SNI
SURGICAL NEUROLOGY INTERNATIONAL
SNI: Trauma
OPEN ACCESS
For entire Editorial Board visit : http://www.surgicalneurologyint.com
Editor: Rahul Raj, MD Helsinki University Hospital, Helsinki, Finland

Case Report

Bilateral blunt cerebrovascular injury resulting in direct carotid-cavernous fistulae: A case report and review of the literature

Stephanie V. Avila#, Brooke Van Noy#, Michael Karsy[1], Matthew Alexander[2], John D. Rolston[1]

Departments of Neurobiology and Anatomy, [1]Neurosurgery, Clinical Neurosciences Center, [2]Radiology, University of Utah, Salt Lake City, Utah, USA

E-mail: Stephanie V. Avila - stephanie.avila@neuro.utah.edu; Brooke Van Noy - brookenhansen@gmail.com; Michael Karsy - Michael.karsy@hsc.utah.edu; Matthew Alexander - Matthew.alexander@hsc.utah.edu; *John D. Rolston - john.rolston@hsc.utah.edu
*Corresponding author
#Contributed equally to manuscript

Received: 26 June 18 Accepted: 10 September 18 Published: 19 November 18

Abstract

Background: Bilateral blunt cerebrovascular injury (BCVI) has been documented in 32 patients in the English-language literature and bilateral carotid-cavernous fistulae (CCFs) have been reported in only 1 patient. Here, we present a case of severe, unexpected bilateral BCVI with bilateral direct CCF and review the literature of BCVI, particularly cases of bilateral injury.

Case Description: A 65-year-old woman with episodic bradycardia presented after a motor vehicle accident. On arrival, she had a Glasgow Coma Scale of 3T and progressive dilation of her right pupil. Computed tomography imaging showed a 1.8-cm right epidural hematoma (EDH) with 6 mm of right-to-left shift. No acute skull-base fracture or injury in the area of the carotid canal was noted. The patient was treated with 3% hypertonic saline and mannitol before being taken to the operating room for emergent decompression of the hematoma. Although the patient initially presented with an EDH, significant intraoperative hemorrhage was identified during surgical evacuation and later confirmed as bilateral direct CCFs during angiographic evaluation. Because of the patient's devastating injuries, life-extending measures were not continued and the patient died.

Conclusions: A review of the literature indicates that bilateral CCFs are rare, having been reported only once previously. As this case demonstrates, CCFs may occur in high-energy injuries and should be considered even if the patient does not meet traditional screening criteria.

Key Words: Blunt cerebrovascular injury, carotid-cavernous fistula, dissection

Access this article online
Website: www.surgicalneurologyint.com
DOI: 10.4103/sni.sni_210_18
Quick Response Code:

INTRODUCTION

Blunt cerebrovascular injury (BCVI) is a potentially morbid result of traumatic brain injury that can result in dissection of blood vessels.[29,35] Dissections involve injury to the endothelial, intimal, or adventitial layers of the blood vessel,[4] and carotid dissections can be associated with development of carotid-cavernous fistula (CCF). High cervical injuries and significant force of injury are risk factors for traumatic CCF.[27] BCVIs occur in 1% of

This is an open access journal, and articles are distributed under the terms of the Creative Commons Attribution-NonCommercial-ShareAlike 4.0 License, which allows others to remix, tweak, and build upon the work non-commercially, as long as appropriate credit is given and the new creations are licensed under the identical terms.

