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Bilateral Internal Carotid and Left Vertebral Artery Dissection after Blunt Trauma: A Case Report and Literature Review

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Abstract

Multi-vessel cervical arterial injury after blunt trauma is rare, and its pathophysiology is unclear. Although blunt cerebrovascular injury is a common cause of cerebral ischemia, its management is still controversial. We describe a 23-year-old man in previously good health who developed three-vessel cervical arterial dissections due to blunt trauma. He was admitted to our emergency and critical care center after a motor vehicle crash. Computed tomography showed a thin, acute subdural hematoma in the right hemisphere and fractures of the odontoid process (Anderson type III), pelvis, and extremities. He was treated conservatively, and about 1 month later, he developed bleariness. Computed tomography angiography showed bilateral internal carotid and left vertebral artery dissection. Aspirin therapy was started immediately, and then clopidogrel was added to the regimen. Two weeks later, magnetic resonance angiography (MRA) showed improved blood flow of the vessels. Only aspirin therapy was continued. About 3 months after discharge, MRA demonstrated further improvement of the blood flow of both internal carotid arteries, but the dissection flap on the right side remained. Therefore, we extended the duration of antiplatelet therapy. On the basis of our experience with this case, we think that antithrombotic therapy is crucial for the management of multi-vessel cervical arterial injury, and agents should be used properly according to the injury grade and phase; however, further study is needed to confirm this recommendation.

Key words: blunt cerebrovascular injury, motor vehicle crash, CT angiography, antithrombotic therapy, dual antiplatelet therapy

Introduction

Blunt cerebrovascular injury (BCVI) is a rare but potentially devastating complication after trauma, accounting for approximately 1.5–3.5% of patients with blunt trauma admitted to trauma centers.[1,2] Of these, multi-vessel injury is an even rarer entity, and its characteristics and optimal treatment remain unclear.

We describe a patient with bilateral internal carotid and left vertebral artery dissection after blunt trauma, which was successfully treated with dual antiplatelet therapy (DAPT). The pathophysiology, clinical presentation, and treatment options of multi-vessel injury after blunt trauma are also discussed based on a literature review.

Case Report

The patient provided informed consent to publish his case and accompanying images.

The 23-year-old man in previously good health was admitted after a motor vehicle crash in which he was run over by a taxi (25 mph). On admission, he was lethargic with a Glasgow Coma Scale score of 13. The physical examination revealed no focal sign. The computed tomography (CT) scan demonstrated a thin, acute subdural hematoma on the right hemisphere and fractures of the odontoid process (Anderson type III), pelvis, and extremities. He was treated conservatively. About 3 h after admission, his consciousness recovered to normal, and the follow-up CT scan demonstrated that the subdural hemorrhage disappeared.

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About 1 month after admission, he developed bleariness. We performed CT angiography to screen for cerebrovascular disorder and found bilateral internal carotid and left vertebral artery dissection. Subsequent magnetic resonance angiography (MRA) and digital subtraction angiography (DSA) confirmed this finding (Figs. 1A–1D). There was no indication of cerebral infarction. On the left side, we observed dissection from the cervical portion to the petrous portion where an intramural thrombus almost completely filled the vessel lumen. Dissimilar findings were present on the right side where a dissection flap was observed in the cervical portion. In the left vertebral artery, luminal narrowing was not found, but the dissection and thrombus were observed in the atlantal part (i.e., the string sign).

Although 81 mg of aspirin daily was started immediately after diagnosis to inhibit the growth of thrombosis caused by intimal damage, constriction of the vessel lumen of the right side progressed; thus, we added 75 mg of clopidogrel daily to the treatment regimen. Two weeks later, MRA showed improved blood flow of the vessels. The intramural thrombus decreased in size, and the vessel lumen widened (Figs. 2A and 2B). We changed DAPT to single antiplatelet therapy (81 mg of aspirin daily) considering the bleeding risk after the improved blood flow was confirmed. Continuation of oral aspirin (81 mg daily) and outpatient follow-up visits with radiological examinations on a regular basis were planned. The patient was discharged home without any neurological deficits about 3 months following admission.

About 3 months after discharge, MRA demonstrated further improvement of the blood flow of both internal carotid arteries, but the dissection flap on the right side remained. We decided to extend the duration of antiplatelet therapy.

