

Internal carotid artery dissection following blunt head trauma: a pediatric case report and review of the literature

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ABSTRACT

Background. Internal carotid artery dissection (ICAD) is a rare but potentially devastating complication after trauma in the pediatric age group. The diagnosis of traumatic dissection is difficult and is usually recognized only when ischemic symptoms appear. We report a pediatric patient with ICAD due to blunt cerebrovascular injury (BCVI).

Case. A 14-year-old boy suffered major trauma due to a motor vehicle accident. When the first aid team reached the accident site, he was intubated because of his low Glasgow Coma Score (GCS) and then transported to the nearest emergency department. Cranial computed tomography (CT) showed multiple fractures at the skull base and independent bone fragments in both carotid canals. On the 6th day; a brain magnetic resonance imaging (MRI) was performed to detect diffuse axonal injury. There was a loss of signal in the left internal carotid artery (LICA) tract but the limitation of diffusion was not associated with the same side, conversely there was a limitation of diffusion on the other side, affecting a very large area. CT angiography was performed in order to detect a filling defect and showed dissection in the LICA. The patient did not have any specific neurological symptoms associated with ICAD. Low-dose aspirin was utilized as anticoagulant therapy. On the 25th day of admission, the patient's GCS was 14, neurologic examination showed no difference between the right and left sides. He was discharged on the 55th day of the accident and was walking without support.

Conclusion. Our patient was a rare case in pediatrics due to having a clinically silent form of ICAD. It is very difficult to diagnose ICAD dissection during the early phase in cases with BCVI accompanied by multiple trauma. Even in the absence of typical neurological deficit, the possible presence of ICAD should be explored in patients with cranial fractures encompassing the skull base.

Key words: blunt cerebrovascular injury, carotid artery dissection, pediatric intensive care.

Blunt cerebrovascular injury (BCVI) is a rare but potentially devastating complication after trauma, and accounts for approximately 1.5-3.5% of patients admitted to trauma centres.¹ Internal Carotid artery dissections (ICAD) are rare, most of them occur spontaneously but around 4% are secondary to trauma.² These statistics are for adult trauma patients and such traumas are rare in the pediatric age group.

Dissection can be a diagnostic challenge due to the complexity of the clinical examination in children and delayed onset of symptoms in many cases. Usually, patients cannot be diagnosed until ischemic symptoms appear.³

Despite its low incidence, dissecting injury of the supraaortic vessels (carotid artery, vertebral artery etc.) is an important cause of ischemic stroke in children, associated with significant morbidity and mortality rates up to 20%.⁴ Arterial dissection may be caused by direct trauma to the artery, hyperextension-rotation of the head with stretching of vessels, basal skull or mandibular fractures, chest-head injuries

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with carotid stretching, and blunt intra-oral trauma.⁵⁻⁸

In this case report, we report a rarely seen pediatric case of ICAD development due to blunt head trauma. Although the ICAD developed on the left side, ischemic areas were predominantly located on the right cerebral hemisphere due to diffuse axonal injury (DAI) which was secondary to traumatic brain injury (TBI).

Case Report

A previously healthy 14-year-old male patient suffered major trauma due to a motor vehicle accident. The mechanism of this major trauma was a tractor rollover during harvest. When the first aid team reached the accident site, the boy was lying still on the ground and was immediately intubated because of his low Glasgow Coma Score (GCS) (E1M4V1). He was immobilized and then transported to the nearest emergency site. At his arrival, GCS was 6 (E1M4V1) and all immediately necessary interventions were performed by the emergency team. His cranial, cervical, thoracic and abdominal imaging studies were performed after he was stabilized, and then he was transported to our pediatric intensive care unit (PICU).

At his examination in the PICU, the patient was intubated, bilateral pupils were isochoric with positive light reflex, and all four extremities were equally moving with painful stimuli. Babinski sign was negative. Due to the patient being unconscious and intubated, a cranial nerve exam could not be performed conclusively. He was hemodynamically stable. The hemogram, arterial blood gas and biochemical parameters of the patient were normal. His thorax, abdominal and cervical computed tomography (CT) screenings were normal. In the bone window setting, cranial CT images showed multiple fractures at the base of the skull, and a bilateral styloid process fracture and hemotympanum (Fig. 1).

