A 49-year-old man presented with a 1-day history of multiple transient episodes of right-sided weakness. The episodes were stereotypical, precipitated by exertion, and lasted 5 to 10 minutes. He denied any chest pain, palpitation, headache, or injury. He was a chronic smoker (40 cigarettes per day for 20 years) and denied any past or family history of hypertension, diabetes mellitus, dyslipidemia, stroke, or ischemic heart disease.

On arrival, he was fully conscious and oriented and had regular pulse (65 bpm), and blood pressure (145/90 mm Hg). Some word-finding difficulties and mild right-sided weakness (power, Medical Research Council grade 4) were noted (National Institute of Health Stroke Scale score, 4 points). Magnetic resonance imaging of the brain (Figure 1A) revealed multiple areas of restricted diffusion in the left middle cerebral artery (MCA) territory. Although the classic ultrasonographic findings were not seen on carotid duplex, the absence of significant atherosclerotic plaques and smooth tapering with severe luminal narrowing of internal carotid artery (ICA) strongly suggested a possible arterial dissection. The findings were confirmed on digital subtraction angiography (Figure 1B). Transcranial Doppler ultrasonography revealed anterior cross-filling of the left MCA via a patent anterior communicating artery. No spontaneous microembolic signals were noted on extended monitoring. Vasomotor reactivity was evaluated during voluntary breath holding for 30 seconds with simultaneous recording of both MCAs. Normal flow acceleration was noted in the right MCA during breath holding, but at the same time, the left MCA demonstrated a paradoxical reduction in flow velocities resulting from the intracranial steal phenomenon (Figure 2), the so-called reversed Robin Hood syndrome.

Metabolic perfusion and cerebral vasodilatory reserve were evaluated by acetazolamide-challenged technetium-99–hexamethylpropyleneamine oxime single-photon emission computed tomography. Significantly increased perfusion (Figure 1G) in the left MCA territory despite multiple ischemic infarctions and severe ICA steno-occlusive disease suggested “luxury perfusion.” However, a paradoxical reduction in the metabolic perfusion in the left MCA territory (Figure 1H) occurred after the vasodilatory challenge with acetazolamide, consistent with reversed Robin Hood syndrome.
In addition to administering aspirin and statins, we kept our patient in the “head-down” position and provided liberal intravenous fluids to facilitate cerebral perfusion. Although we did not observe sleep apnea, nocturnal desaturation, or significant blood pressure reductions, he continued to have neurological fluctuations during the first week, especially on awakening. We hypothesize that the vicious cycles of transient hypoventilation-hypercapnia-intracranial steal phenomenon, occurring several times during sleep, accounted for his diurnal fluctuations. He made a slow but satisfactory recovery and was discharged after 3 weeks (modified Rankin Scale score, 0). Despite persistent and severe ICA steno-occlusive disease, he has remained symptom free during 4 months of follow-up. Normal vasomotor reactivity (and no reversed Robin Hood syndrome) was noted on transcranial Doppler ultrasonography reassessment at 3 months.

In conclusion, we present a patient with acute ischemic infarctions in the MCA territory caused by spontaneous ICA dissection. Luxury perfusion is an uncommon brain imaging abnormality, usually seen after 3 to 7 days of infarction, and represents the failure of cerebrovascular autoregulation. Reversed Robin Hood syndrome in such cases might aggravate the tissue injury and should be considered if neurological fluctuations are noted during the early poststroke period. 

Disclosures
None.

References
Reversed Robin Hood Syndrome in a Patient With Luxury Perfusion After Acute Ischemic Stroke

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