BALNEO RESEARCH Balneo and PRM Research Journal English Edition
e ISSN 2734 - 8458 p ISSN 2734 - 844X ROMANIAN ASSOCIATION OF BALNEOLOGY

Research article

"Worse than hell": Complex Neuraxial Lesions Resulting in Tetraplegia and Blindness after Polytrauma - Case presentation

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Citation: Anghelescu A., Onose G. Pandrea A., Munteanu C., Magdoiu A.M. - "Worse than hell": Complex Neuraxial Lesions Resulting in Tetraplegia and Blindness after Polytrauma - Case presentation
Balneo and PRM Research Journal **2023**, *14(4): 615*

Academic Editor(s):
Constantin Munteanu

Reviewer Officer:
Viorela Bembea

Production Officer:
Camil Filimon

Received: 12.10.2023
Accepted: 04.12.2023
Published: 20.12.2023

Reviewers:
Liliana Vladareanu
Daniela Profir

Publisher's Note: Balneo and PRM Research Journal stays neutral with regard to jurisdictional claims in published maps and institutional affiliations.

Abstract: Background. Lesions of the vertebral artery (VA) may develop spontaneously or as a result of severe traumatic cervico-cephalic injuries. Most frequent VA injuries (VAI) result from blunt trauma to the spine or may be due to penetrating lesions.
VAI plays a significant role in the broader context of cervical spine trauma by providing insights into the vascular aspects of spinal cord injury (SCI).
Upper cervical spine fractures and accompanying subluxation, as well as transverse process fractures extending into the artery bone foramen, are associated with an elevated risk of VAI.
Clinical case.
A 63-year-old female patient with flaccid C4 AIS-A quadriplegia (global motor score 3/100), vertebral myelopathy, neurogenic bladder, sacral pressure sore, and blindness was transferred from the spinal neurosurgical department.
The patient fell from the same level and suffered a mild brain concussion and, apparently, a minor cervical SCI on October 1, 2022. She was asymptomatic, then quickly became tetraplegic, and then blindness added on in a few days.
She was operated on on October 7, 2022, for a C6 vertebral body fracture and a C5-6 dislocation. The surgical protocol consisted of anterior spinal cord decompression, dislocation reduction, and mixed anterior fusion with bone grafts and fixation screws.
Following the traumatic event, a bilateral posterior cerebral artery (PCA) stroke subsequently produced blindness and partial optic atrophy in both eyes.
Serial clinical and imaging examinations confirmed the diagnosis: a subacute bilateral occipital stroke and a chronic lacunar lesion in the right thalamus associated with a severe spinal cord lesion. Anticoagulation has partially improved the neurological outcomes of stroke in the PCA territory.
Ethical considerations. The patient's next of kin has given written consent to the case study and to use the radiological images related to the case for academic purposes. The personal identity data was anonymized. The presentation of this clinical case has the approval of the ethics commission of TEHBA (no. 40205/01.09.2023).
Discussion. The clinical example is notable for the concurrent development of two severely devastating neurological syndromes, quadriplegia, and blindness, which occurred after an initially minor cervical trauma and progressed gradually over a few days.
VA lesions after cervical trauma are considered rare, and the diagnosis is difficult because the vast majority of individuals do not exhibit cerebral neurological symptoms (due to the collateral compensatory blood supply from the circle of Willis).
Most of the patients with bilateral or dominant VA occlusions are symptomatic, with rapid (and possibly fatal) ischemic damage to the encephalon, cerebellum, or brainstem.
The detection and tracking of evolutionary tendencies required a series of clinical tests, including fundoscopy, and imaging procedures, including native CT, MRI, and MR angiography scans of the brain and spine.
Conclusions. Although VAI is a rare complication, in every polytrauma patient with head and neck trauma, VA damage should be suspected.

Careful clinical and imaging monitoring through detailed serial imaging procedures must be performed for every person with severe cranio-cervical trauma.
The clinical example emphasizes the importance of the interprofessional team's participation in the patient's care.

