Endovascular treatment of intractable epistaxis — results of a 4-year local audit

I C Duncan
FFRad (D)
P A Fourie
MMed Rad (D)
C E le Grange
Nat Dipl Rad
H A van der Walt
BSc (Verpl)
Unitas Interventional Unit
Centurion, Gauteng

Abstract

A total of 57 endovascular embolisation procedures were performed for intractable epistaxis in 51 patients over a 4-year period at the Unitas Interventional Unit near Pretoria. Long-term follow-up was possible in 36 patients. Three cases were due to trauma and 2 directly related to previous facial surgery, 1 patient had hereditary haemorrhagic telangiectasia (HHT), and the remaining 45 cases (88.2%) were classed as idiopathic. Eight patients (15.7%) had a rebleed between 1 and 33 days after the initial embolisation. Four were re-embolised once, 1 was re-embolised twice (the HHT patient), and 2 underwent additional ethmoid artery ligation (with no further bleeding). This gives a primary short-term success rate (in all 51 cases) of 86.3% and a secondary assisted success rate of 94.1% for embolisation alone. Long-term follow-up was obtained in 36 patients, with 35 (97.2%) reporting no further bleeding after the initial procedure(s). Only the patient with HHT developed multiple recurrent bleeds. The mortality rate was 0%, the major morbidity rate 2% (1 stroke), and the minor morbidity rate 25% ($N = 36$), which included transient facial pain, headaches and femoral problems related to access. Our results compare favourably with other published series. In conclusion, endovascular embolisation for intractable epistaxis is available locally as an alternative technique for the treatment of this difficult condition.

Introduction

Epistaxis is a common condition, affecting an estimated 60% of the general population of whom an estimated 6% will seek or require medical assistance.[1] Refractory or intractable epistaxis is defined as recurrent or persistent bleeding after appropriate conservative treatment, or multiple episodes of epistaxis over a short period of time, each requiring medical attention.[2] Intractable epistaxis usually arises from the posterior or superior parts of the nasal cavity, and is therefore not readily controllable by direct pressure, topical cauterisation or anterior nasal packing. Arterial ligation has remained the mainstay of treatment for intractable posterior epistaxis in many centres.[3-6] Percutaneous embolisation of the internal maxillary artery for nasal haemorrhage was first described by Sokoloff *et al.* in 1974.[7] Several other reports describing the efficacy of endovascular embolisation followed.[8-11] Currently, endovascular embolisation is an accepted method of treatment for haemorrhage from the nasal cavity or other craniofacial lesions .[12,13]

Materials and methods

A retrospective audit was done of 57 percutaneous endovascular embolisation procedures in 51 patients performed over the 4-year period July 1999 - June 2003 at the Unitas Interventional Unit in Centurion. All patients were referred by an otorhinolaryngologist after failed conventional treatment, generally involving local cauterisation and/or nasal packing; 12 patients had also received a blood transfusion prior to embolisation. We reviewed our procedural data notes, with further clinical data obtained either from initial referral letters or follow-up notes provided by referring clinicians. Long-term telephonic follow-up was obtained from 38 patients (respondents), with 13 patients being untraceable during the period of the audit. Of the 38 respondents, 2 had died of unrelated causes since the embolisation, with telephonic interviews obtained with the remaining 36. The collection of data was flawed by incomplete clinical record keeping. The most complete data obtained

were for the 36 respondents, for whom much of the recorded data could be cross-checked and who could also provide missing information in addition to long-term follow-up. The long-term follow-up period varied from 1 to 47 months.

All embolisation procedures were performed in the Unitas Interventional Unit vascular laboratory using a Phillips V5000 Integris digital monoplane angiography unit (Phillips Medical Systems, Netherlands BV). Of the procedures 22 (38.6%) were performed under general anaesthesia and 35 (61.4%) under local anaesthesia and sedation. A co-axial microcatheter technique was utilised in 56 procedures (98.2%), with embolisation being performed through a standard 4F diagnostic catheter in only 1 case (1.8%). The co-axial technique involved initial selective catheterisation of the external carotid artery (ECA) with a 4F or 5F 0.038-inch lumen diagnostic catheter, followed by superselective catheterisation of the relevant ECA branches by placement of a microcatheter through the diagnostic catheter. The type or make of diagnostic catheter and microcatheter/microguidewire system used varied according to operator preference, vascular anatomy and stock availability. Both the guiding catheter and microcatheter were continuously irrigated with heparinised saline (2000 u in 1 litre) using a pressure bag system via Y-connectors attached to each catheter. Systemic heparin was generally not given in uncomplicated cases.

