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Case Report

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Post-Traumatic Vasospasm after Mild Brain Trauma, a Lesson Learned.

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Abstract

Background: *Post-traumatic vasospasm is a frequent secondary injury in patients with traumatic brain injury, occurring in up to 63% of patients and with associated deleterious consequences. Most studies have reported post-traumatic vasospasm in patients with severe or moderate brain trauma; however, there are documented cases of post-traumatic vasospasm in patients with mild traumatic brain injury that support a more aggressive search to prevent deleterious neurological outcomes.*

Observation(s): *The pertinent literature has been reviewed, and an exemplary case has been reported (post-traumatic vasospasm associated with mild brain trauma in an adult patient that subsequently transformed into cerebral ischemia that was managed with endovascular therapy and oral nimodipine without subsequent neurological deterioration). To date, only 4 cases have been described, to our knowledge this is the first literature review so far.*

Lessons: *Given the possibility of post-traumatic vasospasm associated with mild brain trauma, the authors believe that GCS score alone may be an inadequate risk predictor of symptomatic cerebral vasospasm and a more aggressive search should be performed in patients with mild trauma who present with neurological deterioration without apparent cause.*

Keywords: Posttraumatic Vasospasm, Traumatic Brain Injury, Mild Brain Trauma, Balloon Angioplasty, Cerebral Ischemia.

Introduction

Post-traumatic cerebral vasospasm (PTV) is a secondary injury in brain trauma patients [1]; its epidemiology to date is unknown due to the limited studies in the literature. However, it is known that the presence of cerebral vasospasm is associated with worse neurological outcomes in patients with TBI [2,3]. Usually, vasospasm studies are not performed in patients with mild brain trauma, being overlooked on many occasions of neurological impairment without apparent cause.

We present a rare case of an adult patient with mild TBI associated with PTV and subsequent cerebral ischemia that was managed with endovascular therapy with a proper neurological outcome, emphasizing the need to suspect this secondary injury in all patients with brain trauma and neurological deterioration without apparent cause.

Illustrative Case

A 23-year-old male patient presented to our neurosurgical service with a history of falling from a height, 2 hours before during alcohol consumption. On initial neurological examination a Glasgow coma scale (GCS) of 14 was documented, due to a partial and passing disorientation that improved after 1 hour. His main complaint during his stay in the emergency room was a mild left temporal headache. It was decided to perform a brain CT to rule out secondary injuries associated with skull trauma, a small subarachnoid hemorrhage was found in the left Sylvian fissure (Figure 1), no mass effects and no apparent signs of brain hypertension were seen.

The patient was left on neurological surveillance and intravenous analgesics were given to manage his headache. At the second day of hospitalization, the patient remained with his headache, a second tomography was performed (Figure 2) and a slight SAH on the interhemispheric fissure was seen, a digital subtraction angiography (DSA) was made to rule out associated vascular lesions. No aneurysms were found in the cerebral angiography; however, a narrowing in the left cerebral vessels (MCA-ICA) was evident (Figure 3), so the patient was sent to the neurological care unit for constant supervision. The next day, the patient began with speech difficulties and was rushed to the endovascular service, where a second DSA and endovascular therapy was performed with bal-

loon angioplasty and intra-arterial nimodipine to manage his symptomatic vasospasm.

In its postoperative stage, the patient was treated with the protocol of subarachnoid hemorrhage (SAH) with oral nimodipine for 21 days, being fully recovered 12 days after his hospital stage. After 20 days of stay, a brain MRI was performed, in which brain ischemia was seen on left temporal lobe (Figure 4), and a final DSA was performed to rule out a brain aneurysm not seen in the first study, which remains negative.

