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Case Report

Concomitant Pontine and Cerebellar Infarction Due to Traumatic Vertebral Artery Dissection Secondary to a Cervical C2 Fracture

Coneys Ulysse*, Lauper Nicolas, Dominguez Dennis Enrique and Al Taha Khalid

Spine Team, Division of Orthopedics and Trauma Surgery, Geneva University Hospitals and University of Geneva, Faculty of Medicine, Geneva, Switzerland

***Corresponding Author:** Coneys Ulysse, Spine Team, Division of Orthopedics and Trauma Surgery, Geneva University Hospitals and University of Geneva, Faculty of Medicine, Geneva, Switzerland.

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Abstract

We report a case of acute pontine ischemic lesion concomitant with a traumatic vertebral artery dissection (VAD) after cervical fracture. A patient in their 80s was referred to our hospital with a right brachio-crural hemiparesis and dysarthria after multiple low-energy falls without witness and cervical pain from a previous fall two weeks ago. A cervical computed tomography (CT) scan undergone at admission showed a traumatic C2 fracture Anderson Alonzo type 2 without involvement of transverse foramina. A neck angio-CT showed a left vertebral artery dissection at the fracture level which was confirmed by a cervical magnetic resonance imaging (MRI) revealing a loss of signal void on the left vertebral artery at V2-V3 junction. No cervical medullary injury was found. A complementary brain MRI was executed and showed an acute left pontine ischemic lesion and bilateral cerebellar infarction. The patient C2 fracture was treated conservatively by a cervical collar and referred to our stroke unit for the ischemic brain injury management. The purpose of our case report is to attract clinician attention to rare occurrences of pontine infarction in the setting of a traumatic VAD following cervical fracture.

Keywords: *Vertebral Artery Dissection (VAD); Computed Tomography (CT); Magnetic Resonance Imaging (MRI)*

Introduction

In rare cases of cervical spine injury, traumatic vertebral artery dissection (VAD) has been described [1-4]. When bilateral, VAD can occasionally causes vertebrobasilar stroke; however, unilateral VAD is often asymptomatic and therefore treated conservatively. Nevertheless, a particular attention should be given to silent vertebral artery injury because of tendency of theses dissection to cause symptomatic cerebral infarction in delayed fashion or due to an unexpected triggering factor [4]. We report a case of unilateral VAD presenting with symptomatic, cerebellar and pontine infarction and discuss the clinical course.

Case Report

A patient in their 80s was referred to our hospital with a right hemiparesis and dysarthria after multiple low-energy falls without witness and cervical pain from a previous fall two weeks ago. The patient could not stand up and stayed on the floor for at least 3 hours. The patient was known for treated hypertension, auricular fibrillation treated by apixaban and diabetes.

The initial assessment showed variation in systolic arterial pressure and the neurological examination revealed a NIHSS score of 11, severe dysarthria, M1/5 paresis for the right upper limb, M2/5 paresis for the right lower limb and a right Babinski sign.

A cervical computed tomography (CT) scan at admission showed a traumatic C2 fracture Anderson Alonzo type 2 without involvement of transverse foramen (Figure 1). A neck angio-CT showed a left vertebral artery dissection at the fracture level confirmed by a cervical magnetic resonance imaging showing a loss of void signal on the left vertebral artery at V2-V3 junction. The cervical imaging also showed a potential C2 C3 ligamentum flavum rupture and C1 C2 anterior longitudinal ligament rupture calling for a flexion-extension traumatism mechanism (Figure 2). There was a beginning of a fibrosis ring around the fracture lines showing the subacute-chronic timing of the fracture.