Initial diagnostic arteriograms of the internal carotid artery (ICA) and ECA were obtained on each side. The former were done in order to exclude an intracranial aneurysm, arteriovenous shunt or frontal tumour as a possible cause of the bleeding as well as to identify any arterial supply to the nasal cavity from ethmoidal branches of each ophthalmic artery (Fig. 1). The ipsilateral internal maxillary artery (IMAX), defined as the one supplying the nasal cavity from which the bleeding was visualised or assumed to arise, was then superselectively catheterised and embolised in all cases except 1, in which the ipsilateral ECA origin was occluded due to atheromatous disease and a contralateral IMAX embolisation was performed. Other vessels superselectively catheterised and embolised included the ipsilateral facial artery (N = 29 (51%)), the contralateral IMAX (N = 27 (47.4%)), the contralateral facial artery (N = 8 (14%)), the ascending pharyngeal artery (N = 1 (1.7%)) and the accessory meningeal artery (N = 1 (1.7%)). The decision to embolise arteries other than the ipsilateral IMAX was based upon the relative contributions of each vessel to the supply of the nasal cavity as well as the potential for collateral supply to the nasal cavity (Fig. 2a). During arteriography the actual site of the bleeding can usually not be identified and often no abnormal vessels are seen in the nasal cavities, although in several cases a nonspecific generalised nasal mucosal 'blush' due to nasal mucosal congestion or arterial tortuosity may be seen (Figs 2b - 2d). The embolic agents used were microparticles in 56 procedures (98.2%), with cyanoacrylate (B Braun, Melsungen, Germany) and platinum microcoils (Target Therapeutics, Fremont, Calif., USA) used as the sole agents in 1. Of the 56 procedures performed using microparticles, 47 (77.2%) were done using polyvinyl alcohol (PVA) microparticles (Trufill, Cordis Johnson and Johnson, Miami, Fla., USA) and 9 (15.8%) using trisacryl gelatine microspheres (Embospheres, Guerbet, Paris, France). Particle sizes ranged from 150 to 700 µm in diameter and were usually between 250 and 500 µm. Other adjunctive embolic materials used included cyanoacrylate (N = 5 (8.8%)), platinum microcoils (N = 4 (7%)) and shredded Spongistan (Cordis, Johnson and Johnson, Miami, Fla., USA) (N = 4 (7%)). In all cases where no macroscopic cause of the bleeding such as a pseudoaneurysm could be identified, and where microparticulate embolisation was performed, the desired end-point of embolisation was a significant visible reduction or cessation of flow in the target vessel(s) (Fig. 3). The nasal packing was removed in theatre in 56% of cases in which this was documented and afterwards in 44% (N = 36).

Results

The male/female ratio of our patients was 1.2: 1 (28 men and 23 women), and the mean age was 54.4 years (range 17 - 83 years). Only 15 patients were referred from the Pretoria area, 36 being transferred from other centres. Thirty two (63%) were referred between the months of May and September. The causes of epistaxis in our cases are summarised in Table I. Of the trauma-related cases 1 occurred shortly after a direct blow to the nose and 1 following a Le Fort fracture, whereas the 3rd patient (who suffered the intraprocedural stroke) had been severely assaulted 3 weeks before presentation. Of the 2 patients whose epistaxis was related to prior surgery, 1 had recently undergone

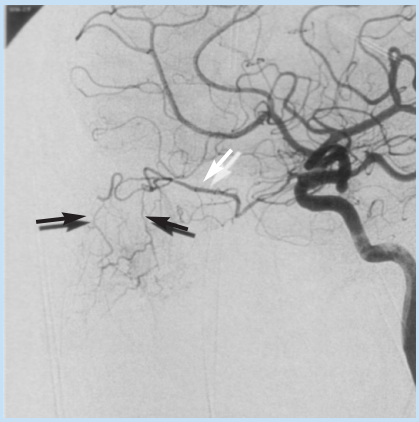

Fig 1. Selective internal carotid digital subtraction arteriogram showing supply to the superior nasal cavity via the ethmoidal branches (black arrows) of the ophthalmic artery (white arrow).

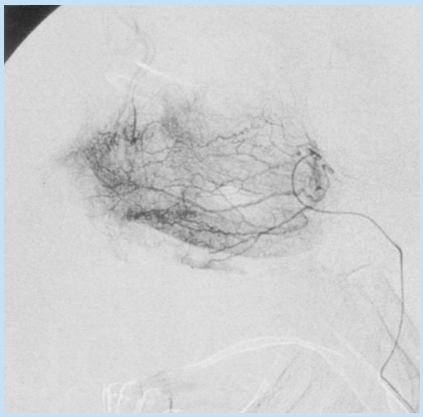

Fig 2c. In some cases a prominent nasal capillary blush may be seen in keeping with mucosal congestion.

