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Case Report

A multidisciplinary approach to the treatment of severe cerebral vasospasm following bacterial meningitis: A case report and literature review

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Abstract

Background: Although cerebrovascular complications of bacterial meningitis are common, postmeningitic cerebral vasospasm significant enough to result in ischemic injury has been reported in only limited fashion.

Case Description: We describe a case of severe cerebral vasospasm following streptococcal meningitis managed successfully with emergency suboccipital decompression, extracranial-intracranial bypass, intra-arterial vasodilator infusion, and maximal medical therapy. To our knowledge, this may be the first case in which surgical cerebral revascularization has been utilized to limit ischemic injury in the setting of postmeningitic cerebral vasospasm.

Conclusions: Patients presenting with abrupt neurological decline following recent treatment for bacterial meningitis may be suffering from a reversible vasoconstriction of the cerebral arteries, and prompt aggressive treatment can result in a favorable outcome even in patients who present in very poor neurological condition.

Key Words: Bypass, ischemia, meningitis, stroke, vasospasm

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INTRODUCTION

Although cerebrovascular complications of bacterial meningitis are common, postmeningitic cerebral vasospasm significant enough to result in ischemic injury has been reported in only limited fashion.[2,5,8,10,12,14,16] In rare instances, endovascular therapy has been used to improve the cerebral blood flow in such cases.[2,3,7,15] We describe an unusual case of severe spasm following streptococcal meningitis resulting in multiple areas of cerebral infarction and necessitating emergency suboccipital decompression. Simultaneous occipital artery – posterior cerebral artery bypass was performed to improve regional cerebral blood flow followed by multiple rounds of cerebral angiography with intra-arterial vasodilator infusion. To our knowledge, this may be the first case in which surgical cerebral revascularization has been utilized to limit ischemic injury in the setting of postmeningitic cerebral vasospasm.

CASE DESCRIPTION

This 46-year-old cattle rancher presented with headaches, fevers, and sinusitis. A lumbar puncture revealed elevated

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white blood cell count, and gram-stain and cultures demonstrated *Streptococcus viridans*. The patient was started on intravenous antibiotic therapy and responded with prompt improvement. He was subsequently switched to an oral antibiotic regimen but returned to the hospital 2 weeks later with visual disturbance, worsening headache, and intermittent dysarthria. He experienced a rapid decline in level of consciousness necessitating endotracheal intubation and was then transferred to our facility for further care.

On arrival, the patient was deeply comatose. A magnetic resonance imaging (MRI) examination of the brain revealed bilateral posterior cerebral artery (PCA) territory ischemic changes with a sizeable right posterior inferior cerebellar artery (PICA) infarction [Figure 1]. There was no evidence of hydrocephalus, and there was limited local mass effect related to the areas of infarction. Broad-spectrum parenteral antibiotics were started, and decadron was administered.

An emergency angiogram demonstrated severe spasm of the posterior circulation with the basilar artery having an almost thread-like appearance and moderate vasospasm of the anterior circulation [Figure 2]. The patient was treated with intra-arterial verapamil infusion resulting in a limited improvement in the caliber of the intracranial vessels. Pentobarbital-induced coma with continuous electroencephalogram monitoring was initiated. Therapeutic hypothermia at a temperature of 35°C was instituted, the cardiac index was improved, and the blood pressure was elevated with pressor medications. Over the ensuing 24 hours, he developed progressive cerebellar edema related to the PICA infarction and then demonstrated pupil dilation responsive to hyperosmolar therapy.

The patient was taken to the operating room where a right frontal ventriculostomy was performed revealing moderately elevated intracranial pressure. The patient then underwent a suboccipital craniectomy and duraplasty for posterior fossa decompression. An intrathecal microcatheter was left in place at the end of this procedure, advanced into the cerebellopontine angle cistern to reach the basilar artery. At this point, the occipital artery (OA) was dissected, and a small occipital supratentorial parasagittal craniotomy was performed through which the OA was anastomosed to a cortical branch of the PCA with 11–0 interrupted suture [Figure 3].

The patient was maintained in barbiturate coma for 5 days with daily angiographic examinations demonstrating persistent severe spasm of the cerebral arteries which were repeatedly treated with intra-arterial verapamil infusion. Intrathecal papaverine infusions through the surgically placed microcatheter were performed on a daily basis as well. Repeated angiographic examinations demonstrated slow improvement in the degree of arterial narrowing and filling of the posterior

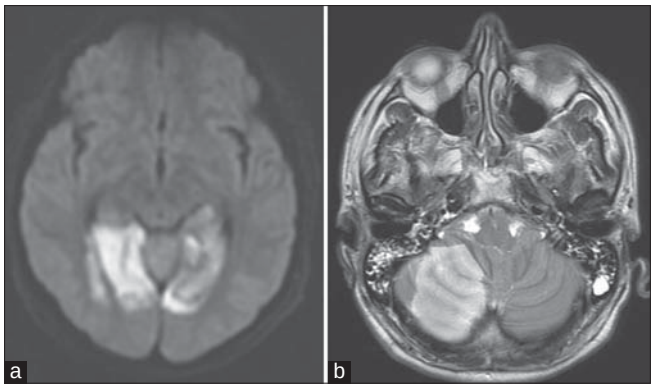

Figure 1: (a) Axial diffusion-weighted magnetic resonance image of the brain reveals bilateral posterior cerebral artery territory ischemic change. (b) T2-weighted imaging demonstrates a sizeable right posterior inferior cerebellar artery infarction with early mass effect

