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Case Report

Delayed Post Traumatic Vasospasm Leading to Ischemia in a Patient with Mild Traumatic Brain Injury

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Abstract

This article describes ischemic symptoms following post-traumatic vasospasm in a patient with mild traumatic brain injury. A 17-year-old female presented with left hemiparesia, confusion and right mydriasis 14 days after a moderate head injury resulting in brain contusion and basal cisternal sub-arachnoid hemorrhage. Bilateral supraclinoidal internal carotid artery vasospasm and a right anterior and middle cerebral arteries stroke were diagnosed. She underwent decompressive craniectomy and balloon angioplasty. Patients with mild traumatic brain injury and serious lesions on initial computed tomography should be evaluated by warrant screening strategy using early computed tomographic angiography and transcranial doppler to diagnose post-traumatic vasospam.

INTRODUCTION

Traumatic sub-arachnoid hemorrhage (tSAH) has been recognized as the most frequent traumatic brain lesion. However, to the best of our knowledge, the present case would be the first case of symptomatic ischemic post-traumatic vasospasm (PTV) following mild traumatic brain injury (TBI), which is described as TBI with Glasgow Coma Scale (GCS) score not worse than 13.

CASE REPORT

A 17 year old female fell down the stairs and presented head injury with transient loss of consciousness. On admission, her Glasgow Coma Scale (GCS) score was 14. Computed Tomography (CT) scan revealed a left basifrontal contusion, thin subdural hematomas of the right frontal convexity and the left frontal falx cerebri, a midline shift less than 5 mm, a tSAH with prepontine, chiasmatic and sylvian cisternal blood (Figure 1). The Rotterdam CT score [1] was 3. During next days, she was alert, fully oriented, without focal neurological deficit, and presented severe headache eased by analgesic. Follow up CT scan was similar. She was discharged 9 days later without any neurological deficit. On day 14, she suddenly developed left hemiparesia, deterioration of consciousness and a fluctuating right mydriasis. GCS score was 10. CT scan demonstrated new hypodensity in the right frontal hemisphere (Figure 2). CT angiography (CTA) showed a bilateral narrowing of the supraclinoidal internal carotid arteries (ICA) (Figure 3). Diffusion Weighted Magnetic Resonance Imaging (MRI) confirmed acute ischemia in territories of the right anterior and right middle cerebral arteries (Figure 4). The infarct volume was 145 cm^3. The patient did not take any medication which could induce vasospasm.

Given the patient's worsening condition and radiological findings, it was decided to realise decompressive craniectomy. A large right frontotemporoparietal craniectomy removing a bone

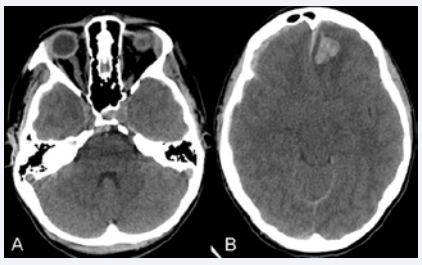

Figure 1 Axial initial CT scan showing a prepontine sub-arachnoid hemorrhage (a) and left basifrontal contusion, thin subdural hematomas of the right frontal convexity and the left frontal falx cerebri, midline shift < 5 mm (b).

flap of 13 cm diameter reaching the floor of middle cerebral fossa with dural opening was performed. After surgery, the patient underwent endovascular management of internal carotid arteries vasospasm. The femoral artery was cannulated, and a 6-French Envoy NPD™ Catheter (Cordis Warren, NJ, USA) was placed in the internal carotid artery. Digital Substraction Angiography (DSA) confirmed severe vasospasm affecting bilateral C4 segment of supraclinoidal internal carotid artery (Figure 5a and 5b). Nimodipine was infused *in situ* and 2000 units of heparin was administered intravenously. Angioplasty was performed, using a 4*10mm HyperGlide™ balloon catheter (ev3 Inc., Plymouth, MN, USA), to dilate the stenotic segment of both ICAs (Figure 5c and 5d).