For reprints contact: reprints@medknow.com

How to cite this article: Avila SV, Van Noy B, Karsy M, Alexander M, Rolston JD. Bilateral blunt cerebrovascular injury resulting in direct carotid-cavernous fistulae: A case report and review of the literature. Surg Neurol Int 2018;9:229.
http://surgicalneurologyint.com/Bilateral-blunt-cerebrovascular-injury-resulting-in-direct-carotid-cavernous-fistulae:-A-case-report-and-review-of-the-literature/

© 2018 Surgical Neurology International | Published by Wolters Kluwer - Medknow

patients with blunt trauma, and CCFs are seen in 0.2% of traumatic brain injury and 4% of patients with sustained basilar skull fractures.[14] Bilateral injures represent a subset of BCVI, accounting for at most 10% of highly selected case series;[28] however, traumatic bilateral CCF has only been reported once before.[16]

The use of computerized tomography angiography has enabled rapid evaluation of patients with BCVI; however, a high index of clinical suspicion is required because BCVI and CCF can be missed. For instance, the antiplatelet treatment compared with anticoagulation treatment for cervical artery dissection (CADISS) trial, which evaluated BCVI treatment modalities, demonstrated difficulty in diagnosing BCVI in up to 20% of cases.[7] These results and various guidelines suggest challenges in identifying BCVI even with advanced imaging techniques.[7,33,40]

We present a case of severe, unexpected bilateral BCVI with bilateral direct CCF and provide a review of the literature describing the diagnosis, management, and outcomes of bilateral BCVI.

CLINICAL PRESENTATION

A 65-year-old woman with unknown past medical history presented after a motor vehicle rollover in which she was restrained with a seatbelt. The patient was intubated at the scene, stabilized at an outside hospital, and transferred to our hospital. She exhibited episodic bradycardia en route but retained reactive pupils. On arrival, the patient's Glasgow Coma Scale (GCS) score was 3T with progressive dilation of her right pupil. Computed tomography (CT) imaging showed a 1.8-cm right epidural hematoma (EDH) with 6-mm right-to-left shift but no acute skull-base fracture or injury in the area of the carotid canal [Figure 1a, b]. Other injuries included extensive osseous and soft tissue injuries in the thorax. The patient was treated with 3% hypertonic saline and mannitol before undergoing emergent hematoma decompression.

We completed evacuation of the EDH without difficulty; however, significant unexpected bleeding was uncovered from the skull-base and carotid-cavernous triangle. There was worsening bleeding from deep within the Sylvian fissure, prompting us to open the dura to achieve additional decompression. Extradural coagulation of the middle meningeal artery and ipsilateral neck pressure reduced the bleeding, but incompletely. We packed the wound and proceeded for emergent angiography to identify the source of bleeding.

Digital subtraction angiography (DSA) showed a large dissecting pseudoaneurysm of the cavernous segment of the right internal carotid artery (ICA), with arteriovenous shunting into the cavernous sinus consistent with a direct CCF. Outflow was noted through the bilateral inferior

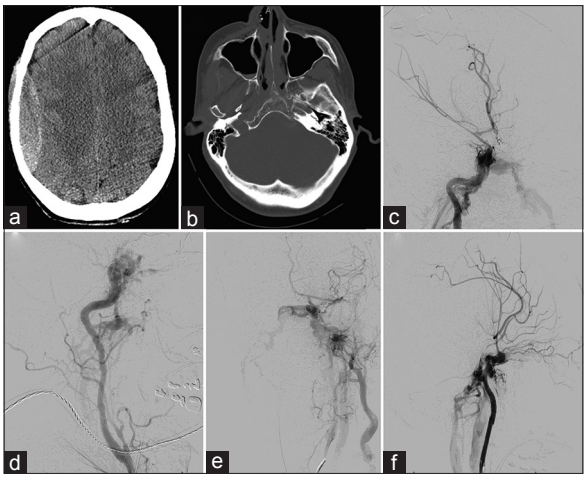

Figure 1: Bilateral blunt cerebrovascular injury (BCVI) with associated high-flow caroticocavernous fistulas (CCFs) is shown. (a) Non-contrast axial CT demonstrates a 1.8-cm right EDH with 6 mm of right-to-left shift. (b) Axial bone window CT shows no acute skull-base fracture or injury in the area of the carotid canal. (c) Townes and (d) lateral digital subtraction angiographic (DSA) images during right internal carotid artery (ICA) injection undertaken after the patient continued to have uncontrolled bleeding intraoperatively show a direct CCF with drainage through the bilateral inferior petrosal sinuses. Flow through the anterior cerebral (ACA) and middle cerebral (MCA) arteries was significantly slow, with displacement of the MCA branches due to mass effect. (e) Townes and (f) lateral DSA images of a left ICA injection show a direct CCF with outflow through the inferior petrosal sinuses, as well as egress through the left external jugular system. Flow through the ACA and MCA was diminished

