

Case Report

DOI: <https://dx.doi.org/10.18203/2349-2902.ijssurgery20253458>

Bilateral inferior turbinate necrosis and septal perforation as a complication of nasal packing and sphenopalatine artery ligation for intractable epistaxis

Peter J. Eves^{1,2*}, Brendan Wright^{1,2}, Robin A. Adair^{1,2}

¹Belfast Health and Social Care Trust, Belfast, Northern Ireland, United Kingdom

²Royal Victoria Hospital, Belfast, Northern Ireland, United Kingdom

Received: 02 August 2025

Revised: 06 September 2025

Accepted: 22 September 2025

*Correspondence:

Dr. Peter J. Eves,

E-mail: pevesSR-eves@hotmail.co.uk

Copyright: © the author(s), publisher and licensee Medip Academy. This is an open-access article distributed under the terms of the Creative Commons Attribution Non-Commercial License, which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

ABSTRACT

A male patient in his mid-70s was referred to ENT services at a regional centre with severe epistaxis. After failing conservative management, including nasal cauterity and bilateral packing, the patient proceeded to have an uncomplicated left sphenopalatine artery (SPA) ligation. Six months later he developed nasal blockage and crusting and blew a large amount of foul-smelling necrotic tissue from his nose. Examination and investigation findings revealed loss of most of his nasal septum and both inferior turbinates. This occurred as a rare complication of nasal packing and SPA ligation causing ischaemic necrosis.

Keywords: SPA ligation, Ischaemic necrosis, Septal perforation, Inferior turbinate, Rapid rhino

INTRODUCTION

Epistaxis is a very common presentation to emergency departments and source of referrals to ear nose and throat (ENT) services with 5-15% of admissions requiring surgical intervention.¹ The main blood supply to the posterior aspect of the nose is via the sphenopalatine artery, its ligation remains the favoured operative approach to treat posterior epistaxis resistant to conservative treatment options.² The first reports of transnasal sphenopalatine artery (SPA) ligations were reported in 1992 as nasal endoscopy overtook the operating microscope.³ Since, the procedure has seen widespread adoption into UK practice with recent years demonstrating increased and earlier use despite anatomical variation and a lack of strong supporting evidence.⁴ Success rates are reported to be up to 95% with few significant complications.¹

Complications reported include nasal crusting, numbness of teeth, palate and upper lip, acute sinusitis, decreased

lacrimation and septal perforation.⁵ Incidence of ischaemic necrosis complicating epistaxis management however are very rare with only a few case reports in the literature. In this case report we present a patient who had bilateral necrosis of his inferior turbinates and an enormous septal perforation after nasal packing and a left SPA ligation for epistaxis.

CASE REPORT

A male in his mid-70s presented with recurrent left sided epistaxis, he initially had a bleed that was controlled by emergency services. The patient received silver nitrate cauterity to Little's area several days after in an ENT outpatient clinic. He then represented with ongoing bleeding a few days later, again this was managed in ED and the patient discharged with advice to return if bleeding continued. The patient returned to the department the same evening before being referred to the ENT team. His past medical history included an extensive cardiac history with aortic stenosis, mitral regurgitation and ischaemic heart

disease which required aortic valve replacement and single vessel coronary artery bypass graft 10-15 years prior to presentation and as a result he was taking Aspirin and Simvastatin. He also was under surveillance for a low-grade urothelial carcinoma of the bladder. No history of previous problems or surgical procedures to his nose.

On presentation his haemoglobin was 105 g/dl (153 several years prior), later that evening this had dropped to 85. His urea increased to 10.7 mmol/l from 9.5 and a mild acute kidney injury was noted, progressing from a creatinine of 113 to 120 μmol/l and an estimated glomerular filtration rate (eGFR) of 55 to 51 (with a baseline creatinine of 92 and eGFR >60). Coagulation screen was also checked at the same times and on both occasions were completely normal.

Treatment

After referral to ENT the patient had his left nostril packed with a rapid rhino and 7 ml of air was insufflated. He was admitted for observation. A couple of hours later further bleeding was noted and an extra 3 ml of air was placed into the left side and a second rapid rhino was inserted to the right side. Given the haemoglobin drop from 105 g/l to 81 g/l the patient received a transfusion of two units of packed red cells. The packs remained in situ for 36 hours before attempt was made at removing the right side. Unfortunately, fresh blood from the right nostril was seen, the pack reinserted, inflated with 14 ml of air and left in-situ for a further 24 hours while arrangements were made for SPA ligation. The patient reported in retrospect having had significant nasal discomfort during this period. The pain was managed at the time with oral or intravenous paracetamol as required with no complaints of unmanaged pain reported in either medical or nursing notes.