We noticed clear secretions in the mouth, which seemed similar to cerebrospinal fluid (CSF). These were tested with the beta-2 transferrin test, confirming our suspicions. To detect the basilar skull fracture that was causing CSF leak, a maxillofacial sinus CT was also performed. There were independent bony fragments in both carotid canals, hemorrhagic collections in the sphenoid and ethmoid sinuses, and fractured fragments of the left temporal bone causing independent bone fragments (Fig. 2).

After discontinuing all sedative medications, GCS was reevaluated. Because GCS was under 8 (E1M4V1), intracranial pressure catheterization (ICP) was performed at the bedside. He was followed-up with ICP catheter for five days to minimize TBI. Our goals were to keep ICP <20 mmHg and cerebral perfusion pressure >55 mmHg. On the 6th day of PICU follow-up, his ICP catheter was removed and his neurologic examination was checked after discontinuing sedation. His GCS was 6 (E1M4V1), pupils were isochoric, light reflex was bilateral positive, and all four extremities were moving equally.

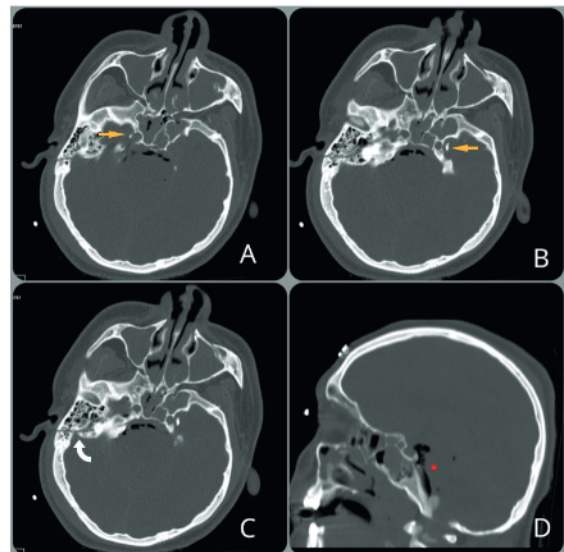


Fig. 1. Initial head CT reveals multiple fractures in skull base including lateral walls of carotid canals on both sides. On image A and B yellow arrows depict the separated osseous fragments. On image C, curved arrow shows longitudinal fracture of right temporal bone. On image D, pneumocephalus (red star) is demonstrated.

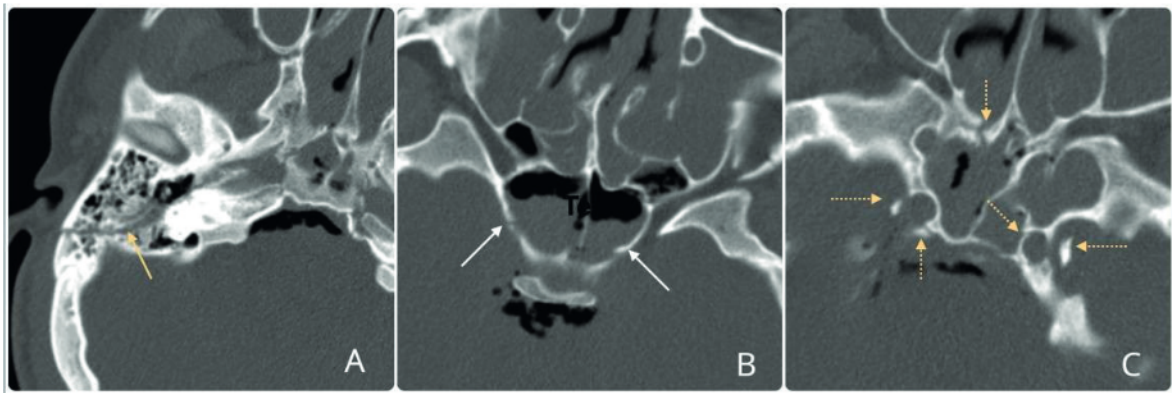


Fig. 2. On image A, fracture of the right temporal bone (yellow arrow). On image B, intense hemorrhagic collections of fractures in the sphenoid sinus and ethmoid cellulae posterior to the nasal septum and filling the secondary sinuses (white arrow), and on image C, free osseous fragment in both carotid channels (yellow arrow).

