Three months after admission, the patient was well and only minor signal abnormalities were found on the brain stem, either on T1-weighted images or on T2-weighted images. In the left extremities, moderate dysmetria, dysdiadochokinesia, and hypotonia were seen. Blood chemistry, hematologic, coagulation, and immunologic studies, as well as an electrocardiogram and an echocardiogram were normal. Anticardiolipin antibodies, syphilis, and human immunodeficiency virus (HIV) tests were negative. Cerebrospinal fluid was clear, with an opening pressure of 210 mm H2O. There were 8 WBC/mm3 (all mononuclear), glucose was 79 mg/dl, and protein 55 mg/dl (improperly reported as 55 mg/l). The patient was treated with heparin and developed no further deficits. One week later, a plain MRI was performed 20 hours after the onset of the acute symptoms using a 0.5-T superconducting magnet unit with and without gadolinium. A digital subtraction angiography was performed and showed resolution of the previous angiographic abnormalities.

Magnetic resonance imaging is more sensitive than CT for detection of strokes, particularly in the vertebrobasilar system.1–3 Only a few reports of MRI imaging of vertebrobasilar artery occlusions have been made.4–6 In two of them,5,6 nonenhanced MRI was performed in the acute stage, within 24 hours of the ictal event, and showed absence of a flow void in the affected vessel on axial T2-weighted images and areas of iso- or mild hyperintensity signal relative to brain parenchyma on T1-weighted or proton density images. None of them used a contrast-enhanced MRI scan.

In our patient, gadolinium demonstrated contrast enhancement of the abnormal signal over the left VA, suggesting slow flow within this artery, and thus aided the diagnosis of dissection. Although changes in signal intensity within the vessel may be attributed to thrombus formation, altered (i.e., slow) flow dynamics is the more likely possibility in this case because of the hyperintense signal on T1-weighted imaging enhanced with gadolinium. This phenomenon occurs more frequently in vessels with slow flow, usually in the venous system, probably because of the enhancement of the hydrogen nuclei within the slowly flowing vertebral artery.6,7

This case supports the usefulness of enhanced MRI as a noninvasive method for early diagnosis of patients with suspected acute intracranial vertebral artery dissection, a situation in which plain MRI may not show conclusive abnormalities.

References


Platelet Aggregation in Patients With Parkinson’s Disease

To the Editor:

In their article, Struck et al1 commented on the lower incidence of ischemic stroke in patients with Parkinson’s disease as compared with controls and attributed it to decreased tobacco intake, decrease in generalized atherosclerosis, and dopamine deficiency.

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FIGURE 1. Sagittal T1-weighted magnetic resonance imaging scan (TR 500 msec, TE 40 msec) performed 24 hours after admission. A hyperintense tubular signal enhanced with gadolinium at the level of intracranial left vertebral artery is noted (arrow). Extremities. Past medical history and review of the systems was unremarkable except for a 1–2 pack-per-day smoking history for 20 years.

Upon examination, the patient appeared ill. Blood pressure, pulse, respirations, and temperature were normal. He was fully oriented, without neck rigidity or meningeal signs. Fundi appeared normal. A left Horner syndrome and bilateral rotatory nystagmus were seen. Remaining cranial nerve functions were intact. A moderate right hemiparesis, right hyperreflexia, and hypotonia were seen. Blood chemistry, hematologic, coagulation, and immunologic studies, as well as an electrocardiogram and an echocardiogram were normal. Anticardiolipin antibodies, syphilis, and human immunodeficiency virus (HIV) tests were negative. Cerebrospinal fluid was clear, with an opening pressure of 210 mm H2O. There were 8 WBC/mm3 (all mononuclear), glucose was 79 mg/dl, and protein 55 mg/dl (improperly reported as 55 mg/l). The patient was treated with heparin and developed no further deficits. One week later, a plain MRI was performed 20 hours after the onset of the acute symptoms using a 0.5-T superconducting magnet unit with and without gadolinium. A digital subtraction angiography was performed and showed resolution of the previous angiographic abnormalities.

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To the Editor:

In their article, Struck et al1 commented on the lower incidence of ischemic stroke in patients with Parkinson’s disease as compared with controls and attributed it to decreased tobacco intake, decrease in generalized atherosclerosis, and dopamine deficiency.
We would like to suggest another factor, namely, that of decreased platelet aggregation, which may contribute to their observation. We studied platelet aggregation in 25 newly diagnosed patients with Parkinson's disease and 25 age- and sex-matched controls. None were diabetic, hypertensive, smokers, or on any treatment. Citrated blood was collected by venipuncture and centrifuged at 200g for 10 minutes, and the supernatant platelet-rich plasma was obtained. Platelet counts were measured using a platelet counter (Contraves, Switzerland). No patient had a low platelet count. Platelet aggregation was measured by the Born method2 using a four-channel aggregrecorder (Daiichi-PA320, Kyoto, Japan). Adenosine diphosphate (2.5 μM/ml), collagen (2.5 μM/ml), and epinephrine (3 μM/ml) were used as inducers. The stock solution was diluted with 0.85% saline. The aggregation was studied at fixed intervals of 5 minutes for collagen and 10 minutes each for adenosine diphosphate and epinephrine. The absorbance was measured as % aggregation. The results were analyzed statistically using Student's t test.

Platelet aggregation induced by adenosine diphosphate and epinephrine was significantly decreased (32% and 60%, respectively) in Parkinson's disease cases, while collagen-induced aggregation was unchanged.

To the best of our knowledge, these findings have not been recorded elsewhere. We hypothesize that decreased platelet aggregation in Parkinson's patients may be a significant contributory factor for the reduced incidence of ischemic stroke.

References

Glycerol Infusion Rates Warrant Caution

To the Editor:

We wish to comment on the assertion by Nau and colleagues1 that very high intravenous infusion rates of glycerol (>500 ml of 10% solutions given over 4 hours) are required to exert any significant osmotic effect on cerebral edema associated with acute stroke.

The most important side effect of such dosages is intravascular hemolysis.2 We conducted a critical evaluation of the latter adverse effect and its possible mechanism in the course of a large, randomized, double-blind clinical trial of intravenous glycerol treatment in patients with acute stroke. This in vivo and in vitro study3 suggested that hemolysis resulted from glycerol at the site of infusion rapidly entering red cells and from destruction of the latter in more central veins due to osmotically induced swelling beyond a critical limit. Moreover, so long as the infusion rate was not allowed to exceed 125 ml/hr, even temporarily, clinically significant hemolysis was avoidable. Japanese investigators and clinicians have reported that use of glycerol solutions containing small amounts of fructose can overcome this problem associated with more rapid infusion rates.4,5 Moreover, in vitro studies in our institution (Figure 1) are also consistent with such a possibility.

Thus, regardless of the possible benefits before embarking on further studies to evaluate the value of more rapid glycerol infusions in managing acute stroke, it is important to address this anticipated and alarming degree of intravascular hemolysis.

References

Multiple Aneurysms Caused by Hemodynamic Stress and Hypertension

To the Editor:

Increased hemodynamic stress is possibly a factor in the development of cerebral aneurysms. The clinical basis for this possibility includes the development of aneurysms in feeders of arteriovenous
Platelet aggregation in patients with Parkinson's disease.
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