petrosal sinuses with no visualized cortical venous reflux [Figure 1c, d]. Flow in the right anterior (ACA) and middle (MCA) cerebral artery circulation was sluggish, and there was displacement of the right MCA branches because of mass effect from residual extra-axial blood and packing material. No abnormal fistula was seen during external carotid artery (ECA) injection (not shown). The left ICA demonstrated extensive dissection and arteriovenous shunting into the cavernous sinus, consistent with a direct CCF. Outflow from the left-sided CCF was noted into the petrosal sinuses and left external jugular veins [Figure 1e, f]. The left ACA and MCA flows were diminished, but no abnormality of the ECA was seen. Endovascular treatment and open surgical carotid bypass were discussed, but the patient proved to be hemodynamically unstable for treatment, and the family chose not to pursue further life-extending measures. This case report does not require patient consent per our institution.

DISCUSSION

CT, magnetic resonance angiography, or DSA can be used to screen for BCVI.[33] Various sets of criteria have been designed to predict the need for imaging to screen for BCVI, including the Denver and Memphis scores in adults and the Utah and McGovern scores in

Table 1: Summary of literature involving bilateral, traumatic blunt cerebrovascular injury (BCVI)

Reference	Age/Sex (*n*=33)	Presentation	Vessel (s) involved	Management	Follow-up (months)	Outcome
Fukuda *et al*. (1989)[17]	17/M	Polytrauma, GCS11	Vertebral; carotid	Heparin, antiplatelet	6	Improved exam, improved vessel anatomy
Galetta *et al*. (1991)[19]	48/M	Visual loss, facial droop	Carotid	Heparin, warfarin	2	Loss of vision, unchanged facial droop
Ballard *et al*. (1992)[3]	20/F	Facial weakness, hemiparesis, expressive aphasia, tongue deviation	Carotid	Heparin	5	Alive
Storrow and Smith (1995)[38]	37/F	Polytrauma, hemiparesis	Carotid	Heparin, warfarin	0	NA
Janjua *et al*. (1996)[21]	41/F	Lower extremity paraparesis	Carotid	Anticoagulant	NA	NA
Duncan *et al*. (2000)[13]	39/M	Numbness, hemiplegia, facial weakness	Carotid	Heparin	1	Hemiplegia
Lee and Jensen 2000[26]	43/M	Headache, visual disturbances	Carotid	LMWH, aspirin	2	Returned to baseline
Babovic *et al*. (2000)[2]	43/F	Ecchymosis, enophthalmos, facial fractures, visual decline	Carotid	Anticoagulant	0	Visual loss
Malek *et al*. (2000)[28]	37/F	Hemiparesis	Carotid	Clopidogrel	8	Returned to baseline
	43/F	Hemiparesis	Carotid	Angioplasty, aspirin, ticlopidine	20	Returned to baseline
	24/F	Hemiparesis, aphasia, dysphagia	Carotid	Heparin, warfarin	3	Residual dysphagia and hemiplegia
Busch *et al*. 2000[6]	27/F	Progressive coma	Vertebral; carotid	Surgical reconstruction	NA	NA
Pittock *et al*. (2001)[32]	40/M	GCS12, facial weakness, hemiparesis, hemineglect	Carotid	Heparin	12	Mobile with assistance
Fanelli *et al*. (2004)[15]	17/M	GCS9, hemiplegia	Carotid	Stent, ticlopidine, heparin	13	Mild, improving weakness
Clarot *et al*. (2005)[10]	38/M	GCS9, hemiparesis	Carotid	None	0	Death
	42/F	Headache	Carotid	Heparin, fluindione	5	Returned to baseline
Yong and Heran (2005)[41]	25/M	GCS6, dilated right pupil	Vertebral; carotid	Heparin bridge to warfarin	3	Improved
Chokyu *et al*. (2006)[9]	61/F	Tetraparesis	Carotid	Stent, aspirin	2	Alive
de Borst *et al*. (2006)[12]	13/F	GSC 3	Carotid	LMWH, aspirin	6	Patency of bilateral ICA
Adaletli *et al*. (2006)[1]	10/M	Headache, hemiparesis	Vertebral	None	2	Occlusion of posterior circulation
Furtner *et al*. (2006)[18]	43/M	Pain, amaurosis fugax	Carotid	Heparin, warfarin, aspirin	12	Alive
Fang (2008)[16]	50/M	Pulsatile proptosis, chemosis, intracranial bruit, decreased visual acuity	Carotid	Stent, anticoagulation	24	Resolution of dissecting pseudoaneurysms and CCFs
Srivastava *et al*. (2008)[37]	58/M	GCS11, hemiparesis, aphasia	Carotid	Heparin, warfarin	NA	NA
Leach and Malham (2009)[25]	23/F	SAH, atlantooccipital dislocation	Vertebral; carotid	Enoxaparin sodium, warfarin	0	Ambulating, following commands in all extremities
Molacek *et al*. (2010)[31]	49/F	GCS3, seizures	Carotid	Angioplasty	0	Returned to baseline
Keilani *et al*. (2010)[23]	52/F	Respiratory distress	Vertebral; carotid	Bilateral stents, aspirin	3	Patency of bilateral ICA
Samra *et al*. (2011)[36]	24/M	Respiratory distress, upper and lower extremity fracture, GCS6	Carotid	Aspirin	NA	NA
Chiba *et al*. (2014)[8]	60/F	Unconscious	Carotid	None	0	Died
Koleilat *et al*. (2014)[24]	23/F	Basal ganglia infarction, mandibular fracture, temporal contusion, hemiparesis	Vertebral; carotid	Heparin, warfarin	32	Independent ambulation, persistent left side weakness, persistent non-occlusive ICA dissection

