



FATAL DELAYED COMPLICATION OF CERVICAL SPINE INJURY IN A CHILD: A RARE CLINICAL OBSERVATION AND LITERATURE REVIEW

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Objective. To present a rare case of severe cervical spine injury in a child, complicated by the development of fatal ischemic stroke.

Material and Methods. The course of complicated cervical spine injury in a 12-year-old child with ischemic stroke secondary to arteriopathy, manifested by cerebral artery dissection is described. The review and discussion present current understanding of the etiology, pathogenesis, clinical manifestations and diagnostic methods of both complicated cervical spine injury and the peculiarities of ischemic stroke in children. A search of scientific sources was conducted in the eLibrary, PubMed, and Google Scholar databases using the following keywords: injury, children, cervical spine, ischemic stroke, arterial dissection, and fibromuscular dysplasia.

Results. The patient sustained a cervical spine injury while playing. His neurological status upon admission was tetraplegia. Given the absence of spinal cord compression, no indications for surgical treatment were identified. Intensive care was administered in the intensive care unit. Cerebrovascular accident developed on the 12th day after the injury, rapidly progressing from coma I to coma III. The diagnosis of ischemic stroke due to severe arteriopathy was established based on the circumstances of the injury, clinical manifestations, and MRI and MSCT results. Despite intensive care, the patient died on the 28th day.

Conclusion. The presented clinical case confirms the association between cervical spine injury and the development of cerebral stroke in pediatric patients. The presence of connective tissue dysplasia is a significant risk factor for vascular complications. The development of acute cerebrovascular accident in the setting of traumatic injury that limits neurological assessment may contribute to a delay in the timely diagnosis of vascular events and increase the likelihood of an unfavorable outcome. Increasing awareness among specialists involved in the treatment of complicated spinal injuries in pediatric patients about the risk of cerebral vascular complications, as well as developing guidelines for the treatment of complicated cervical spine injuries adapted to different stages of childhood, can improve clinical outcomes in terms of stroke prevention.

Key Words: injury; children; cervical spine; ischemic stroke; arterial dissection; fibromuscular dysplasia.

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Complicated cervical spine injury in children is rare, accounting for no more than 1.5% of all severe traumas. In the structure of spinal injuries, unstable fractures of various locations and spinal cord injuries represent potentially hazardous conditions. According to a study published in 2017, the incidence of unstable and complicated spinal injuries in the Russian Federation was reported at only 0.2 ± 0.2 cases per 10,000 child population. Specifically, such injuries account for 46% of cases in children under 12 years of age and 37% in those over 12 years [1–3]. The primary causes of spine injuries in children include motor vehicle accidents, falls from heights, sports-related injuries, and domestic accidents. Mortality rates for pediatric spine and spinal cord injury reach up to 5.7% [4, 5].

The vast majority of pediatric patients with unstable and complicated spinal injuries require surgical treatment. However, evidence suggests that the majority of unstable fractures go undiagnosed, and necessary surgeries are not performed due to various reasons, particularly those related to establishing indications for an active surgical approach [6, 7].

Complicated cervical spine injury causes sensorimotor dysfunction below the level of injury. The severity of developing neurological impairment is directly dependent on the patient's age, with younger children experiencing more severe neurological deficits [8]. However, spinal cord injury is not only accompanied by neurological deficits but also leads to other complications [9]. Thus, even minor head and neck injuries, or a

combination thereof, in children can act as triggers for stroke, which, according to WHO criteria, is defined as rapidly developing clinical signs of focal or global disturbance of cerebral function lasting more than 24 hours [10–12]. Strokes are one of the leading causes of mortality in children. Ischemic stroke (IS) case-fatality rates have been reported to reach as high as 30% [13–15].

Currently, there is a growing recognition of cerebral arteriopathies as the predominant etiology of pediatric stroke across all age groups, with a primary male predominance [16, 17]. One of the most common cerebral arteriopathies associated with arterial ischemic stroke is craniocervical arterial dissection, which is characterized by a tear or separation of the layers of the arterial wall, creating a false lumen for blood penetration. Cra-

niocervical artery dissection accounts for 5% to 22% of all cases of IS in children. Extracranial dissection is more common than intracranial dissection. Large-vessel intracranial arteriopathies refer to arterial wall abnormalities that typically manifest as luminal stenosis and represent a leading cause of IS [18–20].

