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Case Report

Extensive bilateral anterior and posterior circulation ischemic stroke caused by severe vasospasm (Delayed cerebral ischemia) due to aneurysmal subarachnoid haemorrhage (aSAH) - A case report

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ABSTRACT

Aneurysmal subarachnoid haemorrhage (aSAH) is a disease with high morbidity and mortality even in this modern era. Since treating vasospasm is a challenging task, we emphasize in developing newer aganets for the treatment of vasospasm. In the hyper acute phase, patients with SAH can have catecholamine surge-related arrhythmia, neurogenic pulmonary edema, and irreversible damage to the hypothalamus and brainstem. Delayed cerebral ischemia (DCI) is a serious complication of aneurysmal subarachnoid haemorrhage (aSAH). Though there are many clinical trials to look upon the therapies for DCI and vasospasm in aSAH, none have led to an improvement in outcome of the patient.The prognosis of patients with SAH is grave even with the recent advancements in medical treatments. One of the main cause for this situation, that there are no usable neuroprotective drugs that can be used as soon as SAH is diagnosed. We report a case of ruptured Post Communicating Artery (PCOM) aneurysm with diffuse SAH causing severe vasospasm leading to extensive bilateral anterior and posterior ischemic stroke causing severe morbidity. Since there is no therapeutic breakthrough in treatment of aneurysmal SAH, we emphasize in developing a same.

1. Introduction

Aneurysmal subarachnoid hemorrhage (aSAH) is accounting for about 5% of all strokes. Aneursmal SAH causes multimodel injury to brain and can also lead to other systemic complications such as neurogenic pulmonary edema etc. Angiographic cerebral vasospasm (CVS) occurs in 70% of patients during the first 2 weeks after aSAH, but the incidence of delayed cerebral ischemia (DCI) is only around 30%.[1–5] The mechanisms which cause DCI include neuroinflammation, microthrombosis, cortical spreading depolarizations, disrupted integrity of the blood–brain barrier (BBB), microvascular dysfunction and metabolic derangement.[6–9]

2. Case Report

A 59 old female admitted with two days history of headache, altered sensorium and difficulty in breathing. On examination patient was conscious, disoriented, dyspnoeic and tachypneic. Patient was intubated suspecting pulmonary edema? Neurogenic and connected with ventilator. CT brain was done, showing diffuse SAH with bilateral IVH present. CT chest showing bilateral pulmonary edema. Patient was initially stabilised by the ICU intensivists. Brain CT angiography was done which showed a large wide neck PCoA aneurysm measuring 15*14 mm with neck measuring 6 mm in size, pointing inferiorly, Hypoplastic right vertebral and basilar artery. Patient's clinical condition and prognosis well explained to the patient attainders.

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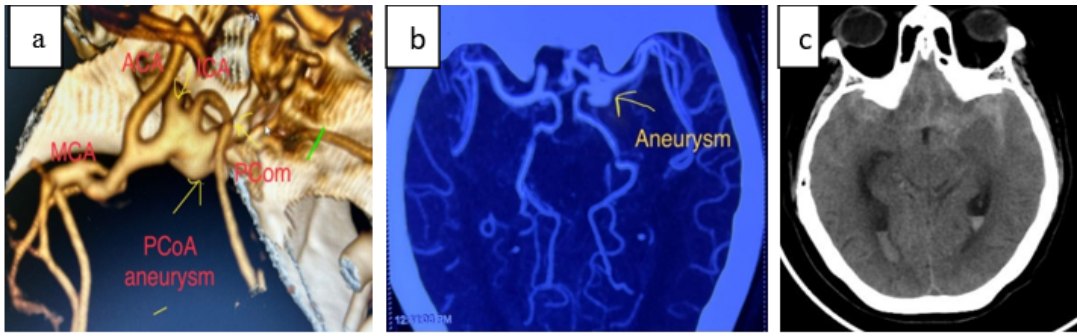

Fig. 1: **a**: CT brain angiography 3D showing large wide neckPCoA aneurysm projecting inferiorly; **b**: CT brain angiography showing PcoA aneurysm with wide neck; **c**: CT brain plain showing diffuse SAH with bilateral IVH

