Epiglottic hematoma after carotid endarterectomy with cervical block

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ABSTRACT

There are few trials assessing the risks and benefits of performing a cervical plexus block (CPB) in urgent carotid endarterectomies (CEA). We describe a case of a patient who underwent urgent CEA under CPB and suffered a complication of postoperative epiglottic hematoma. There were clinical findings that helped to distinguish the hematoma from other, more common postoperative complications. The mainstay of treatment was steroids and observation. Epiglottic hematomas after cervical blocks for CEAs are rare but potentially lethal complications. More research is needed investigating complications related to CPBs performed for CEAs. (J Vasc Surg Cases and Innovative Techniques 2020;6:528-30.)

Keywords: Epiglottic hematoma; Cervical plexus block; Carotid endarterectomy

The patient has consented to the case report and images being published and has been provided a full copy of the manuscript. Carotid endarterectomy (CEA) greatly decreases the risk of stroke in patients with symptomatic moderate to severe carotid artery disease.[1] With numerous medical comorbidities present in afflicted patients,[2] it is important to evaluate methods of anesthesia during CEA and the associated potential complications. This factor is particularly important in high-risk patients who require an urgent CEA where decreased total operative time and avoidance of general anesthesia offsets risks of surgery.[3] There are dew trials assessing the risks of performing a cervical plexus block (CPB) in urgent settings, and although a subset of complications have been described, they have yet to be analyzed in detail.[4] Although the benefits of performing CEA under regional anesthesia have been noted, including monitoring neurologic function during cross-clamping,[5,6] lower shunting requirements,[7] and shorter hospital stay,[8] data suggest there is no difference in outcomes of stroke, myocardial infarction, or death.[9] It is necessary to recognize potentially life-threatening complications before accepting favor of this technique.[7] We describe a case of a patient who underwent urgent CEA under CBP and suffered a rare complication of postoperative epiglottic hematoma.

CASE REPORT

A 65-year-old man presented with a 2-week history of intermittent episodes of right-sided weakness and numbness as well as several episodes of amaurosis fugax on the left side. His last attack caused word-finding difficulty and confusion for 30 seconds before resolving. He denied any dizziness, fainting, other neurologic deficits, or difficulty swallowing liquids or solids. His medical history included diabetes mellitus, hypertension, hyperlipidemia, and coronary artery disease status post triple coronary artery bypass grafting 3 years prior. His medications included metformin, metoprolol, amlodipine, ramipril, and aspirin.

On presentation, his vitals, physical examination, and laboratory tests were within normal limits. A computed tomography angiogram of the head and neck was obtained and revealed bilateral carotid stenosis; the right proximal internal carotid artery (ICA) had 90% stenosis, and the left proximal ICA had 80% to 90%stenosis (Fig 1). Magnetic resonance imaging of the head showed a watershed pattern consistent with areas of acute punctate infarcts and diminished flow at the level of the left cavernous carotid. A carotid duplex showed bilateral ICA high-grade (80%-99%) stenosis, with velocities in the proximal right ICA of 670/306 mL/min and in the left ICA of 713/252 mL/min.

Owing to the patient's significant comorbidities and recent symptoms, an urgent left CEA with a CPB was indicated. Before the operation, an ultrasound-guided left superficial and deep CPB was performed by an anesthesiologist with training in procedural blocks. For the superficial component of the CBP, local anesthetic was injected into subcutaneous tissue along the posterior border of the sternocleidomastoid. For the deep component, the transverse processes of upper cervical vertebrae C2 to C4 were identified and local anesthetic was injected directly into the prevertebral cervical space.

Surgical dissection of the carotid artery was performed without difficulty. The hypoglossal and vagus nerves were visualized and preserved. The patient did not have any neurologic

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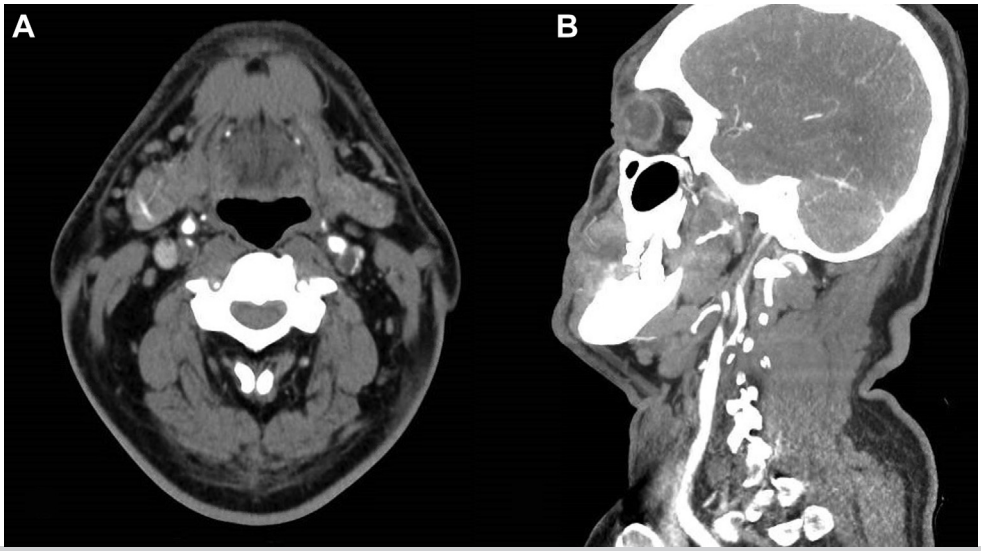