The development of cerebrovascular complications in the presence of a competing traumatic injury – specifically, complicated cervical spine trauma that limits a detailed neurological assessment – can significantly increase morbidity and mortality [21]. Raising physician awareness regarding the detection of cerebrovascular complications, especially fatal ones, drives the necessity to study and describe each occurrence, given that these insights are insufficiently covered in the scientific literature.

This study aims to report a rare case of severe cervical spine injury in a child complicated by a fatal IS.

Case report

A 12-year-old child presented to the emergency department of the Novosibirsk Research Institute of Traumatology and Orthopaedics n.a. Y.L. Tsivyan 7 hours after sustaining a trauma, having been transferred by a critical care transport team from a district hospital. According to the patient, after jumping from the bed of a cargo truck into a snowdrift, he landed on his feet and experienced sudden acute flexion of the cervical spine, immediately followed by the onset of weakness in both the upper and lower extremities. Prehospital/transport therapy included crystalloid infusion (1,000 mL), analginum (500 mg), tramadol (50 mg), and a single bolus of prednisolone (100 mg). Urine output during transit was 600 mL.

In the emergency department, the patient was evaluated by an orthopedic trauma surgeon, a neurosurgeon, and an anesthesiologist-intensivist. The patient is conscious, cooperative, and oriented in all three spheres. The medical history revealed normal growth and development; has a history of bronchial asthma.

Complaints of nausea and lack of movement in the upper and lower extremities. Spontaneous diaphragmatic breathing at a rate of 20 breaths per minute, with an SpO₂ of 98%. Heart sounds are regular, with a heart rate (HR) of 75 bpm; blood pressure (BP) is 80/30 mmHg, with a mean arterial pressure (MAP) of 47 mmHg. The abdomen is soft, and bowel sounds are audible but sluggish. Urinary output via urethral catheter. Neurological status: tetraplegia. Accompanied by an anesthesiologist-resuscitator and under ongoing intensive care measures (infusion of 500 mL of crystalloids and 0.5% dopamine at 5 mcg/kg/min), the patient underwent an imaging work-up, including MRI of the cervical spine, MSCT of the cervicothoracic spine, and X-rays of the thoracic and lumbar spine, pelvic bones, and chest. Chest and pelvic radiographs revealed no pathological changes. MSCT of the cervicothoracic spine reveals comminuted sagittal split fractures of the C3, C4, and C5 vertebral bodies, a frontal split fracture of the anterior aspect of the C4 vertebral body, and bilateral non-displaced laminar fractures of the C3 and C4 vertebrae. Post-traumatic kyphotic deformity at the C3–C4 level of 9° and compression fractures of the T4, T5, and T7 vertebral bodies (Fig. 1).

Cervical spine MRI revealed a focal spinal cord contusion at the C4 level and spinal cord edema at the C2–C6 vertebral levels (Fig. 2).

Diagnosis established upon admission: Cervical spinal cord injury (CSCI); closed complicated fractures of the C3, C4, and C5 vertebral bodies with a sagittal split, and a frontal split of the anterior aspect of the C4 vertebral body; bilateral fractures of laminar parts of C3 and C4 vertebrae without displacement of fragments; closed uncomplicated compression wedge fractures of the T4, T5, and T7 vertebral bodies; spinal cord contusion; tetraplegia; neurogenic bladder and bowel dysfunction (central type); hypoesthesia from the C4 level by a conductive type affecting all sensory modalities; ASIA A; spinal shock; neurogenic shock.

Given the absence of spinal canal stenosis and spinal cord compression, combined with severe neurological deficits, spinal shock, neurogenic shock, the patient's critical condition, and extensive spinal cord edema, the medical board has determined the management strategy: to forgo emergent surgery due to a lack of indications, with subsequent monitoring and treatment in the Intensive Care Unit (ICU).

In the ICU, vital signs monitoring was performed while the patient's cervical spine was immobilized in a Philadelphia collar. Hemodynamic support was continued; fluid, gastroprotective, prokinetic, anti-edema, and nutritional-metabolic therapies were initiated, along with physical therapy and breathing exercises. Pharmacological thromboprophylaxis and pressure injury prevention have been initiated.