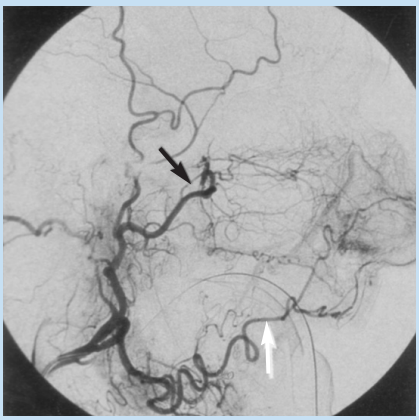

Fig 2a. Selective external carotid digital subtraction arteriogram showing the arterial supply to the nasal cavity via terminal branches of the internal maxillary artery (black arrow) and facial artery (white arrow).

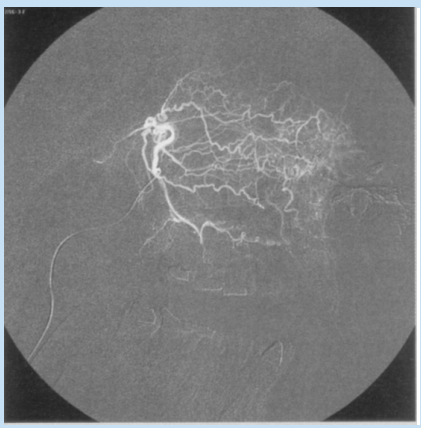

Fig 2d. In others tortuosity of the intranasal arteries may be seen.

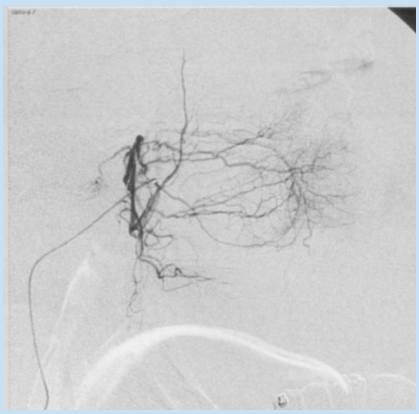

Fig 2b. Selective internal maxillary arteriogram (same case as Fig. 2a) showing the typical appearance of the nasal arteries prior to embolisation. No bleeding site is identifiable and the arteries have a normal appearance.

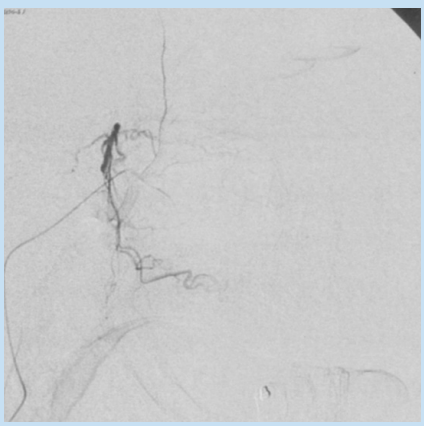

Fig 3. In the same case as shown in Figs 2a and 2b, a selective internal maxillary arteriogram following microparticulate embolisation shows very little contrast opacification of the nasal branches, indicating a satisfactory angiographic end-point for embolisation.

removal of an osteoma from the sphenoid sinus (with ipsilateral IMAX occlusion seen at angiography) and the other presented with severe bleeding from a large pseudo-aneurysm of the distal IMAX following a recent maxillary osteotomy. Identifiable risk factors in the respondent group ($N = 36$) are listed in Table II. None of the respondents volunteered any history of alcohol or illicit drug abuse. One patient had previously undergone radiation therapy for a maxillary tumour considered inactive at the time of the embolisation. One of the non-respondents developed severe epistaxis during the 25th week of pregnancy. No underlying cause for this was found. A 17-year-old girl with sideroblastic anaemia for which she had received multiple blood transfusions over a period of several years was referred for a semi-elective procedure, the only patient in our series not treated on an emergency basis.