Keywords: vertebral artery; SCI; tetraplegia; stroke; posterior cerebral artery; blindness.

1. Introduction

Background. The vertebral artery (VA) can be damaged either traumatically or spontaneously. Vertebral artery injury (VAI) is most frequently the consequence of blunt traumatic spinal cord injuries (SCI) or may be due to penetrating lesions in the spine. [1-6]
VAI plays a significant role in the broader context of cervical spine trauma by providing insights into the vascular aspects of SCI.
A retrospective polytrauma study found that 94.6% of 1084 subjects sustained blunt cervical trauma. CT neck screening angiography was performed in 11.7% of cases and detected VAI in 8.3% of subjects. [6]
To further illustrate the clinical case presentation, allow us to take a glance at anatomy (Fig. 1)

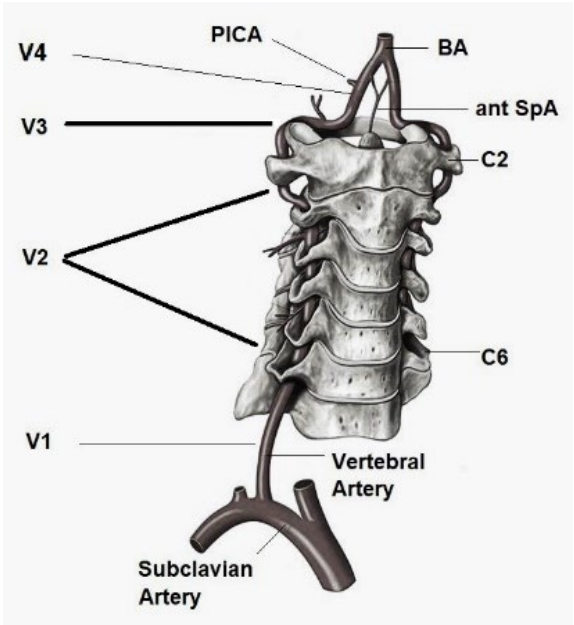

Fig.1 The vertebral artery (VA) and its topographic segments. The majority of VAIs occur in the V2 foraminal arterial segment and are caused by cervical vertebral fractures.

The VA originates from the subclavian artery. Its anatomical path can be divided into four distinct topographic regions. [7]

- The V1 extraosseous arterial segment is adjacent to the first rib. V1 includes the portion from its origin in the subclavian artery to the C7 transverse process and the entry point of the C6 transverse foramen. A major thoracic injury or first-rib fracture is associated with a high risk of VAI.

- The V2 (foraminal segment) is situated within the transverse foramina of the C6-C2 cervical vertebrae. AV crosses the foraminal bony channel formed by overlaying the C6-C2 transverse apophyses of the cervical vertebrae.
  V2 is the most vulnerable artery zone, exposed to severe traumatic injuries and/or inadvertent iatrogenic damage during pedicle screw drilling, tapping, or insertion.
  Upper cervical spine injury (C1 to C3) and mid-cervical (C2-C6) facet fractures, destabilized fractures with associated vertebral subluxation, and/or fractures of the transverse process extending into the transverse foramen are all associated with an increased risk of traumatic VAI. [1-4, 8]
  A recent publication noted that VAI occurred in 45.2% of cases with transverse apophyse fractures extending into the transverse foramina. [3]
- The V3 arterial segment is adjacent to the superior part of the atlas arch and the foramen magnum. The VA is susceptible to inadequate surgical procedures (lateral exposure and laminectomy of C1 or drilling C1-2 trans-articular screws oriented caudally and laterally), which may damage the artery.
  The V3 segment was indicated as the second anatomic region commonly injured (44.4%) in cervical spinal fractures. [3]
- V4 intradural extension from the foramen magnum to unite with the contralateral vertebral artery and form the basilar artery.
  Ethical considerations: The patient's next of kin has given written consent to the case study and to use the radiological images related to the case for academic purposes. The patient`s identity data were anonymized. The presentation of this clinical case has the approval of the ethics commission of TEHBA (no.40205/01.09.2023).
  Clinical case: A 63-year-old woman suffered polytrauma by falling from the same level on October 1, 2022, with a minor brain concussion and cervical SCI. She presented at the TEHBA spinal neurosurgery department two days later, complaining of vision issues and flaccid C4 AIS-A quadriplegia. Investigations revealed a C6 vertebral body fracture, a C5-6 dislocation, and severe spinal cord damage (Fig.2).