On day 1 in the ICU, the patient is alert, cooperative, and oriented, with a Glasgow Coma Scale score of 15. Concurrently, there is a negative trend characterized by the progression of cardiovascular and respiratory failure. Continued dopamine infusion (5.5 mcg/kg/min) and initiated norepinephrine infusion (0.22 mcg/kg/min). Implemented non-invasive CPAP ventilation with P_{insp} 15 mbar, PEEP 5 mbar, FiO₂ 35%, TV 320–440 mL, and RR 15–20 bpm. Against the background of these interventions, hemodynamic parameters remain stable: BP 113/51 mmHg, MAP 72 mmHg, and HR 73 bpm. Gas exchange is compensated: SpO₂ – 100%.

On day 5 of ICU admission, chest MSCT demonstrated the development of right lower lobe segmental (S9–S10) pneumonia. Antibiotic therapy (amoxicillin/clavulanate 1200 mg daily) was initiated. On day 10 of ICU stay, the patient developed a febrile temperature, accompanied by an increase in inflammatory markers and the presence of *Klebsiella oxytoca* in urine cultures. Antibiotic coverage was escalated to Meronem (1500 mg per day).

Overall, the first 11 days of the post-traumatic period were consistent with the severity of the sustained spinal cord injury. Hemodynamic and gas exchange

parameters were stabilized against the background of intensive care.

On day 12, amid stable hemodynamic and respiratory parameters and clear sensorium, a transient episode of depressed consciousness was noted: the patient became somnolent, failed to respond to examination, and was unable to follow commands. Glasgow Coma Scale Score was 12. The episode subsided spontaneously within 30 minutes. On the 13th post-injury day, the patient experienced an acute onset of global neurological symptoms, with consciousness deteriorating to a comatose state. Glasgow Coma Scale Score as 8. The patient was transitioned to invasive mechanical ventilation. The patient underwent an MRI of the brain and cervical spine, contrast-enhanced CT angiography of the head and neck vessels, carotid duplex ultrasound, echocardiography, and lower extremity venous duplex ultrasound.

Brain and cervical spine MRI findings: a diffuse zone of hyperacute ischemia is noted in the left hemisphere, extending via the convexital regions into the right hemisphere; a focal area of acute ischemia is present in the right frontal lobe (measuring 27.5×42.5 mm in the axial plane); post-traumatic changes of the spinal cord at the C2–C7 vertebral levels; compression fractures of the C3, C4, and C5 vertebrae; right-sided otitis media with inflammatory extension into the mastoid process; mucosal thickening of the sphenoid sinus (Fig. 3).

MSCT angiography of the intra- and extracranial cerebral vessels: 0.5 mm axial slices were obtained. 3D Volume Rendering and 3D MIP reconstructions were performed. Angiographic findings:

1) Extracranial vessels: The vertebral arteries enter the transverse foramina at their typical anatomical locations. The diameter of the left vertebral artery – 3.7 mm, and the right vertebral artery – 3.8 mm. Angulation of the V1 segment of the left vertebral artery is noted. The V4 segments of both vertebral arteries, as well as the basilar artery, demonstrate a «string-of-beads» appearance due to alternating zones of stenosis and local expansion of the lumen. Left V4 segment

diameter: 1.3 mm – 0.9 mm – 2.2 mm. Right V4 segment diameter: 1.9 mm – 1.1 mm – 2.2 mm. Basilar artery diameter: 3.4 mm – 1.1 mm – 1.9 mm – 2.8 mm. The common and internal carotid arteries show no focal luminal narrowing, pathological dilatation, or vessel wall structural abnormalities. Angulation of the cervical segments of both internal carotid arteries with loop formation is observed (Fig. 4).

