Cerebral vasospasm after selective amygdalohippocampectomy

INTRODUCTION

Symptomatic cerebral vasospasm (CV) is a frequently observed complication after aneurysmal subarachnoid haemorrhage (SAH).[1] Infrequently, it is known to occur after skull base surgeries and surgeries for medically intractable epilepsy (viz., temporal lobectomy and selective amygdalo-hippocampectomy (AHE).[2] Factors predicting the occurrence of CV after AHE have been described.[3] Transcranial Doppler (TCD) has been suggested as an invaluable modality for the detection of CV after epilepsy surgery.[3] We report a case of AHE where the patient developed symptoms of CV even in the absence of predictive factors and normal TCD findings.

CASE REPORT

A 15-year-old male with a height of 172 cm and weighing 65 kg was admitted for epilepsy surgery. The pre-anaesthetic evaluation was unremarkable except for uncontrolled seizures on oral phenytoin sodium 100 mg thrice daily, carbamazepine 400 mg twice daily and sodium valproate 500 mg twice daily. The patient was taken up for left AHE under general anaesthesia. The intra-operative period remained stable during which he had 150 ml blood loss. An external drain was placed intra-operatively to prevent any collection of haematoma. At the end of surgery, the patient was extubated and shifted to the Intensive Care Unit (ICU) for observation. He remained stable for next 24 h. A post-operative computerised tomography (CT) scan of the head did not reveal any significant findings. He was shifted out of the ICU. Almost 14 h thereafter, the patient started to develop weakness of the right side of the body (power 2/5) and global aphasia. He was immediately shifted back to the ICU where a TCD revealed normal flow pattern [Figure 1].

A repeat CT scan of the head was unremarkable. Based on a high index of suspicion, digital subtraction angiography (DSA) was performed which revealed vasospasm of the left middle cerebral artery territory [Figure 2a]. Intra-arterial (IA) vasodilatation was performed with 3 mg of nimodipine as a slow infusion over 30 min, along with oxygen

supplementation and noradrenaline infusion to maintain a mean arterial pressure between 90 and 110 mm Hg. The patient showed immediate clinical and angiographic [Figure 2b] improvement following IA vasodilatation with the near-total return of power, speech and recognition after 4 h.

Oral nimodipine (60 mg q4 h) was started post-IA dilatation along with intravenous (IV) fluids. Oxygen therapy and noradrenaline were continued in the ICU for the next 24 h. He was shifted back to the ward on oral nimodipine and IV fluids. As the patient remained neurologically stable, he was discharged after 2 days with advice to continue oral nimodipine for 3 weeks.

DISCUSSION

Cerebral vasospasm after SAH and intracranial surgeries is multi-factorial.[4] Routine use of oral nimodipine is recommended as prophylaxis against CV in case of aneurysmal SAH.[5] However, no such recommendation exists for its use after intracranial surgeries. Intra-operative prophylactic instillation of papaverine and placement of an external drain have been used to decrease the incidence of post-operative CVS after intracranial surgeries.[6,7]

Selective AHE restricts the resection of mesial temporal structures via a pterional craniotomy and trans-sylvian approach. This minimises the possibility of neurocognitive deficits, which otherwise occurs due to resection of anterior and lateral temporal structures in the classical anterior temporal lobectomy for the treatment of epilepsy.[8] Factors which have been correlated with increased incidence of CV after AHE are female gender and presence of significant bleed in the post-operative CT scan.[3] As our patient was a male, and the post-operative CT had not shown increased intracranial bleed, the occurrence of CV in the post-operative period was not anticipated. Lackner *et al*. examined 119 cases of intractable temporal lobe epilepsy undergoing either selective AHE or temporal lobe resection.[3] All patients were evaluated with pre-operative and post-operative neurological examination, TCD and CT scans. Among all patients, 35 developed post-operative CV. They recommended routinely employing TCD after AHE for detection of CV. However, this recommendation is not universally accepted as a standard of care. O'Brien and Cascino opine that 'the beneficial effects on patients' outcome of detecting and treating CV need to be demonstrated in future research before a firm recommendation can be made that patients should be routinely evaluated for CV following surgery for drug-resistant temporal lobe epilepsy'.[8] Currently, our institutional protocol involves mandatory admission and neurological assessment of all patients undergoing intracranial surgeries in the ICU for 24 h period and applying investigative modalities where indicated. In our patient, CV manifested itself 38 h after surgery. His post-operative CT scans were unremarkable. TCD failed to demonstrate any vasospasm. The specificity of TCD in detecting CV is low, especially for distal vessels. Furthermore, the specificity is operator dependent.[9] DSA is currently the gold standard for radiographic documentation of CV.[9] Where CV is demonstrated, simultaneous IA vasodilatation can be performed during DSA. Where DSA is not readily available, utilizing a modality with high specificity and high