Eight patients (15.7%) developed a rebleed between 1 and 33 days after the initial embolisation (Table III). Of these, 4 patients (7.8%) underwent a second embolisation procedure and 1

Table I. Causes of epistaxis

Idiopathic	45	88.2%
Trauma	3	5.9%
Post surgery	2	3.9%
HHT	1	2%

Table II. Risk factors for epistaxis (36 respondents)

Hypertension	19	52.8%
Smoking	9	25%
Anticoagulation/ASA	5	13.9%
Radiation therapy	1	2.8%

One patient had also received multiple blood transfusions for chronic sideroblastic anaemia.

patient with hereditary haemorrhagic telangiectasia (HHT) underwent two further embolisation procedures in our unit and one at another centre. Two patients (3.9%) underwent additional surgical ligation of the ethmoid arteries, after which no further bleeding was encountered. One of these was the patient with the Le Fort II fracture and the other had an idiopathic-type bleed. One patient had a single post-procedural rebleed for which angiography was repeated but no further embolization performed. Forty-four patients (86.3%) therefore responded well to a single embolisation procedure. No further rebleeds were noted in the 4 patients who underwent a second embolisation procedure, giving a cumulative success rate for embolisation alone of 94.1%. With the exception of the patient with HHT, none of the 36 living respondents reported a late rebleed after primary or secondary intervention. The mortality rate in our series was 0%, and we encountered only one major complication (2%), being the development of a right-sided cerebral infarction during attempted guiding catheter replacement in a tortuous carotid artery. Minor complications are listed in Table IV. Those related to the femoral access included transient tenderness in 2 cases, a tender lump (non-aneurysmal) in 1 and prolonged numbness around the puncture site in 1. The total minor complication rate for the respondent group is therefore 25%.

Discussion

Most cases of epistaxis occur in the anterior nasal cavity in the region of

Table III. Rebleeding after initial embolisation

Patient	Age (yrs)	Risk factors	Time of rebleed	Cause of rebleed	Further treatment	Outcome
1	34	Smoker	2 days	Unknown	Repeat angiogram only	No rebleed
2	58	Smoker, HHT	Multiple rebleeds over many years	HHT	Total of 3 embolisations performed (+ 1 at another centre) in 2 years	Eventually well palliated for > 1 yr with intralesional bleomycin
3	50	Warfarin and disprin (cardiac valve replacement)	$^1/_2$ day	Unknown. The PI at time of the rebleed was 75%	Repeat embolisation of ipsilateral IMAX only with microparticles	No rebleed
4	25	Pregnant (25 weeks)	33 days	Unknown	Repeat embolisation of ipsilateral IMAX only with microparticles	No rebleed
5	83	Warfarin (pulmonary embolism), PI on admission of 34%	1 day	Significant collateral supply via accessory meningeal artery	Repeat embolisation of ipsilateral IMAX and accessory meningeal artery	No rebleed
6	36	Le Fort II fracture	2 days	Significant collateral supply via ethmoid arteries	Surgical ligation of ethmoid arteries	No rebleed
7	55	Hypertension	$^1/_2$ day	Significant collateral supply via ethmoid arteries	Surgical ligation of ethmoid arteries	No rebleed
8	18	Maxillary osteotomy with IMAX pseudo-aneurysm	10 days	Coil migration and recannalisation through coils inserted previously in the IMAX	Repeat embolisation using particles, coils and cyanoacrylate	No rebleed

PI = prothrombin index.

Table IV. Complications of embolisation

Major	(*N* = 51)	
CVA	1	2%
Minor	(*N* = 36)	
Headache	3	8.3%
Transient facial pain/ paresthesia	2	5.5%
Local groin complications	4	11%

the antero-inferior septum known as Little's area. Underlying this is a confluence of arterial territories comprising those of the sphenopalatine artery, the greater palatine artery, the facial artery and the anterior and posterior ethmoidal arteries. This arterial confluence is known as Kisselbach's plexus.[14] Anterior epistaxis is usually easily controlled by conservative measures including localised pressure, topical cauterisation and vasocontriction, local infiltration with antifibrinolytic agents and anterior nasal packing.[15] Posterior (and to a lesser degree superior) epistaxis accounts for about 5% of all cases and can be extremely difficult to manage. Posterior nasal packing can be done with gauze or balloon catheters. Nasal packing has a reported failure rate of 26 - 52% and a complication rate of 69%.[14,16] Complications related to packing include nasal trauma, vasovagal response, aspiration, displacement of the packing, persistent bleeding, infection, toxic shock syndrome and hypoxia.[15,17] Surgical approaches include transantral ligation of the distal IMAX and ligation of the ethmoid arteries for superior epistaxis.[3] More recently endoscopic cauterisation and arterial ligation techniques have been described, although these are technically more demanding.[18-20] Since the advent of nasal embolisation in 1974,[7] the endovascular management of epistaxis has become an established alternative to surgical ligation. In a comparison of efficacy between transantral ligation and embolisation in intractable epistaxis, Strong *et al.*[21] reported success rates from the literature of 85% and 90% and average complication rates of 28% and 27% respectively for ligation and embolisation, with success rates of 89% and 94% respectively from their own series. Cullen and Tami[22] showed a failure rate of 21% for embolisation versus 27% for ligation (with or without ethmoid ligation) and an overall complication rate of 16% for embolisation and 18% for IMAX ligation.[22] In their review of the literature, which expanded on that by Strong *et al.*, they reported a slightly higher failure rate for embolisation (20%) than for IMAX ligation (18%) but a significantly higher complication rate for IMAX ligation (26%) than for embolisation (14%). No difference in major complications rates (5% v. 4%) was noted between the two methods.