2) Intracranial vessels: The Circle of Willis is complete; the internal carotid artery (ICA) siphons demonstrate normal configuration without localized stenosis. The anterior cerebral arteries (ACAs) are clearly visualized throughout their course; the right A1 segment is asymmetrically narrower than the left (measuring 1.0 mm on the right versus 1.3–1.6 mm on the left). The A2 segment of the right anterior cerebral artery is hypoplas-

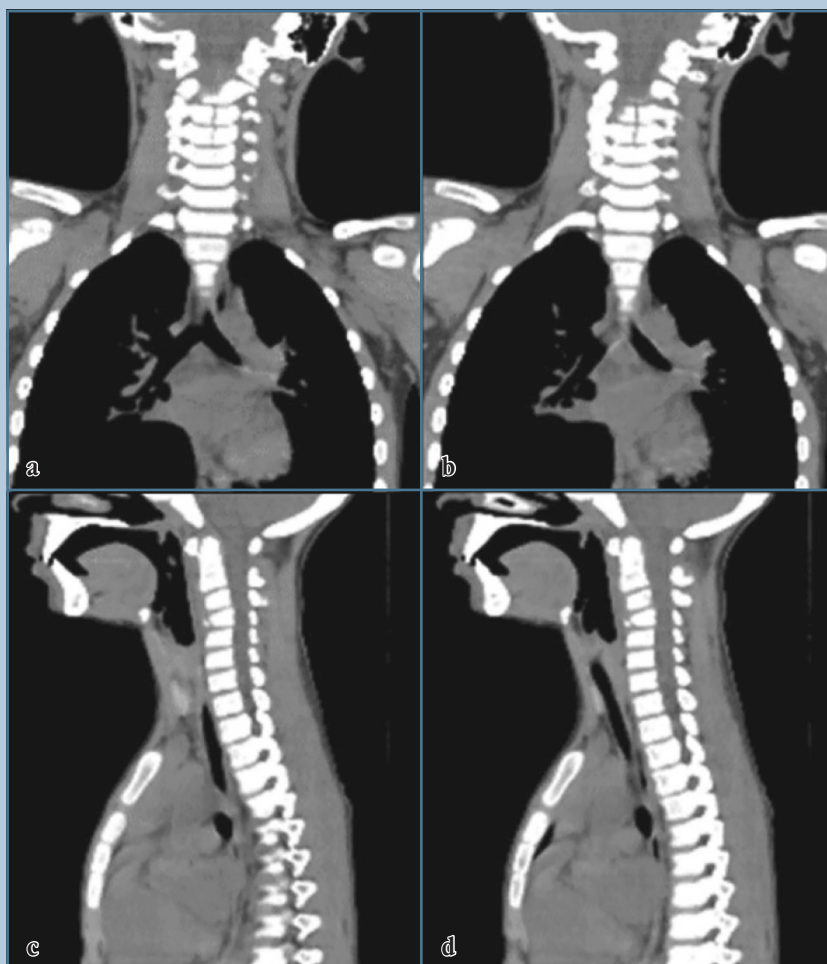


Fig. 1

Initial MSCT scan of the spine in a 12-year-old patient (level C0–T8) upon admission: **a, b** (frontal view): compression-comminuted fractures of the C3, C4, and C5 vertebral bodies with sagittal splitting, and a frontal split fracture of the anterior aspect of the C4 vertebral body; **c, d** (sagittal view): post-traumatic cervical kyphosis of C3–C4 at 18° with the apex at the intervertebral disc. Posterior subluxation of the inferior endplate of the C4 vertebral body by up to 2.0 mm. Spinal canal deformity at C3–C4 (~10.9 mm) and C4–C5 (~11.4 mm). Wedge compression fractures of the T4, T5, and T7 vertebral bodies



Fig. 2

Sagittal MRI of the cervical spine of a 12-year-old patient upon admission demonstrates acute compression fractures of the C3, C4, and C5 vertebrae. Narrowing of the spinal cord reserve spaces at the C3–C4 and C4–C5 levels. Post-contusive spinal cord changes at the C3–C5 vertebral levels. Spinal cord edema at the C2–C6 levels

tic (poorly developed). The anterior communicating artery (ACoM) is well-defined. Flow through the middle cerebral arteries (MCAs), posterior cerebral arteries (PCAs), and the basilar artery is clearly visualized. There is a marked reduction (paucity) of perfusion/blood flow in the left temporal and occipital cortex, as well as the cortex of the left frontal lobe (Fig 5).