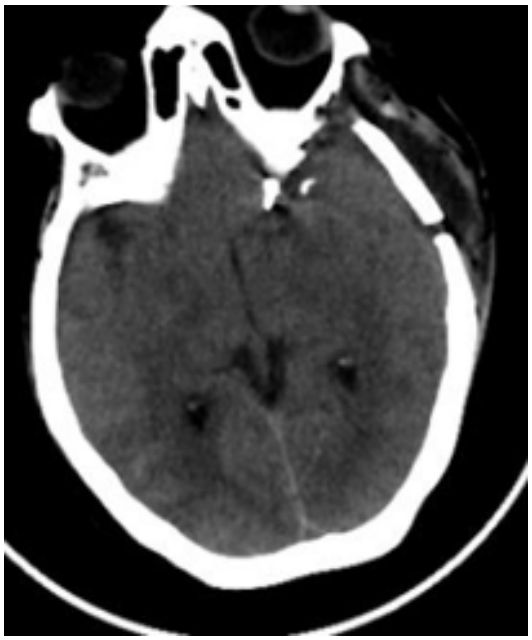

Fig. 2: CT brain post op showing aneurysm clip in situ with resolving SAH

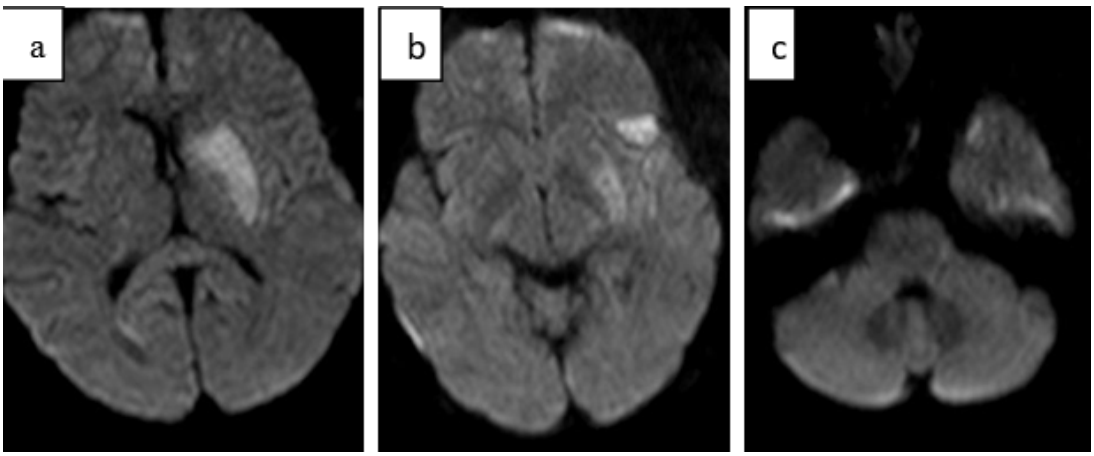

Fig. 3: **a**: MRI brain DWI showing acute infarct in left ganglio capsular region; **b**: MRI brain DWI showing acute infarct in left frontal lobe; **c**: MRI brain DWI showing acute infarct in left temporal lobe.

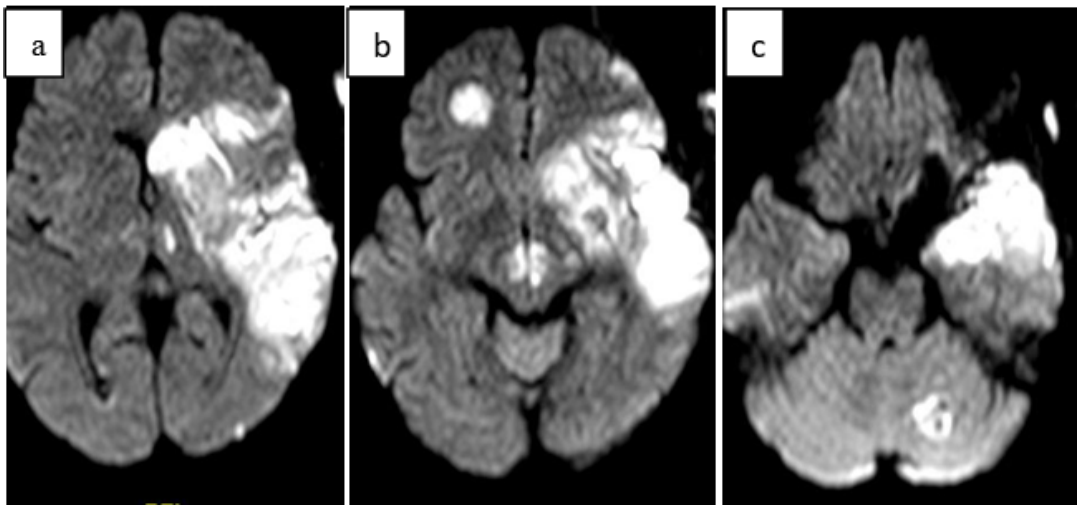

Fig. 4: a: MRI brain DWI showing extensive infarct involving left basal ganglia, fronto temporo parietal region; **b**: MRI brain DWI showing infarct seen in right frontal lobe and bilateral thalamus and hypothalamus, **c**: MRI brain DWI showing infarct seen in left cerebellum and left temporal lobe