The goal of embolisation is simply to reduce the arterial pressure head to the affected region without causing any ischaemic damage to the nasal soft tissues,[13] so allowing the body to heal itself. The posterior nasal cavity is supplied mainly via the sphenopalatine artery and greater palatine artery, both of which are terminal branches of the IMAX. This means that the management of posterior epistaxis largely hinges on control of the sphenopalatine artery,[23] and explains the equally high rate of success of ipsilateral IMAX embolisation alone. However, there is still a failure rate of 10 - 15% for both embolisation and IMAX/sphenopalatine ligation. In some cases embolisation may succeed where IMAX ligation fails.[24] Furthermore, as shown by Vitek[11] the technical success rate of embolisation increases significantly when additional embolisation of the ipsilateral facial and contralateral IMAX arteries is performed as well. This attests to the importance of collateral vessels in the facial region with collateral blood supply to the posterior nasal cavity, which probably accounts for a significant percentage of failed IMAX ligation or embolisation procedures.[25-27] The advantage of arteriography is that it can often identify these collateral sources, allowing them to be treated simultaneously. Collateral supply to the nasal cavity was important in 4 of our cases, leading to recurrent haemorrhage. In 1 case this involved a collateral pathway via the accessory meningeal artery, a hitherto undescribed variant of importance in the treatment of epistaxis.[28] Collateral supply via the ethmoid arteries led to secondary surgical intervention in 2 cases. Another potential complication related to collateral circulation is the presence of intracranial-extracranial arterial anastomoses, as inadvertent embolisation through these pathways can lead to ophthalmological or neurological complications.[12] It is therefore vital to identify these communications during the initial angiographic investigation. Although we routinely check both internal and external carotid arteries during each proce-

dure, we do not follow a rigid treatment protocol with regard to the number of vessels embolised but rather tailor the approach in each patient on the basis of the vascular anatomy, ease or difficulty of selective and superselective catheterisation, and the presence or absence of other associated extracranial vascular disease. Nearly 50% of our cases involved single-vessel (ipsilateral IMAX) embolisation only.

Two of our cases with a rebleed following initial embolisation of the ipsilateral IMAX underwent a second procedure involving a repeat microparticulate embolisation of the same vessel, with good outcome. Inadequate embolisation, like incomplete IMAX ligation, can therefore lead to recurrent bleeding. One patient who had a rebleed 2 days after embolisation underwent a repeat arteriogram that showed adequate occlusion of the ipsilateral distal IMAX branches. No significant collateral vessels were identified, so no further embolisation was performed. No further bleeding was experienced thereafter. False rebleeds following embolisation may occasionally be encountered and may be due to retained blood in and around the nasal packing or blood draining from the paranasal sinuses.

We used microcatheter techniques in all cases but 1. Although leading to increased expense, procedural duration and complexity, the use of microcatheters has been reported to reduce the number of local cranial complications although to date this has not been proven in any randomised study.[29,30] In Table V we review four studies conducted during or after 1995 and reporting the routine use of microcatheter techniques and calculate a cumulative success rate of 90.2% and overall complication rate of 13.4% (11.4% minor and 2% major) with microcatheter use. Although the use of microcatheter techniques does not improve the overall success rate, there is a tendency towards a lower complication rate. This factor should be weighed up against the additional procedural costs involved. We have also noted that when microcatheters are used, arterial vasospasm, which can prolong and complicate a procedure, is less frequent. One problem apparent in all of the embolisations reported to date is inconsistency in the reporting of minor and major complications and hence variation in the reported figures. Other factors such as improvements in catheter and guidewire design and increased operator experience have also probably contributed to the reduction in complication rates in more recent times.