Results of ultrasound examination of the neck vessels shows no structural abnormalities of the brachiocephalic arteries. Signs of severe bilateral increase in peripheral resistance within the carotid basin (most pronounced in the left internal carotid artery). Bilateral accelerated flow velocities in the middle cerebral arteries (MCAs) with Lindegaard ratios (adjusted for age) indicative of mild cerebral angiospasm, more pronounced on the left. Signs of decreased blood flow in the right anterior cerebral artery (ACA).

Chest MSCT demonstrates resolution of consolidations in the right S9–S10 segments. Duplex ultrasonography of the low-

er extremity veins demonstrated patency of both deep and superficial veins.

To determine the prognosis and treatment strategy, a multidisciplinary consultation involving external specialists was held, concluding with a primary clinical diagnosis of spinal cord injury. Closed complicated fractures of the C3, C4, and C5 vertebral bodies with a sagittal split, and a frontal split of the anterior aspect of the C4 vertebral body. Bilateral undisplaced laminar fractures of the C3 and C4 vertebrae. Closed uncomplicated wedge compression fractures of T4, T5, and T7 vertebral bodies. Cervical spinal cord contusion. Tetraplegia. Central-type pelvic organ dysfunction. Total sensory loss below T4. ASIA A. Spinal shock. Complications: acute IS with development of ischemic foci within the left middle, left posterior, and right anterior cerebral artery territories secondary to craniocerebral artery dissection associated with mitochondrial cytopathy as part of a systemic dysplastic process. Comorbidities: mild controlled allergic asthma. Respiratory failure. Secondary osteoporosis (?). Multiple drug and food allergies. Mild anemic syndrome. Stress-induced hyperglycemia (SIH).

It as recommended to continue anticoagulant, vascular, and neuroprotective therapy.

On the 14th day, the patient's condition is extremely critical. This severity is due to acute cerebrovascular accident in the middle cerebral artery, left posterior cerebral artery, and right anterior cerebral artery systems, cerebral edema, and worsening general cerebral symptoms leading to coma grade III. The Glasgow Coma Scale (GCS) level is 3. Worsening central cardiovascular failure, tolerant to hemodynamic support, and fluid and electrolyte imbalances are noted.

A follow-up medical board review was conducted to determine the patient's prognosis and management strategy. Conclusion of the medical board: given the onset of brainstem signs due to worsening cerebral edema, the prognosis is poor. Continue non-operative management in the ICU.

Days 13–27: Patient in critical condition with clinical deterioration due to

progressive multiorgan failure (cerebral, vascular, respiratory, and intestinal). Fluid and electrolyte imbalances, hypermetabolic state, and systemic inflammatory response syndrome (SIRS) are present. Neurological status remains unchanged: coma scale III, Glasgow Coma Scale (GCS) score is 3.

Despite ongoing intensive care, the patient expired on post-operative day 28 due to progressive cardiovascular insufficiency.

Discussion

In children under 8 years of age, the upper cervical spine (C0–C2) is most susceptible to injury, whereas after age 12, cervical spine trauma mirrors adult patterns, primarily affecting the lower cervical spine (C3–C7) [3]. The leading mechanism of injury in cases involving cervical and thoracic spine trauma is compressive flexion. This specific mechanism of injury is associated with a severe neurological deficit, with the final outcome dictated by the presence and severity of spinal cord changes [22, 23].

Given the mechanism of injury, the patient's age, the nature of the cervical spine osseous lesions, the presence of vertebral body fractures in three thoracic vertebrae, and the severity of neurological deficits, it can be concluded that compression flexion was undoubtedly the leading mechanism of injury in our presented case.

Cervical spine fractures are known to be a risk factor for severe arteriopathies in 14% of cases, specifically cerebrovascular arterial dissection, which may present as a primary lesion or as an extension of an extracranial vascular wall dissection. Intracranial dissections lead to IS in 30–84% of cases [12, 16, 19].