On the 6th day after the accident, a brain magnetic resonance imaging (MRI) was performed to detect DAI. When evaluating arteries, we determined a loss of signal in the left internal carotid artery (LICA) tract but diffusion limitation area was not correlated with the region supplied by the LICA. Surprisingly, the diffusion restriction of the right hemisphere was worse than that of the left side (Fig. 3). To verify this loss of signal in the LICA tract, CT angiography was performed and revealed filling defect and presence of intimal flap on the left side of the LICA's cavernous segment and LICA's petrous part, which was verifying results with MRI screening (Fig. 4).

A detailed neurological exam could not be performed due to low GCS and intubation;

nevertheless, as far as examination could show, there was no specific neurological deficit (hemiplegia, hemiparesis, facial palsy etc.) that was conclusive for left ICAD. As such, ICAD was only detected during a routine MRI performed on the 6th day of admission. Because it was late for the endovascular intervention and the use of heparin were not suitable due to trauma presence, we only used low-dose aspirin (100 mg/day) as an anticoagulant.

Due to his low GCS (E2M4V1), tracheostomy and percutaneous enteral gastrostomy operations were performed on the 15th day of hospital stay. His neurological state was progressively getting better. On the 25th day of PICU stay, GCS was 14, muscle strength was 2/5 for upper extremities and 3/5 for lower extremities. Surprisingly,

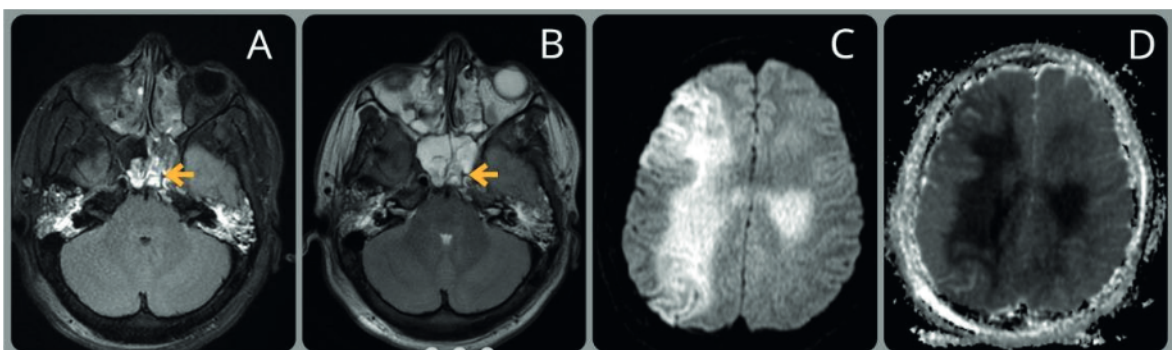


Fig. 3. Diffusion restriction in bilateral watershed areas of brain predominantly on right side is seen on image C and D. Retrospectively we also noticed loss of signal void on left internal carotid artery (yellow arrow) which is in favor of arterial dissection on image A and B.