Pros and cons of both coiling and clipping treatment was explained and finally agreed to proceed for clipping. Patient underwent left pterional craniotomy and clipping of aneurysm using 11 mm curved clip. Intra op ICG showed almost near total occlusion of the aneurysm with small residual neck. Patient was extubated after the surgery with no deficits and shifted to ICU for further care. On first post operative (POD) patient again developed tachypneic, suspected pulmonary edema, treated and became stable. Patient was continuously monitored for MAP, ICP and neurological status. Transcranial Doppler (TCD) was done daily. Post op CT brain showed aneurysm clip in situ with resolving SAH. On POD 6 patient suddenly became aphasic and developed dense hemiplegia on right side, suspecting vasospasm confirmed with TCD, shifted for MRI brain showed acute infarct in left basal ganglia (Figure 3). Planned for Digital subtraction angiography (DSA) with intra arterial spasmolysis using Nimodipine under IV sedation. DSA showed significant vasospasm in left ICA, Left M1 and M2 segments with <0.5mm patency. Slow infusion of Nimodipine into the catheter was done for about 90 minutes after which there was a minimal relief of vasospasm was noted. On POD 7 patient developed high grade fever with hyperglycaemia treated accordingly. On POD 9 patient was reintubated as patient developed tachpneic and dyspnoeic, DVT screening done showed left calf vein thrombosis with no pulmonary embolism. On POD 10 patient developed hypotension with persistent fever and drop in Glasgow coma scale (GCS). Inotropes was started and MRI brain was taken which showed extensive infarct seen in left basal ganglia, left fronto temporo parietal region, left occipital region, left cerebellum, right frontal, bilateral thalamus and hypothalamus (Figure 4) suggestive of severe vasospasm involving basilar and bilateral ICA due to aneurysmal subarachnoid haemorrhage.

3. Discussion

Delayed cerebral ischemia (DCI) is complication of aSAH,. DCI is classically defined as a development of a new neurological deficit(s), impaired consciousness, or infarct on imaging that commences 3 to 4 days after the initial insult and peaks around 7 to 8 days post-bleed.[10] An earliy onset DCI (prior to day 7) has been associated with higher mortality[11] Large vessel vasospasm was the recognized complication of DCI. Clazosentan, which was used to treat and reverse vasospasm successfully, failed to improve outcomes clinically.[8] DCI has also been reported among patients with none or only mild angiographic evidence of vasospasm.[8,12] Hence, the focus has shifted to early brain injury (EBI) and its association with autoregulatory failure, neuroinflammation, and eventually delayed injury (also defined as DCI), with the thought that the treatment of large vessel vasospasms alone might be reactionary and does not address the inciting mechanisms of injury. In our case we continuously monitored the patient and treated with neuroprotective measures, DCI causes devastating severe vasospasm involving basilar and bilateral ICA due to aneurysmal subarachnoid haemorrhage leads due to severe morbidity to the patient. Early focus on EBI and global cerebral edema are essential in managing aSAH. Our research goals should continue to change and attempt to address these newer pathomechanisms in a search for improved outcomes that can be translated into routine clinical practice.

4. Conclusion

The primary goal of neurointensivists in clinical practice is to ameliorate the burden of morbidity and mortality associated with aSAH. DCI is a complex multifactorial pathophysiological process that starts early post-aSAH, with risk factors present before and at ictus. The pathophysiology of EBI and the mechanisms act as a substrate for the development of DCI. This includes the well-known mechanism of vasoconstriction and spasm, focusing on the sympathetic surge and cytokine release, microthrombosis, and BBB breakdown. Since no therapeutical breakthrough in aSAH has been made to date, and as expected further research is needed, it is vital to develop an idea of its consequences in terms of its outcome and developing potential therapies efficiently targeting brain injury.

5. Source of Funding

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6. Conflict of Interest

There is no conflict of interest.

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