The development of pathological intracranial sequelae following flexion injury of the cervical spine can be conceptualized through the following mechanism: cervical flexion, particularly when associated with an unstable injury, induces pathological tension and kinking of the vertebral artery at its point of entry into the cranium (V3–V4 seg-

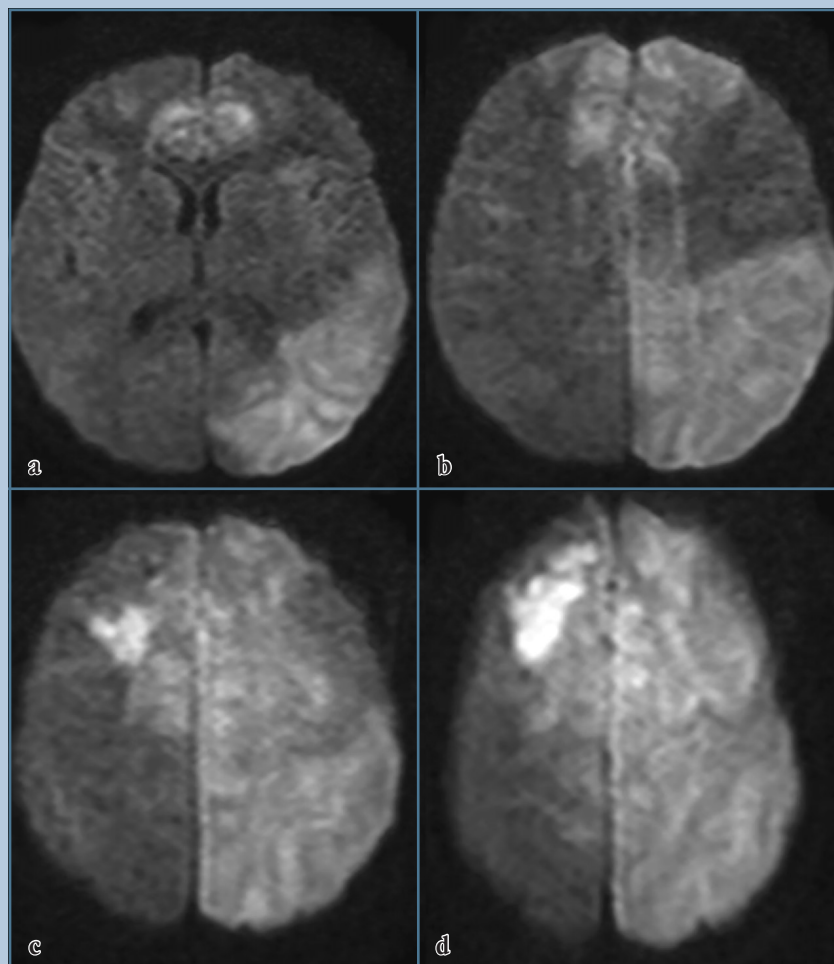


Fig. 3

Brain MRI of a 12-year-old patient on day 13, fluid-attenuated inversion recovery (FLAIR) images: **a, b** – Diffuse zone of hyperacute ischemia in the left cerebral hemisphere, extending to the right hemisphere via the convexital regions; **c, d** – area of acute ischemia in the right frontal lobe (measuring approximately 27.5 × 42.5 mm in the axial plane)

ments). Due to the anatomical tethering of the intracranial V4 segment and the lack of redundant length, this tension leads to an intimal tear followed by vessel wall dissection. The damaged vessel wall triggers thrombus formation via two concurrent mechanisms: the activation of the coagulation cascade in response to wall injury, and the induction of turbulent flow downstream of luminal narrowing caused by wall dissection. When a thrombus forms in the vascular lumen, it tends to propagate anterogradely with the blood flow into the basilar artery,

and from there into the arteries of the Circle of Willis. The described subtotal bihemispheric cerebral ischemia in this case is most likely a consequence of the specified thromboembolic mechanism. Based on MSCTA findings, the A2 segment of the right ACA is hypoplastic, likely secondary to occlusion of the right ACA, correlating with the MRI presentation of an acute ischemic stroke in the right frontal lobe. Given the documented pattern of severely diminished cortical perfusion in the left temporal and occipital lobes, microthromboem-

bolism within the territories of both the left middle cerebral artery (MCA) and left posterior cerebral artery (PCA) should be considered.

However, apart from traumatic arterial dissections, spontaneous arterial dissections can also develop. Current scientific evidence supports the hypothesis that spontaneous arterial dissection is facilitated by an underlying connective tissue disorder, manifesting either as various constitutional forms of dysplasia or as active conditions, including vasculitis and coagulopathies. Anatomical and morphological vascular features associated with this factor are present in 20% of cases [24–26].