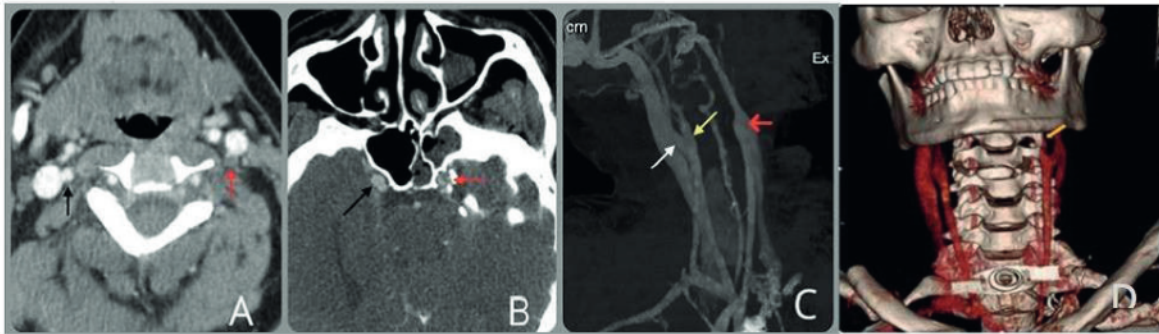


Fig. 4. CT angiography reveals filling defect in cervical portion of the left internal carotid artery (LICA), indicated with red arrow (Image A). At the level of the carotid canal, an intimal flap on the left side is demonstrated (Image B, red arrow). On maximum intensity projection images (Image C and D), it is clearly appreciated that there is no luminal enhancement at neither left common carotid artery from the level of bifurcation nor LICA (red arrows). Only external carotid artery is filled with contrast media which are highly suggestive findings for dissection of LICA. 3D CT angiography reveals filling defect in cervical portion of the LICA, indicated with yellow arrow (Image D).

there was no difference between the right and left sided neurological examinations. There were no findings of cranial nerve paralysis. Daily physiotherapy was performed. During his follow-up, muscle strength reached 4/5 and 5/5 for upper and lower extremities, respectively. When his GCS rose to E4M6V5 on the 51st day of hospitalization, tracheostomy and gastrostomy were closed. As his neurological improvement was very fast he was discharged from the hospital safely on the 55th day of the accident, and he was walking without support.

Informed consent was obtained from the patient's parents for publication.

Discussion

Traumatic carotid artery injuries (CAI) are rare; however, because of the anatomy, there may be important life-threatening complications. It occurs especially in patients with multiple trauma and direct blunt trauma to the neck. Delayed clinical presentation of traumatic ICAD may occur after weeks, months, even years following injury and it has important clinical outcomes.³ Our case demonstrates that ICAD can be easily missed during the initial trauma survey in patients with multiple trauma, especially in cases of additional TBI. Knowledge of the mechanism and risk factors

of the traumatic ICAD is important because it could have consequences for the treatment of these patients. A literature review revealed 21 published cases of ICAD caused by trauma.⁶⁻²¹ These cases are summarized in Table I.

Pathophysiology

Dissections start with a tear in the intima/media layer of the vessel wall, after which a hematoma develops. Ischemic strokes after ICAD may occur because of thromboembolism, or, less frequently, via artery occlusion due to the hematoma in the vessel wall.^{1,22} Dissection can also progress in the subintimal or subadventitial layer, leading to local compression resulting in a loss of function of the adjacent cranial nerves, presenting as Horner syndrome or facial nerve palsy.^{1,22}

Dissections may be traumatic or spontaneous. In addition to severe traumas like motor vehicle accidents, direct neck trauma and mild trauma like minor shoulder trauma can also be associated.^{12,15,20} Traumatic ICAD may develop in association with the following mechanisms: I- hyperextension and contralateral rotation of the head and neck, II- blunt intraoral trauma that affects the internal carotid artery at the angle of the jaw, III- laceration of the artery due to the skull base and mandible fractures,

Table I. Summary of published case reports of traumatic internal carotid artery dissection.

