CASE REPORT

Surgical repair following trauma to vascular graft causing spinal cord infarction

Sayinthen Vivekanantham,¹ Gokulan Phoenix,² Chetan Khatri,¹ Saroj Das³¹Faculty of Medicine, Imperial College London, London, UK²Department of General Surgery, Chelsea and Westminster Hospital, London, UK³Department of General and Vascular Surgery, The Hillingdon Hospital, Uxbridge, UK**Correspondence to**

Sayinthen Vivekanantham, sayinthen.vivekanantham@gmail.com

Accepted 27 March 2014

SUMMARY

A 55-year-old woman with a background of vascular disease presented with signs of bilateral limb ischaemia. Following elective axillobifemoral bypass and hospital discharge, accidental axillary trauma causing a chest wall haematoma, the patient underwent an emergency graft repair. Postextubation, she reported with absent sensation in her legs. Spinal cord infarction was diagnosed through clinical assessment and exclusion of other causes. The aetiology of compromise to the spinal cord blood supply is unclear. Possibilities include intraoperative hypotension, inadvertent compromise to blood supply of thoracic radicular arteries, dislodged atherosclerotic emboli or a combination of these factors. Spinal cord infarction recognised early can be treated. Sedation to assist ventilation had obscured the problem early enough to consider treatment. Patients with vascular risk factors should be carefully managed intraoperatively to minimise hypotensive episodes and care should also be taken not to compromise blood flow of radicular arteries.

BACKGROUND

Paraplegia secondary to spinal cord infarction is rare and accounts for only 1% of all strokes,¹ with sparse literature describing the clinical phenomenon and few large clinical investigations.² The condition is very disabling, causing previously well patients to become unable to walk and often incontinent too. Perhaps due to its low incidence, our understanding of the pathogenesis and natural progression remains largely unknown, with the exact aetiologies behind many cases unidentified.² We report a case of a middle-aged woman who developed spinal cord infarction postoperatively after vascular surgery, where the exact aetiology is unclear. Our case highlights that vast atherosclerotic disease is likely to place patients at higher risk of developing spinal cord infarction intraoperatively. Care should be taken to minimise hypotensive episodes and interference with other blood vessels when managing such patients.

CASE PRESENTATION

A 55-year-old Caucasian woman with multiple comorbidities presented to our vascular department with rest pain and signs of bilateral limb ischaemia in July 2011. Her history included type 2 insulin-dependent diabetes mellitus, drug-resistant hyperlipidaemia, polyvascular atherosclerotic disease, hypertension due to bilateral renal artery stenosis and recurrent venous thromboembolism (treated by indefinite warfarin). Since undergoing a left iliac

angioplasty and a femorofemoral crossover externally-supported Dacron graft (EXS Dacron) for right iliac occlusion in July 1996, she has had multiple angioplasties and stentings to her left iliac artery.

A CT angiogram following her recent presentation demonstrated that her femoral-femoral bypass graft had become occluded. In addition, both iliac arteries and the distal aorta were partially occluded up to the level of the left renal artery, which itself was perfusing through a stent. Furthermore, aneurysmal changes with severe thrombotic disease involving the superior mesenteric artery, coeliac artery and lower descending thoracic aorta were seen. The vast vascular pathology left few treatment options bar an elective open left axillobifemoral bypass graft to treat rest pain, which was performed in September 2011. Following an uncomplicated postoperative period with successful revascularisation of both lower limbs, she was discharged on the 11th postoperative day.

That same day, she returned to the hospital's emergency services with syncope, a large chest wall haematoma and bilateral ischaemic legs following a blunt traumatic injury, from a swinging car door in windy conditions, to her left chest wall. On re-presentation, her vital signs were initially preserved, but within 30 min her blood pressure began to fall and emergency surgical intervention to repair the graft was started. The acute presentation did not allow time for further investigations such as Doppler or CT angiography imaging. Endovascular repair via interventional radiology was considered; however, the rapid decline in vital signs favoured an open emergency repair.

She underwent exploration and evacuation of the haematoma, thrombectomy of the graft and refashioning of the axillary anastomosis under general anaesthetic. A left transverse infraclavicular incision and Fogarty catheter were used for the procedure. Intraoperative findings showed that the graft had become partially detached at the anastomosis to the subclavian artery. Intraoperatively, there were three separate episodes of hypotension, with the lowest blood pressure recorded being 82/52 mm Hg lasting between two and 5 min, which were managed with fluid boluses of Hartmann's solution. Having been kept ventilated on the intensive trauma unit for 72 h for correction of respiratory insufficiency and acute kidney injury, on extubation she presented with absent sensation in her legs. She had faecal and urinary incontinence, with flaccid tone in both lower limbs. Sensation to pain and temperature were absent below the level of T10, in

CrossMark

To cite: Vivekanantham S, Phoenix G, Khatri C, *et al.* *BMJ Case Rep* Published online: [please include Day Month Year] doi:10.1136/bcr-2013-202283

addition to fine touch and proprioception. The patient had a Medical Research Council grade III power on hip flexion, knee flexion and extension, with absent knee and ankle reflexes

bilaterally. The remainder of the neurological examination was normal including the upper limb, anal tone and perineal sensation.