In the presented case, the development of arteriopathy was likely multifactorial, involving several causes of varying significance. Trauma acted as a trigger for the development of arteriopathy. Concurrently, the patient was found to have connective tissue insufficiency, presenting as fibromuscular dysplasia as confirmed by MSCT angiography of the intra- and extracranial cerebral vessels. These findings align with literature data demonstrating a high prevalence of fibromuscular dysplasia in the extracranial carotid and vertebral arteries, where diagnosis is predominantly established via vascular imaging. Recently, fibromuscular dysplasia has been recognized as a significant predictor of cervical artery dissection, accounting for up to 65% of all arterial dissections [27–29]. Moreover, reports indicate that connective tissue disorders are linked to mitochondrial insufficiency. These data derive from histological and histochemical studies of muscle biopsies in patients with cervical artery dissection [30, 31].

In this case, a contributing role of the patient's concurrent bacterial infection to the development of arteriopathy cannot be ruled out, as infectious and inflammatory processes are known factors involved in the pathogenesis of IS in children. Data from scientific sources indicate that this pathogenic pathway is fairly common in IS development, accounting for 8.8% of cases [10]. Existing literature demonstrates that immune-mediated reactions and subsequent local

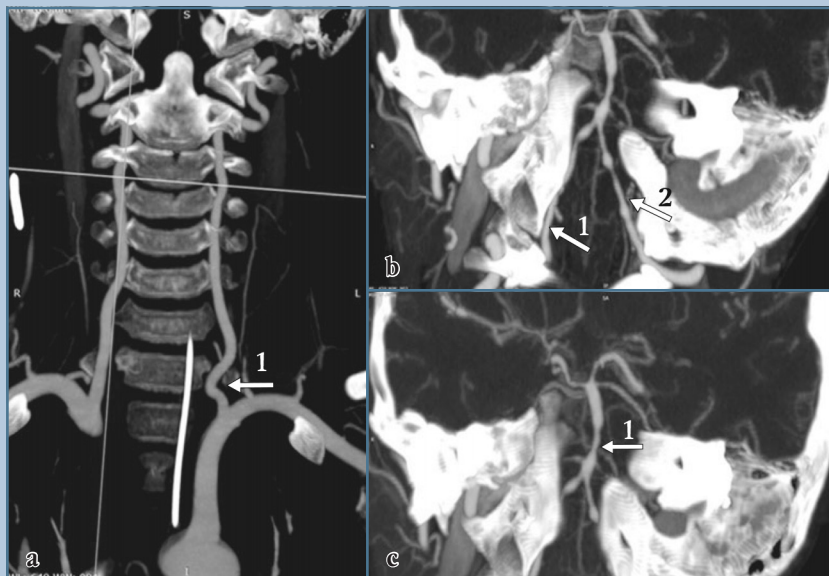


Fig. 4

MSCT angiography of the extracranial vessels: **a** – angulation of the V1 segment of the left vertebral artery (1); **b** – right vertebral artery (1); left vertebral artery (2); **c** – basilar artery (1)



Fig. 5

MSCT angiography of the intracranial vessels: **a** – the right A1 segment is asymmetrically narrower than the left (1, 2), with the right diameter measuring 1.0 mm and the left measuring 1.3–1.6 mm; the A2 segment of the right anterior cerebral artery is hypoplastic (poorly developed); **b** – there is a pronounced reduction of blood flow in the cortex of the left temporal and occipital lobes, as well as the cortex of the left frontal lobe

inflammation contribute to the pathogenesis of childhood stroke [32–34].

In the presented case, the clinical presentation of stroke masked by tetraplegia precluded a definitive determination of the stroke type. However, considering the patient's age and gender, the lack

of prior cardiovascular history, and the established fact that young-onset stroke frequently stems from cervical or cerebral artery dissection, along with the documented cervical spine trauma and diagnostic findings, a multidisciplinary medical board with external consultants

established the primary etiology. Guided by current scientific literature and the Standardized Classification of IS in Children, and despite the unavailability of molecular genetic testing, the panel concluded that the acute IS was primarily caused by cerebral artery dissection secondary to mitochondrial insufficiency within a systemic dysplastic process.