Discussion

Characteristics and mechanisms of BCVI

Regarding the appearance of BCVI, some studies have shown nearly an equal number of blunt carotid injuries and blunt vertebral injuries,[3,4] and bilateral injuries occur in 18–25% of patients with BCVI.[2,5,6] Three- or four-vessel BCVIs are extremely rare entities, and to the best of our knowledge, only

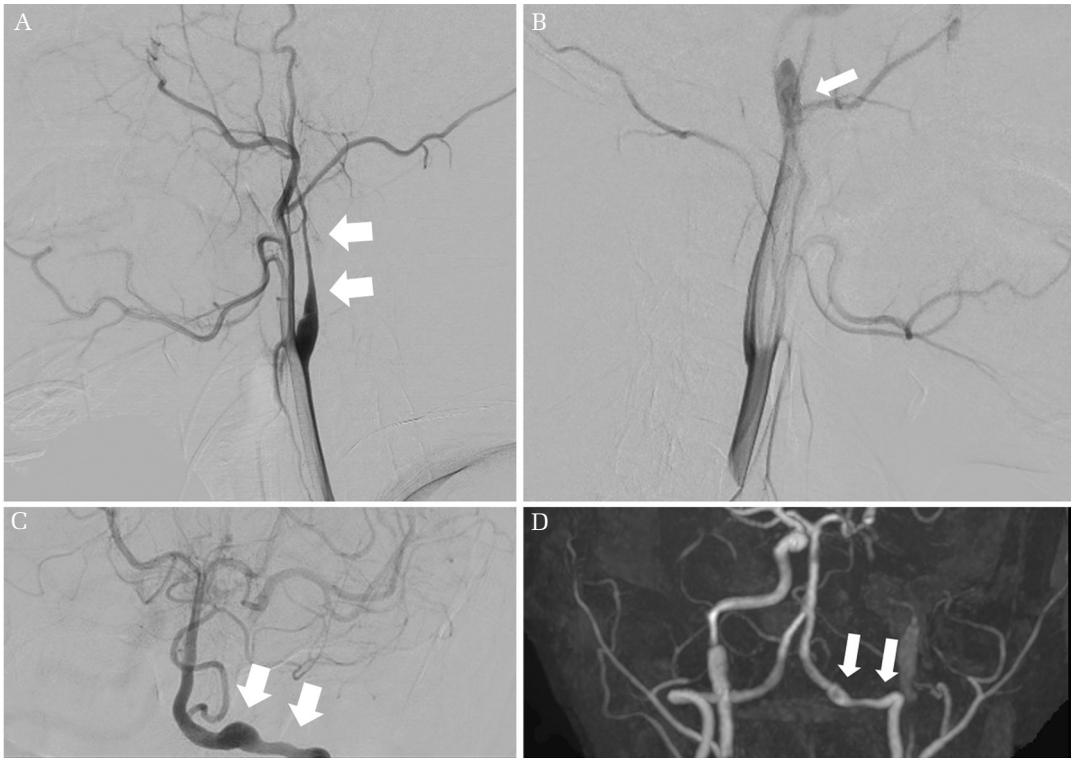

Fig. 1 (A) On the left side, digital subtraction angiography shows the dissection from the cervical portion to the petrous portion where an intramural thrombus almost completely fills the vessel lumen (*white arrows*). (B) Dissimilar findings are present on the right side where a dissection flap is observed in the cervical portion (*white arrow*). (C) In the left vertebral artery, luminal narrowing is not observed, but the string sign is observed in the atlantal part (*white arrows*). (D) Magnetic resonance angiogram also shows the above finding of the left vertebral artery (*white arrows*).