Fig 1. Computed tomography angiogram of the head and neck. Two images showing carotid stenosis. **A,** Axial slice of a computed tomography angiogram of the neck. It shows bilateral high-grade carotid stenosis. **B,** Sagittal slice of the same study showing left carotid stenosis.

changes after clamping the carotid artery and the surgical team proceeded with the endarterectomy with patch angioplasty using a Hemashield patch. There was good distal flow in the internal and external carotids after the repair and the incision was closed.

The patient recovered in the postanesthesia care unit and was hemodynamically stable. On examination, he did not exhibit any respiratory compromise or focal neurologic deficits; however, the patient was noted to have difficulty swallowing and hoarseness in his voice. Owing to these symptoms, the otolaryngology service was consulted for further investigation. A bedside nasolaryngoscopy was performed and revealed a left aryepiglottic fold hematoma (Fig 2).

The patient was started on dexamethasone, a proton pump inhibitor, and placed on airway precautions. Over the following 5 days, the patient's voice and swallowing ability improved. He underwent daily bedside laryngoscopies, which showed interval improvement of the hematoma over time. He was discharged on postoperative day 5 tolerating a regular diet. At a 6-month follow-up appointment, the patient was doing well, but stated his voice had only returned to 85% of its strength before the procedure.

DISCUSSION

Despite the advantages to performing a CEA under regional anesthesia,[10] there are few studies detailing the complications. Pandit et al[4] conducted a systematic review comparing complication rates with superficial and combined (deep and superficial) regional CBPs. In their review, they classified complications into three groups—block-related, conversion to general anesthesia, and serious systemic complications. Rates of block-related complications were higher for deep CBPs, yet specific causes for these complications were speculative. The incidence of block-related complications was low in both groups, limiting strength of interpretation. In the 83 papers analyzed, including 7558 individual deep CBPs, the complication of an epiglottic hematoma was not described.

A majority of CEAs in our institution are done under general anesthesia. Regional block is used for patients who have a high cardiovascular risk for general anesthesia. At our institution, there are no dedicated teams for CBPs, but regional blocks are performed with ultrasound guidance by anesthesiologists who have completed additional training in regional anesthesia.

Epiglottic hematomas have been described in case reports of patients on therapeutic anticoagulation in different settings. The mechanisms for epiglottic hematoma include traumatic or difficult endotracheal intubations. One case describes such a complication after repeated endotracheal intubation attempts.[11] Another case describes a patient who suffered postoperative epiglottic hematoma after routine, atraumatic endotracheal intubation.[12] The authors propose the forces applied during routine intubation may become significant enough to cause hematoma in patients anticoagulation. An additional case describes a patient on warfarin who suffered spontaneous sublingual and laryngeal hematomas, suggesting that anticoagulation alone was a significant risk factor.[13]

In regard to our patient, it is likely that aspirin therapy and intraoperative heparin played a role in the formation of the epiglottic hematoma. We propose the mechanism for epiglottic hematoma formation in this case includes a technical component related to deep CBP in combination with anticoagulation and antiplatelet therapy.

Epiglottic hematomas can be managed conservatively. Close airway monitoring is essential, and surgeons

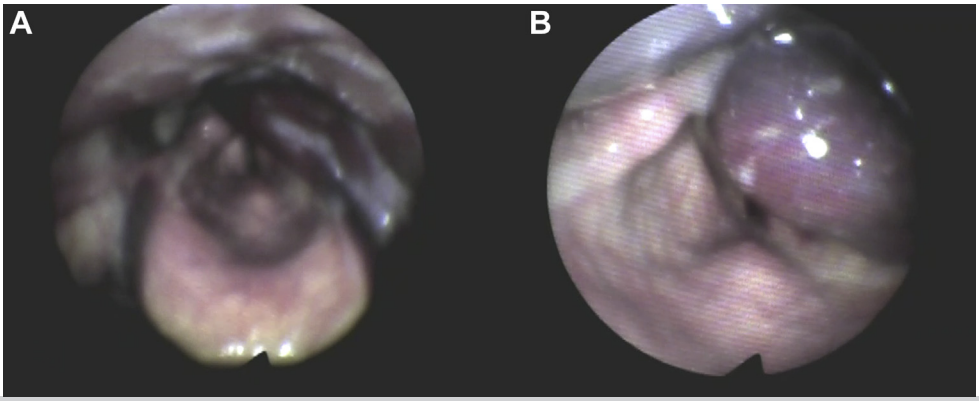

Fig 2. Aryepiglottic fold hematoma. **A** and **B,** Left epiglottic hematoma under flexible nasolaryngoscopy.