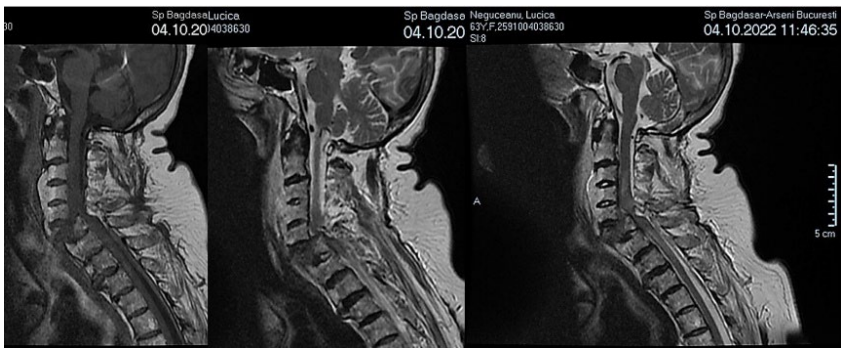

Fig.2 Cervical MRI scan before surgery: C6 vertebral fracture, C5-6 dislocation, and severe spinal cord damage.

On October 7, 2022, neurosurgical intervention was performed: anterior spinal cord decompression, dislocation reduction, corpectomy, mixed anterior osteosynthesis with a bone graft, and metallic plate with three fixation screws were performed (Fig.3).

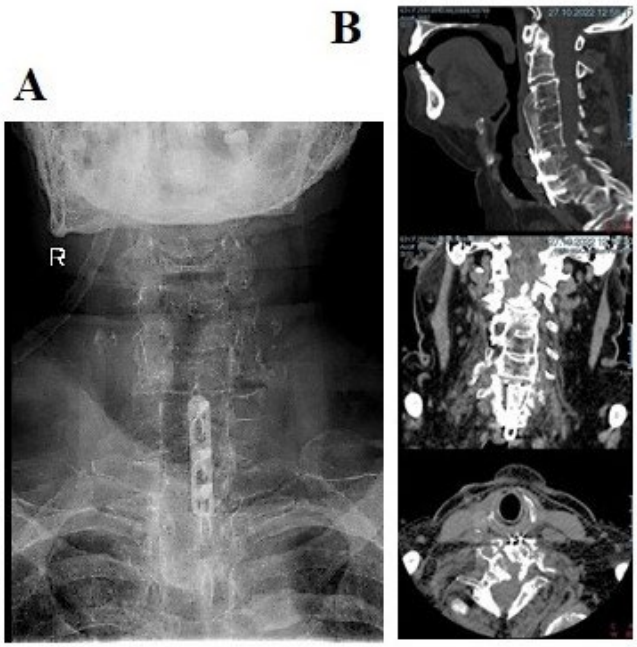

Fig.3 Post-surgical control radiography (A - October 8, 2022) and cervical spine CT scan (B - October 27, 2022; Rehabilitation Clinic): anterior mixed osteosynthesis, C5 bone graft, and plate with three screws. Accentuation of the cervical-thoracic lordosis.

The preoperative protocol includes a systematic imaging assessment of the axial skeletal segments (head, spine, and thorax). A brain CT scan (October 3, 2022) highlighted cerebral ischemic lesions at the level of the occipital lobes with an arterial topographic pattern (stroke in the bilateral PCA territory). The radiological aspect took shape gradually with the evolution, and the arterial topography of the ischemic lesions (Fig. 4) paralleled the severe reduction of her visual acuity.