Figure 1 Arterial blood supply to the spinal cord. Reproduced with permission.⁵

INVESTIGATIONS

MR angiography of the spine was performed urgently to rule out any reversible causes of continuing ischaemia. This demonstrated a normal blood supply to the spinal cord throughout without any evidence of impingement of the cord or conus medullaris, cord compression, haemorrhage or cauda equina at any spinal level. No further imaging was conducted to comply with the patient's wishes.

DIFFERENTIAL DIAGNOSIS

The dissociation of sensory loss was characterised by loss of pain and temperature sensation without preservation of kinesthesia and discriminative touch sensation. This would suggest a global spinal cord infarction, as opposed to a localised infarction affecting just the anterior or posterior spinal cord. Other potential differentials include trauma directly to the spinal cord. However, there was no radiological imaging to support this. Neither was there any direct impingement of the spinal cord during the blunt trauma or during her hospital care with us.

TREATMENT

Had the patient not been under sedation and ischaemic insult to the spinal cord been identified straight away, the cause of the ischaemia may have been more apparent. In addition, factors that increase ischaemic tolerance of the spinal cord while perfusion is low, such as hypothermia, cerebrospinal fluid drainage³ and drugs that mitigate ischaemic injury (steroids, excitatory neurotransmitter inhibitors and free radical scavengers), could also have been trialled.⁴ The patient was admitted to an intensive rehabilitation unit at the Hillingdon Hospital to improve functional capacity of the spinal cord.

OUTCOME AND FOLLOW-UP

A diagnosis of spinal cord infarction was made through exclusion of other common causes on MR angiography. No further imaging was performed on follow-up of the patient to follow her wishes and as neurologists felt that this would not change her clinical management. The patient gradually regained full continence and was able to walk 50 m while supported following 5 months of intensive therapy. Twelve months later, the patient is a wheelchair user. However, she has a well functioning vascular graft and is maintaining adequate perfusion to her lower limbs.

DISCUSSION

The vascular supply of the spinal cord is through three main arteries that run along the axis of the spinal cord (figure 1).⁵ The single anterior spinal artery supplies the anterior two-thirds of the spinal cord and two posterior spinal arteries supply the posterior one-third of the spinal cord. Radicular arteries anastomose with either the anterior or posterior spinal arteries at variable levels, with the posterior receiving input from six–nine unpaired radicular arteries in locations that are highly variable along its axis.⁶ The T1–T4 levels of the thoracic cord are particularly vulnerable to infarction even when a single radicular artery is compromised. The largest radicular artery supporting the anterior spinal artery is the artery of Adamkiewicz, also known as the *arteria radicularis magna*, which anastomoses between T9 and T12 (figure 2).²

The distribution of our patient's neurological symptoms would support global ischaemic injury of the spinal cord below the level of T10. A diagnosis of global infarction of the spinal cord is supported, as function is lost to anteriorly and posteriorly situated spinal tracts. Function below T10 is lost for the

Figure 2 Arterial blood supply to the spinal cord in relation to the aorta. Reproduced with permission.²

anteriorly situated spinothalamic spinal tract (mediating sensation of pain and temperature) and corticospinal tract (mediating motor movement to the lower trunk). In addition, function is also lost for the posteriorly situated dorsal columns tract (mediating sensation of fine touch and proprioception; figure 3).⁷

Had the arterial supply to the artery of Adamkiewicz been solely compromised, our patient's presentation would be expected to present with symptoms of an anterior spinal cord syndrome. This may have occurred secondary to the inadvertently compromising blood flow intraoperatively, or alternatively, a thrombosis or embolism may have occurred within the vessel. Because our operative intervention focused on the shoulder region and postoperative MR angiography failed to identify any vascular abnormalities in the artery of Adamkiewicz, it is unlikely that damage to this artery would be responsible, although the potential remains that the responsible pathology may have cleared in this artery by the time the imaging was taken.

