

# 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.

*Arch Neurol.* 2006;63:891-893

**S**UBCORTICAL 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 features, and is referred to as *capsular warning syndrome*.<sup>1</sup> It can be an elusive 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 ostia is the presumed mechanism in older individuals.<sup>2</sup> We present an unusual case of protracted and recurrent pure motor hemiparetic attacks consistent with capsular warning syndrome in a young man with postcoital middle cerebral artery (MCA) dissection.

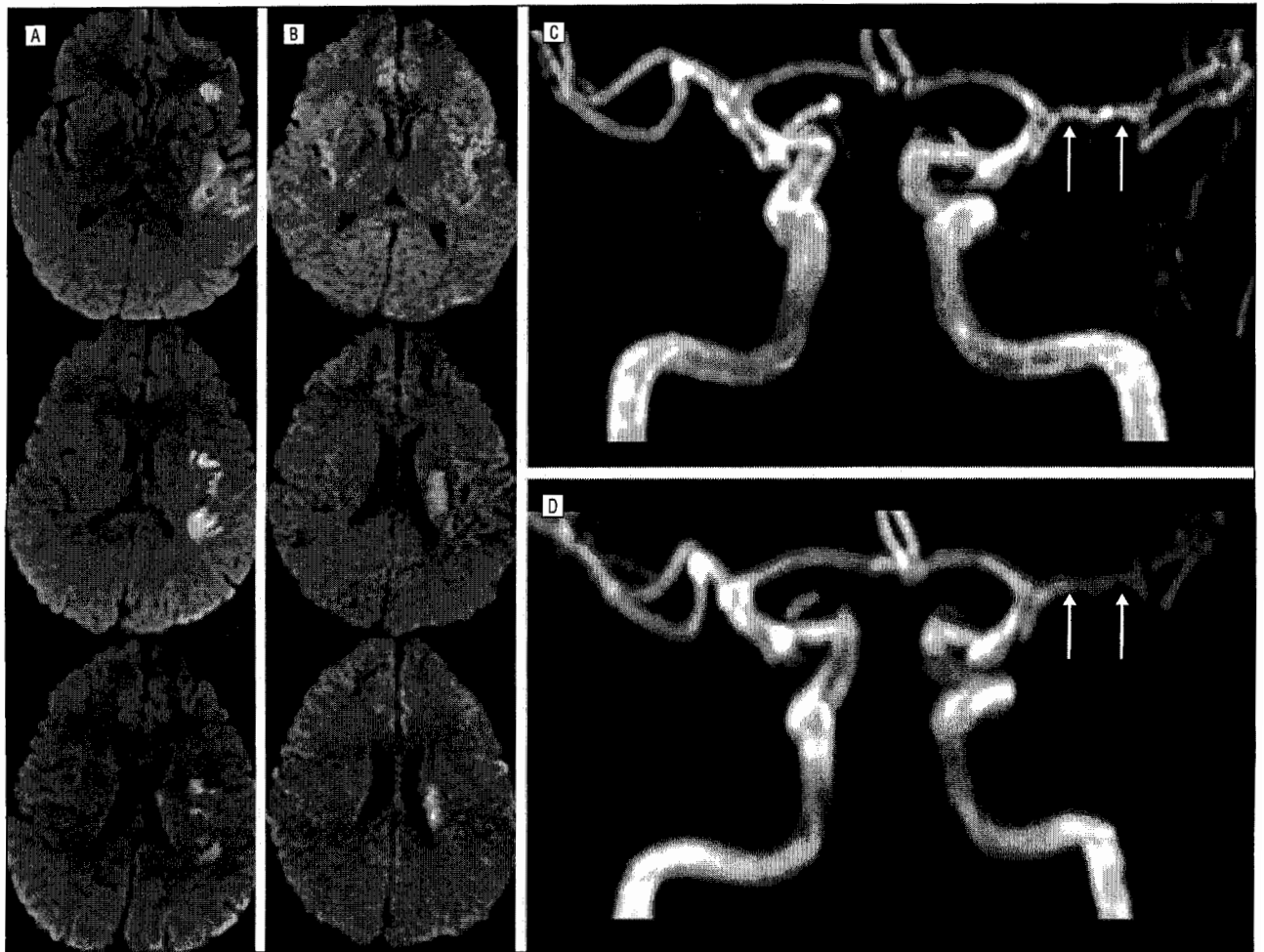
## REPORT OF A CASE

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 at-

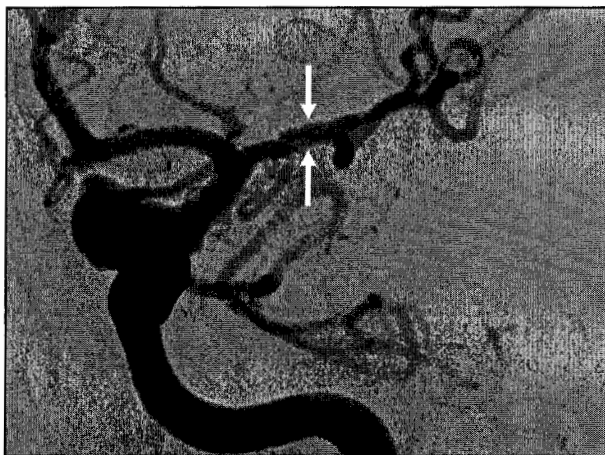
tack. Magnetic resonance imaging (MRI) of the brain revealed restricted diffusion in the left insula and left frontotemporal opercula (**Figure 1A**). A cerebral angiogram on hospital day 3 showed normal intracranial and extracranial vasculature. The patient was transferred to our institution, where 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 perioral 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-

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**Figure 1.** Diffusion-weighted imaging (DWI) on day 4 demonstrates acute infarcts in the left insula and frontotemporal opercula (A), DWI on day 18 shows new infarcts in the left corona radiata and internal capsule (B), magnetic resonance angiography (MRA) of the head on day 15 shows left M1 irregularity (arrows) (C), and MRA of the head at 1 month shows stable M1 irregularity (arrows) (D).



**Figure 2.** A cerebral angiogram of the left middle cerebral artery demonstrates a "double-barrel"-type lesion in the M1 segment, with moderate stenosis consistent with dissection (arrows).

lay in time to peak in the left MCA territory. On day 5, 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.

The patient remained asymptomatic for the next 8 days, until day 13, 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 within 2 hours to right hemiplegia and marked dysarthria. Urgent brain MRI showed new areas of infarction involving the left posterior limb of the internal capsule and the paraventricular corona radiata (Figure 1B); 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 pure mo-

tor hemiplegia, with persistent severe paresis in the hand only. He has had no recurrent symptoms or worsening. Brain MRI 1 month after the first stroke revealed no new regions of restricted diffusion and no change in the caliber of the MCA (Figure 1D).

