Multiple Reversible Episodes of Subcortical Ischemia Following Postcoital Middle Cerebral Artery Dissection

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**Background:** Subcortical transient ischemic attacks are usually seen in individuals with small-vessel disease. They are often refractory to medical treatment and progress to infarction in many cases.

**Patient:** We describe a young man with an unusual and protracted case of recurrent capsular ischemia due to postcoital middle cerebral artery dissection.

**Conclusion:** Spontaneous middle cerebral artery dissection should be considered in young patients presenting with subcortical transient ischemic attacks.

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**SUBCORTICAL TRANSIENT ISCHEMIC ATTACKS**

Transient ischemic attacks are marked by episodes of hemiparesis without cortical dysfunction. A subset of these transient ischemic attacks presents as multiple stereotyped attacks, usually in individuals with lacunar pathological foci, and is referred to as *capsular warning syndrome*. It can be an evasive and challenging diagnosis, with spectacular “on-off” episodes of subcortical ischemia that can leave the treating physician incredulous. High-grade in situ penetrator vessel stenosis or large-vessel atheroma at lenticulostriate or thalamoperforate sites is the presumed mechanism in older individuals. We present an unusual case of protracted and recurrent pure motor hemiparesis attacks consistent with capsular warning syndrome in a young man with postcoital middle cerebral artery (MCA) dissection.

**REPORT OF A CASE**

A 33-year-old right-handed man without a medical history was admitted to another institution with right hemiparesis and motor aphasia following sexual intercourse. He recovered completely within hours. Another transient episode of right hemiparesis and dysarthria occurred the next day, which prompted treatment with aspirin, clopidogrel, and heparin for a possible crescendo transient ischemic attack. Magnetic resonance imaging (MRI) of the brain revealed restricted diffusion in the left insula and left frontotemporal opercula (Figure A). A cerebral angiogram on hospital day 3 showed normal intracranial and extracranial vasculature. The patient was transferred to our institution, where the results of a further diagnostic workup for stroke in the young were unremarkable; the workup included echocardiography, urine toxicology screening, inflammatory and hypercoagulable tests, and cerebrospinal fluid analysis.

Over the ensuing 3 days, multiple recurrent episodes of stereotyped right hemiparesis and severe dysarthria occurred 3 to 4 times daily, lasting 1 to 20 minutes, each with complete resolution. A typical episode began with mild dysesthesia in the right hand or right-sided personal area, followed rapidly, within seconds to minutes, by progressive right hemiparesis and marked dysarthria, there were no cortical symptoms or signs; no headache before, during, or after the episode and no alteration in consciousness. The patient would describe the course of symptoms as "descending in and out of a trough." His blood pressure remained normotensive throughout. Continuous electroencephalography during several of these episodes showed only mild left hemispheric attenuation, but no epileptiform activity. Repeat brain MRI on day 4 showed no new infarctions or hemorrhages. Magnetic resonance perfusion showed no significant de-
The patient remained asymptomatic for the next 8 days, until day 17, when he had 3 recurrent transient episodes of right hemiparesis and dysarthria, lasting less than 10 minutes each. On day 14, he had 6 more stereotyped episodes, one lasting 45 minutes, all of which resolved completely. Repeat brain MRI on days 15 and 16 showed no new infarcts; magnetic resonance angiography showed a subtle area of irregularity in the left MCA stem (Figure 1C). Five more episodes occurred on day 17, and a continuous electroencephalogram again showed no seizure activity.

The last ictus occurred on day 18 and began with right arm weakness that fluctuated for a few minutes before progressing with bilateral right hemiparesis and marked dysarthria. Urgent brain MRI showed new areas of infarction involving the left posterior limb of the internal capsule and the parieto-occipital convexity (Figure 1D); magnetic resonance angiography again showed irregularity of the right MCA stem. Repeat cerebral angiography demonstrated an area of irregularity and moderate stenosis in the MCA stem, with a "double-barrel" lesion suggestive of dissection (Figure 2). Aspirin and clopidogrel were administered. Follow-up MRI on day 20 showed no new infarctions. Over the following month, the patient made a moderate recovery from his previous condition.

lay in time to peak in the left MCA territory. On day 3, after being symptom free for 24 hours, the patient was discharged home with the diagnosis of cryptogenic stroke; he was advised to take aspirin daily.
This case has several important and unique features. First, MCA dissection is rare and, to our knowledge, has not been previously reported in the setting of coitus. Second, it offers insights into the mechanism of ischemia due to intracranial dissection. Third, MCA dissection in this case led to a form of capsular warping syndrome with a markedly protracted time course. Fourth, imaging of the intracranial vasculature was surprisingly benign despite the dramatic clinical syndrome.

Ischemic stroke during sexual intercourse is rare. Possible mechanisms include patent foramen ovale, 1 migraines, 2 and vertebral artery dissection. 3 To our knowledge, this is the first reported case of intracranial dissection as a cause of postcoital stroke. The exact pathophysiological features whereby coitus might cause arterial dissection are not known but might include formal injury due to acute blood pressure surges and/or trivial rotational or mechanical vessel trauma during intercourse.

As is known for cerebral dissections, 4 an intracranial vessel can dissect, remain patent on angiogram and still serve as a nidus for thromboembolism. The topography of the initial injuries was consistent with emboli to M2 branches of the MCA. Uniquely, an intracranial dissection has the added potential to occlude local perfators directly. The presumed mechanism of the recurrent capsular ischemia in this case was obstruction of lenticulostriate arteries due to MCA stem dissection. We postulate that dissection of the MCA stem created a dynamic intramural “flip” or thrombus over the ostia of the lenticulostriate, capable of producing recurrent episodes of subcortical ischemia.

The time course of recurrent ischemic events in this case is also remarkable. Moreover, the protracted course with 30 episodes of transient capsular ischemia occurring over 2 weeks before the final infarction attests to a highly unsalable perforator vessel. To our knowledge, this case is the current record in duration (18 days) and frequency (30 episodes) of fully reversible attacks of subcortical ischemia. Although prior cases document stroke due to capsular infarction due to isolated MCA dissection, preceding subcortical ischemic episodes have not been described.15

The diagnosis of intracranial dissection can be difficult and requires a high degree of clinical suspicion because vascular imaging results may be normal. In our case, the final diagnosis was only made and confirmed on repeat angiography after the patient’s condition progressed to capsular infarction despite maximal medical therapy. Treatment in traditional cases of capsular warping syndrome is likewise disappointing, with 40% of patients progressing to infarction despite antplatelet, anticoagulant, thrombolytic, and hemodiagnosis therapies. 1 A recent report, 7 however, lends some hope by suggesting that clopidogrel loading plus aspirin might prevent subsequent capsular infarction.

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