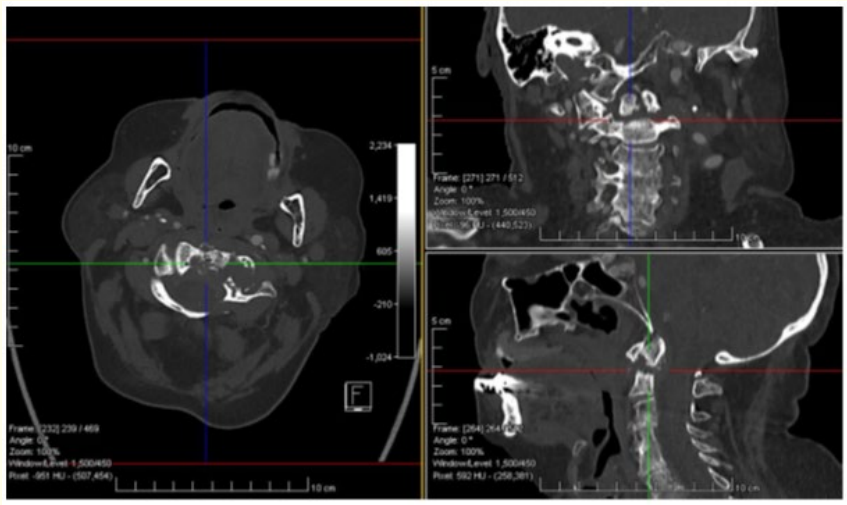


Figure 1: *Native cervical CT scanner showing a C2 odontoid Alonzo-Anderson type 2 fracture in the axial plan (left image), coronal plan (upper right image) and sagittal plan (lower right image).*

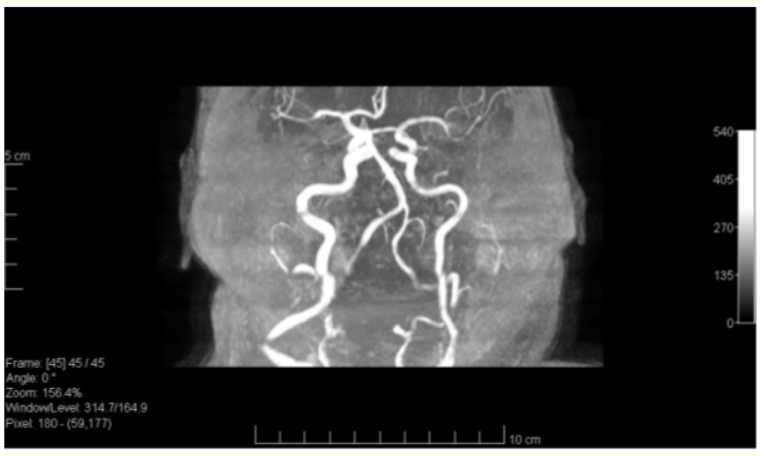


Figure 2: *3D angiogram showing a gap at V2-V3 junction of the right VA.*

Faced with the neurologic symptoms, we proceeded with a brain MRI that revealed an acute left pontine ischemic lesion associated with bilateral cerebellar lesion (Figure 3-5).

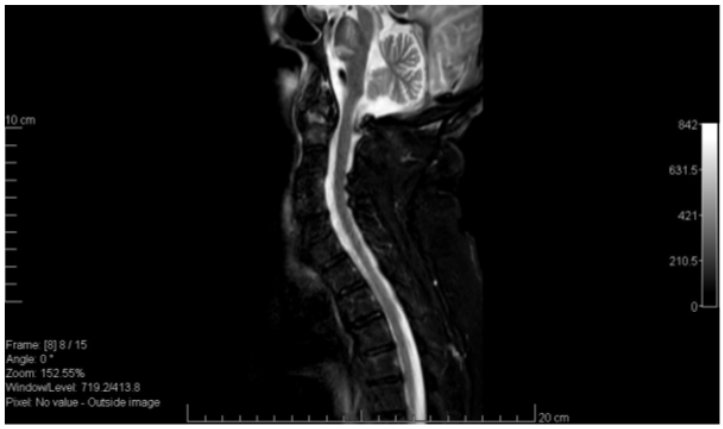


Figure 3: *Sagittal T2-STIR sequence cervical MRI showing no cervical medullary insult and potential C2 C3 ligamentum flavum rupture and C1 C2 anterior longitudinal ligament rupture.*