Author(s) / year of publication	Age (years)	Mechanism of injury	Unilateral/ Bilateral	Symptoms possibly linked with dissection	Neuroimaging	Dissection segment	Specific treatment	Outcomes
1 O'Sullivan ¹⁰ , 1990	17	Blunt head trauma	Unilateral	Hemiplegia, aphasia	CTA	Intracranial	None	Death
2 Borges ¹¹ , 2000	16	Intraoral trauma	Unilateral	Somnolence, seizure, hemiparesis	CTA	Extracranial	None	Hemiparesia
3 Borges ¹¹ , 2000	4	Intraoral trauma	Unilateral	Somnolence, motor aphasia, hemiplegia, hemianopia, eyes deviation	CTA, MRA	Intracranial, Extracranial	None	Hemianopia
4 Payton ⁶ , 2004	11	Laser tag accident	Unilateral	Lethargy, dysarthria, eye deviation, tongue deviation, hemiplegia, absence of corneal reflex	MRA	Intracranial	Heparin, aspirin, warfarin	Speech impairment
5 Agner ⁷ , 2006	0.3	Child abuse	Unilateral	Seizure, stiffening	MRA	Intracranial	None	Decreased vision in the left eye
6 de Bros ⁸ , 2006	13	Motor vehicle accident	Bilateral	Loss of consciousness, facial palsy, hemianopia, hemiparesis	MRA	Extracranial	Heparin, aspirin	Complete recovery
7 Pierrot ⁹ , 2006	4.5	Intraoral trauma	Unilateral	Loss of consciousness, hemiplegia, facial palsy	MRA	Extracranial	Heparin, aspirin	Complete recovery
8 Pierrot ⁹ , 2006	3.5	Intraoral trauma	Unilateral	Asymptomatic	MRA	Intracranial, Extracranial	Heparin	Complete recovery
9 Jariwala ¹² , 2006	17	Motor vehicle accident	Unilateral	Confusion, unipareses	MRA	Intracranial	Aspirin, clopidogrel	Unipareses
10 Lin ¹³ , 2007	7	Water slide injury	Unilateral	Headache, vomiting, facial palsy, hemiplegia, uvula deviation, slurred speech	MRA	Extracranial	Warfarin	Hemiparesis
11 Moriarty ¹⁴ , 2009	0.8	Spoon trauma	Unilateral	Decreased level of consciousness, hemiplegia	MRA	Intracranial	Heparin	Hemiparesis
12 Tsurukiri ¹⁵ , 2013	4	Motor vehicle accident	Unilateral	Loss of consciousness, no specific symptom	CTA	Intracranial	None	Complete recovery
13 Orman ¹⁶ , 2013	3.6	Fall from stroller	Unilateral	Hemiplegia, aphasia	CTA, MRI	Intracranial	Aspirin	Hemiplegia
14 Orman ¹⁶ , 2013	3.1	Motor vehicle accident	Unilateral	Seizure	CTA, MRI	Intracranial	Aspirin	Complete recovery

CTA: computed tomography angiography, MRA: magnetic resonance angiography, MRI: magnetic resonance imaging.

Table I. Continued.

Author(s) / year of publication	Age (years)	Mechanism of injury	Unilateral/ Bilateral	Symptoms possibly linked with dissection	Neuroimaging	Dissection segment	Specific treatment	Outcomes
15 Orman ¹⁶ , 2013	1.9	TV fell on the head	Unilateral	Loss of consciousness	magnetic resonance imaging	Intracranial	None	Complete recovery
16 Orman ¹⁶ , 2013	1	Fall from mother's arm	Unilateral	Hypoesthesia	magnetic resonance imaging	Extracranial	Heparin, Aspirin	Hemiparesis
17 Akbaş ¹⁷ , 2016	5	Water slide injury	Unilateral	Slurring of speech, right-sided weakness, facial palsy	MRA	Intracranial	Heparin, aspirin	Complete recovery
18 Bent ¹⁸ , 2016	1.5	Intraoral trauma	Unilateral	Vomiting, lethargia, diminished left extremity movement,	CTA, MRA	Extracranial	None	Hemiparesis, speech impairment
19 Esianor ¹⁹ , 2017	14	Blunt head trauma	Unilateral	Loss of consciousness, seizure, hemiparesis	CTA	Intracranial	Aspirin	Hemiplegia
20 Zant ²⁰ , 2017	0.3	Minor head trauma	Unilateral	Eye deviation, encephalopathy	MRA	Intracranial	Heparin	Unipareses
21 Cebeç ²¹ , 2018	10	Minor shoulder trauma	Unilateral	Headache, nausea, vomiting, dysphasia, facial palsy	MRA	Extracranial	Heparin	Complete recovery
22 Present case	14	motor vehicle accident	Unilateral	Loss of consciousness, no specific symptom	CTA, MRA	Intracranial	Aspirin	Complete recovery

CTA: computed tomography angiography, MRA: magnetic resonance angiography, MRI: magnetic resonance imaging.

IV- a combination of head-thorax trauma with overstretching of the internal carotid artery, V- direct application of force to the neck.¹ The frequency of ICAD is especially high in cases with accompanying carotid or petrous bone fractures in association with skull base fractures due to blunt trauma.²³ We also identified a skull base fracture extending to the lateral wall of the carotid canal in our patient.

Spontaneous ICADs are more common in the intracranial region, and traumatic dissections of the ICA in the head and neck region are most frequently located in the extracranial segments of the vessel. Concerning the ICA, most of the traumatic dissections occur below the skull base.²⁴⁻²⁶ In our case, blunt trauma-related ICAD was observed in the intracranial segment of the ICA.