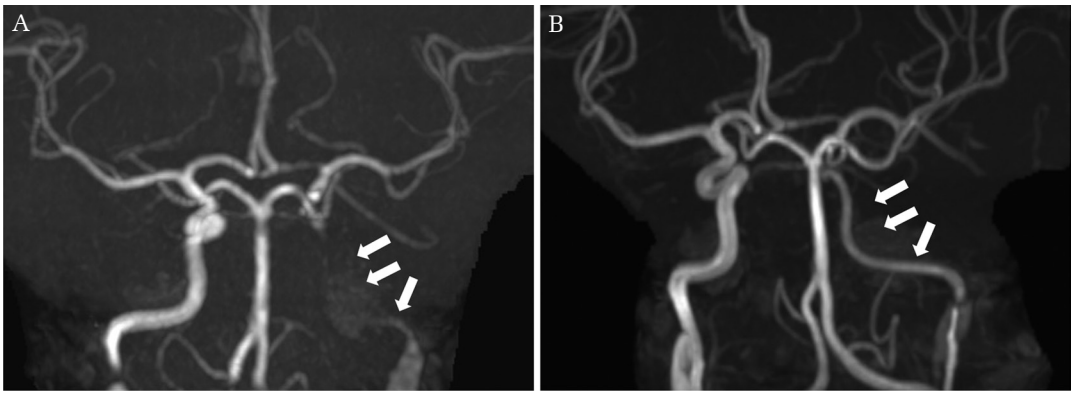

Fig. 2 (A) Magnetic resonance angiography showing luminal narrowing of the left internal carotid artery before dual antiplatelet therapy (DAPT). (B) After DAPT, the blood flow is improved 2 weeks later (*white arrows*).

nine case reports of such injuries were published in the literature.[7–15] Mechanisms of injury and associated injuries of patients with multiple BCVIs are summarized and compared in Table 1. In the patients with multi-vessel injury, motor vehicle crash was the most common cause of trauma, and severe injuries to the neck and chest were frequently observed.

Combined hyperextension and contralateral rotation of the head and neck was the common etiology of blunt carotid injuries. External segments are susceptible to being stretched over the lateral masses of the cervical vertebrae (particularly the C1–3 vertebrae).[16,17] Other etiologies were a direct blow to the anteromedial neck[12] and direct intraoral trauma.[18] Blunt vertebral injuries commonly occurred in the atlantal part (V2 segment) or foraminal part (V3 segment) as a result of laceration from fracture fragments or stretching.[19] Because the V2 segment winds around the C1 and C2 vertebrae, upper cervical injuries can cause severe blunt vertebral injuries. Considering these factors associated with BCVI, a case of an odontoid process fracture, like our patient, is highly suggestive of multiple arterial dissections. In addition, chest trauma can cause injuries in vertebral arteries at their origin, and severe chest injuries resulting in blunt vertebral injuries with essentially no filling have been reported.[7,8,15]

The presence of congenital or acquired risk factors (e.g., fibromuscular dysplasia, Marfan syndrome, hematological disorders, and collagen disease) can affect the occurrence of BCVIs. There is a possibility that patients with these risk factors are likely to develop multiple arterial dissections following lesser degrees of trauma. We were unable to confirm the presence of such risk factors in our patient. In only one case described by Eachempati et al.,[8] fibromuscular dysplasia was considered as a predisposing factor to the occurrence of multiple BCVIs.

Screening of BCVI

Blunt cerebrovascular injury is a potentially devastating event with a high stroke rate. Subsequent strokes occur between 10 and 72 h after injury;[20,21] thus, early recognition of BCVI is essential. The Modified Denver Criteria are the most studied and commonly used.[16] If BCVI is suspected, screening using 16-section or higher CT scanners is recommended.[22] MRA is also helpful in diagnosing arterial dissections. This modality enables noninvasive visualization of the vessel walls. The advantages of MRA include the avoidance of iodinated contrast agents, lack of bony artifacts, and earlier detection of cerebral infarction. DSA used to be the preferred method in diagnosing BCVI; however, DSA has been replaced by CT angiography because it requires transportation of a critically injured patient to the angiography suite and procedural time, which delays the diagnosis.[16]

Management of BCVI

Blunt cerebrovascular injury is always graded based on the Denver Scale,[23] and increasing risk of stroke and worse prognosis are associated with an increasing grade.[2,24] Antithrombotic and endovascular therapy are suggested as management strategies, which should be modified based on the grade, location of injury, and patients' symptoms.[22,25] The management of BCVI, as recommended based on the Denver Scale, is also highlighted in Table 2.[16]

Medical therapy is generally accepted as first-line therapy since prudent antithrombotic therapy has been shown to decrease the incidence of stroke after BCVI to <1%.[24,26] Therapy is available for all patients with BCVI, except those with grade V BCVI. The use of weight-based unfractionated heparin is