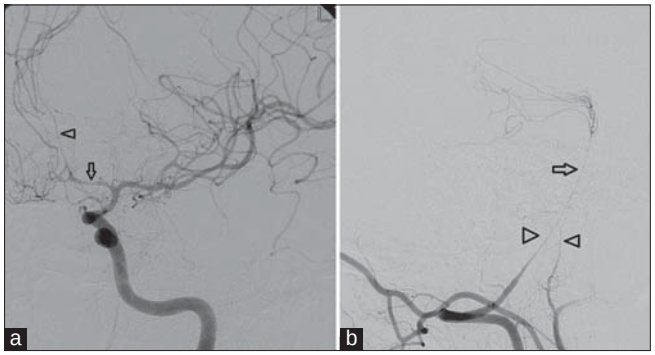

Figure 2: (a) Right internal carotid arteriogram demonstrates moderate spasm of the A1 (arrow) and more severe narrowing of the A2 (arrowhead) segments of the anterior cerebral artery. (b) Vertebral arteriographic image shows severe narrowing of the distal vertebral arteries (arrowheads) with thread-like appearance (arrow) of the basilar artery itself

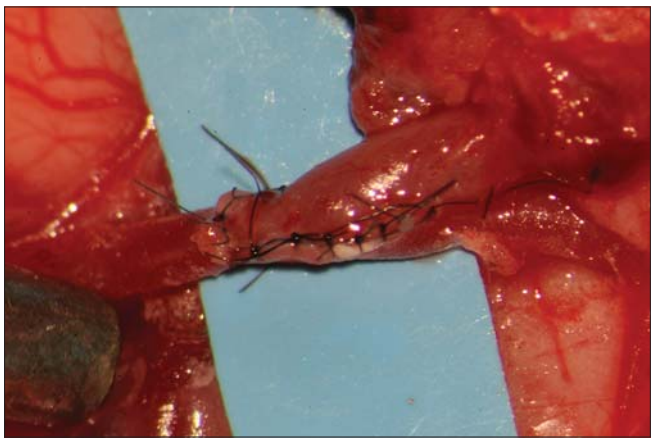

Figure 3: Intraoperative photomicrographic image demonstrating completed anastomosis of the occipital artery to a cortical branch of the posterior cerebral artery

cerebral territory through the bypass [Figure 4]. The patient remained stable and repeated MRI examinations demonstrated no further ischemic injury. Tracheostomy and jejunostomy were performed.

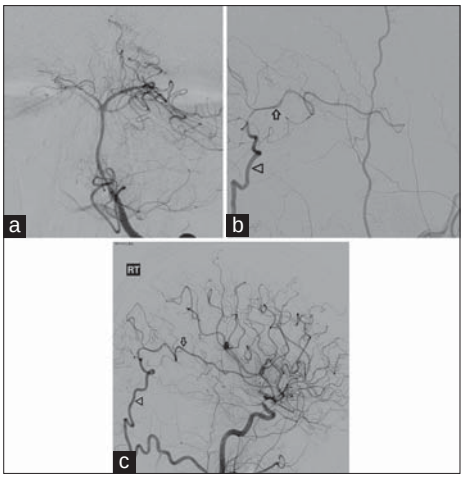

Figure 4: (a) Late angiographic follow-up imaging demonstrates significant improvement in the caliber of the basilar artery. (b and c) Delayed arteriographic images showing the occipital artery (arrowhead) filling the posterior cerebral artery (arrow)

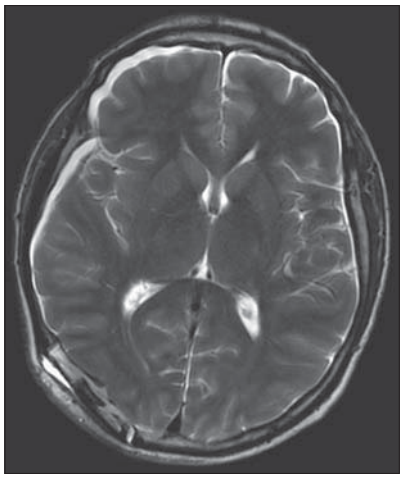

Figure 5: Delayed, magnetic resonance image, obtained 6 months after presentation shows dramatic normalization of the previously visualized ischemic changes corresponding to the patients' ultimately favorable outcome

After 2 weeks, the patient began to regain consciousness; and by the 3rd week, he was clearly following simple commands. The ventriculostomy was discontinued, and the patient was subsequently transferred to an inpatient rehabilitation facility. He was maintained on antibiotic therapy for 3 months, prednisone for 6 weeks, and aspirin indefinitely. Six months later, the patient had made a striking recovery with only limited visual field impairment and was back to work on a near full-time basis [Figure 5].