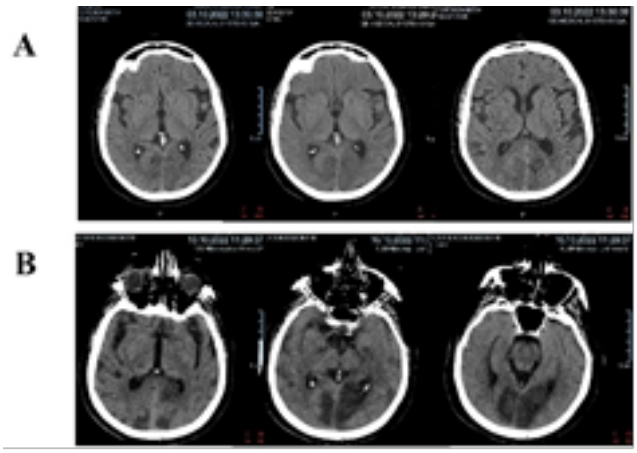

Fig.4 Non-contrast cerebral CT scan: acute cerebral ischemia in the PCAs territory and neuroimaging evolution stages of stroke. Comparative imagery from October 3, 2022 (A) and October 10, 2022 (B). Bilateral occipital hypodense intraparenchymal regions, with parasagittal topography.

Sequential imagery confirmed the diagnosis and the outcomes: bilateral occipital stroke and a subacute lacunar lesion in the right thalamus (Fig.5) secondary to traumatic VAI (Fig.6)

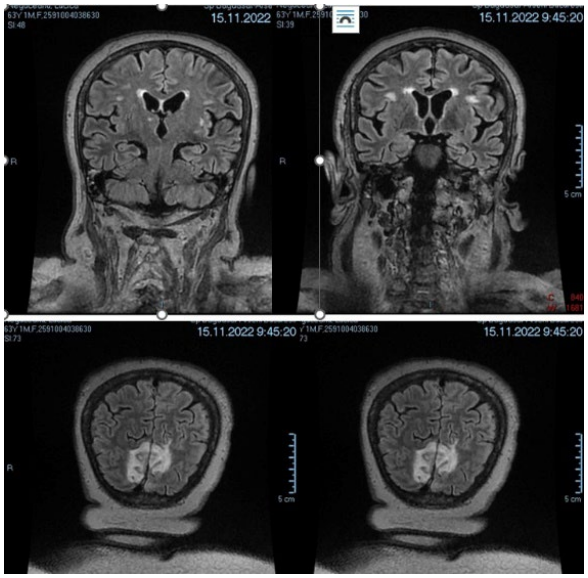

Fig.5 Cranial MRI scan, coronal sections. Thalamic lacunar and occipital subacute stroke (November 15, 2022, one and a half months from the traumatic event).

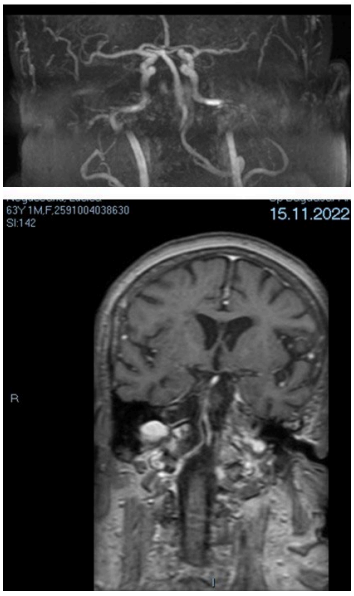

Fig.6 Vertebral artery imaging, angio-MRI cerebral scan (October 15, 2022). Thrombosis or vascular injury at the level of the left VA, possibly permeabilized, probably in a post-traumatic context.
Subacute stroke in bilateral PCA territory, right thalamic lacuna.
On the 3D TOF angiographic sequence or post-contrast sequences, there are no signal or permeability abnormalities in the basilar artery or the proximal bilateral PCA areas. The cortical branches of the PCA were difficult to discern.
Signal anomalies are maintained at the level of the left VA in the V3 and V4 segments until the formation of the basilar artery.