should have a low threshold for securing the airway via tracheostomy in cases with airway compromise.[14] Securing the airway via intubation is not ideal, because laryngoscopy can be difficult due to obstruction and intubation can cause trauma and bleeding.[15,16] For overall management of airway edema, steroids are used to decrease laryngeal edema, especially in settings of recent extubation.[17]

The recent literature surrounding CEA focuses on comparative techniques for general verse regional anesthesia, and within regional techniques deep verse superficial verse combined CPBs. Deep CPBs are associated with higher rates of serious complications and conversion to general anesthesia when compared to superficial blocks, but they are also associated with better pain control.[4,18] Recording complications for deep CPB and aggregating data will enable surgeons to make informed decisions regarding the risks of CPBs for CEA. Once elucidated, the probability of complications may prohibit deep CPBs for asymptomatic CEA and nonurgent symptomatic CEAs. Although these studies helped to shape the current practice in vascular surgery, more investigation into complications is warranted.

REFERENCES

1. Barnett HJM, Taylor DW, Eliasziw M, Fox AJ, Ferguson GG, Haynes RB, et al. Benefit of carotid endarterectomy in patients with symptomatic moderate or severe stenosis. N Engl J Med 1998;339:1415-25.
2. Woo SY, Joh JH, Han SA, Park HC. Prevalence and risk factors for atherosclerotic carotid stenosis and plaque: a population-based screening study. Medicine (Baltimore) 2017;96:e5999.
3. Bowyer MW, Zierold D, Loftus JP, Egan JC, Inglis JK, Halow KD. Carotid endarterectomy: a comparison of regional versus general anesthesia in 500 operations. Ann Vasc Surg 2000;14:145-51.
4. Pandit JJ, Satya-Krishna R, Gration P. Superficial or deep cervical plexus block for carotid endarterectomy: a systematic review of complications. Br J Anaesth 2007;99:159-69.
5. Agrifoglio G, Bonalumi FA. Prediction of cerebral ischaemia using loco-regional anaesthesia. Eur J Vasc Surg 1993;7(Suppl A):13-5.
6. Anderson CA, Rich NM, Collins GJ, McDonald PT. Carotid endarterectomy: regional versus general anesthesia. Am Surg 1980;46:323-7.
7. Carling A, Simmonds M. Complications from regional anaesthesia for carotid endarterectomy. Br J Anesth 2000;84:797-800.
8. McCarthy RJ, Walker R, McAteer P, Budd JS, Horrocks M. Patient and hospital benefits of local anaesthesia for carotid endarterectomy. Eur J Vasc Endovasc Surg 2001;22:13-8.
9. Gough MJ, Bodenham A, Horrocks M, Colam B, Lewis SC, Rothwell PM, et al. General anaesthesia versus local anaesthesia for carotid surgery (GALA): a multicentre, randomised controlled trial. Lancet 2008;9:28.
10. Stoneham MD, Stamou D, Mason J. Regional anaesthesia for carotid endarterectomy. Br J Anaesth 2015;114:372-83.
11. Cheng KS, Li HY. Epiglottic hematoma secondary to endotracheal intubation. Acta Anaesthesiol Sin 1999;37:49-52.
12. Brown I, Kleinman B. Epiglottic hematoma leading to airway obstruction after general anesthesia. J Clin Anesth 2002;14:34-5.
13. Parvizi S, Mackeith S, Draper M. A rare cause of upper airway obstruction: spontaneous synchronous sublingual and laryngeal haematomas. BMJ Case Rep 2011;27:1-3.
14. Koch CA, Olsen SM, Saleh AM, Orvidas LJ. Spontaneous epiglottic hematoma secondary to supratherapeutic anticoagulation. Int J Otolaryngol 2010;2010:201806.
15. Lepore ML. Upper airway obstruction induced by warfarin sodium. Arch Otolaryngol 1976;102:505-6.
16. Cohen AF, Warman SP. Upper airway obstruction secondary to warfarin-induced sublingual hematoma. Arch Otolaryngol Head Neck Surg 1989;115:718-20.
17. Pluijms WA, van Mook WN, Wittekamp BH, Bergmans DC. Postextubation laryngeal edema and stridor resulting in respiratory failure in critically ill adult patients: updated review. Crit Care 2015;19:295.
18. Stoneham MD, Doyle AR, Knighton JD, Dorje P, Stanley JC. Prospective, randomized comparison of deep or superficial cervical plexus block for carotid endarterectomy surgery. Anesthesiology 1998;89:907-12.

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