#### COMMENT

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 warning 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,<sup>3</sup> migraine,<sup>4</sup> and vertebral artery dissection.<sup>5</sup> 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 intimal injury due to acute blood pressure surges and/or trivial rotational or mechanical vessel trauma during intercourse.

As is known for cervical dissections,<sup>6</sup> an intracranial vessel can dissect, remain patent on angiogram, and still serve as a nidus for thromboembolism. The topography of the initial infarcts was consistent with emboli to M2 branches of the MCA. Uniquely, an intracranial dissection has the added potential to occlude local perforators directly. The presumed mechanism of the recurrent capsular ischemia in this case was obstruction of lenticulostriate ostia due to MCA stem dissection. We postulate that dissection of the MCA stem created a dynamic intimal "flap" or thrombus over the ostia of the lenticulostriate, capable of producing recurrent episodes of subcortical ischemia.

The time course of recurrent ischemia in this case is also remarkable. Moreover, the protracted course with 30 episodes of transient capsular ischemia occurring across 2 weeks before the final infarction attests to a highly unstable 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 striatocapsular infarction due to isolated MCA dissection, preceding subcortical ischemic episodes have not been described.<sup>7,8</sup>

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 warning syndrome is likewise disappointing, with 40% of patients progressing to infarction despite antiplatelet, anticoagulant, thrombolytic, and hemodilution therapies.<sup>1</sup> A recent report,<sup>9</sup> however, lends some hope by suggesting that clopidogrel loading plus aspirin might prevent subsequent capsular infarction.

**Accepted for Publication:** January 27, 2006.

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**Author Contributions:** *Study concept and design:* Prabhakaran and Krakauer. *Acquisition of data:* Prabhakaran and Krakauer. *Analysis and interpretation of data:* Prabhakaran and Krakauer. *Drafting of the manuscript:* Prabhakaran and Krakauer. *Critical revision of the manuscript for important intellectual content:* Prabhakaran and Krakauer. *Study supervision:* Prabhakaran and Krakauer.

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