The patient tolerated surgery and endovascular procedure without any immediate worsening of her neurological status. Few days later, hemiparesia almost completely resolved. She was discharged to physical medicine and rehabilitation department 2 weeks later with left facial weakness, discreet palsy in the left upper extremity and a mild frontal syndrome. The final neurological outcome according to the Extended Glasgow Outcome Scale at 3 months was an upper good recovery (GOS-E 8). She resumed school on the same level. Cranioplasty was performed two months after craniectomy.

DISCUSSION

In the pre-CT scan era, tSAH was identified as the most frequent traumatic brain lesion by autopsy [2]. It was also identified as an independant predictor of worse clinical outcome [3-8] and has been proposed as a causal factor of delayed PTV [7,9-11]. However this association has not been fully elucidated.

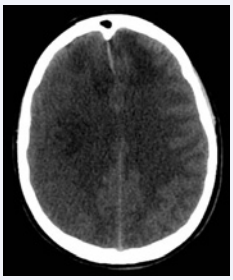

Figure 2 Axial CT scan on day 14 showing new hypodensity in the right frontal lobe.

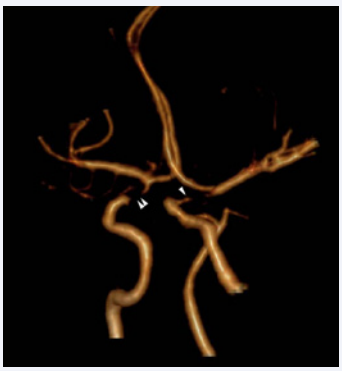

Figure 3 Left anterolateral view of the circle of Willis by 3D reconstructed CTA showing a narrowing of the supraclinoidal left (arrow head) and right (double arrow head) internal carotid arteries.

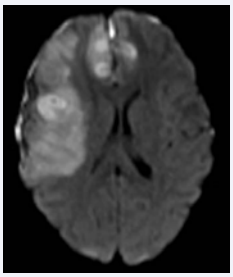

Figure 4 Diffusion weighted MRI (b1000) on day 14 confirming ischemia in territories of the right anterior and the right middle cerebral arteries.

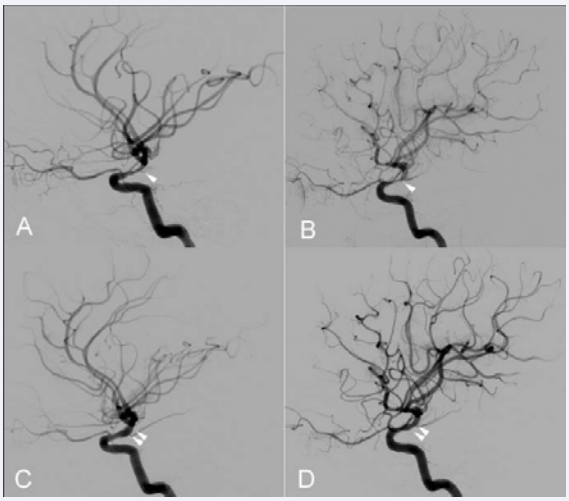

Figure 5 Left lateral views of Digital Substraction Angiography of the left (a) and right (b) internal carotid arteries showing severe bilateral vasospasm of C4 segment of supraclinoidal intenal carotid arteries (simple arrowheads). After balloon angioplasty, left (c) and right (d) internal carotid arteries have recovered a normal shape (double arrowheads).

Vasospasm resulting from aneurysmal SAH is a well known complication, which occurs up to 40% of patients but results in ischemic neurological symptoms only in 50% of vasospasm cases [12,13], and usually starts around day 3 after SAH onset. Maximal risk of vasospam is about day 7, but risk can persist up to 2 weeks [7,13,14]. On the contrary, the reported incidence of PTV has varied widely, probably because of differences in patient selection criteria, in definition of vasospasm or in method for detecting spasm, and in sample size of studies [6,8,13-15]. The true incidence of PTV is thus uncertain, but with severe head injury, systematic and repeated transcranial doppler (TCD) measurements and use of Lindegaard ratio [16], it is about 40% [8]. PTV seems to be more common in patients with hyperthermia on admission [6], low initial GCS scores [13,14] and in younger patients [13]. According to Zubkov & al [14], none of the patients with initial GCS score up to 12, suffered PTV.