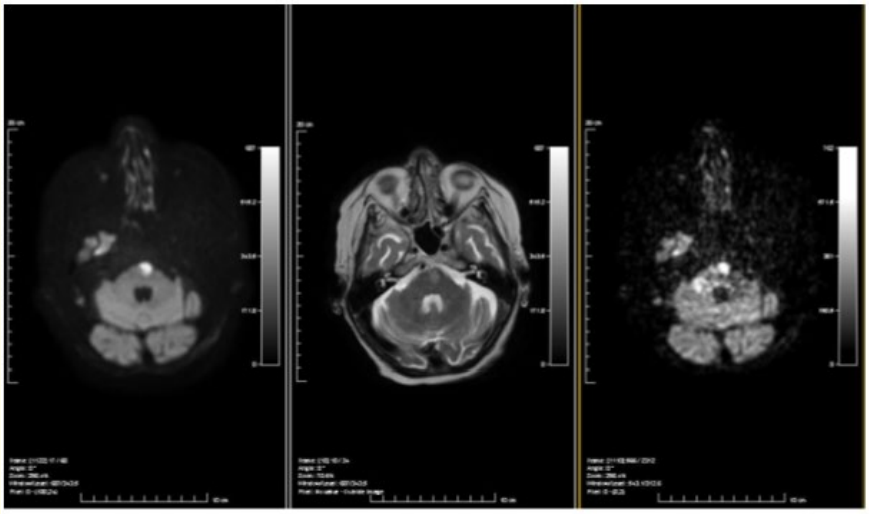


Figure 4: *Left pontine acute ischemic infraction with on the left image FLAIR hyperintensity, on the middle image T2 hyperintensity and on the right image diffusion weighted restriction.*

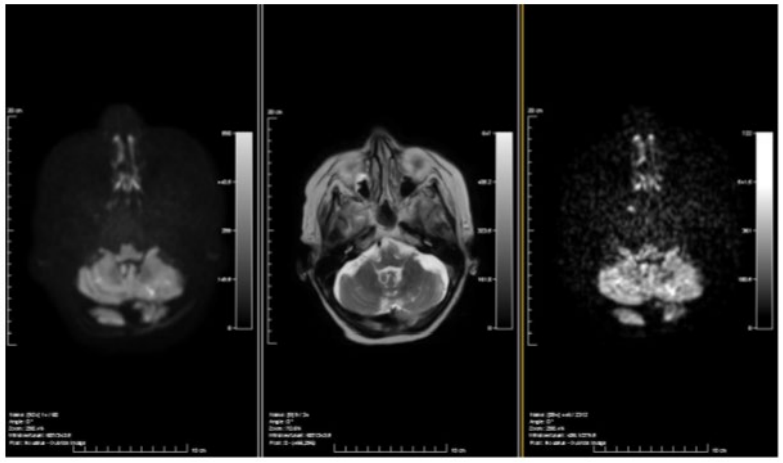


Figure 5: *Bilateral cerebellar punctiform acute ischemic infraction with on the left image FLAIR hyperintensity, on the middle image T2 hyperintensity and on the right image diffusion weighted restriction.*

The patient was treated conservatively by a cervical collar for the cervical C2 fracture. In fact, there was near anatomical alignment, a less than 5 mm gap and a beginning of a fibrose non-union [5]. The patient was managed at our stroke unit by anticoagulation therapy, clinical and radiological follow-up showing stability in terms of neurological deficits and repeated brain and cervical MRI obtained at day 1 showed a stability of the acute left pontine ischemic lesion and of the bilateral cerebellar lesion. The patient was transferred afterward to a neuro rehabilitation facility after a last brain and cervical MRI obtained at day 5 confirmed the stability of the lesion.

At 3 months follow-up the clinical examination was the same and the fracture CT scan showed a fibrous non-union. A surgical treatment by C0-C4 posterior fixation was proposed, but the patient refused that option.

Discussion

Because it is rarely symptomatic and easily unnoticed, the incidence of vertebral artery injury after blunt trauma of the cervical spine is unknown [2]. Vertebral artery injury may be more prevalent than commonly believed after cervical spine fracture.

Traumatic VAD following in the context of a severe neck trauma was first described in 1955, and most cases result from motor vehicle accident [3]. The potential for vertebral artery injury should be considered in case of blunt trauma of the cervical spine [6]. Although the exact mechanisms aren't fully understood, there appears to be a significant link between arterial dissection and cervical trauma. Giacobetti., *et al*. [1] stated that flexion-distraction type injuries are the most common cause of non-penetrating vertebral artery damage, followed by a flexion compression injuries.

Due to their unique path through four or five transverse foramina, the vertebral arteries are especially vulnerable to direct trauma [3]. Fractures of the lateral masses, particularly those involving the transverse foramen, can injure the artery within its bony borders. The initial arterial injury likely involves intimal disruption caused by excessive stretching and distraction of the artery between two adjacent transverse foramina [6]. A unilateral occlusion of the vertebral artery occasionally results in symptomatic ischemic stroke.

Blunt trauma to the vertebral artery can lead to occlusion, which may extend beyond the site of bony injury. The site of dissection can serve as a site of thrombosis development or artery-to-artery emboli, the latter may result with occlusion of the distal branch artery

[7,8]. Symptoms of VAD following neck trauma typically include neck and/or head pain, often localized to the initial injury site, which are common warning symptoms of dissection. These symptoms are usually mild, and there might be no neurological deficits [3].