Furthermore, based on the Standardized Classification and Diagnostic Evaluation of Arterial IS in Children, it can be hypothesized that the presented clinical case involves a multi-source stroke, as it meets the criteria for multiple types within this classification system, with no single dominant source identifiable [35].

Traumatic cervical vascular injury is frequently underdiagnosed due to its asymptomatic clinical presentation. Additionally, severe neurological deficits caused by spinal cord injury can obscure the early diagnosis of acute cerebrovascular accidents. Therefore, diagnosing vascular injury requires a high index of suspicion and detailed neurovascular imaging, encompassing both the cranial and cervical regions. If there is even the slightest suspicion of a spinal injury, both spinal and cranial MRI or MSCT must be performed. In the pediatric population, MRI is the preferred modality for neurovascular imaging [3, 19].

It is well established that the highest risk of stroke occurs within the first 24 hours of trauma, although cases have been reported developing two weeks or more following the initial arterial vascular injury [36]. Regrettably, the exact onset and location of the primary vascular lesion could not be determined in the presented case, given that the neck vasculature was not evaluated at the time of the patient's admission. Additionally, the presence of a baseline severe neurological deficit, presenting as tetraplegia, may have mitigated or masked the initial onset of global cerebral symptoms. The fact that a distinct clinical manifestation of stroke occurred on post-injury day 13 does not rule out a delayed onset of ischemic or hemorrhagic event following the primary vascular injury sustained during trauma [21, 25, 36]. At the same time, spontaneous dissection cannot be

ruled out, as its clinical signs are known to appear hours or days prior to the onset of cerebral ischemia [37]. Therefore, in the presented case, the brief episode of impaired consciousness observed on post-traumatic day 12 can be considered the onset of the cerebral vascular complication.

Management of patients with unstable cervical spine trauma requires ICU admission, focusing on vital sign stabilization and optimization of cerebral perfusion pressure. Mortality in this patient cohort remains high, reaching up to 7%. In intensive care management, the critical priorities are surgical decompression, spinal stabilization, and respiratory and hemodynamic support. The presence of concomitant injuries or secondary complications can complicate the management of acute spinal cord injury due to competing priorities regarding target blood pressure, mechanical ventilation requirements, and antithrombotic therapy. In the presented case, intensive care management largely adhered to current guidelines, with the exception of respiratory support; noninvasive positive-pressure ventilation was avoided due to compromised airway protection and inadequate secretion control [21, 38].

Clinical studies present conflicting evidence on whether surgical spinal sta-

bilization is required when spinal cord compression is absent. At the same time, a number of researchers maintain that spinal stabilization must be performed routinely to avert secondary spinal cord damage secondary to spinal instability [7, 38]. Furthermore, the current 2024 AOSpine guidelines recommend early surgical intervention performed within 24 hours of injury, which enhances neurological recovery. Other authors also report the efficacy of early surgical treatment for spinal cord injury [7, 39, 40].

Of note, a surgical approach involving cervical spine stabilization could have been justified in this clinical scenario. However, the absence of spinal cord compression, the presence of extensive spinal cord edema, and the fact that any surgical intervention could significantly exacerbate the edema or even cause iatrogenic cervical artery dissection served as limiting factors for an aggressive surgical strategy. At this level of risk, the benefit of an aggressive surgical approach compared to ongoing conservative management was questionable. At this level of risk, the benefit of an aggressive surgical approach compared to ongoing conservative therapy was questionable. Answering this question necessitates the development of clinical guidelines tailored to the specific characteristics of

different pediatric age groups regarding treatment approaches for complicated spinal cord injury.

A limitation of this study is that it only addresses the clinical aspect of the case, as the diagnosis could not be pathomorphologically verified due to objective reasons.

Conclusion

The presented case report confirms the association between cervical spine injury and the development of ischemic stroke in pediatric patients. The presence of connective tissue dysplasia features serves as a significant risk factor for the development of vascular complications. The onset of vascular complications amidst existing traumatic injuries – which limit a detailed neurological assessment – can delay the timely diagnosis of a vascular catastrophe and increase the likelihood of an unfavorable outcome. Increasing awareness among specialists involved in managing complicated spinal cord injuries in pediatric patients regarding the risk of cerebral vascular complications, alongside developing age-specific guidelines for treating complicated cervical spine trauma, could improve clinical outcomes by preventing stroke.

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