Similarly, CT scan risk factors are not clearly identified. Many reports confirmed that the presence of subarachnoid blood after

head injury significantly influenced the incidence of PTV [3,13]. The Rotterdam CT Score [1] considered the items proposed by Marshall et al. [17], and the presence of tSAH or intraventricular hemorrhage. Studies have confirmed the predictive value of the Marshall CT classification and the international guidelines on prognosis include that classification as a major predictor [1]. The amount and location of subarachnoid blood may also play a role in TBI prognosis [5,7]. Furthermore PTV can be seen in patients with intraventricular hemorrhage, subdural hematoma and parenchymal contusions [3,6,10,15] but not in those with normal CT scan, cerebral edema or extradural hematoma. In summary, young patients (<45 years old), with tSAH of basal cisterns and basifrontal or basitemporal contusions on initial head CT scan, might develop PTV. The aim of defining high-risk patients of PTV would be to offer preventive care to that population. Oral nimodipine (60 mg every 4 hours) should be administrated after aneurysmal SAH for a period of 21 days to prevent vasospasm [18]. Nevertheless there is no evidence that this preventive treatment should be proposed after tSAH.

Thus we need to provide special screening for these patients. TCD may be used for detection of large artery vasospasm [18-20]. However, CTA is actually the investigation of choice to diagnose cerebral arteries vasospasm [20]. A high degree of correlation between CTA and DSA for diagnosis of delayed cerebral vasospasm in patients with aneurysmal SAH has been demonstrated [20]. CTA seems to be less precise than DSA for quantifying the degree of vasospasm and for assessment of distal arteries but may be a screening tool for patients with suspected symptomatic vasospasm, and may reduce the need for DSA. Initial head CT scan showing tSAH should include CTA in order to have comparative imaging and diagnose cervical arteries injury. The best timing and repetition of CTA should be at least on admission and before being discharged. Moreover, early CT Perfusion measurement of Cerebral Blood Flow was proposed to predict delayed cerebral ischemia in patients with aneurysmal SAH [20]. These recommandations may be applied to tSAH management, but exposure to X-rays has to be considered.

Once PTV is diagnosed, one should discuss the appropriate treatment, which is complicated by multiple factors and cannot follow recommandations for treatment of aneurysmal SAH vasospasm. Triple-H therapy (hypertension, hypervolemia, hemodilution) may be deleterious for patients with diffuse edema, hyperhemia, mass lesions. However, hypertension may be necessary in patients with hypoperfusion [21]. Intra-arterial papaverine and balloon angioplasty have been shown to successfully reduce vasospasm and ischemic neurological deficits. Nevertheless, superiority of one treatment has not been demonstrated because of predominance of retrospective analyses and small sample size in most series [22]. In our case, the choice of balloon angioplasty was led by the segmental and proximal characteristics of vasospasm.

Finally, early decompressive craniectomy in malignant middle cerebral artery infarction reduced significantly mortality and disability, in patients with diffusion weighted MRI infarct volume > 145cm^3 who were treated within 48 hours of stroke onset [23]. In HAMLET (Hemicraniectomy After Middle cerebral artery infarction with Life-threatening Edema Trial), 22% of patients who were treated surgically presented a middle cerebral artery plus posterior or anterior cerebral artery infarct [24]. Therefore, considering age of the patient, infarct volume and delayed neurologic deterioration we would recommand the use of hemicraniectomy in such cases of ischemic PTV.

CONCLUSION

In conclusion, warrant screening strategies may be useful to early diagnose PTV and to prevent ischemic damage in young patients with TBI (even mild or moderate) and basal cisternal tSAH or basal cerebral contusion on initial CT scan. Although risk factors of PTV are not clearly identify, and efficiency of any preventive medication is currently uncertain, studies are necessary to assess which patients really need this complete screening and a preventive medical treatment of post-traumatic cerebral vasospasm.

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