Table 1 Case reports of multiple BCVIs

Case no.	Type of BCVI	First author	Year of publication	Sex	Age (years)	Mechanism of injury	Denver grade	Complication	Stroke	Treatment	Outcome
1	Four-vessel	Fukuda et al.[7]	1989	Male	17	Motor vehicle crash: he crashed into the rear end of a truck while driving a motorcycle and sustained blunt injury to his neck and chest	Right ICA: II Left ICA: I Right VA: IV Left VA: V	Fracture of both first ribs Disruption of the left thyrocervical trunk	Multiple cerebral ischemia in the frontal lobe and right occipital lobe	Median sternotomy for ligation of both VAs Heparin	He was discharged on postoperative day 70 in a satisfactory neurological condition.
2	Three-vessel	Eachempati et al.[8]	1998	Female	33	Motor vehicle crash: her automobile was travelling at 55 mph during a head-on collision	Right ICA: III Left ICA: II Right VA: IV	Left parietal and temporal contusions Grade III/IV liver laceration Pubic ramus fracture Multiple rib fractures Left hemopneumothorax Multiple facial fractures Right perinephric hematoma Extensive lacerations	Bilateral watershed infarcts	Heparin and ticlopidine in the acute stage Aspirin in the chronic stage	Her deficits on discharge included difficulty with fine finger movements bilaterally, memory disorder and possessing only 80% of her strength in her right upper and lower extremities.
3	Three-vessel	Busch et al.[9]	2000	Female	27	Motor vehicle crash: details unknown	Right ICA: II Left ICA: II Right VA: II	Fracture of the right scapula Fracture of the fourth thoracic vertebral body	Extensive cerebral ischemia in the territory of the right middle cerebral artery	Revascularization	She was discharged to a neurological rehabilitation facility because of persistent left-sided hemiparesis.
4	Four-vessel	Nadgir et al.[10]	2003	Male	34	Chiropractic treatment	Right ICA: II Left ICA: I Right VA: III Left VA: II	None	Right thalamic infarct	Heparin Coumadin and aspirin	He returned home with minimal residual left-sided hemianesthesia and dysesthesia of the left hand.
5	Four-vessel	Yong and Heran[11]	2005	Female	25	Motor vehicle crash: she was the belted driver of a pickup truck traveling at a high speed when another truck skidded and struck her vehicle on the driver's side	Right ICA: III Left ICA: III Right VA: III Left VA: III	Direct carotid cavernous fistula Interhemispheric subdural hematoma Intraventricular blood Interpeduncular cistern blood Right pulmonary contusion	Multiple cerebral ischemia in the cerebellum and left frontal subcortical white matter	Heparin in the acute stage Warfarin for 14 weeks Aspirin in the chronic stage	The patient made a good neurological recovery, exhibiting only some persistent attention deficits and mild word finding difficulties at her 2-year follow-up evaluation.

(*Continued*)

Table 1 Case reports of multiple BCVIs—Continued

Case no.	Type of BCVI	First author	Year of publication	Sex	Age (years)	Mechanism of injury	Denver grade	Complication	Stroke	Treatment	Outcome
6	Four-vessel	Chakrapani et al.[12]	2008	Female	50	Facial massage	Right ICA: II Left ICA: II Right VA: II Left VA: II	None	No cerebral ischemia	Heparin in the acute stage Warfarin for 6 months, clopidogrel for 18 months, and aspirin indefinitely	Follow-up examinations at 1 year showed progressive improvement in all vessels; over a period of weeks, the patient's ptosis and miosis resolved.
7	Four-vessel	Leach and Malham[13]	2009	Female	23	Motor vehicle crash: she was involved in a head-on motor vehicle crash that occurred at 70 mph	Right ICA: II Left ICA: I Right VA: III Left VA: II	Bilateral C2 pars fractures with angulation of C2 on C3 and disruption of the C2/3 disc	Left thalamic infarct	Clexane for 48 h Warfarin	She was ambulating in the ward, wore a rigid cervical collar, and was discharged to a rehabilitation unit.
8	Four-vessel	Keilani et al.[14]	2010	Female	52	Horse-riding injury: she fell off a horse, and the horse jumped up and landed on her chest	Right ICA: II Left ICA: III Right VA: III Left VA: III	Multiple bilaterally displaced rib fractures with bilateral hemopneumothorax Pneumoperitoneum Multiple thoracic, lumbar, and vertebral fractures	Multiple areas of ischemic strokes in the left frontal and occipital lobes as well as in the left cerebellar hemisphere	Heparin Aspirin and clopidogrel Stent placement in both ICAs clopidogrel Coil embolization in the slightly enlarged right VA pseudoaneurysm Low-dose aspirin	She regained full cognition but had a persistent mild right-sided weakness at the time of discharge.
9	Three-vessel	Abuzayed[15]	2012	Female	33	Horse-riding injury: she fell from a horse, and the horse kicked her and bit her neck	Right CCA: II Right VA: IV Left VA: IV	Traumatic disc herniation at the D12–L1 level	Cerebral ischemia in the right parietal lobe	Endovascular stent placement in the CCA Right decompressive craniectomy	At the time of discharge, she was independently mobile with left hemiparesis.