Clinical Symptoms

The most common signs and symptoms are hemipareses, headache, aphasia, dysphasia, cranial nerve palsy, anopsia and an altered level of consciousness. CAI may present with an ischemic event such as transient ischemic attack (TIA) or stroke before reaching the hospital. Other manifestations may include ipsilateral headache, Horner syndrome, neck pain, bruit, and tinnitus.²⁷ In cases with traumatic mechanism, bleeding through the oral cavity, nostrils or ears can be detected as well. The diagnosis is relatively late due to the presence of other lesions related to trauma and this causes important sequelae.²⁸ In our case, no specific neurological findings could be detected, and a detailed neurological evaluation could not be performed because of TBI and unconsciousness. In the 21-case literature review we have reported, it is evident that cranial nerve paralysis and neurological findings such as hemiplegia, hemiparesis are at the forefront. However, due to the lack of clinical findings suggestive of ICAD, the diagnosis was delayed in our case.

There is usually a significant time interval between trauma and neurological symptoms in such patients, even though some cases have

been shown to have immediate findings. The interval between blunt trauma and neurological deficits can be hours, days, or even months.^{22,29} Especially in children, the dissection may remain in the subadventitial layer rather than the subintimal layer, which may account for a delay in presentation.²⁹ This delay between the moment of dissection and the occurrence of neurological symptoms is the critical factor that makes diagnosis difficult. Further, it is not at all clear that early detection would have found a treatable lesion after a stroke that is clinically evident hours or days after the injury. Reports of strokes developing after initial negative screening have been made.³⁰ On the other hand, the time interval between injury and onset of symptoms offers the possibility of screening for CAD and initiating therapy before the neurological symptoms become clear.

Diagnosis

Whilst the demonstration of an initial flap, double lumen sign or string sign (areas of adjacent stenosis and dilatation) on conventional catheter angiography remains the gold standard for diagnosis of CAD; non-invasive diagnosis with duplex ultrasound (USG), CT angiography (CTA) or MR angiography (MRA) is being increasingly used. Duplex USG has poor vision of the intracranial aspects of a dissection and it also provides limited information about small intimal tears and it is difficult to interpret when there is a hematoma in the neck.¹

CTA is prominently used as diagnostic modalities for the detection of BCVI, especially considering the acute situation in the emergency room of severely injured patients. Further development of CT scanners (>64 slices) shows comparable results in sensitivity to MRA scans.² However, to get additional information about a possible CVI, it can be necessary to include CTA into the protocol of the whole-body CT. To rule out patients that need an additional CTA, the modified "Denver Screening Criteria" (Table II) can be helpful.³²

Table II. Modified Denver screening criteria for BCVI.

Sign of BCVI	Risk factors for BCVI
Arterial bleeding	High-energy trauma mechanism combined with:
Cervical hematoma	Le Fort II/III fractures
Focal neurological deficiency	Cervical spine fractures
Neurological findings not matching with CT findings	Basilar skull fracture with carotid canal involvement
Ischemic insult seen on a secondary CT scan	Diffuse axonal injury with GCS $\leq$ 6
Cervical heart murmur	Near hanging with anoxic brain injury
	Combination of TBI and thorax trauma

BCVI: blunt cerebrovascular injury, CT: computed tomography, GCS: Glasgow coma scale, TBI: traumatic brain injury.

Imaging with an MRI scan combined with the MRA is considered to be the gold standard for the detection of carotid artery dissections.³² It determines dissections in up to 99% of cases and provides additional information about concomitant injuries (i.e. diffuse axonal injuries, acute infarction, fractures, etc.).² But it also has certain limits, especially during the diagnosis of critically injured patients with trauma. Besides the long duration of the scan, on the one hand, there exist restrictions in the use of potentially life supporting devices (i.e. pacemakers/iron-based metal implants, etc.), while on the other hand, there is a possibility that MRI availability is limited, especially in smaller trauma centers.³¹ The choice of the screening method may change with regard to the clinical setting and patient condition. In our 16-case literature review, it was determined that diagnosis was most commonly performed via MRA. In our case however, the diagnosis was confirmed via CTA after suspicious signal-loss findings were identified in MRI imaging.