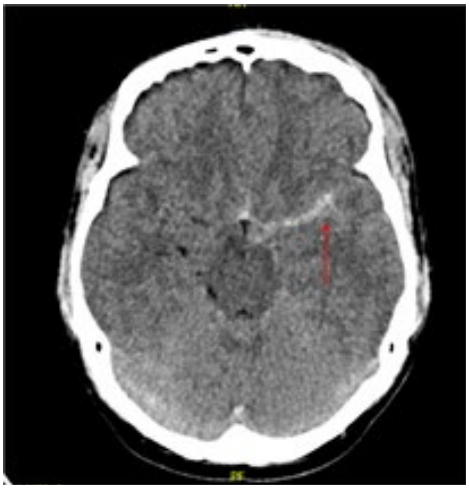

Figure 1: Axial Brain CT with traumatic subarachnoid hemorrhage (red arrow) on left Sylvian fissure

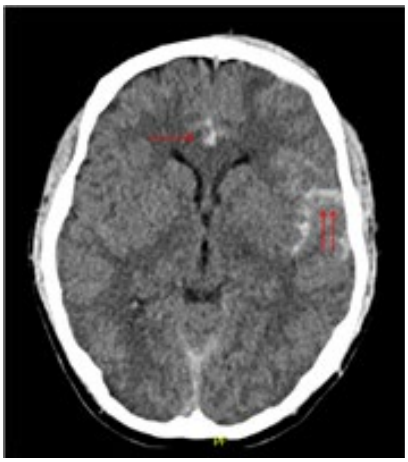

Figure 2: Axial CT with traumatic subarachnoid hemorrhage with blood clot on interhemispheric fissure and left Sylvian fissure (red arrows)

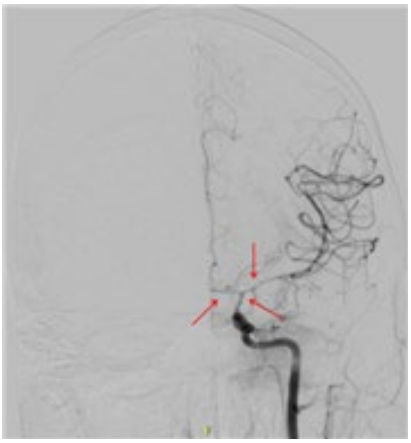

Figure 3: Digital subtraction angiography showing vasospasm on left middle cerebral artery, anterior cerebral artery and left internal carotid artery (red arrows)

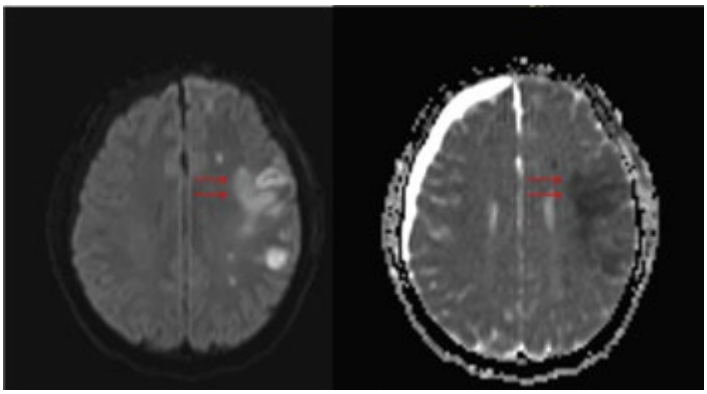

Figure 4: Increased DWI signal (left) with a reduction on ADC sequence (right) on the left temporal lobe, indicating cerebral ischemia

The patient was in total 23 days hospitalized, with an improvement of his clinical status and without neurological impairments, he was visited in the hospital by the speech therapy specialist and by the neurology team, showing a proper recovery during his hospital stay, he was discharged without any neurological pattern and reviewed 3 and 6 months after his trauma with a proper neurological recovery

Discussion

Observations: Traumatic brain injury (TBI) is a major cause of disability among young patients worldwide [4-6], with primary lesions that can be deleterious and secondary lesions that must be managed to prevent associated complications [7,8]. Post-traumatic cerebral vasospasm (PTV) is one of these secondary lesions with deleterious neurological outcomes in TBI patients, its pathophysiology is currently subject of study, however, an association with vessel compression during blunt trauma and the irritative factor of subarachnoid hemorrhage has been documented [9, 10], similar to what occurred in SHA from an aneurysm.