Contd...

Table 1: Contd...

Reference	Age/Sex (*n*=33)	Presentation	Vessel (s) involved	Management	Follow-up (months)	Outcome
Cronlein *et al*. (2015)[11]	28/F	Anisocoria	Carotid	Heparin	6	Alive
Jenkins *et al*. (2016)[22]	55/M	Headache, weakness, numbness	Carotid	Aspirin	NA	NA
Taoussi *et al*. (2017)[39]	29/F	Dysphagia	Carotid	LMWH, warfarin	NA	NA
Our case	65/F	GCS3T, ipsilateral fixed/dilated pupil	Carotid	None	0	Death

GCS: Glasgow Coma Scale; LMWH: Low molecular weight heparin; ICA: Internal Carotid artery; CCF: Carotid-cavernous fistula; NA: Not available

children.[5,20,30,34] The Denver screening criteria include arterial hemorrhage, cervical bruit in patients <50 years, expanding cervical hematoma, focal neurological deficit, neurological examination incongruous with CT findings, and stroke on secondary CT. Other high-risk factors that often prompt screening are Le Fort type 2 and 3 fractures, basilar skull fracture involving the carotid canal, diffuse axonal injury with GCS < 6, cervical spine fracture involving C1–2 or the transverse foramen, cervical spine subluxation, and near-hanging with hypoxic–ischemic brain injury. In our patient, the observed neurological decline with concordant clinical and radiological findings was presumed to be due to an EDH, prompting surgical treatment without additional imaging, which might have been obtained had the patient been more stable. It is unlikely that this case would have been managed differently had the bilateral CCFs been discovered preoperatively, although this knowledge might have led to more rapid endovascular therapy after the EDH removal.