Therapeutic options

In general, asymptomatic patients with low-grade dissections are treated conservatively with medical management and close imaging. There are several therapeutic options to treat cerebrovascular dissections, including open surgery, endovascular management (stenting, endovascular thrombolysis, thrombectomy), thrombolysis, anticoagulant and antithrombotic therapy.³¹

Surgical options are difficult and risky in the acute phase of the dissection.³³ This is why they usually do not belong in the first line of therapy in the acute phase. However, there are certain cases where operative treatment is necessary (i.e. to restore the blood flow in a case of distinct tear of the vessels or complete occlusions without ischemic intracranial lesions).³¹

The literature on the endovascular treatment (stenting) of traumatic ICAD is still limited to small series based on adult patients with relatively short follow-up. A proportion of the dissections occur at the skull base or in the intracranial region, and are therefore inaccessible to surgical or endovascular therapy. However, recent studies of stenting for CAD show excellent early and one-year patency rates and a low major adverse event rate.³⁴ Endovascular local thrombolysis is thought to be useful especially to prevent thromboembolic cerebral insult, but they still present high risks in the setting of a severely injured patient with multiple fractures or TBI, as presented in our case.^{2,31}

Antithrombotic or antiplatelet treatments are recommended in patients with neurological symptoms; whereas endovascular intervention is suggested only when the neurological status of a patient deteriorates under conservative medical treatment.³⁵ Antithrombotic treatment consists of either anticoagulation, typically with intravenous heparin followed by warfarin, or antiplatelet therapy with aspirin. Anticoagulation is typically preferred over

antiplatelet therapy in severe stenosis or pseudoaneurysm –assuming that it prevents thromboembolic complications more effectively. Patients who are unable to receive anticoagulants were treated with antiplatelet therapy.³⁶ In adults, anticoagulation therapy is suggested for 3 to 6 months to prevent injuries progressing to a higher injury grade, and to reduce the number of strokes.³⁶ Thrombolysis regimens, including recombinant tissue plasminogen activator or urokinase, are used to restore blood flow in some cases. But these approaches require an early diagnosis. The rationale of antithrombotic therapy is that the mechanism of the majority of strokes in CAD is due to thromboembolism.³⁶

In the pediatric population, the treatment of CAD is still controversial and more studies are needed. The use of antiplatelet therapy is even more controversial for patients who suffer a stroke following an intracranial dissection because of a very real risk of intracranial haemorrhage.²¹ Unfortunately, patients who suffered from blunt carotid injuries typically have closed head injuries, solid organ injuries, and/or pelvic fractures that prevent the use of early anticoagulation. When other concomitant injuries are present, then the optimal type and time of treatment should be considered with an interdisciplinary approach.³⁷ Pandey et al.³⁸ recommended 3 months of anticoagulation therapy and then antiplatelet therapy. In our case, as there were no specific symptoms and the diagnosis was made in the later period, we only used low-dose aspirin as an antiaggregant therapy. Anticoagulant therapy was not suitable for our patient due to multiple trauma.

Prognosis

Blunt trauma-related ICAD is known to carry a high rate of devastating neurologic morbidity (60%) and mortality (19–43%). Interestingly, a significant number of patients (66–73%) may be asymptomatic upon initial presentation, developing delayed neurologic symptoms anywhere from 1 hour to 7 days after injury.^{8,9,22,29}

CAD patients (33.7%) may present with an ischemic event at the time of presentation. As the severity of vessel injury worsens, stroke and mortality rates consequently increase.³¹ In our review of the 21 pediatric patients with ICAD, 47.6% (n=11) of cases did not have neurological symptoms at admission. The death rate was 4.8% (n=1) and 40% (n=8) of survivors had a complete recovery. However, it must be noted that ICAD was accompanied by TBI –which may have influenced neurological outcomes.

It is difficult to diagnose ICAD in the early phase in patients with TBI accompanied by multiple trauma. Even in the absence of typical neurological deficit (as was the case in our patient), the possible presence of ICAD should be explored in those with cranial fractures encompassing the skull base. Regarding therapeutic options, bleeding risk is one of the most challenging aspects of treatment considerations, especially in the presence of multiple trauma (i.e. pelvic fractures, or haemorrhagic traumatic contusion).

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