groups after the induction of either hemodilution or hypoxic hypoxia. Although it would be inappropriate to interolate results obtained under normal conditions to those that may be applicable under ischemic conditions, we must agree that the magnitude of hypoxic insult caused by hemodilution or hypoxia may be different between the two groups. However, this does not imply that the blood flow augmentation recorded in the hemodilution group was caused by hypoxic stimulation. If this were true, blood flow in the hypoxic hypoxia group should have increased after the induction of hypoxia. On the contrary, we observed a decrease in blood flow after the induction of hypoxic hypoxia. Therefore, it seems quite appropriate to speculate that the blood flow augmentation observed in the hemodilution group was caused by a mechanism other than hypoxia.

Kazu Korosue, MD
Roberto C. Heros, MD
Department of Neurosurgery
University of Minnesota Medical School
Minneapolis, Minn.

References

Capsular Warning Syndrome Preceding Pontine Infarction

Repete bursts of a focal neurological deficit due to cerebral ischemia have been described as “crescendo transient ischemic attacks” and a subset clinically localized to the internal capsule as the “capsular warning syndrome.” The latter is characterized by three or more episodes affecting face, arm, and leg, without cortical symptoms, occurring within 24 hours. A high proportion of individuals develop subsequent capsular stroke. It has been postulated that the mechanism for such strokes is that of small-vessel single-penetrator disease. We would like to report a case fitting the criteria for the capsular warning syndrome that progressed to anterior pontine infarction.

The patient was a 72-year-old right-handed man. While watching television he was suddenly unable to move his right arm or leg and noted severe dysarthria. The initial episode lasted 15 minutes, with total recovery such that the results of neurological examination on admission to the hospital 1 hour later were entirely normal. Over the course of the next 24 hours, he had at least 15 episodes of complete paralyses of the right face, arm, and leg, with associated dysarthria with no sensory or cortical signs. The episodes lasted from 5 to 30 minutes, with recovery between. These episodes persisted despite anticoagulation with heparin. After 3 days the fluctuations stopped, and he was left with 0/5 power in his face, arm, and leg, with persisting dysarthria. This improved over the following 3 weeks to 3/5 power in his arm and leg, mild facial paresis, and dysarthria. His medical history included treated hypertension and hypercholesterolemia, and he was an ex-smoker.

A cerebral angiogram revealed a hypoplastic left vertebral artery, a dominant right vertebral artery, and a normal basilar artery. The anterior circulation was normal. Transesophageal echocardiography was normal. A computed tomographic (CT) scan of the brain was normal on admission and remained normal when repeated 8 days after the completed stroke. Magnetic resonance imaging (MRI) performed 7 days postictus showed an infarction in the left anterior pons on T2-weighted images (Figure 1). There were no lesions elsewhere.

The repeated stereotypic nature of these episodes clearly fits with the description of the capsular warning syndrome. The pontine location of subsequent infarction is, however, at variance with that previously reported (capsular), although it was postulated as an alternative site. In a series of 50 patients, abnormalities on CT scan were found in 25, with deep capsular infarctions being the most common. The rest were normal. It may be that with the superior imaging of the brain stem achieved with MRI, more of these patients have pontine infarctions than were previously recognized, and the presence of profound dysarthria during the episodes may suggest this location. The risk of stroke after capsular warning syndrome is extremely high, and may reach 42% within 3 weeks of the onset of symptoms. The failure of heparin to prevent stroke in this case emphasizes probable resistance of this condition to current forms of therapy. We believe that a prospective trial of intervention is warranted and that future classification should make use of MRI scanning to accurately determine the location of infarction.

Jeremy Farrar, MRCP
Geoffrey A. Donnan, MD
Department of Neurology
Austin Hospital
Heidelberg, Australia

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Capsular warning syndrome preceding pontine infarction.

J Farrar and G A Donnan

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