DISCUSSSION

Cerebrovascular complications have been described in 15–20% of adults with community-acquired bacterial meningitis.[16] In this setting, acute cerebral infarction may involve large arterial territories potentially resulting in brain swelling and a decline in the level of consciousness.[16] In general, such infarction has been felt to result from septic arterial emboli directly occluding the cerebral blood vessels, so called "endarteritis obliterans," although venous thrombophlebitis resulting in venous hypertension and infarction has also been described.[4,9,16,17] The finding of clinically relevant, reversible vasospasm following meningitis is much less common.[3]

Several studies have shown that transcranial Doppler ultrasound evaluation of patients with acute bacterial meningitis reveals elevated velocities suggesting some degree of vascular narrowing in a surprisingly significant percentage of patients studied, but reversible vascoconstriction severe enough to result in ischemic injury is quite unusual.[6,7,10,13,14] Older studies utilizing cerebral angiography to evaluate patients with focal neurological deficits resulting from bacterial meningitis identified irregularity and narrowing of the intracranial vasculature in a high percentage of cases.[12] Not surprisingly, these findings appeared to be associated with a poor prognosis.[12]

Recently, a number of investigators have described the use of endovascular therapies to manage patients with cerebral vasospasm resulting from infectious meningitis.[2,3,7,11] Techniques utilized have included intra-arterial infusion of vasodilator medications as in our patient, as well as percutaneous transluminal angioplasty. Although we and others have used extracranial-intracranial bypass to augment cerebral blood flow in extremely rare cases of severe vasospasm following aneurysmal subarachnoid hemorrhage, we are not aware of a prior case in which surgical revascularization has been used in the setting of postmeningitic vasoconstriction.[1]

In our case, the severe narrowing of the basilar artery and its branches likely resulted in the patients' rapid neurological decline and associated areas of ischemic change on MRI. Once the patient developed edema related to his PICA infarction, it became evident a decompressive procedure was required to prevent a fatal herniation syndrome, and with the patient already in the operating room, a decision was made to attempt to augment intracranial flow in the posterior circulation with a bypass procedure. It is impossible to know for sure whether the patient would have recovered as well without the bypass. We suspect that the maximally aggressive multidisciplinary approach to optimize cerebral blood flow including optimization of cardiac output and the cerebral microenvironment by the neurocritical care team, the use of intra-arterial vasodilator therapy by our interventional neuroradiology colleagues, and the use of bypass combined with intrathecal infusion of papaverine likely all contributed to the ultimately favorable outcome.

Finally, it should be noted that narrowing and irregularity of the cerebral arteries in the setting of infectious

meningitis have been variably described as vasospasm, vasculitis, and vasculopathy.[2,3,8,13] The exact underlying etiology of this narrowing is uncertain and likely represents a combination of direct irritation of the vessels as well as exposure of the vessels to various inflammatory mediators within the cerebrospinal fluid.[13] Because of this, anti-inflammatory and immunosuppressive agents may well represent an important component of therapy, and the angiographic response to treatment may be slower than that encountered in the more common setting of subarachnoid hemorrhage-induced cerebral vasospasm.

CONCLUSIONS

We report an unusual case of particularly severe cerebral vasospasm following bacterial meningitis. This may be the first such case managed in part with surgical cerebral revascularization as one aspect of therapy. Patients presenting with abrupt neurological decline following recent treatment for bacterial meningitis may be suffering from a reversible vasoconstriction of the cerebral arteries, and prompt aggressive treatment can result in a favorable outcome even in patients who present in very poor neurological condition.

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Conflicts of interest

There are no conflicts of interest.

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Commentary

This is an outstanding paper with many lessons for the reader and neurosurgeons. First, the patient presented with widespread vasospasm and infarction. A very creative, skilled, and talented neurosurgeon who was willing to break convention and aggressively treat this patient performed a suboccipital craniotomy to relieve the swelling in the posterior fossa, and at the same time did an occipital to posterior cerebral artery territory bypass to provide blood to the posterior circulation that was compromised. Over time, the bypass increased as the vasospasm relented and provided perfusion to the vascular territory. The patient was placed in barbiturate coma in the interval, a technique that has its own complications. What is also dramatic is the disappearance 6 months later of the "infarcts" as shown by diffusion-weighted imaging (DWI) on magnetic resonance, which few would believe. Hence, the DWI changes were not indicative of infarction but ischemia. This is a seminal paper on a new approach to cerebral vasospasm in multiple modality treatments by an aggressive, creative, innovative neurosurgeon.

Many years ago, I had a case of extreme meningitis that was not responsive to antibiotics. From work I had done in the laboratory using hypothermic cerebral ventricular perfusion to isolate various parts of the brain, I thought that this idea might apply to my patient. Hence, I placed a ventricular catheter and a lumbar catheter and perfused ringer's solution through the ventricles and the cerebrospinal fluid. I do not think that the patient made it. You could not use antibiotics at the time as penicillin was known to

produce seizures. I have often thought that someone ought to do a laboratory experiment in this same manner, with antibiotics to see if these extreme cases of meningitis can be resolved.

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