Immediate Spastic Hemiplegia following Coil Embolization of an Anterior Communicating Artery Aneurysm

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Abstract

Background: Acute ischemia typically results in a flaccid paralysis. An immediate spastic hemiparesis following ischemia has rarely been reported. One case involved a left hemispheric transient ischemic attack and the other cases occurred after intracerebral hemorrhage.

Case Report: A 46-year-old male developed an immediate spastic hemiplegia shortly after coil embolization of an unruptured anterior communicating artery aneurysm. A CT perfusion scan at the time of these symptoms demonstrated hypoperfusion in the right anterior cerebral artery (ACA) territory. The patient was emergently taken back to the angiography suite where he was found to have right A2 ACA occlusion. Following abciximab infusion, the artery recanalized and the patient’s symptoms resolved. Repeat CT perfusion demonstrated resolution of the perfusion deficit in the right ACA territory and the patient’s neurological exam remained normal.

Conclusion: Acute ACA ischemia can present with spasticity in the hyperacute period instead of flaccid paralysis.

Introduction

Acute ischemia causes paresis or plegia with decreased tone and decreased deep tendon reflexes. The upper motor neuron signs of spasticity and increased deep tendon reflexes do not typically develop until a few days after stroke onset [1]. Immediate spasticity at the onset of an ischemic stroke or transient ischemic attack (TIA) is extremely rare. When immediate spasticity occurs, it is typically associated with
intracerebral hematoma and not ischemia [2]. However, there has been one prior report documenting acute spastic paresis after a TIA [3].

The current management recommendation of large unruptured, intracranial, saccular aneurysms is surgical clipping or coil embolization. Coil embolization has been shown to be a durable treatment with low morbidity [4–6]. Developments such as balloon-assisted coiling and stent-assisted coiling have further enhanced the capabilities of neuro-interventionalists to coil previously untreatable, wide-necked, complex aneurysms with reduced morbidity [7, 8]. A possible complication associated with coil embolization is ischemic stroke secondary to parent vessel occlusion, branch or perforator occlusion, or a dislodged coil.

We present a rare case of immediate spastic hemiplegia secondary to anterior cerebral artery (ACA) occlusion and ischemia after coil embolization of a complex anterior communicating artery (AComm) aneurysm. Hemiplegia may occur with ACA occlusion as a result of ischemia in the posterior limb of the internal capsule (supplied by the recurrent artery of Heubner) or medial precentral gyrus (supplied by terminal ACA branches). The hyperacute spastic hemiplegia in this patient was successfully reversed after selective intra-arterial (IA) thrombolysis.

Case Report

A 46-year-old male presented to the emergency department with severe left frontal and retro-orbital headache. On presentation his neurological exam was normal. A computed tomography (CT) scan of the brain showed no subarachnoid or intracerebral hemorrhage (ICH) but demonstrated a rim-calcified mass in the suprasellar cistern (fig. 1a). CSF analysis from a lumbar puncture ruled out a radiographically occult hemorrhage. A subsequent CT angiogram demonstrated an 11-mm, anteriorly directed, rim-calcified, saccular AComm aneurysm. Though the aneurysm was not ruptured, treatment of the aneurysm was recommended to the patient given the size of the aneurysm [4].

Consequently, the patient underwent a diagnostic cerebral angiogram, which demonstrated a multilobulated AComm aneurysm that measured 10.3 × 11.6 mm with a wide neck that incorporated the A2 segments of both ACAs (fig. 1b). The A1 segment of the left ACA was noted to be hypoplastic. Given the degree of dense calcification in the aneurysm neck and dome wall, which significantly hinders surgical aneurysm clipping [9], it was decided to proceed with balloon-assisted coil embolization of the aneurysm. The patient was placed under general anesthesia and systemic heparin was administered. Two 4 × 10 mm Ascent balloons (Micrus Endovascular, San Jose, Calif., USA) were advanced through guide catheters into the right internal carotid artery (ICA). One balloon was advanced through the right A1 ACA segment to the right A2 segment so it was centered over the neck of the aneurysm. The second balloon was advanced into the right A1 ACA segment with its guide wire in the left A2 segment and therefore positioned so it could be readily advanced as needed. A microcatheter with a J-shaped tip was advanced into the aneurysm neck. With the balloon in the right A2 segment inflated, multiple coils were advanced into the aneurysm dome and neck. With the balloon in the right A2 segment inflated, multiple coils were advanced into the aneurysm dome and neck. Prior to detachment, the balloon was deflated with no prolapse of coil loops noted. After deployment of the last coil, a 3D rotational angiogram was performed that demonstrated anterograde flow through the right ACA and bilateral A2 segments with a minimal degree of vasospasm in the right A2 segment, likely secondary to balloon and microwire manipulation. IA verapamil was infused, which significantly improved the focal vasospasm (fig. 1c, d). At the conclusion of the procedure the patient received 300 mg of aspirin per rectum and was started on a heparin drip to reduce the risk of thrombus formation at the interface between the aneurysm and parent vessel.