The clinical picture suggests global ischaemia as opposed to just anterior cord syndrome. Hence, we believe that intraoperative hypotension is the most likely cause of the spinal cord

LONG TRACTS IN THE SPINAL CORD			
Key	Tract	Location	Function
Fasciculus gracile: dorsal columns (posterior)	Medial dorsal column	Proprioception from the leg Light touch Vibration
Same as above	Fasciculus cuneate: dorsal columns (posterior)	Lateral dorsal column	Proprioception from the arm Light touch Vibration
Spinocerebellar	Superficial lateral column	Muscular position and tone, unconscious proprioception
Lateral spinothalamic	Ventrolateral column	Pain and thermal sensation
	Ventral spinothalamic	Ventral column	Tactile sensation of crude touch and pressure
Lateral corticospinal tract (pyramidal)	Deep lateral column	Motor: Medial (cervical)-Lateral (sacral) C → S (motor neuron distribution)
Anterior corticospinal tract	Medial ventral column	Motor: Neck and trunk movements

Figure 3 Transverse cross section of the spinal cord showing distribution of the spinal cord tracts. Reproduced with permission.⁷

infarction in our patient. The lumbar enlargement is particularly vulnerable to hypoperfusion when spinal arterial blood flow is compromised⁸ and the advanced atherosclerosis in our patient may have predisposed her to further reductions in blood flow.

Another potential mechanism might have been a disruption to the blood supply of the vertebral artery branching from the subclavian artery. The anterior spinal artery and posterior spinal arteries are downstream from the vertebral arteries; hence, blood supply to the distal spinal cord could have been compromised given this disruption to the blood flow upstream. It is also possible that during emergency exploration and refashioning of the axillary anastomosis, inadvertent damage or blood supply compromise could have been caused to the vertebral artery. Although great care was taken when handling the vasculature during the operative process, we believe hypotension to be the most probable cause. Atherosclerotic emboli may have been equally responsible for a partial block in these vessels supplying the spinal cord that later resolved before imaging of the vasculature was taken.

The exact aetiology of many cases with spinal cord infarction is unclear.² There have been various cases where spinal cord infarction has occurred secondary to vascular surgery, both on surgery of the aorta^{9–11} and other prominent vasculature.^{12–14}

However, this is the first case documented in the literature, to the best of our knowledge, where surgery on the subclavian artery has resulted in spinal cord infarction, bringing into question the possible involvement of the vertebral arteries. There are similarities between our case and existing spinal cord infarction cases documented in the literature, with severe atherosclerotic disease being a consistent feature^{2 15–17} and hypotensive episodes.^{8 18–22} It should be noted that cases of non-vascular surgical procedures have also caused spinal cord infarction as a complication,¹⁸ without episodes of hypotension.¹⁷ This clinical phenomenon has also occurred after the use of epidurals.^{6 8}

Our case, among others, highlights varied presentations that can result in this extremely disabling complication. Unfortunately, as our patient was sedated to assist ventilation, there was no opportunity to recognise the problem early enough for potential treatment. Our case also highlights the importance of neurological assessment following major vascular surgery, managing blood pressure in vascular patients with vast atherosclerotic disease intraoperatively and trying to minimise disruption to anastomosing radicular arteries. Intensive rehabilitation therapy through a multidisciplinary team has been crucial to our patient regaining continence and acquiring some limited

mobility. A multidisciplinary approach has been successful in treating other cases of spinal cord infarction too.²³

Learning points

- Mechanisms of spinal cord infarction in vascular surgery potentially include hypotensive episodes, block of vasculature by atherosclerotic emboli or disruption to the blood supply in the vertebral or radicular arteries.
- Patients with severe atherosclerosis and vascular risk factors should be carefully managed to avoid hypotensive episodes and minimise disruption to vasculature intraoperatively.
- Postoperatively after vascular surgeries, neurological examination is important to detect potentially modifiable causes of neurological injury.
- A multidisciplinary approach is effective in regaining some functional recovery in patients with spinal cord infarction.

Acknowledgements The authors would like to thank Mr Kumaran Shanmugarajah for his kind advice on the manuscript.

Contributors SV was responsible for design, extrapolating the lessons learnt from the initial case, complete drafting and final approval of the case report. GP, CK and SD were involved in advising about the case and reviewing the drafts.

