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Vertebro-carotid anastomosis as a cause of uncontrollable epistaxis

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Abstract
Uncontrolled epistaxis may require a long hospital stay, multiple blood transfusions and multiple arterial ligations. This case report highlights vertebro-carotid anastomosis as a rare cause of uncontrolled epistaxis. The importance of angiography in the identification of rare arterial anastomoses is stressed.

Key words: Epistaxis; Vertebral artery; Carotid artery, external

Case report

A 23-year-old male patient underwent functional endoscopic sinus surgery, inferior turbinate reduction and bilateral maxillary antral washout for chronic sinusitis, in November 1992. A septoplasty had been performed in 1991 for nasal obstruction.

Eight days post-operatively the patient presented with profuse nasal bleeding. He was admitted and hospitalized for 17 days. During this period the patient underwent four operative procedures and was transfused 12 units of blood, for recurrent epistaxis. Surgical procedures performed included: (1) left external carotid artery (ECA) ligation and left anterior ethmoidal artery ligation; (2) examination of the nose and nasal packing (anterior and posterior); (3) ligation of right ECA and left maxillary arteries (MA); (4) examination of the nose and nasal packing (anterior and posterior). Throughout this period the clotting factors were normal.

On day 17 the patient was transferred to the Queen Elizabeth Hospital for angiography. Four-vessel angiography was performed and showed (Figure 1): (a) ligated right and left ECAs; (b) a substantial left occipito-vertebral anastomosis which filled the ECA above the level of ligation. The retrograde flow from this anastomosis indirectly supplied a prominent patent left maxillary artery via the left ECA distal to site of ligation; (c) the right vertebral artery was small, and fed the posterior inferior cerebellar artery; (d) scant recurrent bilateral ethmoidal arteries, supplied by the ophthalmic arteries.

The patient underwent his fifth general anaesthetic and the neck was reopened on the left side. The ECA and the abnormal anastomotic occipital artery were ligated. The left maxillary artery was embolized with spongistan and ligated. Examination of the nose revealed general bruising. Both sphenopalatine foramina were diathermied. No further epistaxis occurred and the patient was discharged one week post-operatively.

Discussion

Welsh *et al.* (1990) quote that the craniofacial collatereal circulation including the anastomoses between the internal carotid artery (ICA) and the external carotid artery (ECA) were described by Soemmerring (1818) and Du Brueil (1847) and that Meyer (1887), further characterized ECA/ICA anastomoses via the facial, maxillary, and temporal branches of the ECA and the ethmoidal, periorbital and ophthalmic branches of the ICA.

Lasjaunias *et al.* (1978) have established that additional potential anastomoses can occur between the carotid and vertebral arteries, due to persistence of foetal vascular anatomy. During early foetal development a number of arterial bridges form between the dorsal and ventral foetal aortas. The cranial end of these immature aortas contribute to the formation of the carotid and anterior longitudinal neural vascular (vertebral) systems respectively. Persistence of these metameric or segmental vessels would lead to the formation of vertebro-carotid anastomoses. These anastomoses are usually temporary and classically they regress by the end of the second stage of foetal life (Padget, 1968). The first four, rostrally located, arteries anastomose the ICA and vertebrobasilar systems, while the fifth metameric artery connects the external carotid and vertebral arteries. The three most rostrally located arteries are intracranial. The fourth and fifth arteries represent the arteries of the first and second foetal somites. Persistence of these vessels results in arteries designated as proatlantal arteries type 1 (Pa1) and type 2 (Pa2) respectively (Figure 2).

In the case presented in this paper the abnormal ECA/ver-

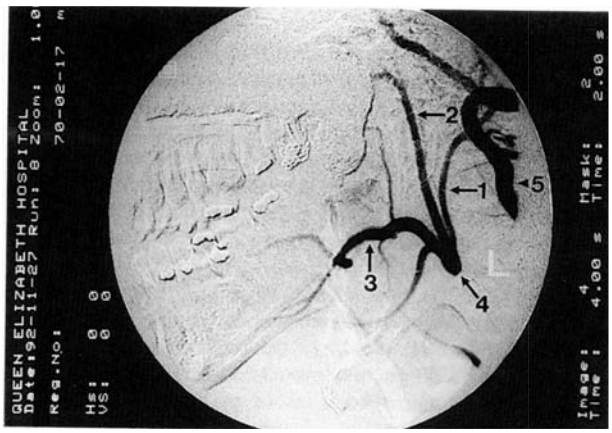

Fig. 1

Showing the occipital artery (1); maxillary artery (2); facial artery (3); external carotid artery at the site of ligation (4); vertebral artery (5).

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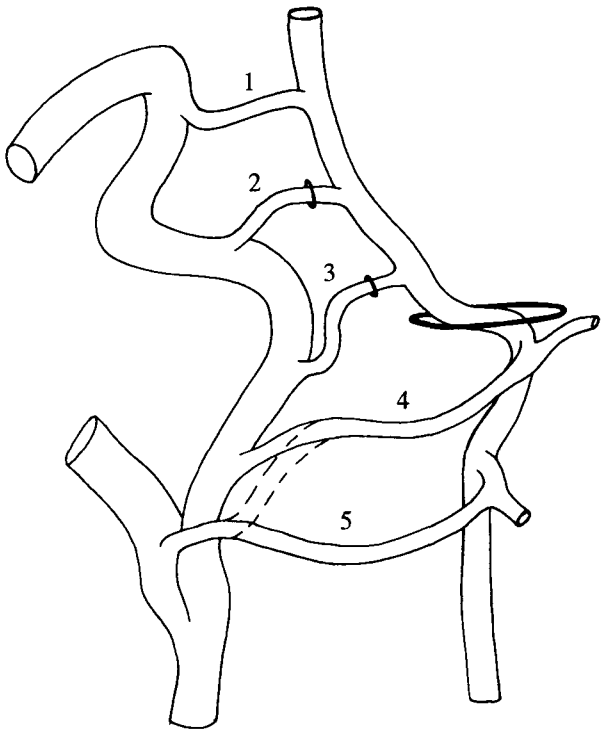

FIG. 2

Carotid-vertebral anastomosis. Showing the trigeminal artery (1); otic artery (2); hypoglossal artery (3); proatlantal artery type 1 (4); proatlantal artery type 2 (5).

tebral anastomosis represents a proatlantal type 2 artery (Pa2) i.e. the abnormal occipital artery. Knowledge of its foetal origin allows accurate prediction of the anatomical course of the vessel. Arising from the proximal ECA it courses posteriorly through the second cervical space to the vertebral canal (Lasjaunias and Berenstein, 1989). In this patient persistence of the Pa2 artery created a vertebrocarotid anastomosis. Retrograde flow along this abnormal feeding artery filled the ECA distal to the site of the previous ligation. The epistaxis continued despite ligation of the ECAs because blood flow continued to the patent and bleeding ipsilateral MA.

This rare type 2 anatomical variant was not recognized until demonstrated by angiography. The angiogram demonstrated that an ICA/ECA anastomosis was not the cause of the refractory epistaxis in this case.

Continued bleeding from the left MA, despite a previous ligation attempt, demonstrates that ligation of this artery is not as simple as suggested in some texts. A rebleeding rate of between 10 and 22 per cent after MA ligation has been noted by Breda *et al.* (1989).

Conclusion

When epistaxis, unsuccessfully managed by standard arterial ligation procedures, occurs the investigation of choice should be angiography. This investigation successfully highlighted a vertebro-carotid anastomosis as the cause for the intractable epistaxis in this case, permitting appropriate ligation of the patent artery.

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