the Caribbean. This would offer an excellent opportunity for us to understand the etiology of hypertension.4

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References

Wallenberg's Syndrome Secondary to Bullet Injury of the Vertebral Artery

Lateral medullary ischemic infarction (Wallenberg’s syndrome)1 is probably the most frequent stroke in the vertebrobasilar system distribution.2 Recognized etiologies include thrombosis, embolism, spontaneous dissections (aneurysm, chronic hypertension and atherosclerosis, fibromuscular dysplasia, arteritis, migraine, and oral contraceptives use), neck manipulation, fitness exercises, and trauma.3-6 We report a patient with a lateral medullary syndrome (LMS) following a gunshot wound in the neck and subsequent occlusion of the right vertebral artery. To the best of our knowledge, no such case has ever been described.

A healthy, 51-year-old right-handed man received a gunshot wound in the right dorsal aspect of the neck. No exit wound was present. He lost consciousness for the next 7 hours, then regained consciousness with nausea, vomiting, and dizziness. Neurological evaluation revealed spontaneous horizontal and clockwise torsional nystagmus in all positions of gaze, numbness in the right side of the face and left arm and leg, a right Horner’s syndrome, ataxia of the right limbs, hoarseness, and decreased movement of the right soft palate. On cervical spine x-ray, a metallic fragment was observed on the C1 lateral mass. Computed tomographic scan of the head showed no parenchymal abnormalities. Magnetic resonance imaging (MRI) showed increased T2 signal compatible with infarction in the right lateral medulla and inferior cerebellum (Figure 1). An angiogram showed occlusion of the distal third segment of the right vertebral artery and the lateral medullary segment of the right posterior inferior cerebellar artery (PICA) (Figure 2). The patient received 325 mg aspirin per day. He made a slow recovery, with residual right Horner’s syndrome, hoarseness, and mild incoordination on the right side as well as slight numbness on the left side of his body. When seen 4 months later, his neurological status was essentially unchanged.

The clinical and radiological features of our patient were similar to those in previously reported cases of LMS. Our patient showed medullary as well as cerebellar infarction in the PICA territory, as previously described.7

Injury from the 5.2-mm bullet resulted in occlusion of the distal right vertebral artery just after its emergence from the first transverse foramina, presumably due to intramural hematoma with secondary thrombosis.8 The T1-weighted axial MRI revealed a hypointense lesion in the region of the right lateral medulla and basal cerebellum. These areas showed increased signal intensity in T2-weighted images consistent with acute ischemic infarction.

Our case report adds one more cause to the list of documented etiologic conditions resulting in Wallenberg’s syndrome.

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References

Figure 1. T2-weighted axial image showing high-intensity signal in right lateral medulla and cerebellum.

Figure 2. Four-vessel cerebral angiogram. Complete occlusion of right vertebral artery (arrowhead) and posterior inferior cerebellar artery (arrow). Bullet is demarcated with three small arrowheads.
Response
The authors report yet another cause of direct trauma to the vertebral artery with resultant occlusion. While claims of primacy in observation and reporting are subject to contradiction (and frequently are), their case may well be the first such instance to be reported. The authors do not comment on the presumed cause of the prolonged initial period of unconsciousness, a most unusual feature in patients with acute ischemic lateral medullary syndrome. The occlusion of the PICA at some distance from the site of vertebral artery occlusion suggests that embolism to the PICA may have occurred. This case sheds no further light on the continuing debate over the relative roles of occlusion of the vertebral artery and the PICA as the cause of this syndrome. It also sheds no light on the long-standing dispute as to the role of anticoagulant drugs in acute vertebral basilar ischemic disease; one aspirin per day was associated with a good clinical outcome.

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Pure Motor Monoparesis Due to Intracerebral Hemorrhage

Pure motor monoparesis (PMM), characterized by motor involvement limited to one limb without sensory deficits, is a rare symptom complex in central nervous system diseases. Recently, we had a chance to treat a patient with persistent PMM caused by an intracerebral hemorrhage whose lesion (confirmed by magnetic resonance imaging [MRI]) was restricted to the primary motor area in the opposite hemisphere.

The patient was a 49-year-old right-handed man. He suddenly realized that he was unable to move his left arm and was immediately admitted to our hospital. His history included aortic valvuloplasty for aortic regurgitation (AR) 10 years earlier and chronic hepatitis. On admission, he was alert and oriented. Cranial nerves, including the facial nerve, were normal. His left upper limb was flaccid, with 0/5 strength by manual muscle testing. However, the left lower limb retained completely normal strength. Sensation was normal in all modalities. Deep tendon reflexes were weak in the left upper limb. Babinski sign was negative bilaterally. Although slight liver dysfunction was noted on laboratory examinations, results of the coagulation tests were within normal limits. An echocardiogram revealed mild AR but no embolic sources. A brain computed tomographic (CT) scan demonstrated a high-density lesion in the right frontoparietal region, suggesting hemorrhage. A right carotid arteriogram was normal (including the venous phase). Somatosensory evoked responses were normal. Deep tendon reflexes in the left upper limb became hyperactive in a few days. The left arm weakness improved gradually. One month after the onset, the proximal strength of the left arm improved to 4/5 and those of the distal muscles to 3/5. Another CT scan performed at this time showed a shrinkage of the high-density area. A T1-weighted MRI at this time demonstrated a small circumscribed high signal intensity area in the upper portion of the right precentral gyrus (Figure 1). The lesion was restricted to the cortical and partially subcortical regions in the primary motor area. After the rehabilitation the patient was discharged with mild residual forearm weakness.

Reported etiologies of PMM originating from intracranial lesions include brain tumor,1,2 brain abscess,3 and ischemic stroke.4-7 Although Sossin and colleagues,8 in their series of patients with pure motor hemiplegia, described a patient showing PMM caused by an intracerebral hemorrhage in the internal capsule, the symptom extended to hemiparesis in a few hours. Thus, to our knowledge, our patient is the first case of stable PMM due to intracerebral hemorrhage. Theoretically, any suitably placed lesions along the course of the corticospinal tract could produce PMM. However, the lesion most likely to produce this sign would be in the cortical or near-cortical area because somatomotor representation is most widely separated at this level. At the origin of motor innervation, i.e., in the primary motor cortex, the pattern of motor representation has been well known as the “motor homunculus.”9 The T1-weighted MRI of our patient obtained 1 month after stroke onset demonstrated a restricted lesion in the upper portion of the right precentral gyrus. This lesion location corresponded well with the site of the contralateral upper limb in the motor homunculus. The nature of hemorrhage in our patient was different from the usual hemorrhagic stroke in its location and size. The results of the neuroradiological and other examinations could not disclose the exact causes of the unusual hemorrhage. However, we considered the possibility of the hemorrhage following the rupture of the small angioma.

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References

FIGURE 1. T1-weighted magnetic resonance image (1.0-T; spin-echo; repetition time, 500 msec; echo time, 34 msec) obtained 1 month after stroke onset demonstrating a small high signal intensity area in upper portion of right precentral gyrus. Arrow 1, right precentral sulcus joining to superior frontal sulcus; arrow 2, right central sulcus.
Wallenberg's syndrome secondary to bullet injury of the vertebral artery.
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