Intracranial vasculopathy is a well-known complication of bacterial, fungal, and tuberculous meningitis.4,7,10 Despite advances in antimicrobial therapy, the current primary treatment, the overall mortality rate for bacterial meningitis remains at 25%.1,12,15 While cerebral ischemia as a result of meningitis-related vasculopathy is closely related to the high mortality rates,8,13,14 actual angiographic demonstration of meningitis-associated vasculopathy and vasospasm has only rarely been reported.4,7 Intracranial PTA as a treatment modality for infection-related vasculopathy and vasospasm has only been previously described in a single case of coccidioidal meningitis.9 There have been no prior descriptions of intracranial PTA in the setting of bacterial meningitis.

We recently used angiographic demonstration and successful intracranial PTA for the treatment of staphylococcal meningitis with associated severe vasospasm and saw improvement both anatomically and in cerebral ischemic symptoms.

**Case Report**

**History and Presentation.** This 20-year-old man presented with a 5-day history of severe frontal headache. The patient’s medical history was remarkable only for recent sphenoid sinusitis treated with oral antibiotics. He did not drink alcohol, smoke cigarettes, or use illicit drugs. He was afebrile with normal vital signs. Physical examination revealed a stiff neck, but the remainder of the examination (including neurological systems) was unremarkable.

Testing for the HIV antibody was negative, and a urine drug screen was negative for cocaine and amphetamines. Computed tomography scanning showed bilateral sphenoid sinusitis but no brain abnormalities. A lumbar puncture provided cloudy CSF with a white blood cell count of 68/mm³ (98% neutrophils), a red blood cell count of 3/mm³, protein of 41 mg/dl, and glucose of 63 mg/dl. Gram staining of the CSF showed many white blood cells and gram-positive cocci in clusters. Culture of the CSF grew β-lactamase-positive *Staphylococcus aureus*. Intravenous nafcillin was begun. Endoscopic sinus surgery performed on the 3rd hospital day revealed purulent sinus material, which also grew the same organism from culture. Transesophageal echocardiography findings were unremarkable. Because of continued headache on the 5th hospital day, MRI with and without intravenous contrast was performed and showed normal-appearing brain and ventricles. On the 11th hospital day, right hemiparesis was noted, and findings from repeat MRI with contrast were consistent with meningitis, demonstrating diffuse basilar leptomeningeal enhancement extending along the outer surface of both the carotid terminus and proximal MCAs in the sylvian fissure (Fig. 1), more severe on the left. In addition, diffusion-restrictive change...
representing acute infarction was present in the left basal ganglia.

**Treatment.** The following day, the patient continued to worsen neurologically and had a decreased level of consciousness and aphasia. Emergency angiography demonstrated severe stenosis to a diameter of 1–1.5 mm (reference range 3–4 mm) and vasospasm in the paraciloid and supraclaudioin segments of the left ICA and the M1 and M2 segments of the left MCA (Fig. 2). Milder vasospasm of the right MCA and both anterior cerebral arteries was also noted. Infusion of nitroglycerin (200 μg) performed selectively into each ICA failed to result in significant angiographic improvement (Fig. 3A).

With the left-sided vasospasm appearing more severe and being accompanied by worsening right-sided weakness, it was decided to proceed with left ICA and MCA PTA. Initially 3000 U of heparin was administered intravenously. While utilizing local anesthesia and low-dose “conscious sedation,” a 6-Fr standard guide catheter (Envoy, Codman Neurovascular/DePuy Orthopedics, Inc.) was placed selectively into the midcervical ICA. A 4 × 7–mm Hyperform microballoon catheter (ev3, Inc.) was navigated into the distal left ICA and MCA using a magnified digital fluoroscopic “road-mapping” technique. Careful low-pressure dilations to a diameter of 2–2.5 mm were performed in the M1 and proximal M2 segments as well as carotid terminus and supraclaudioin paraciloid ICA segments. Only a single pass with a single balloon dilation was performed for each arterial segment. Because of the inflammatory meningitis and possible associated vasculitis, minimal balloon diameters were used as opposed to what might be performed in the setting of vasospasm induced by subarachnoid hemorrhage.