A severe SCI with flaccid C4 AIS-A tetraplegia (global motor score 3/100), neurogenic bladder and colon, post-stroke blindness, and partial optic atrophy in both eyes were all part of the clinical picture. Despite the complex injuries, she was afebrile and cardio-respiratory balanced.

The ophthalmologic evaluation revealed severe vision reduction in both eyes and partial optic atrophy. The patient couldn't distinguish at 50 cm. The optic papillae were flat, well-demarcated, and discolored. Arteries had a slightly reduced caliber, and veins had a standard caliber.

The therapeutic program consisted of rehabilitative nursing procedures, prophylactic anticoagulant medication (Clexane 0.6 ml/daily), sedatives and anxiolytics, antibiotic therapy and urinary disinfectants, poly-vitamins, and neurotrophic products, synergically associated. The sacral pressure sore was dressed with Hydrocol and Grasolind compresses.

The *prognosis ad vitam* was uncertain, and the prognostic *ad functionem* was null. The patient was dependent on care for all the usual and instrumental daily activities. The IADL, BADL, and Barthel scores were zero. She needed recuperative care for life.

Discussion

The clinical case discusses the etiology, pathophysiology, and diagnosis of two interrelated complex neuraxial lesions: a bilateral stroke in the PCA area brought on by traumatic VAI in the setting of SCI.

VAI is an important element in the broader picture of cervical trauma as it offers perspective on the vascular elements of spinal fractures.

VAIs may lead to spinal cord and cerebral vascular compromise, reducing blood supply downwards to the spine and upwards to the brain. It can result in ischemia, contributing to secondary injury cascades and cellular death.

The etiology of injuries to the VA can be traumatic or spontaneous. [1, 2, 4, 6, 8, 9]

Blunt injuries to the head and neck or penetrating trauma to the cervical spine (such as a gunshot wound) are the most frequent etiologic causes of VAI.

VA stenosis, occlusion, intimal tear, dissection, and pseudoaneurysm development are associated with SCI. [1, 3, 9, 10]

Spontaneous VAI is caused by intrinsic vessel wall weakening induced by a vascular or connective tissue condition, resulting in vessel dissection. Associated metabolic imbalances can favor the susceptibility and fragility of the vascular endothelium. [11]

Usually, minimal trauma is the precipitating factor, such as hyperextension associated with lateral neck flexion and/or rotation of the head. [7] These forced postures are related to chiropractic treatment, yoga, and swimming or plunging. [1, 2, 4, 12]

VAI incidence is variable in different studies. Due to the absence of randomized controlled trials, the heterogeneity of study populations, and the methodology of the studies, [3] VAI rates are highly variable, with literature reports ranging from 3 to 88%. Prospective studies suggest rates of 17–27% [10]

A review of the literature noticed 0.09%–0.5% incidence of VAI in blunt neck traumas. About 70% of all cases had an associated cervical spine fracture. [4]

A recent review of 24 studies, including 48,744 patients, revealed an overall 5.19% incidence of VAI. [3]

VAI might have an iatrogenic etiology secondary to anterior cervical spine surgery (0.3%) or posterior cervical spine surgery (4.1–8.2%), mainly after insertion of C1-2 trans-articular screws. [9]

As in our patient, many subjects are initially asymptomatic; some others progress to cerebral ischemia or stroke with permanent neurologic deficits. [2, 4, 7, 9, 13]

Most non-symptomatic individuals have unilateral VA occlusion because the circle of Willis and the contralateral VA compensate for blood flow.

Excluding the rare peculiar cases of VA atresia or hypoplasia [14] and abnormal vertebral anatomy, [7] in normal physiological conditions, the polygon of Willis shunts the blood between the carotid arterial axis and the vertebrobasilar system.