The occurrence of vertebrobasilar ischemia after cervical spine injury was first documented in 1955 [9] when posterior inferior cerebellar artery (PICA) syndrome manifested several days after a C5 fracture. Interestingly, Unilateral VAD with subsequent thrombosis might not present clinical symptoms due to sufficient blood supply from the contralateral side via the PICA and/or collateral circulation through intramuscular vessels of the thyrocervical trunk [10]. Willis., *et al*. [6] reported findings from a prospective clinical study where none of 26 patients with a vertebral artery injury exhibited clear neurological dysfunction or other sequelae.

Parent., *et al*. [10] described a retrospective analysis of 640 patients with cervical spine fractures revealing that five patients experienced lateral dislocation of the cervical spine along with injuries to the vertebral arteries. Three of these patients showed symptoms of vertebrobasilar ischemia, prompting a diagnosis of VAD confirmed though angiographic or postmortem findings. While conventional angiography remains the gold standard, Doppler ultrasound, duplex sonography, MRI, and MR angiography can reveal vessel stenosis or occlusion, and hematoma may be detected within in the vessel wall [11].

Our case had a systolic pressure variation and a normal patent non dominant contralateral vertebral artery and developed neurological deficits to ischemic infarction manifested by his clinical neurological presentation. According to 'the selfish brain' hypothesis of hypertension, aging leads to significant reduction in vertebral artery blood flow, with this decline being four times greater than the reduction in internal carotid artery flow. This substantial decrease in vertebral artery flow may significantly contribute to the age-related increase in mean arterial pressure (MAP) [12]. Since cerebral autoregulation is often impaired in the stroke-affected area, even minor fluctuations in blood pressure can cause under or over perfusion. In the hours following the onset of brain ischemia, sudden drops in blood pressure decrease the likelihood of reperfusing the potentially viable penumbra around the ischemic core, increasing the risk of tissue ischemia and the expansion of the lesion size [13]. These changes in cerebral autoregulation could be an explanation to our patient's ischemic manifestation during the compensatory process of taking in charge the occluded VA territories by the non-dominant patent VA of the contralateral side facing a concomitant blood pressure variation in our hypertension known patient.

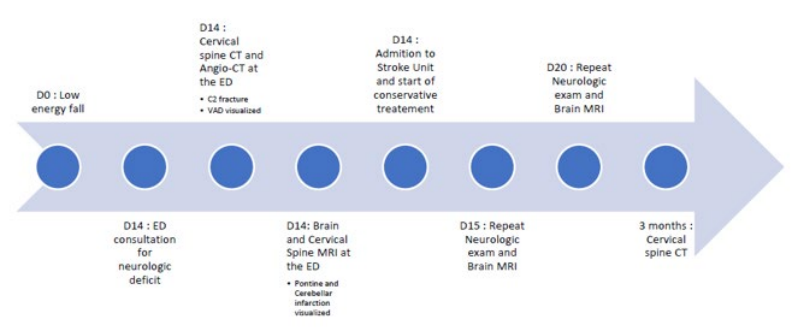
The treatment of traumatic vertebral artery dissection remains a subject of debate, with no established guidelines for the timing or methods of anticoagulation or interventional therapy. The primary goal during the acute phase is to prevent ischemic stroke [4]. Antiplatelet or anticoagulation therapy is generally recommended for intimal disruption [14]. Initial management typically involves antiplatelet or anticoagulation therapy, though anticoagulation poses a significant hemorrhagic risk in trauma patients. Interventional therapy is considered for patients with severe vertebral artery injuries or symptoms of vertebrobasilar embolism [15]. The prognosis for vertebral artery dissection is generally favorable, contingent on the extent of the damage and the adequacy of collateral circulation. Typically, vertebral artery dissection does not result in critical manifestations [4].

The incidence of ischemia associated with VAD in patients with cervical spine trauma is low due to the presence of the contralateral vertebral artery. However, if collateral blood flow is insufficient, an occlusion can result in a severe vertebrobasilar stroke [4]. Early recognition of VAD following cervical injury and an understanding of its natural history can lead to more effective management and improved survival outcomes.

Conclusion

We report a rare case of symptomatic, pontine and cerebellar infarctions concomitant with the setting of a unilateral VAD after C2 cervical fracture. Physicians should remain vigilant for the possibility of cerebral infarction due to unilateral VAD following cervical injury, even though its incidence is reported to be low in the literature.

Timeline



Figure

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