Intraoperatively the right side of the patient's nose had no active bleeding after washout, however bleeding was noted from the left septum, left middle turbinate and SPA area. Packing trauma was noted bilaterally, but no evidence of necrosis seen. A single dose of Co-Amoxiclav was given intraoperatively. The SPA main branch was visualized adequately, isolated with a ball probe and cauterized with bipolar forceps. The patient's post-operative recovery was smooth and was discharged the following day without any follow up. On discharge he was given Iron supplementation and paracetamol.

Outcome and follow-up

Just over six months later the patient presented to his general practitioner (GP) with a 6-month history of nasal blockage. He had felt a sensation of something sliding down his left nostril and then removed a large quantity of brown foul-smelling material. On examination the GP noted partially obstructed nasal passages with crust, a foul odour and a perforated nasal septum and referred the patient back to ENT services assuming there was a foreign body left behind post-operatively. Sending the material for

histopathology the pathologist noted the presence of a central plate of cartilage and bone of the nasal septum. The attached soft tissues were impressive, entirely necrotic and overrun by colonies of commensal bacterial and fungal organisms including aspergillus. With ENT input he was started on saline and then budesonide rinses and seen in clinic with the pathology report. The patient at this stage was feeling much improved with minimal symptomatology, he had some minor crusting but no foul smell, bleeding or pain.

On examination an enormous subtotal septal perforation was noted with loss of his inferior turbinates and the right middle turbinate adherent to the lateral nasal wall. There was a large foul-smelling crust on the nasal floor which was removed to reveal a slightly inflamed mucosa. The appearance was not in keeping with malignancy or an invasive fungal infection.

A remnant of the posterior left inferior turbinate was biopsied but only demonstrated ulcer slough with active chronic inflammation, no features of malignancy, infection or vasculitis were noted. A vasculitis screen was completed including ANA, ANCA and ESR which returned normal and a CT of his sinus corroborated examination findings demonstrating the absence of the inferior turbinates and most of the nasal septum with no bony sclerosis or destructive process and no features suggestive of malignancy (Figure 1).



Figure 1: Coronal CT image.

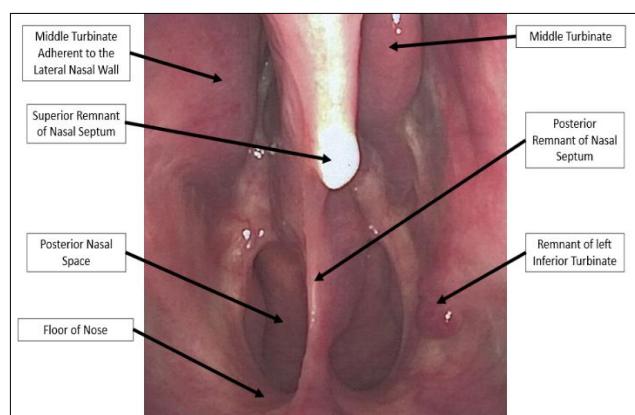


Figure 2: Flexible nasal endoscopy image.

The patient was reviewed one month after his initial post-operative referral and found to be totally asymptomatic; flexible nasal endoscopy revealed the area of mucosal inflammation to have completely resolved (Figure 2).

DISCUSSION

There are just a few case reports of similar complications describing ischaemic necrosis after SPA ligation, and only one affecting the inferior turbinates.⁶ In this report a 70 male who had had his nose packed on at least 5 occasions in a 10–11-week period with at least 4 of the nasal packings within a 4–5-day period. The patient also had a left sided SPA ligation but only developed necrosis of the ipsilateral turbinate and without any septal perforation. This patient also experienced an uneventful recovery post operatively with exception of persistent nasal crusting which in this case was removed during EUA where it was discovered that the inferior turbinate had indeed necrosed.⁶ In our case there was bilateral turbinate necrosis with septal perforation after unilateral SPA ligation. It may be that in this case extensive atherosclerotic disease contributed to his developing such extensive and bilateral destruction of his intranasal structures, perhaps vessel narrowing even resulted in a collateral supply of the right inferior turbinate by the left SPA. Interestingly however this is not the only case of bilateral necrosis. A case report of a 45-year-old male with minimal medical history but significant smoking history presented with intractable posterior epistaxis, he received nasal packing only once and on failure received bilateral SPA ligation. He represented 4 months later with bilateral nasal crusting and nasal obstruction, again after EUA nasal crusting was removed to reveal necrosis of the inferior aspects of both middle turbinates and a posterior septal perforation.⁷ In another case report a 53 year-old patient who had bilateral SPA embolization resulted in necrosis of the posterior 2/3 of the hard palate demonstrating rare anatomical variability in blood supply or perhaps inadvertent embolization of more than one branch of the maxillary artery.⁸