After severe traumatic cervical SCI, VA occlusion with cerebrovascular consequences in the posterior circulation seems to be less common. VAI may lead to potentially severe cerebral complications, as noted in up to 33%–39% of cervical trauma. [2-5, 7, 9]
Most of the cervical spine fractures associated with double neuraxial lesions (tetraplegia and stroke) result from injuries and compromised perfusion to the basilar or vertebral arteries. [9, 2, 4, 6, 9, 10, 13]
Few reported clinical cases have disabling and permanent cerebral neurological sequelae caused by cervical spine fractures. Posterior circulation infarction linked to high-grade stenosis or VAI occlusions was noticed in 7% of patients. [10]
Only 20% of people with non-dominant VA injuries are symptomatic. Most of the subjects with bilateral or dominant VA occlusions are symptomatic and may experience rapid and fatal ischemic damage to the cerebellum and/or brainstem. [2, 3, 9, 10, 13]
Bilateral VA dissection, or retro- and anterograde thrombosis, may occur either at the spine fracture site or at the level where the VA pierces the dura and enters the foramen magnum.
The arterial damage can extend further into the vertebrobasilar system and has a high acute mortality rate, ranging from 0-26.2% across different studies. [2-4, 6, 9]
An unexplained central or lateralizing neurologic deficit, evidence of acute cerebral infarct on CT scan, associated with cervical mid-spine fractures (extended into the transverse foramen) or subluxation, C1-3 fractures (example hanging injuries), or severe thoracic injury or first-rib fracture are indications for vascular exploratory imagery. [2, 4, 5, 8]
Advances in imaging techniques increased the rate of VAI diagnosis. CT angiography and magnetic resonance angiography (MRA) both have a sensitivity of 93.9% for detecting arterial occlusions and stenosis. [5, 7]
The clinical case underlined the possibility of severe complications from an apparent minor polytrauma, which gradually induced a VAI.
Although VAI is a rare complication, in every polytrauma patient with head and neck trauma, VA damage should be suspected. Every patient with severe SCI requires careful clinical and imaging monitoring through complex serial studies (spinal and cerebral CT, MRI, and MRA scans).
There is currently no uniform evidence-based management protocol. [3, 5, 9]
Diagnosing and treating VAI can frequently be challenging because, at the time of damage, many patients may be asymptomatic.
Post-operative prophylactic anticoagulation is routinely administered immediately after spinal surgery. It is continued throughout the subacute phase in our rehabilitation clinic to prevent thromboembolic complications in the peripheral venous system and, accordingly, the central encephalic ischemic stroke attributed to VAI.
Due to the heterogeneity of study populations, study design, and outcome measures, there have been no randomized controlled trials regarding management strategies to date. [3, 9]
Under these conditions, the real VAI-attributable mortality rate is not exactly estimated.

Conclusions.

What disability could be worse than blindness and severe tetraplegia?
The clinical case illustrated the concurrent development of two severely devastating neurological syndromes, quadriplegia, and blindness, which occurred after a neglected SCI that progressed gradually over a few days.
VAI was linked to significant cervical lesions and severe bilateral ischemic stroke in the PCA territory in the context of a minor polytrauma.
There was consideration of the etiology, pathophysiology, and diagnosis of two associated (and linked) severe neuraxial lesions.
The evolution was unpredictable and gradual in two stages (like a camel's humps). The outcomes of VAI were challenging to predict.

VAI might have disastrous consequences, such as basilar territory infarction and death.
Although VAI is a rare complication, in every polytrauma patient with head and neck trauma, VA damage should be suspected.
Every subject with severe SCI requires careful clinical and imaging monitoring through complex serial studies (CT, MRI, and MRA spinal and cerebral scans).
Anticoagulation, systematically administered to all AIS-A subacute tetraplegics, has partially improved the neurological outcomes of stroke in the PCA territory.
The clinical example highlighted the value of the multi- or interprofessional team's participation in the patient's care. It provided a framework for focused investigations and therapies to enhance the prognosis of our patients.
Prospective controlled and randomized studies are further needed to improve diagnostic and treatment protocols.

Authors declare no conflict of interests

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