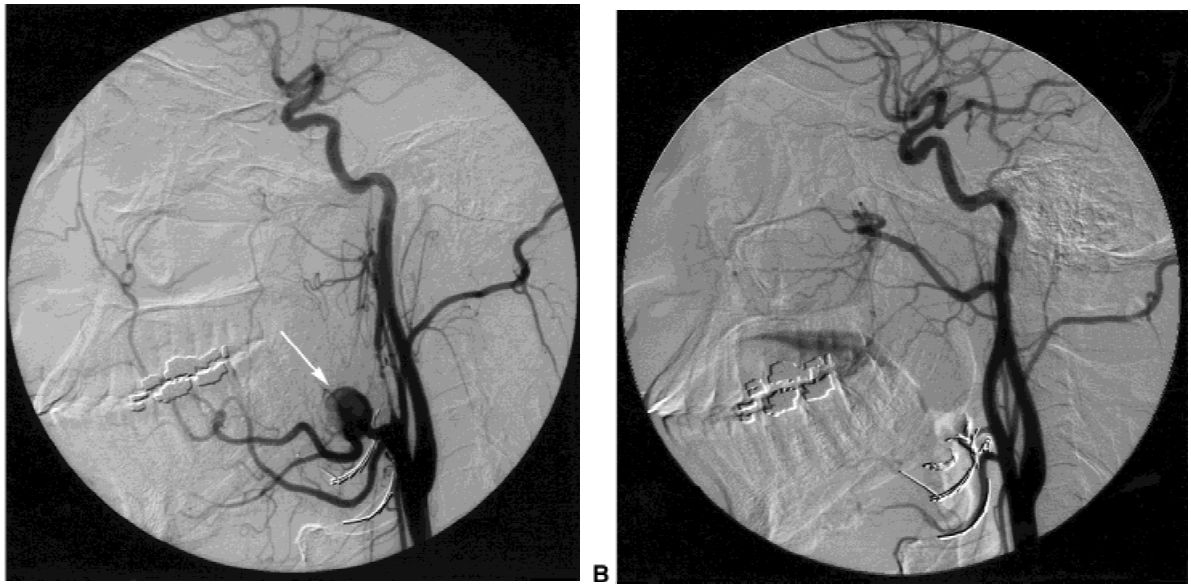

FIGURE 2. Carotid Arteriography 5 weeks after gunshot wound. **(A)**, left carotid, lateral view. A pseudoaneurysm is seen at the proximal left facial artery (arrow). **(B)**, left carotid tree immediately after embolization. No flow is seen either in the PA or the proximal facial artery.

tion. In addition, the deeper, larger vessels are protected by more soft tissue.[8,19,20] The vast majority of reported extracranial PAs arose from the superficial temporal, distal facial, and distal internal maxillary arteries, which is thought to be a result of their superficial position in the face and their crossing bony structures.[8,16,19,20,27,28] Although there are several reports of facial artery PA, nearly all involved the juxtamandibular segment or the distal branches in the face. Batten[3] reported a facial artery PA in the floor of mouth after alveoloplasty, but to our knowledge there are no reports of PA more proximally.

Our patient developed a PA at a deep location along the facial artery, close to its origin, and it presented as delayed rupture and hemorrhage into the oropharynx. The site of hemorrhage, at the lateral pharyngeal wall and tongue base, correlates with the position of the proximal facial artery and the patient's PA. Moreover, mucosal injury in the oropharynx had been noted at laryngoscopy at the time of his initial injury, and this area may have represented a weak point in the capsule of the developing PA over the ensuing weeks. Oropharyngeal hemorrhage is a distinctly rare presentation of PA of the head and neck. A review of the world literature reveals only three other cases reported; two from PAs of the internal carotid[1,29] and one from external carotid.[9] In contrast, the vast majority of PAs present as masses, which are often pulsatile, and often unstable in size.

Treatment of PAs is generally considered mandatory because of their unstable wall and tendency toward spontaneous expansion and rupture.[7] Historically, treatment has been surgical, with isolation and ligation of vessels supplying the PA, with or without excision of the mass. More recently, however, advances in interventional radiology have allowed successful treatment of the PA using endovascular embolization, with excellent results.[3,19,30] These techniques are particularly useful for aneurysms for which surgical accessibility is difficult.[9,19,23] Moreover, at many larger centers, endovascular treatment is available in the angiography suite immediately upon diagnosis.

At the time of his initial injury, our patient probably sustained a small laceration of the left facial artery that went undetected in his initial arteriogram at the outside hospital, perhaps because of the spasm noted in the left external carotid tree. Thus, our patient's lesion likely represents a "missed" arterial injury, despite the appropriate use of arteriography. An argument could be made that immediate neck exploration in our patient would have detected a vessel wall defect and prevented the nearly catastrophic events 5 weeks later. Historically, treatment algorithms for penetrating injuries to the neck have incorporated neck exploration to assess large vessel injury,[14] but as angiographic imaging and treatment methods have advanced this concept has become more controversial.[11,13,14,31] Patients with normal initial arteriograms have a less than 2% incidence of missed injuries.[32] Despite this low false negative rate, further workup despite negative arteriogram may be indicated in certain clinical situations. Cogbill[11] presented a case of delayed carotid pseudoaneurysm one week after a gunshot wound and an initially negative arteriogram. He proposed that delayed, repeat arteriography should be considered in cases of penetrating trauma near major vessels in an effort to detect "missed" injuries. It is unclear whether this would have helped in our patient, since the precise time his vasospasm resolved and flow restored to his facial artery and PA is unknown.

CONCLUSION

PAs are rare entities that may result from penetrating injury to the neck. PAs usually involve the distal aspects of the ECAS and present as expansile masses. We report a case of a man who sustained a penetrating neck injury resulting in a PA of the proximal facial artery, which presented as oropharyngeal hemorrhage. The PA was completely controlled by arterial embolization. The physician should be aware of the delayed sequela of PAs. Evaluation of unexplained hemorrhage and/or an expanding neck mass should always include a complete history, including all prior neck trauma even months or years earlier.

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