There is a wide range of anatomical variation of the SPA and its branches which have been well documented with excellent collateral supply with other vessels originating from both internal and external carotid arteries making necrosis after SPA ligation very unusual, especially after a unilateral procedure.^{9–12} In reference specifically to the inferior turbinate Padgham et al in a cadaveric study demonstrated that the two terminal branches of the posterior lateral nasal artery that supply the inferior turbinate increase in diameter as they approach the anterior aspect of the turbinate. They felt this likely represented anterior anastomosis with the facial artery or other intranasal vessels. Rare anatomical variations in the dominance of SPA blood supply were suggested in these case reports, and in this case left sided dominance with unusually absent or insufficient arterial anastomosis and minimal right sided contribution. Interestingly one case report describes an anomaly where SPA contribution was

negligible bilaterally with the majority of blood supply to the nasal mucosa being supplied by the anterior ethmoidal arteries.¹³ In this case the SPA could not be found with endoscopic dissection, angiography was then performed to identify dominant anterior ethmoidal vessels.

CONCLUSION

This case study demonstrates that nasal packing and SPA ligation for epistaxis may have ischaemic complications with loss of nasal structures such as the turbinates and septum. Prolonged nasal packing for epistaxis with associated pain may be a risk factor for ischaemic complications. Clinicians should consider the possibility of necrosis and loss of nasal structures in patients presenting with nasal blockage and crusting after nasal packing and SPA ligation.

Funding: No funding sources

Conflict of interest: None declared

Ethical approval: Not required

REFERENCES

1. Basnet M, Ghimire B, Shrestha A, Aryal GR. Success rate of endoscopic sphenopalatine artery ligation for the management of refractory posterior epistaxis patients in a tertiary care hospital: A descriptive cross-sectional study. *J Nepal Med Assoc*. 2020;58(232):1056-60.
2. Agreda B, Urpegui Á, Alfonso JI, Valles H. Ligation of the Sphenopalatine Artery in Posterior Epistaxis. Retrospective Study of 50 Patients. *Acta Otorrinolaringologica (English Edition)*. 2011;62(3):194-8.
3. Romeo B, Eiccardo S. Microscopic and endoscopic ligation of the sphenopalatine artery. *Laryngoscope*. 1992;102(12):1390-4.
4. Ellinas A, Jervis P, Kenyon G, Flood LM. Endoscopic sphenopalatine artery ligation for acute idiopathic epistaxis. Do anatomical variation and a limited evidence base raise questions regarding its place in management? *J Laryngol Otol*. 2017;131:290-7.
5. Snyderman CH, Goldman SA, Carrau RL, Ferguson BJ, Grandis JR. Endoscopic sphenopalatine artery ligation is an effective method of treatment for posterior epistaxis. *Am J Rhinol*. 1999;13(2):137-40.
6. Moorthy R, Anand R, Prior M, Scott PM. Inferior turbinate necrosis following endoscopic sphenopalatine artery ligation. *Otolaryngol-Head and Neck Surg*. 2003;129(1):159-60.
7. Elsheikh E, El-Anwar MW. Septal perforation and bilateral partial middle turbinate necrosis after bilateral sphenopalatine artery ligation. *J Laryngol Otol*. 2013;127(10):1025-7.
8. Adam S, Sama HD, Chossegros C, Bedrune B, Chesnier I, Pradier JP. Nécrose palatine iatrogène par embolisation des artères sphéno-palatines au décours

du traitement d'une épistaxis rebelle. Rev Stomatol Chir Maxillofac Chir Orale. 2015;116(3):170-2.

9. Pádua FGM, Voegels RL. Severe posterior epistaxis-endoscopic surgical anatomy. Laryngoscope. 2008;118(1):156-61.

10. Simmen DB, Raghavan U, Briner HR, Manestar M, Groscurth P, Jones NS. The anatomy of the sphenopalatine artery for the endoscopic sinus surgeon. Am J Rhinol. 2006;20(5):502-5.

11. Lee HY, Kim HU, Kim SS, Son EJ, Kim JW, Cho NH, et al. Surgical anatomy of the sphenopalatine artery in lateral nasal wall. Laryngoscope. 2002;112(10):1813-8.

12. Padgham N, Vaughan-Jones R. Cadaver studies of the anatomy of arterial supply to the inferior turbinates. J R Soc Med. 1991;84(12):728-30.

13. Biswas D, Ross SK, Sama A, Thomas A. Non-sphenopalatine dominant arterial supply of the nasal cavity: An unusual anatomical variation. J Laryngol Otol. 2009;123(6):689-91.

Cite this article as: Eves PJ, Wright B, Adair RA. Bilateral inferior turbinate necrosis and septal perforation as a complication of nasal packing and sphenopalatine artery ligation for intractable epistaxis. Int Surg J 2025;12:1976-9.