BCVIs: blunt cerebrovascular injuries, CCA: common carotid artery, ICA: Internal carotid artery, no.: number VA: vertebral artery.

Table 2 Grade-based treatment of BCVIs

Grade of injury	Initial treatment	Long-term treatment
I	Antithrombotic therapy or observation	Antiplatelet therapy until healing
II	Antithrombotic therapy	Long-term antiplatelet therapy until healing or definitive treatment
	Endovascular treatment (if neurologic symptoms or the progression of dissection is present)	
III	Antithrombotic therapy	Long-term antiplatelet therapy until healing or definitive treatment
	Endovascular treatment (if symptomatic or the thrombus measures >1 cm)	
IV	Antithrombotic therapy	Life-long antiplatelet therapy
V	Emergent intervention/surgery	No data (symptomatic)

BCVIs: blunt cerebrovascular injuries.

recommended in the acute stage because it is reversible compared with antiplatelets.[2,22] The regimen usually starts with heparin (10 U/kg/h with a low activated partial thromboplastin time goal of 40–50 s) for 7–10 days, followed by 3–6 months of oral warfarin or antiplatelet agents (75 mg of clopidogrel daily or 325 mg of aspirin daily). If the disease progresses, the addition of a second antiplatelet agent can be considered.[27] Recent studies preferred the use of an antiplatelet for the long term compared with the use of warfarin because of its better safety profile, equivalent efficiency, and lower cost.[26] A few research studies indicated that a direct oral anticoagulant (DOAC) can be safer and more reasonable than warfarin.[28,29] Use of a DOAC for treating BCVI seems to be more widely known, but further studies are required to investigate this treatment.

Endovascular therapy remains controversial, and it is usually used on a case-by-case basis. Results of its safety and efficacy vary between reports.[14] Endovascular techniques include stenting and embolizing traumatic pseudoaneurysms and endovascular vessel occlusions. If symptomatology or imaging findings worsen over a few days, patients with grade II BCVIs may be treated with stenting. It is preferable to treat grade III BCVIs with coiling. Grade V BCVIs with active bleeding require immediate attempts to control bleeding (e.g., direct pressure and emergent intervention or surgery).[16]

Multi-vessel injury may require more active treatment than a single BCVI to prevent stroke. The results of previous reports implied that multiple BCVIs had a very high incidence of stroke complication despite various management strategies described (Table 1). In fact, eight out of nine patients had cerebral ischemia while receiving the best medical care as possible in each facility. Although there is no multi-vessel-injury-specific treatment recommendation, antithrombotic therapy tends to be immediately started in cases of grades I–III BCVIs. In patients who received heparin in the acute stage and an antiplatelet agent in the chronic stage, the recurrence of severe ischemia and progression of the neurological disorder at the very least could be prevented. In our patient, we could not start heparin despite recommendations because for such treatment he was not diagnosed with BCVIs in the acute stage. After the appropriate diagnoses were made, grade II bilateral internal carotid artery dissections and grade I left vertebral artery dissection, we decided to use a single antiplatelet agent initially because of some of the advantages of this therapy. Then we had to add a second antiplatelet agent shortly thereafter because of rapid luminal narrowing. As a result of this therapy, follow-up examinations showed progressive improvement in all vessels, like in case number 6 described by Chakrapani et al.[12] Despite no report of bleeding complications, DAPT was finished within a short period in our patient because luminal narrowing improved and the bleeding risk decreased. Therefore, we believe that the use of antithrombotic therapy should be emphasized, especially for multi-vessel injuries, although using the agents may be difficult if the patient has severe concomitant injuries.

Conclusion

In summary, BCVIs are a rare but important cause of stroke. In particular, three- or four-vessels BCVIs are unusual and should be recognized and treated early because of the higher rate of stroke. We think that antithrombotic therapy is crucial for management, and agents should be used properly based on the injury grade and phase; however, further study is needed to confirm this recommendation.

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Conflicts of Interest Disclosure

All authors declare no conflict of interest.

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