Different risk factors have been documented for the appearance of

PTV, such as the presence of intracerebral hematoma, subarachnoid hemorrhage, fever, the severity of TBI measured by GCS, and the number of affected lobes [11].

Table 1: Post-Traumatic Vasospasm in Mild Brain Trauma, Literature Review

Authors	Number of patients reported	Clinical characteristics	CT findings	On-set of vasospasm	Vasospasm diagnosis	Treatment	Outcomes
Loret et al,2013[29]	1	17 y-o female Fell down the stairs GCS (14)	tSAH SDH	14 days after TBI	CTA	Intraarterial nimodipine Heparine IV Balloon angioplasty decompressive craniectomy due to brain swelling	GOS –E: 8 after 3 months
Ogami et al,2017[28]	1	63 y-o female ground-level mechanical fall GCS (14) On rivaroxaban due to Atrial fibrillation	tSAH SDH	4 days after TBI	DSA	Intraarterial verapamile	mild word-finding difficulty.
Dessai & Morris,2018[27]	1	27 y-o male struck by a car while walking GCS (14)	tSAH SDH Temporal contusion	5 days after TBI	CTA	Intraarterial verapamile Controlled hypertension	paraphasic
Borsotti et al,2019[26]	1	55 y-o male Motor vehicle accident GCS(15)	tSAH SDH	2 days after TBI	CTA with perfussion	Intraarterial nimodipine	Complete neurological improve

Computed tomography (CT),Glasgow coma scale (GCS), Traumatic subarachnoid hemorrhage (tSHA),Subdural hematoma (SDH),Traumatic brain injury (TBI),CT angiogram (CTA),intravenous (IV), Glasgow Outcome Scale Extended (GOS-E)

In terms of epidemiology, most of the studies to date are case series of patients with severe and moderate TBI, documented an incidence varyingly reported from 35.6 to 61%% [12, 13], however, PTV is rarely reported in patients with mild brain trauma (Table 1), with few reports associated with important neurological impairtment such as cerebral ischemia [14].

PTV presentation varies over time compared to vasospasm associated with cerebral aneurysm, with posttraumatic vasospasm presenting earlier (2-3 days post-trauma vs 3-5 days post-aneurysmal vasospasm) [15], the diagnostic aids for cerebral vasospasm are the same as for aneurysm-associated vasospasm, with brain angiography as the Gold- standard for his diagnosis and CT angiogram being a good less invasive option, but with the disadvantage of not observing mild or moderate vasospasm [16-19].

The management of this secondary lesion has not been well defined, some studies have shown the usefulness of nimodipine in preventing deleterious neurological outcomes in patients with SAH when its associated with brain trauma, with good results at 6 months in patients managed with oral nimodipine [20-22]. Balloon angioplasty has also been associated with neurological improvement in brain trauma patients and PTV, showing an improvement in cerebral blood flow after endovascular treatment [23].

The outcome of patients with TBI is worse in patients with PTV than in patients with TBI without it, the management with oral nimodipine or endovascular therapy had shown to reduce mortality and vegetative status rates in brain trauma patients associated with PTV, however, few studies had documented this report on mild trauma, being this the first to complete a literature review of the subject [24].

Lessons

Although cerebral vasospasm has been studied in SAH of aneurysm origin with different studies about its pathophysiology, post-traumatic vasospasm has scarcely been studied in the literature, being the majority case reports and small studies,where their

higher incidence is on severe and moderate TBI [25].

However, cases where this secondary lesion is present in mild TBI patients had been documented [26-29], which warns of the importance of not relying solely on GCS as a predictor of PTV and neurological deterioration in patients with brain trauma, having to date different tools to diagnose this entity in any patient with mild TBI with neurological deterioration without apparent cause. To date it is important to report and know the association between the different PTV management strategies that are being made known in order to provide safe and timely care to patients with this rare complication of TBI.

Disclosures

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