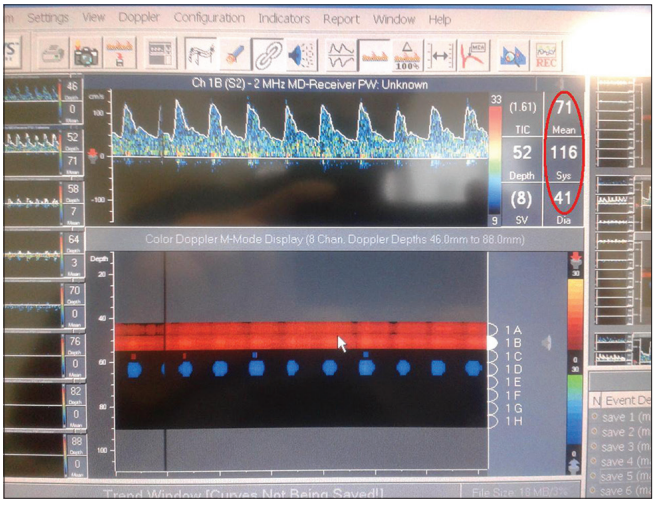

Figure 1: Transcranial Doppler of the left middle cerebral artery showing normal flow pattern (see encircled values)

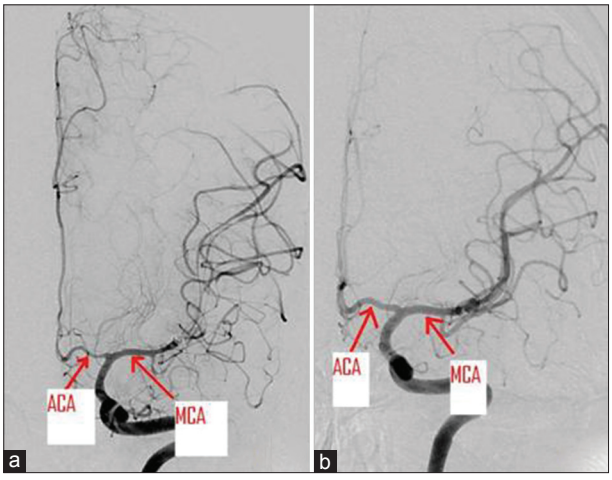

Figure 2: Digital subtraction angiography showing left anterior cerebral artery (ACA) and left middle cerebral artery (MCA) in the predilated state (a) and postdilated state with 3 mg intra-arterial nimodipine (b)

positive predictive value like perfusion CT for detection and management of CV can be beneficial.[9] In their absence, a high index of suspicion for the possibility of occurrence of CV and its prompt and aggressive management can be rewarding.

The role of nimodipine for prophylaxis and management of CV is well-established. There have been many reports of successful management of refractory CV with IV milrinone.[10] As per our institutional protocol, we use oral nimodipine for prophylaxis against CV. Oral nimodipine and IV milrinone either alone or in combination (depending on the clinical response) is used for established CV. If there are no signs of improvement, development of focal neurological deficit or decrease of at least two points on the Glasgow Coma Scale, IA dilatation using nimodipine and milrinone (either alone or in combination, depending upon the clinical and angiographic response) is performed.

CONCLUSION

Currently, no firm recommendation exists regarding routine evaluation for CV following surgeries for drug-resistant temporal lobe epilepsy. High index of suspicion for its occurrence and development of an individual institutional protocol for its recognition and management based on current literature and evidence is suggested.

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