Competing interests None.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

REFERENCES

- 1 Sandson TA, Friedman JH. Spinal cord infarction. Report of 8 cases and review of the literature. *Medicine* 1989;68:282–92.
- 2 Novy J, Carruzzo A, Maeder P, et al. Spinal cord ischemia: clinical and imaging patterns, pathogenesis, and outcomes in 27 patients. *Arch Neurol* 2006;63:1113–20.
- 3 Ullery BW, Cheung AT, Fairman RM, et al. Risk factors, outcomes, and clinical manifestations of spinal cord ischemia following thoracic endovascular aortic repair. *J Vasc Surg* 2011;54:677–84.
- 4 Acher C, Wynn M. Paraplegia after thoracoabdominal aortic surgery: not just assisted circulation, hypothermic arrest, clamp and sew, or TEVAR. *Ann Cardiothorac Surg* 2012;1:365–72.
- 5 Goshgarian H. *Blood supply of the spinal cord*. New York: Demos Medical Publishing, 2003.
- 6 Al-Asmi A, John R, Nandhagopal R, et al. Spinal cord infarction following abdominal surgery and postoperative epidural analgesia. *Sultan Qaboos Univ Med J* 2010;10:396–400.
- 7 Kirshblum SGP, Cuccurullo S. *Spinal cord injuries: transverse section of the spinal cord*. New York: Demos Medical Publishing, 2004.
- 8 Cekic G, De Kock M, Kremer Y, et al. Paraplegia after combined general and epidural anesthesia: a case report. *Acta Anaesthesiol Belg* 2008;59:95–8.
- 9 Wong DR, Coselli JS, Amerman K, et al. Delayed spinal cord deficits after thoracoabdominal aortic aneurysm repair. *Ann Thorac Surg* 2007;83:1345–55.
- 10 Winnerkvist A, Anderson RE, Hansson L-O, et al. Multilevel somatosensory evoked potentials and cerebrospinal proteins: indicators of spinal cord injury in thoracoabdominal aortic aneurysm surgery. *Eur J Cardiothorac Surg* 2007;31:637–42.
- 11 Marini CP, Cunningham JN. Issues surrounding spinal cord protection. *Adv Card Surg* 1993;4:89–107.
- 12 Delank KS, Delank HW, König DP, et al. Iatrogenic paraplegia in spinal surgery. *Arch Orthop Trauma Surg* 2005;125:33–41.
- 13 Stöckl B, Wimmer C, Innerhofer P, et al. Delayed anterior spinal artery syndrome following posterior scoliosis correction. *Eur Spine J* 2005;14:906–9.
- 14 Picone AL, Green RM, Ricotta JR, et al. Spinal cord ischemia following operations on the abdominal aorta. *J Vasc Surg* 1986;3:94–103.
- 15 Weidauer S, Nichtweiss M, Lanfermann H, et al. Spinal cord infarction: MR imaging and clinical features in 16 cases. *Neuroradiology* 2002;44:851–7.
- 16 Afshinmajd S, Khalaj A, Roohani Y, et al. Acute paraplegia after general anesthesia. *Acta Med Iran* 2011;49:560–4.
- 17 Saxena P, Abu Hasan F, Merry C. Spinal cord ischemia following coronary artery bypass surgery. *J Card Surg* 2011;27:45–6.
- 18 Singh U, Silver JR, Welpy NC. Hypotensive infarction of the spinal cord. *Paraplegia* 1994;32:314–22.
- 19 Duggal N, Lach B. Selective vulnerability of the lumbosacral spinal cord after cardiac arrest and hypotension. *Stroke* 2002;33:116–21.
- 20 Pashkovskaya I, Smith CE. Spinal cord infarction and paraplegia after peripheral vascular surgery with spinal anesthesia. *J Clin Anesth* 2004;16:440–4.
- 21 Weinberg L, Harvey WR, Marshall RJ. Post-operative paraplegia following spinal cord infarction. *Acta Anaesthesiol Scand* 2002;46:469–72.
- 22 Drummond JC, Lee RR, Owens EL. Case report: spinal cord ischemia occurring in association with induced hypotension for colonic surgery. *Anesth Analg* 2012;114:1297–300.
- 23 Matsuda H, Ogino H, Fukuda T, et al. Multidisciplinary approach to prevent spinal cord ischemia after thoracic endovascular aneurysm repair for distal descending aorta. *Ann Thorac Surg* 2010;90:561–5.

Copyright 2014 BMJ Publishing Group. All rights reserved. For permission to reuse any of this content visit <http://group.bmj.com/group/rights-licensing/permissions>.
BMJ Case Report Fellows may re-use this article for personal use and teaching without any further permission.

Become a Fellow of BMJ Case Reports today and you can:

- Submit as many cases as you like
- Enjoy fast sympathetic peer review and rapid publication of accepted articles
- Access all the published articles
- Re-use any of the published material for personal use and teaching without further permission

For information on Institutional Fellowships contact consortiasales@bmjgroup.com

Visit casereports.bmj.com for more articles like this and to become a Fellow