The Canadian Stroke Best Practice Recommendations for the Secondary Prevention of Stroke[40] and 2018 Guidelines for the Early Management of Patients With Acute Ischemic Stroke[33] recommended use of anticoagulation for low-grade (Biffl grade 1 and 2) injuries and combined endovascular stenting, vessel sacrifice, and surgical reconstruction for higher grade (Biffl grade 3–5) lesions. A meta-analysis of 34 clinical trials and 762 patients showed no difference between antiplatelet and anticoagulation treatment in terms of risk of death or stroke.[29] Similarly, the rate of ipsilateral stroke or death in the CADISS trial[7] did not differ significantly between antiplatelets and anticoagulants. Although the role of antiplatelet treatment for BCVI is established, anticoagulation in the setting of active intracranial bleeding is contraindicated. The presence of bilateral CCF in our case presented a treatment dilemma.

We identified 29 studies with 32 patients who had bilateral BCVI [Table 1]. Seven patients had nonmedical intervention (stent, angioplasty, surgical reconstruction) and 4 had no treatment; antiplatelet or anticoagulant treatment was used in 25 patients (some patients had both medical and nonmedical intervention). Previous reports of bilateral BCVI indicated that cases involved either focal or polytrauma often with concomitant injuries. CCFs represent arteriovenous shunting from the ICA or ECA into the cavernous sinus. Direct CCFs involve shunting directly from the ICA into the cavernous sinus. These are usually the result of trauma, although they can occur after rupture of a cavernous segment aneurysm. Endovascular therapy is the treatment of choice for direct CCFs. Coil embolization of the recipient cavernous is now the preferred first-line treatment. In rare cases, venous occlusion cannot be achieved, and vessel sacrifice is needed.

Bilateral CCFs are rare, with only 1 prior case reported after traumatic ICA dissection. Fang[16] reported a good 2-year outcome in a case of bilateral BCVI with bilateral high-flow CCF treated with bilateral detachable balloon occlusion. In our patient, hemodynamic instability prevented endovascular treatment. Among other reported cases with bilateral BCVI but not CCF, several patients died during the hospital course, often because of other polytrauma injuries.[8,10] In addition, most prior studies demonstrated significant neurological deficit on presentation, with many patients showing little improvement despite treatment. Various treatments were also performed for these patients, including medical, endovascular, and surgical therapies.

CONCLUSION

Bilateral CCFs due to trauma are exceedingly rare, having been reported only once previously. Although the injury was fatal in our case, we believe that continued improvements in the diagnosis and treatment of BCVI, as well as awareness that CCFs may occur in high-energy injuries and should be considered even if the patient does not meet traditional screening criteria, will improve future outcomes in cases of bilateral CCF.

Financial support and sponsorship
Nil.

Conflicts of interest
There are no conflicts of interest.

REFERENCES

1. Adaletli I, Sirikci A, Ulus S, Yilmaz MH, Kervancioglu S, Kurugoglu S. Traumatic bilateral vertebral artery dissection at the dural entry point site in a 10-year-old boy. Pediatr Surg Int 2006;22:468-70.
2. Babovic S, Zietlow SP, Garrity JA, Kasperbauer JL, Bower TC, Bite U.