Other complications related to embolisation for epistaxis reported in other local institutions include ischaemic necrosis of the upper lip (facial arterial embolisation), unilateral trismus (related to deep temporal arterial occlusion) and ischaemic sialadenitis (facial artery embolisation).[31] Further complications described in the literature include facial nerve paralysis, tongue necrosis, tonsillar ulceration, facial atrophy and transient submandibular gland swelling.[32] Other potential complications can be related to the femoral access (pain, bruising, pseudoaneurysm or arteriovenous fistula) or to the use of contrast media (allergic reaction, renal failure).[33] Our single major complication was a stroke related to attempted replacement of a guiding catheter in a very tortuous carotid artery. Despite our recent success in treating hyperacute iatrogenic strokes during neuro-interventional procedures with intra-arterial abciximab, we were unable to prevent major cerebral infarction in this case.[34]

Table V. Reported success and complication rates since 1995

Study	No. of patients	Success rate	Minor complications	Major complications	Overall complication
Elahi *et al.*, 1995[32]	57	52 (96%)	None reported	3 (6%)	3 (6%)
Tseng *et al.*, 1998[2]	114	101 (88%)	17 (15%)	2 (1.8%)	19 (16.8%)
Leppanen *et al.*, 1999[29]	37	33 (89%)	4 (8%)	0 (0%)	4 (8%)
Oguni *et al.*, 2000[33]	37	35 (94.6%)	7 (45%)	0 (0%)	7 (45%)
Total	245	221 (90.2%)	28 (11.4%)	5 (2%)	33 (13.4%)

All head and neck embolisation procedures should therefore ideally be performed by a team capable of managing serious cerebrovascular complications.

One advantage of endovascular treatment is that it can be performed under local anaesthesia with sedation if required. Although only 38.6% of our cases were performed under general anaesthesia, we now routinely utilise general anaesthesia for two reasons arising from our accumulated experience. Firstly, patients are generally uncomfortable, with nasal packing *in situ* and occasionally also active bleeding during the procedure. Prolonged immobilisation on the angiography table may be required during difficult cases. This results in further patient discomfort and reduced co-operation with movement (voluntary and involuntary), rendering the use of electronic vascular roadmapping useless, further prolonging the procedure and adding to the risk of complications or an incomplete or inadequate procedure. Secondly, we now insist on full airway protection by means of a cuffed endotracheal tube before commencing any embolisation procedure in the head and neck region where active bleeding is an issue.[28] Regardless of whether or not a general anaesthetic is administered, an anaesthetist is always present for patient monitoring and administration of analgesic or sedative drugs as required.

One subgroup of patients who develop recurrent epistaxis are those with HHT (Osler-Weber-Rendu disease). HHT is a genetic multisystemic angiodysplasia. These patients develop fragile nasal mucosal telangiectasias and more than 90% are are prone to repeated and intractable epistaxis[18] (Figs 4a and 4b). The ENT surgeon can miss the diagnosis of HHT during the acute presentation.[35] Embolisation is not a definitive treatment for epistaxis in these patients, but can control an acute bleeding episode. Because of the known propensity for epistaxis to recur in these patients, Elden *et al.*[30] found that their long-term success rate for embolisation increased from 82% to 90% once the HHT patients in their series were excluded. There is still no definitive treatment available for the nasal manifestations of HHT. Palliative treatment with intramucosal injections of bleomycin (Blenoxane, Bristol-Myers Squibb) has been successful in our patient for more than 18 months.[36]

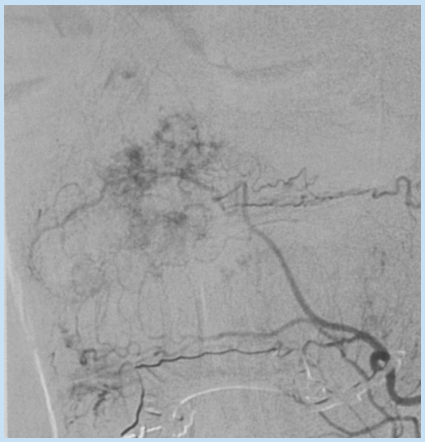

Fig 4a. Selective left facial arteriogram in our HHT patient showing multiple enhancing lesions representing small nasal mucosal telangiectasias.

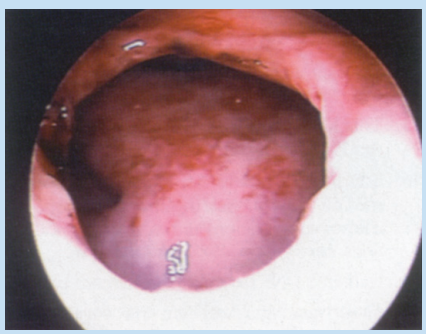

Fig 4b. Endoscopic nasal view showing multiple small red telangiectasias seen through a perforation of the nasal septum, a complication of repeated surgical attempts at managing the recurrent epistaxis in this patient.