The patient was successfully extubated after the procedure but on arrival to the neurointensive care unit (NICU), was noted to have a spastic hemiplegia of the left arm and leg. He had increased deep tendon reflexes of the left biceps and patella. He was awake, alert, and briskly following commands at the time. An emergent non-contrast head CT demonstrated no hemorrhage or infarct. A CT perfusion
scan (CTP), however, demonstrated increased mean transit time (fig. 2a), correspondingly decreased cerebral blood flow (fig. 2b), but relatively preserved cerebral blood volume in the right ACA territory, which was consistent with ischemia but not infarction. Based on the exam and CT findings, the patient was taken emergently back to the interventional neuroradiology suite for an angiogram and IA thrombolysis. The angiogram demonstrated a right A2 ACA segment occlusion (fig. 2c) with good collateral filling from the right middle cerebral artery (fig. 2d). The right A1 segment was selectively catheterized and a total of 20 mg of abciximab was infused, which recanalized the right A2 segment (fig. 2e, f). The procedure was performed with moderate conscious sedation to follow the patient’s neurological exam. There was residual stenosis of the right A2 segment after the abciximab infusion, but the patient’s neurological deficits had resolved. After transfer back to the NICU, the patient was found to have 5/5 strength in the left arm and leg with markedly decreased spasticity and normal deep tendon reflexes. The patient was continued on aspirin and a heparin drip, and his blood pressure was kept elevated to facilitate collateral filling. Over the next 3 days, the blood pressure parameters were weaned and the patient was taken off the heparin drip.

A follow-up CTP study 5 days later demonstrated resolution of the large ACA territory hypoperfusion (fig. 3a, b) and only a few small foci of hypodensity in the inferior right frontal lobe. The patient was discharged on hospital day 7 with no neurologic deficits. At 5 months’ follow-up, the patient continued to do well neurologically. A follow-up MRA demonstrated patency of the bilateral ACAs. However, a small recurrence of the embolized aneurysm neck was visualized (fig. 3c). The patient will be closely followed and re-embolization of the aneurysm will be considered if progression of the aneurysm recurrence is detected.

**Discussion**

Immediate spasticity in the setting of TIA or ischemic stroke is exceedingly rare. It is most commonly seen with basal ganglia hemorrhages. In a report by Steiner et al. [2] of 150 patients that were hospitalized consecutively for stroke, 8 patients presented with acute spastic hemiparesis or hemiplegia, all of which had acute ICH. Of the 8 patients, 7 presented with basal ganglia ICH and one presented with ICH of the temporal lobe. Besides noting the association with basal ganglia ICH, the authors were not able to postulate a neurophysiologic basis for the acute spastic hemiparesis/-plegia. There is only one published report of acute spasticity associated with a clinical TIA syndrome. Ciucci et al. [3] reported the case of a 64-year-old man who presented with acute weakness, spasticity, and aphasia. CT scans were normal at admission and on follow-up. Electroencephalogram demonstrated moderate slow-wave activity over the left hemisphere that resolved on follow-up. And lastly, angiography demonstrated atheromatous plaque in both ICAs near the bifurcation. The patient’s symptoms resolved after 20 h with no evidence of stroke on repeat CT 20 days later, making this an acute spastic left hemispheric TIA.

This is the first report of spastic hemiplegia as a result of ACA territory hypoperfusion that is corroborated by CTP imaging and cerebral angiography. This case is unique in that we not only demonstrate hypoperfusion and vessel occlusion with multiple imaging modalities but also immediate resolution of the spastic hemiparesis after reperfusion. The fact that the CTP showed hypoperfusion and not hyperperfusion, as is seen during a seizure, argued against a focal seizure as a cause of the stiffness. Additionally, follow-up imaging showed a small ACA territory stroke, demonstrating that this was an ischemic event. Decerebrate posturing was also an unlikely explanation given the lack of mass effect on the head CT and the patient’s clinical exam. Also, the patient did not receive any medications associated with dystonia or spasticity that may have argued against an ischemic cause of his presentation.
The mechanism for ischemia in this case was thrombus formation after aneurysm coil deployment with a contribution from focal vasospasm. It is possible that this different mechanism of ischemia, as opposed to ischemic stroke secondary to embolus or atherothrombosis, may have contributed to the unusual presentation. The combination of thrombus formation and vasospasm could have caused atypical upper motor neuron injury that resulted in immediate spasticity and exaggerated reflexes. Injury to the motor system does not produce a homogenous clinical syndrome and there is a wide range of signs and symptoms that accompany upper motor neuron injury. Not all patients develop spasticity after an upper motor neuron injury and, in those that do develop spasticity, there is a wide time course of when spasticity develops. In most patients it occurs after a few days to a week but some patients may be predisposed to develop spasticity sooner. This patient may have been more likely to develop spasticity sooner after upper motor neuron injury and, combined with his mechanism of stroke, the spasticity occurred even earlier. This case demonstrates the possibility of immediate spastic plegia as a clinical manifestation of acute ACA territory ischemia. All practitioners should be cognizant of this presentation, especially after a neuro-interventional procedure such as aneurysm coil embolization.

**Fig. 1.** CT scan of the brain shows a rim-calcified mass in the suprasellar cistern (a). Digital subtraction angiogram demonstrates a multi-lobulated AComm aneurysm with a wide neck that incorporated the A2 segments of both ACAs (b). Digital subtraction angiogram after IA verapamil infusion (c, d). AP = Anterior-posterior view, LAT = lateral view.
Fig. 2. CTP demonstrates increased mean transit time (MTT; a) and decreased cerebral blood flow (CBF; b) in the right ACA territory. Digital subtraction angiogram demonstrates right A2 segment occlusion (c) with good collateral filling from the right middle cerebral artery (d) and recanalization of the right A2 after abciximab infusion (e, f). AP = Anterior-posterior view, LAT = lateral view.
Fig. 3. CTP study 5 days later demonstrated resolution of the large ACA territory perfusion deficit (a, b). Five-month follow-up MRA depicts a small recurrence of the embolized aneurysm neck (c).

References