including brainstem signs, headache, and absence of hemorrhage on head CT scan, together with an opacified basilar artery suggestive of basilar artery thrombosis.3-4

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 vertebrobasilar circulation and the occlusive process truly benefits in cases of suspected acute basilar artery occlusion.

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 heart beats 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.

Figure 1. Digital subtraction angiography showing large atrioventricular fistula in left lower lobe.
After admission, the patient received 100 mg of dipyridamole by mouth three times a day and gradually recovered from his neurological deficit. Two months after the initial attack, an embolization of the pulmonary fistula with isobutyl 2-cyanoacrylate resulted in complete occlusion.

Embolism through a pulmonary A-V fistula is a very unusual cause of cerebral infarction. In a retrospective study of 144 patients over 10 years, Adams et al did not find a single case while Hart and Miller reported only two cases in a review of 100 patients. In a multicenter study carried out in Spain on 1,200 patients, only one case associated with Rendu-Osler disease was reported.

Pulmonary A-V fistulas are usually asymptomatic, occasionally causing hemoptysis and exertion dyspnea. The presence of cerebral symptoms associated with this type of lesion may be due to cerebral abscess, embolism, hypoxemia, thrombosis secondary to polycythemia, or brain hemorrhage from an A-V malformation at the level of the central nervous system.

From 40 to 65% of patients with a pulmonary A-V fistula have further vascular malformations in places such as skin, mucous membrane, digestive tract, or brain. In our case, there was no evidence of vascular malformations at other levels. The lack of cardiovascular risk factors, combined with normal results in the other tests, would seem to preclude any etiology other than paradoxical embolism. The presence in this case of large varicose veins in the lower limbs suggests that these may have been the origin of the embolism, despite the absence of thrombi in the phlebographic study performed a few days later. Nevertheless, we cannot rule out the possibility that the embolus was formed in the pulmonary arteriovenous malformation.

This case demonstrates that embolization of pulmonary A-V fistulae may in fact be an efficient way of preventing further episodes of paradoxical embolism.

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