Although embolisation is a good alternative to surgical IMAX ligation (or related procedures), interventional expertise is only available in the major centres in South Africa. Of our patients 70% were referred from outside of the Pretoria area, although most were from within a 150 km radius of the Unitas Interventional Unit. In their 1998 study of the attitudes of practising otorhinolaryngologists in Ohio, Cullen and Tami[22] found that only 16% had referred patients for embolisation; of these 75% had urban practices and the rest were from rural areas. They also found that more hospitals in urban areas than rural ones had embolisation facilities. This situation is mirrored and undoubtedly amplified in South Africa. In their comparison of the respective costs of IMAX ligation and embolisation, Strong *et al.*[21] found the average cost per case in 1994 for antral ligation to be US$5 941 versus US$6 783 for embolisation.[21] Cullen and Tami[22] calculated the average costs in their institution in 1998 to be US$ 6 184.55 for IMAX ligation and US$ 4 544.85 for embolisation. These figures include the combined costs of hospitalisation, treatment and consumable items. We were only able to calculate the radiological costs per case, which included the embolisation procedure and interventional theatre costs, as shown in Table VI. Not included in these figures are the costs of hospitalisation and private anaesthetist and ENT specialist fees. The overall cost of hospitalisation would be extremely difficult to calculate, as many patients had already been treated or hospitalised at other institutions before transfer to our own. In general,

Table VI. Average costs per embolisation procedure

Year	Rands	US dollars ($1 = R8)
1999	R 16 628	US$2 078
2000	R 15 993	US$1 999
2001	R 18 551	US$2 319
2002	R 22 761	US$2 845
2003	R 22 136	US$2 767

The above excludes all hospitalisation costs and professional private fees for the referring ENT specialist and anaesthetist.

uncomplicated cases would be admitted overnight for observation either in a high-care facility or more usually a general ward, with discharge or transfer back to the referring institution during the following day.

Conclusion

Intractable epistaxis is a debilitating condition, the treatment of which can be extremely difficult. Percutaneous transcatheter embolisation of the nasal arteries is an accepted alternative to surgical intervention, with comparable major and minor complication rates, success rates and overall costs.

Most cases will respond well to a single embolisation procedure, with approximately 10% failing to respond due to factors such as collateral arterial supply and HHT. Success and complication rates for procedures performed at the Unitas Interventional Unit compare favourably with those reported in the literature to date. These procedures should be performed by suitably experienced interventional radiologists, either under general anaesthesia or local anaesthesia with sedation, with full monitoring and anaesthetic backing.

