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Accessibility
Fibrin Glue Injection for Cavernous Sinus Hemostasis Associated with Cranial Nerve Deficit: A Case Report

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Abstract
Fibrin glue injection has been used to control intraoperative cavernous sinus (CS) venous bleeding. There have been no reported complications related to this maneuver.¹,² We present a case where a patient developed a sensory trigeminal nerve deficit after injection of fibrin glue into the posterior CS during resection of a petrosal meningioma. We believe that this deficit was due to the compression of the trigeminal ganglion (TG) similar to balloon compression procedures. Although fibrin glue injection may achieve satisfactory cavernous sinus homeostasis, the volume and rate of injection should be kept in mind to avoid a compressive lesion on traversing cranial nerves and surrounding structures, or retrograde filling of the venous tributaries.

Keywords
► fibrin glue
► cavernous sinus
► cranial nerve

Introduction
Fibrin glue injection (Baxter Healthcare Corp., Deerfield, Illinois, United States) has been used to control intraoperative cavernous sinus (CS) venous bleeding. There have been no reported complications related to this maneuver.¹,² We present a case in which a patient developed a sensory trigeminal nerve (TN) deficit after injection of fibrin glue into the posterior CS during resection of a petrosal meningioma. We believe this deficit was due to the compression of the trigeminal ganglion (TG) similar to balloon compression procedures but persistent until the glue resolves.

Case Report
A 40-year-old woman with radiation-induced petrous apex meningioma underwent resection of the lesion via a posterior fossa approach. After complete resection of the lesion, the involved dura was excised to the junction of the superior petrosal sinus and the posterior wall of the CS, where venous hemorrhage ensued. This was easily controlled with injection of fibrin glue into the posterior CS. The remaining of the procedure was uneventful. Postoperatively the patient developed new-onset ipsilateral facial numbness. There was dense hypoalgesia and hypoesthesia in the trigeminal V1 and V2 distributions, and less affected, in V3. The motor trigeminal function was intact. Postoperative magnetic resonance imaging (MRI) demonstrated total gross resection; however, there was expansion of the CS by the fibrin glue. Repeated postoperative MRI at 3 months demonstrated resolution of the CS expansion (► Fig. 1). However, the patient’s sensory TN deficit persisted after the 18-month follow-up.

Discussion
Multiple intraoperative techniques have been described for cavernous sinus hemostasis that include the use of Surgicel
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follow-up, patients with a shorter duration of compression had lower rates of facial numbness, with similar rates of efficacy. Brown and Pilitsis also demonstrated an association between duration of PBC compression (1.18 versus 1.06 minutes) and facial numbness in 65 PBC procedures. Zanusso et al performed PBC with intraoperative pressure monitoring and demonstrated an association between high PBC pressure with greater side effects but with lower recurrence rates at 1 year. Similarly, Brown and Pilitsis demonstrated a trend toward facial numbness with higher PBC pressures.

Drawing analogies from the preceding discussion regarding PBC and TN sensory deficits, the fibrin glue injection produced a compressive lesion on the TN, similar to PBC, leading to the patient’s facial numbness. In this case, the duration of TG compression was sustained for days (evident by the postoperative MRI), as compared with seconds for PBC. The amount of pressure applied to the TG by the fibrin glue injection is unknown, although it was sufficient to produce the sensory deficit without a motor deficit.

Conclusion

Although fibrin glue injection may achieve satisfactory cavernous sinus homeostasis, the volume and rate of injection should be kept in mind to avoid a compressive lesion on traversing cranial nerves and surrounding structures or retrograde filling of the venous tributaries.

References

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