Traumatic carotid artery dissection causing blindness. Mayo Clin Proc 2000;75:296-8.
3. Ballard JL, Bunt TJ, Fitzpatrick B, Malone JM. Bilateral traumatic internal carotid artery dissections: case report. J Vasc Surg 1992;15:431-5.
4. Biffl WL, Moore EE, Offner PJ, Brega KE, Franciose RJ, Burch JM. Blunt carotid arterial injuries: Implications of a new grading scale. J Trauma 1999;47:845-853.
5. Burlew CC, Biffl WL, Moore EE, Barnett CC, Johnson JL, Bensard DD. Blunt cerebrovascular injuries: Redefining screening criteria in the era of noninvasive diagnosis. J Trauma Acute Care Surg 2012;72:330-5; discussion 336-7, quiz 539.
6. Busch T, Aleksic I, Sirbu H, Kersten J, Dalichau H. Complex traumatic dissection of right vertebral and bilateral carotid arteries: a case report and literature review. Cardiovasc Surg 2000;8:72-4.
7. CADISS trial investigators, Markus HS, Hayter E, Levi C, Feldman A, Venables G, *et al.* Antiplatelet treatment compared with anticoagulation treatment for cervical artery dissection (CADISS): A randomised trial. Lancet Neurol 2015;14:361-7.
8. Chiba F, Makino Y, Motomura A, Inokuchi G, Ishii N, Torimitsu S, *et al.* Bilateral middle cerebral artery infarction associated with traumatic common carotid artery dissection: A case report and review of literature. Forensic Sci Int 2014;236:e1-4.
9. Chokyu I, Tsumoto T, Miyamoto T, Yamaga H, Terada T, Itakura T. Traumatic bilateral common carotid artery dissection due to strangulation. A case report. Interv Neuroradiol 2006;12:149-54.
10. Clarot F, Vaz E, Papin F, Proust B. Fatal and non-fatal bilateral delayed carotid artery dissection after manual strangulation. Forensic Sci Int 2005;149:143-150.
11. Cronlein M, Sandmann GH, Beirer M, Wunderlich S, Biberthaler P, Huber-Wagner S. Traumatic bilateral carotid artery dissection following severe blunt trauma: A case report on the difficulties in diagnosis and therapy of an often overlooked life-threatening injury. Eur J Med Res 2015;20:62.
12. de Borst GJ, Slieker MG, Monteiro LM, Moll FL, Braun KP. Bilateral traumatic carotid artery dissection in a child. Pediatr Neurol 2006;34:408-411.
13. Duncan MA, Dowd N, Rawluk D, Cunningham AJ. Traumatic bilateral internal carotid artery dissection following airbag deployment in a patient with fibromuscular dysplasia. Br J Anaesth 2000;85:476-8.
14. Ellis JA, Goldstein H, Connolly ES, Jr., Meyers PM. Carotid-cavernous fistulas. Neurosurg Focus 2012;32(5):E9.
15. Fanelli F, Salvatori FM, Ferrari R, Pacella S, Rossi P, Passariello R. Stent repair of bilateral post-traumatic dissections of the internal carotid artery. J Endovasc Ther 2004;11:517-21.
16. Fang C. Endovascular treatment of a high-flow direct traumatic carotid cavernous fistula with a two year follow-up. Interv Neuroradiol 2008;14:297-301.
17 Fukuda I, Meguro K, Matsusita S, Shigeta O, Oohashi N, Nakata Y. Traumatic disruption of bilateral vertebral arteries and internal carotid arteries: case report. J Trauma 1989;29:263-6.
18. Furtner M, Werner P, Felber S, Schmidauer C. Bilateral carotid artery dissection caused by springboard diving. Clin J Sport Med 2006;16:76-8.
19. Galetta SL, Leahey A, Nichols CW, Raps EC. Orbital ischemia, ophthalmoparesis, and carotid dissection. J Clin Neuroophthalmol 1991;11:284-7.
20. Herbert JP, Venkataraman SS, Turkmani AH, Zhu L, Kerr ML, Patel RP, *et al.* Pediatric blunt cerebrovascular injury: The McGovern screening score. J Neurosurg Pediatr 2018;21:639-49.
21. Janjua KJ, Goswami V, Sagar G. Whiplash injury associated with acute bilateral internal carotid arterial dissection. J Trauma 1996;40:456-8.