References

1. Small M, Murray J, Maran AG. A study of patients with epistaxis requiring admission to hospital. *Health Bull (Edinb)* 1982; **40:** 20-29.
2. Tseng EY, Narducci CA, Willing SJ, Silliers MJ. Angiographic embolization for epistaxis. *Laryngoscope* 1998; **108:** 615-619.
3. Chandler JR, Serrins AJ. Transantral ligation of the internal maxillary artery for epistaxis. *Laryngoscope* 1965; **75:** 1151-1160.
4. Wang L, Vogel DH. Posterior epistaxis: Comparison of treatment. *Otolaryngol Head Neck Surg* 1981; **89:** 1001-1006.
5. Small M, Maran AGD. Epistaxis and arterial ligations. *J Laryngol Otol* 1984; **98:** 281-284.
6. Hunter K, Gibson R. Arterial ligation for severe epistaxis. *J Laryngol Otol* 1989; **83:** 1099-1103.
7. Sokoloff J, Wickbom I, McDonald D, Brahme F, Goergen TG, Goldberger LE. Therapeutic percutaneous embolization in intractable epistaxis. *Radiology* 1974; **111:** 285-287.
8. Van Wyck LG, Vinuela F, Hoeneman H. Therapeutic embolization for severe epistaxis *J Otolaryngol* 1982; **11:** 271-274.
9. Parnes LS, Hoeneman H, Vinuela F. Percutaneous embolization for control of nasal blood circulation. *Laryngoscope* 1987; **97:** 1312-1315.
10. Hicks JN, Vitek JJ. Transarterial embolization to control posterior epistaxis. *Laryngoscope* 1989; **99:** 1027-1029.
11. Vitek JJ. Idiopathic intractable epistaxis: Endovascular therapy. *Radiology* 1991; **181:** 113-116.
12. Kagetsu NJ, Berenstein A, Choi IS. Interventional radiology of the extracranial head and neck. *Cardiovasc Intervent Radiol* 1991; **14:** 325-333.
13. Connors JJ III, Wojak JC. Epistaxis. In: Connors JJ III, Wojak JC, eds. *Interventional Neuroradiology: Strategies and Practical Techniques.* Philadelphia: WB Saunders, 1999: 147-156.
14. Koh E, Frazzini VI, Kagetsu NJ. Epistaxis: Vascular anatomy, origins and endovascular treatment. *Am J Roentgenol* 2000; **174:** 845-851.
15. Tan LKS, Calhoun KH. Epistaxis. *Med Clin North Am* 1989; **83:** 43-56.
16. Schaitken B, Strauss M, Houck JR. Epistaxis: medical versus surgical therapy- a comparison of efficacy, complications and economic considerations. *Laryngoscope* 1987; **97:** 1392-1396.
17. Fairbanks DNF. Complications of nasal packing *Otolaryngol Head Neck Surg* 1986; **94:** 412-415.
18. Elwany S, Abdel-Fatah H. Endoscopic control of posterior epistaxis. *J Laryngol Otol* 1996; **110:** 432-434.
19. Winstead W. Sphenopalatine artery ligation: An alternative to internal maxillary ligation for intractable posterior epistaxis. *J Laryngol* 1996; **106:** 667-669.
20. Pritikin JB, Caldarelli DD, Panje WR. Endoscopic ligation of the internal maxillary artery for treatment of intractable posterior epistaxis. *Ann Otol Rhinol Laryngol* 1998; **107:** 85-91.
21. Strong EB, Bell DA, Johnson LP, Jacobs JM. Intractable epistaxis: Transantral ligation versus embolization: Efficacy review and cost analysis. *Otolaryngol Head Neck Surg* 1995; **113:** 674-678.
22. Cullen MM, Tami TA. Comparison of internal maxillary artery ligation versus embolization for refractory posterior epistaxis. *Otolaryngol Head Neck Surg* 1998; **118:** 636-642.
23. Simpson GT, Janfaza P, Becker GD. Transantral sphenopalatine artery ligation. *Laryngoscope* 1982; **92:** 1001-1005.
24. Breda SD, Choi IS, Persky MS, Weiss M. Embolization in the treatment of epistaxis after failure of internal maxillary artery ligation. *Laryngoscope* 1989; **99:** 809-813.
25. Hacien-Bay L, Rosenbloom JS, Pile-Spellman J, *et al.* Anastomoses in recurrent epistaxis. *J Vasc Intervent Radiol* 1997; **8:** 535-538.
26. Lasjaunias P, Marsot-Dupuch K, Doyon D. The radio-anatomical basis of arterial embolization for epistaxis. *J Neuroradiol* 1979; **6:** 45-53.
27. Lasjaunias P, Berenstein A. *Surgical Neuroangiography: Functional Anatomy of Craniofacial Arteries.* Vol 1. Berlin: Springer-Verlag, 1987.
28. Duncan IC, Dos Santos C. Accessory meningeal arterial supply to the posterior nasal cavity: Another reason for failed endovascular treatment of epistaxis. *Cardiovasc Intervent Radiol* 2003; **26:** 488-491.
29. Leppanen M, Seppanen S, Loranne J, Kuoppala K. Microcather embolization of intractable idiopathic epistaxis. *Cardiovasc Intervent Radiol* 1999; **22:** 499-503.
30. Elden L, Montanera W, TerBrugge K, Willinsky R, Lasjaunias P, Charles D. Angiographic embolization for the treatment of epistaxis: A review of 108 cases. *Otolaryngol Head Neck Surg* 1994; **111:** 44-50.
31. Duncan IC, Spiro FI, Van Staden D. Acute ischaemic sialadenitis following facial artery embolization. *Cardiovasc Intervent Radiol* 2004; **27:** 300-302.
32. Elahi MM, Panes LS, Fox AJ, Pelz DM, Lee DM. Therapeutic embolization in the treatment of epistaxis. *Arch Otolaryngol Head Neck Surg* 1995; **121:** 65-69.
33. Oguni T, Korogi Y, Yasunaga T, *et al.* Superselective embolization for intractable idiopathic epistaxis. *Br J Radiol* 2000; **73:** 1148-1153.
34. Duncan IC, Fourie PA. Catheter-directed intra-arterial abciximab administration for acute thrombotic occlusions during neurointerventional procedures. *Interventional Neuroradiology* 2002; **8:** 159-168.
35. Haitjema T, Balder W, Disch FJ, Westermann CJ. Epistaxis in hereditary haemorrhagic telangiectasia. *Rhinology* 1996; **34:** 176-178.
36. Duncan IC, Van der Nest L. Intralesional bleomycin injections (IBI) for the palliation of epistaxis in hereditary haemorrhagic telangiectasia. *Am J Neuroradiol* 2004; **25:** 1144-1146.

A shortened version of this article was published in the *South African Medical Journal* 2004; **94:** 373-378.