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Radiology in Focus

A case of intractable epistaxis

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Abstract
The surgical management of intractable epistaxis by external carotid artery ligation may become complicated if there is a high bifurcation of the common carotid artery. Occlusion of the bleeding vessels by catheter embolization is described in a patient in whom exploration of the neck had failed to locate the external carotid artery.

Key words: Epistaxis; Embolization, therapeutic

Introduction

Epistaxis is common, with most individuals experiencing at least one episode in a life time. Usually the episodes are minor and resolve spontaneously. In only about six per cent of the population are the episodes more severe requiring medical attention. In a very small percentage the bleeding cannot be stopped with the usual packing measures, and these 'intractable epistaxes' require further action. Surgical ligation of the external carotid artery is an established treatment in such cases, however, if there is total or segmental agenesis of the internal carotid artery, this may be impossible. This anomaly has been documented by various authors following the introduction of angiography in the 1970s (Smith *et al.*, 1972; Rosen *et al.*, 1975; Handa *et al.*, 1980; Ueda *et al.*, 1984). One similar case to our own has been described in the otolaryngological literature (Nishimura *et al.*, 1989).

This is an account of a protracted epistaxis in which the anomalous anatomy and haematological disease both influenced subsequent management.

Case report

A 68-year-old woman, with a four-year history of low grade chronic myelomonocytic leukaemia in clinical remission, presented with a three-day history of intermittent left-sided epistaxis. Her general practitioner ascribed this to rhinitis and started her on a course of amoxycillin prior to her referral.

She had been seen three years previously in the Ear Nose and Throat department with an acute onset of deafness in the right ear. This was due to haemotympanum, thought to have occurred spontaneously as a consequence of thrombocytopaenia. Her platelet count at that time was 64×10^9/l (normal range 120–400 $\times 10^9$/l). The haemotympanum resolved with conservative measures.

On examination the left epistaxis appeared to originate from a dark nasal polyp. The following day she underwent a left nasal polypectomy and histology showed 'moderately oedematous polyps in which there is marked telangectasia of the vessels with small aggregates of plasma cells and lymphocytes'. Her pre-operative full blood count was: haemoglobin (Hb): 13 g/dl; platelets: 111×10^9/l, white cell count (WBC): 15.3×10^9/l. Both sides of her nose were packed with Vaseline gauze.

Four hours post-operatively she was noted to be oozing blood bilaterally through her nasal packs, and continued to do so slightly for one day, but remained haemodynamically stable. By the second post-operative day the oozing had settled and the packs were removed, but she immediately bled profusely and was repacked bilaterally with bismuth, iodoform, and paraffin paste (BIPP) packs. She was sedated with diazepam 5 mg tds and continued on oral antibiotics. This stemmed the main flow of blood but she continued to ooze slightly bilaterally. A clotting screen gave normal results and Hb: 10.6 g/dl; platelets: 139×10^9/l, WBC: 18.5×10^9/l. During the following four days she suffered four further episodes of epistaxis through the nasal packs, underwent repacking with BIPP on each occasion and required transfusion of two units of blood. Repeat clotting screen was within normal limits, as were the factor VIII screen and immunoglobulin levels. Despite this she continued to ooze. The post-transfusion Hb was 11.8 g/dl.

On the basis that a platelet dysfunction related to her underlying leukaemia (that would not be revealed by the routine clotting screens) was the underlying cause of her continued bleeding, she received a 200 ml platelet transfusion. Notwithstanding this the oozing continued.

The following day her nasal packs fell out and she was trickling blood from the left nostril. She was immediately taken to theatre for examination under anaesthesia, and was found to be bleeding from the left nasal cavity posteriorly, but no single bleeding point was identified, and although cautery was attempted, the bleeding was not arrested. Not surprisingly, antral washouts revealed blood in both maxillary antra. A firm BIPP pack was applied bilaterally and the surgeon proceeded to ligation of the left

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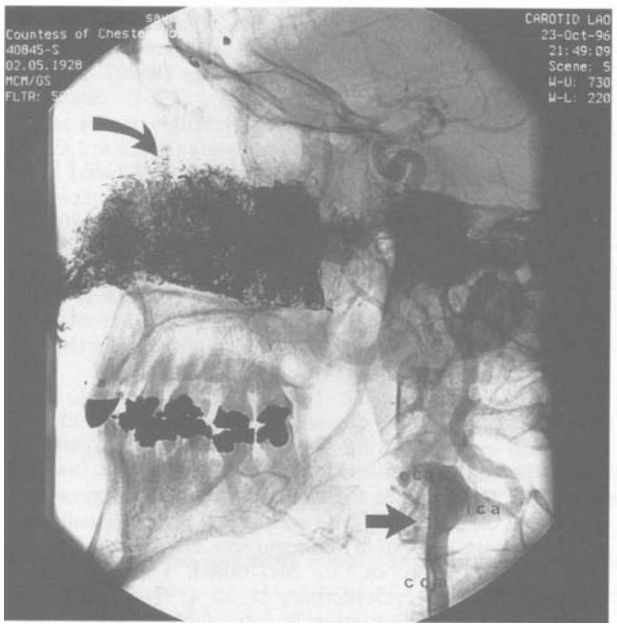