22. Jenkins JM, Norton J, Hampton T, Weeks R. Rare case of bilateral traumatic internal carotid artery dissection. BMJ Case Rep 2016;2016:bcr2016217262.
23. Keilani ZM, Berne JD, Agko M. Bilateral internal carotid and vertebral artery dissection after a horse-riding injury. J Vasc Surg 2010;52:1052-7.
24. Koleilat I, Gandhi R, Boulos A, Bonville D. Traumatic bilateral carotid and vertebral artery dissection. J Emerg Trauma Shock 2014;7:47-8.
25. Leach JC, Malham GM. Complete recovery following atlantoaxial fracture-dislocation with bilateral carotid and vertebral artery injury. Br J Neurosurg 2009;23:92-4.
26. Lee WW, Jensen ER. Bilateral internal carotid artery dissection due to trivial trauma. J Emerg Med 2000;19:35-41.
27. Lin TC, Mao SH, Chen CH, Chen YL, Wong HF, Chang CJ, *et al.* Systematic analysis of the risk factors affecting the recurrence of traumatic carotid-cavernous sinus fistula. World Neurosurg 2016;90:539-45 e531.
28. Malek AM, Higashida RT, Halbach VV, Dowd CF, Phatouros CC, Lempert TE, *et al.* Patient presentation, angiographic features, and treatment of strangulation-induced bilateral dissection of the cervical internal carotid artery. Report of three cases. J Neurosurg 2000;92:481-7.
29. Menon R, Kerry S, Norris JW, Markus HS. Treatment of cervical artery dissection: A systematic review and meta-analysis. J Neurol Neurosurg Psychiatry 2008;79:1122-7.
30. Miller PR, Fabian TC, Croce MA, Cagiannos C, Williams JS, Vang M, *et al.* Prospective screening for blunt cerebrovascular injuries: Analysis of diagnostic modalities and outcomes. Ann Surg 2002;236:386-93; discussion 393-5.
31. Molacek J, Baxa J, Houdek K, Ferda J, Treska V. Bilateral post-traumatic carotid dissection as a result of a strangulation injury. Ann Vasc Surg 2010;24:1133.e9-11.
32. Pittock SJ, Moroney JT, Alexander M, Brennan P, Moorhouse D. Traumatic bilateral carotid dissection with concomitant cerebral infarction. J Emerg Med 2001;20:33-8.
33. Powers WJ, Rabinstein AA, Ackerson T, Adeoye OM, Bambakidis NC, Becker K, *et al.* 2018 Guidelines for the early management of patients with acute ischemic stroke: A guideline for healthcare professionals from the American Heart Association/American Stroke Association. Stroke 2018;49:e46-e110.
34. Ravindra VM, Riva-Cambrin J, Sivakumar W, Metzger RR, Bollo RJ. Risk factors for traumatic blunt cerebrovascular injury diagnosed by computed tomography angiography in the pediatric population: A retrospective cohort study. J Neurosurg Pediatr 2015;15:599-606.
35. Redekop GJ. Extracranial carotid and vertebral artery dissection: A review. Can J Neurol Sci 2008;35:146-52.
36. Samra NS, Ravi AK, Johnson LW, Williams M. Traumatic bilateral carotid artery dissection. J La State Med Soc 2011;163:26-8.
37. Srivastava A, Bradley M, Kelly M. Bilateral carotid artery dissection after high impact road traffic accident. J Radiol Case Rep 2008;2:23-8.
38 Storrow AB, Smith BA. Traumatic bilateral carotid dissection. J Emerg Med 1995;13:169-74.
39. Taoussi N, Alghamdi AJ, Bielewicz J, Luchowski P, Rejdak K. Traumatic bilateral dissection of cervical internal carotid artery in the wake of a car accident: A case report. Neurol Neurochir Pol 2017;51:432-8.
40. Wein T, Lindsay MP, Cote R, Foley N, Berlingieri J, Bhogal S, *et al.* Canadian stroke best practice recommendations: Secondary prevention of stroke, sixth edition practice guidelines, update 2017. Int J Stroke 2018;3:420-43.
41. Yong RL, Heran NS. Traumatic carotid cavernous fistula with bilateral carotid artery and vertebral artery dissections. Acta Neurochir (Wien) 2005;147:1109-13; discussion 1113.