

tion in size, making them amenable to surgery.

We present a case of a 56-year-old man presented with a 6-year history of severe right-sided sharp lancinating pain affecting the mandibular division of the trigeminal sensory distribution. On examination there appeared to be a trigger zone over his right zygoma. There was no evidence of any neurological deficit. He was maintained on 1200 mg of carbamazepine a day with little effect.

High-resolution magnetic resonance imaging (MRI) and gadolinium enhanced magnetic resonance angiography (MRA) revealed a very large vascular loop of the right superior cerebellar artery (SCA) passing inferiorly and causing distortion of the root entry zone of the right trigeminal nerve.

Cerebral angiography confirmed the presence of a postero-superior vermian AVM fed by the right SCA and draining into the vein of Galen [Figure 1]. Endovascular embolization was performed using Berenstein liquid coils. This resulted in significant reduction in blood flow with transit time through the AVM doubled. Follow-up MRI and MRA with gadolinium after 6 months showed partial patency of the AVM with dilatation of tentorial veins bilaterally and a large loop of SCA on the right. Repeat angiography and embolization showed no change in the appearance of the superior vermian AVM with its three feeders (two from the SCA and one from PICA). Using the same technique catheter was inserted into the SCA and the superior of the two feeders. A straight Berenstein liquid coil was injected. This produced gratifying reflux up the pedicle on cheek injection and the flow was clearly reduced further [Figure 2]. Despite many attempts it was not possible to catheterize the second feeder. The procedure was terminated without any neurological complication. The patient was discharged after 3 days. After a follow up of 18 months the patient remain free from pain and there was a significant re-

Arteriovenous malformation presenting with trigeminal neuralgia and treated with endovascular coiling

Sir,

Arteriovenous malformations (AVMs) are known to cause trigeminal neuralgia (TGN).^[1] The incidence of posterior fossa AVM is less than 10% of all intracranial AVMs. The association of an AVM and TGN is rare.^[2] Eisenbrey and Hegarty were the first to describe a case of AVM presenting with TGN in 1956.^[3] Staged endovascular coiling as an adjunct to surgery for large (3 cm) AVMs can achieve satisfactory reduc-

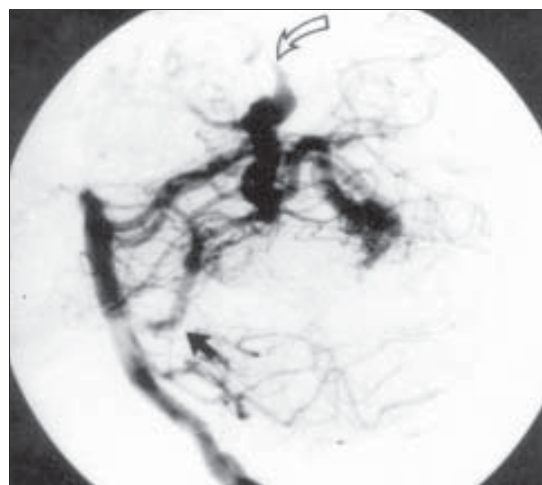


Figure 1: Right vertebral angiogram showing dilated, ectatic right superior cerebellar artery (solid arrows) and occlusion of the vein of Galen (curved open arrow).

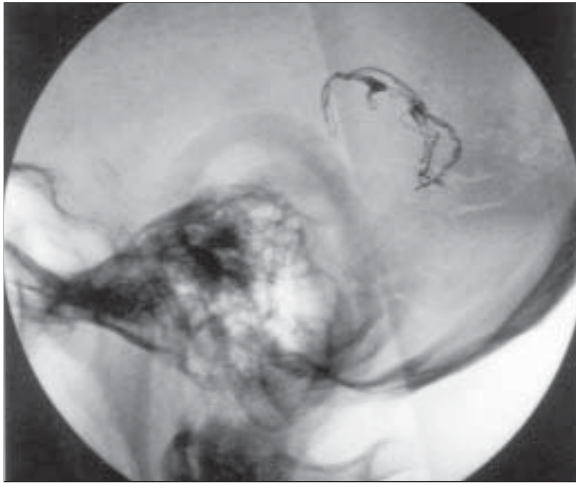


Figure 2: Fight vertebral angiogram following first embolization showing marked reduction in A-V shunting . The deposition of coils is clearly shown.

duction in the volume of the nidus. This case indicates the feasibility of embolization for TGN in selected patients. In this case the angioarchitecture meant that liquid embolic agents such as cyanoacrylate glue were contraindicated. The aim of the procedure was to reduce flow in the AVM with coil embolization. Large AVMs can be treated with staged embolization and surgery (operative surgery or radiosurgery) if needed. A multimodality approach (embolization, radiosurgery, surgical exploration) is the current policy for treating AVMs and takes into account the cumulative risk of bleeding, the location and size of the AVM and the presence of venous anomalies.^{[4],[5]}

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