FIG. 1

Selective common carotid artery (cca) arteriograph showing high bifurcation of the left common carotid artery, behind the level of the angle of the mandible, (solid arrow) into the internal carotid artery (ica) and external carotid artery (eca). The bismuth, iodoform and paraffin paste (BIPP) nasal packs are also marked (curved arrow).

external carotid artery. The left common carotid artery was exposed by an incision along the anterior border of the left sternocleidomastoid muscle, but no bifurcation was identified from the level of the lower border of the thyroid cartilage to the lower border of the mandible. Ligation was therefore abandoned. Examination of the ears revealed bilateral haemotympanum and bilateral Shepherd grommets were inserted. Post-operatively Hb was 8.9 g/dl, platelet count: 170 $\times$ 10^9/l. She received four units of whole blood and suffered no further epistaxis on her first post-operative day. At this stage platelet function studies

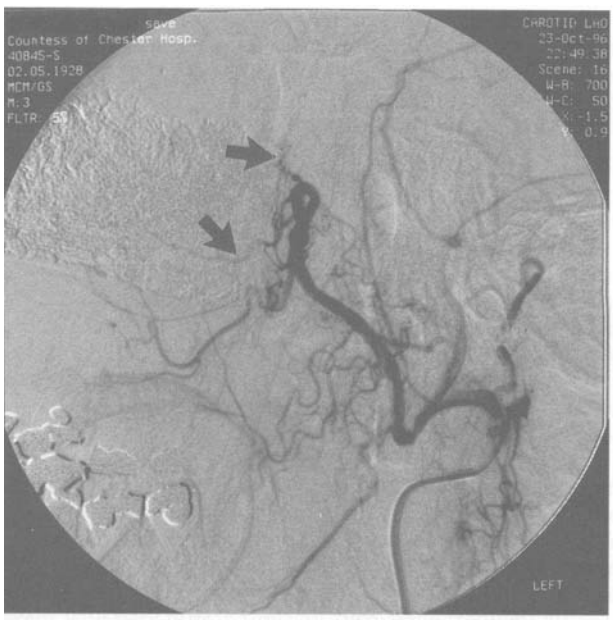

FIG. 2

Arterial branches of nasal and ethmoidal branches of the maxillary artery, (solid arrows).

FIG. 3

Completion arteriograph showing satisfactory occlusion of the posterior nasal vasculature. The 5F guiding catheter (open straight arrow), 3F 'Fast Tracker' catheter (open curved arrow), and single complex coil, (solid closed arrow), are seen.

and the bleeding time were within normal limits but despite this and tight nasal packs there was continuous oozing of blood.

Three weeks after her admission she underwent transfemoral angiography of the left carotid artery to determine the site of bleeding and demonstrate the anatomy of the carotid bifurcation. This showed that the artery bifurcated at a high level behind the angle of the mandible (Figure 1), and revealed multiple vessels in the posterior part of the nasal cavity which were branches of the nasal and ethmoidal branches of the maxillary artery (Figure 2). Arterial embolization was performed by super selective cannulation of the internal maxillary artery from the common femoral approach using a coaxial 3F 'Fast Tracker' catheter (Target Therapeutics, Fremont CA). One thousand μ particles (ITC, Fremont CA) suspended in a water soluble contrast medium (Visipaque, Nycomed, Oslo) were used to occlude the nasal vessels. A single complex coil was also deployed in the distal internal maxillary artery. A completion arteriogram revealed satisfactory occlusion of the posterior nasal vasculature (Figure 3).

The nasal packs were removed the following day and the patient suffered no further epistaxis. Prior to discharge she remained in hospital a further six days for physiotherapy to help her mobilize after three weeks of being confined to her bed. In total she had been hospitalized for 28 days and had undergone nasal packing on six separate occasions. Following discharge she has suffered no further epistaxis.

Discussion

Intractable epistaxis is defined as epistaxis that is refractory to nasal packing. It is occasionally encountered in pregnancy, where it may only resolve on parturition. The management of non-pregnancy related intractable epistaxis consists either of selective arterial ligation, clipping, or arterial embolization. Sokoloff *et al.* in 1974 introduced the method of embolization utilizing Gelfoam particles suspended in normal saline.

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The nasal mucosa is supplied by anastamosing branches of the internal maxillary and external carotid arteries. Embolization of the internal maxillary artery is reported as controlling the epistaxis in 87 per cent of patients, and with supplemental embolization of the facial artery the rate is reported to be 97 per cent. Complication rates of three per cent are reported (Vitek, 1991), consisting mainly of paresis and other neurological complications secondary to reflux of emboli into the internal carotid artery (Metson and Hanson, 1983; De Vries *et al.*, 1986; Parnes *et al.*, 1987).

Procedures described for arterial occlusion are clipping of the maxillary artery in the pterygomaxillary fossa, ethmoidal artery clipping for epistaxis from the upper part of the nose above the middle turbinate, via a medial orbital incision, and ligation of the external carotid artery in the neck.

When attempting ligation of the external carotid artery the existence of the internal carotid artery must be confirmed. The internal carotid artery consists of six segments, the cervical, petrous, vertical, horizontal cavernous, clinoid, and cisternal (Lasjaunias and Santoyo-Vasquez, 1984). Each of the above segments can show agenesis (Altman, 1947). Therefore, the Ear Nose and Throat surgeon, before ligating the external carotid artery for the treatment of intractable epistaxis, should be aware of the possible agenesis of the cervical part of the internal carotid artery.

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