Acute Basilar Artery Occlusive Disease

To the Editor:

Current treatment of basilar artery occlusion is unsatisfactory, with an unacceptably high morbidity and mortality approaching 70%. Fibrinolytic therapy is an exciting approach and has been shown anecdotally to help, but it is still experimental and therefore of unproven value. Heparin alone has anecdotally been described to improve impending basilar artery occlusion. C. Miller Fisher has described two patients with presumed basilar artery occlusion who improved after being turned upside down and given heparin as an intravenous bolus. We recently had the opportunity of implementing Fisher’s novel approach, with a new twist and a surprisingly good outcome.

A 52-year-old white male with a history of heavy cigarette and alcohol use, but no history of illicit drug use including cocaine, arrived at the emergency room in the evening complaining of bioccipital and parietal headache, which first appeared upon awakening that morning and continued steadily for several hours. Early in the afternoon while at work, he had felt a sense of spinning and unsteadiness of gait, with subsequent diplopia and blurred vision. He felt weak on both sides, but more so on the right. These symptoms fluctuated prior to his arrival at the emergency room, but had been present for longer than 1 hour at the time of this evaluation.

Initial neurologic examination revealed the patient to be alert and oriented. He had severe dysarthria. A left sixth nerve palsy and left internuclear ophthalmoplegia, as well as a right facial weakness and decreased hearing on the right side, were present. The gag reflex was diminished. Motor examination revealed a flaccid, plegic right upper extremity and a paretic right lower extremity. Bilateral upper extremity dysmetria was present, as well as bilateral pathologic hyperreflexia, with clonus of the right patella and ankle and bilateral extensor plantar responses. The hemoglobin was 16.3. The head computed tomographic (CT) scan showed no hemorrhage, but did reveal a hyperdense basilar artery, as well as a questionable left dorsomedial pontine infarct (Figure 1). Because of the suspicion of basilar artery occlusion, the patient was initially hydrated and held upside down and shaken for about 5 minutes. He was then started on heparin (intravenous drip), without bolus, and phlebotomized. In about 5 minutes his neurologic deficit resolved with the exception of the left sixth nerve paresis and internuclear ophthalmoplegia.

Transcranial Doppler performed the following day suggested basilar stenosis. The patient’s condition persisted unchanged, and 4 days after admission, cerebral angiography was performed, which demonstrated a 25–30% left midbasilar stenosis (Figures 2 and 3), as well as filling defects in the basilar artery, presumably due to clots. The patient was continued on heparin and then treated with warfarin. Pretreatment search for coagulopathy was not performed. Follow-up at 2 and 6 weeks revealed an intact neurologic examination with the exception of a mild left internuclear ophthalmoplegia.

Our initial clinical impression was that the patient had impending basilar artery occlusion based on fluctuating bilateral findings,
including brainstem signs, headache, and absence of hemorrhage on head CT scan, together with an opacified basilar artery suggestive of basilar artery thrombosis.2,4

Any effective treatment of basilar artery occlusion is welcome. Our experience, together with Fisher’s report of turning the patient upside down,2 suggests that the use of heparin in conjunction with mechanical factors (i.e., gravity or shaking, which we implemented) may be more beneficial than anticoagulation alone. The presence of clot, suggested by the cerebral angiogram, may have made mechanical “treatment” more likely to be successful, perhaps by dislodging the clot and enhancing clot dissolution.

Obviously our case is anecdotal and uncontrolled; however, this approach warrants further investigation in a larger number of patients to determine if manipulating other factors of the vertebral-basilar circulation and the occlusive process truly benefits in cases of suspected acute basilar artery occlusion.

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References

Paradoxical Cerebral Embolism Secondary to Pulmonary Arteriovenous Fistula

To the Editor:

Cerebral infarction in young adults is a disease that has received a great deal of attention in recent years due to its frequency and high rate of functional sequelae. Paradoxical embolism is a well-known but infrequent cause of cerebral infarction in young adults, accounting for just 4% of the total.1,2 It is mostly due to the existence of a right-to-left shunt at the cardiac level, though occasionally the shunt may be at the pulmonary level. We present a patient with varicose veins in the lower limbs and a pulmonary atrioventricular (A-V) fistula that started with an episode of cerebral embolism.

A 37-year-old male with no previous pathology or toxic habits, who was well the day before admission, experienced a sudden headache and difficulty speaking after swimming in very cold sea water. On admission he was conscious and aphasic, with rhythmic heartbeats at 88 beats/min and blood pressure of 130/70 mm Hg. We noted a continuous bruit at the seventh left intercostal space, which increased with inspiration, and severe varicose veins in both lower limbs.

Brain computed tomography (CT) without contrast carried out on admission demonstrated two hypodense lesions in vascular territories of the left anterior and middle arteries with no mass effect, consistent with areas of infarction at these levels. The CT with contrast performed 6 days later confirmed the previous findings, with details suggestive of luxury perfusion in these areas. Angiographic studies of both carotid arteries and basilar-vertebral system were normal.

Chest radiography, electrocardiography, and two-dimensional echocardiography were all normal. A Doppler-echo examination and the phlebography of both lower limbs carried out several days after admission disclosed numerous varicose dilatations in both external saphenous veins, although there was no evidence of thrombi in their interior. Pulmonary arteriography disclosed a large pulmonary arteriovenous fistula in the left lower lobe (Figure 1). Endoscopic examination of the superior digestive tract and colonoscopy revealed no angiomata.

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FIGURE 3. Cerebral angiogram, anteroposterior view. There is a region of left midbasilar stenosis 25–30% (arrow).

FIGURE 1. Digital subtraction angiography showing large atrioventricular fistula in left lower lobe.
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