**Posttreatment Course.** Follow-up angiography demonstrated improvement in vessel caliber and marked improvement in distal cortical flow (Fig. 3B and C). Postprocedure care consisted of standard hypervolemic, hypertensive, and hemodilution therapy. The patient’s neurological symptoms were noted to begin improving within 2–3 hours after the procedure. He was transferred...
to a rehabilitation facility after 1 month of hospitalization and 28 days of intravenous antistaphylococcal therapy. At the time of transfer he was able to ambulate with minimal assistance. Speech, memory, and intellectual capacity were assessed as normal, and there was only minimal residual weakness in his right arm.

**Discussion**

While less than 5% of community-acquired meningitis is due to *S. aureus*, the mortality rate of this infection is on the order of 50%. Accompanying adverse effects on the brain may include direct parenchymal edema, hydrocephalus, and cerebral vasculopathy. Vasculopathy associated with meningitis produces adverse neurological sequelae mainly due to ischemia, and infarcts occur most commonly in the basal ganglia and thalami. Higher mortality rates are seen with greater degrees of vasculopathy. Vasculopathy and its sequelae of intracranial arterial narrowing are likely a combination of inflammatory exudates adjacent to the vessels, thickened meninges, vasculitis, and, very importantly, vasospasm. Vasospasm is a significant factor in producing severe vascular stenosis in basilar large and medium arteries. Transcranial Doppler ultrasonography studies have clearly demonstrated vasospasm in association with meningitis as evidenced by elevated flow velocities. They also demonstrated and reaffirmed that higher cerebral flow velocities were directly related to a poorer prognosis and that fatal outcomes have a high association with vascular involvement.

Intracranial PTA is more commonly performed in the setting of intracranial atherosclerotic disease and severe stenosis associated with subarachnoid hemorrhage–induced vasospasm, where it has been demonstrated to be a viable and effective treatment option. Intracranial PTA as a therapy for vasospasm due to infectious meningitis has been reported only in a single recent case of coccidioidal meningitis. This is the first reported case of intracranial PTA as a therapy for bacterial meningitis–induced vasospasm. A unique and important consideration in performing PTA in this specific situation is that there may indeed be an associated “vasculitis” at the site of vasospasm and stenosis, which would potentially complicate the problem by diminishing vessel wall integrity, rendering it weaker and possibly more prone to PTA injury or even rupture. After weighing this concern and risks with the poor prognosis associated with the severity of the patient’s worsening clinical condition, it was decided to carefully proceed with the PTA. Only single low-pressure and small diameter inflations were performed at each ICA and MCA target site with resultant angiographic improvement in vessel diameter and flow.

Interestingly, in the previous report of coccidioidal meningitis by Hu et al., it was emphasized by the authors that the vasospasm they encountered was somewhat “refractory.” This was discovered after appropriate initial lower pressure PTA attempts were unsatisfactory, necessitating longer, repeated balloon dilations. This could suggest either a difference in vasospasm induced by different organisms or separate “phases” of infectious meningitis–related vasculopathy and vasospasm. This may indeed be a manifestation of an earlier vasospastic period followed by a more “fibrotic” type response. A previous report with radiological and pathological correlation describes in detail an earlier vasculopathy phase consisting primarily of vasospasm induced by local surrounding purulent material and a later phase with arterial stenosis as a result of organization of subendothelial edema, intimal thickening and smooth-muscle proliferation. If this later “phase” starts to occur, then this would potentially be perhaps more “refractory” to PTA and might require longer, repeated dilations.

**Conclusions**

Mortality rates for infectious meningitis are significant and are closely associated with the presence of vas-
culopathy. Vasospasm is an important factor in meningitis-induced cerebral vasculopathy and can result in devastating cerebral ischemic injury. In patients with meningitis, an aggressive management approach that closely monitors for vasospasm, such as with serial surveillance using transcranial Doppler studies, and that is prepared to proceed with treatment modalities such as hypervolemia, hypertension, and hemodilution therapy will be required to hopefully improve outcomes. If cerebral ischemic symptoms occur, cerebral angiography should not be delayed. In the appropriate clinical setting, intracranial PTA is an effective treatment option. In performing PTA it will be important to consider the pathological state of the target vessel due to associated vasculitis. Initial, low-diameter dilations would be suggested only, followed by repeated, prolonged inflations if necessary.

Disclosure

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

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