Letters to the Editor

Plasmapheresis in Heparin-Induced Thrombocytopenia and Thrombosis

To the Editor:
Thrombosis associated with severe thrombocytopenia is an infrequent, but life-threatening side effect of heparin therapy, which can involve any venous or arterial territory including the cerebral circulation. The treatment of this serious complication has not been satisfactorily resolved. Recently, Becker and Miller reviewed heparin-induced thrombocytopenia with thromboembolism and found one case that had been treated successfully with plasmapheresis. In a recent report, we described a similar patient who also satisfactorily responded to plasmapheresis therapy. She was admitted to the hospital for right thrombophlebitis with pulmonary embolism. After 13 days on heparin therapy, new thrombotic events in pulmonary and peripheral circulation became evident, and the platelet count fell to 10,000/mm³. We discontinued heparin administration, but the platelet count remained unchanged and the ischemic lesions increased progressively until plasmapheresis therapy was initiated. Shortly thereafter, the number of platelets returned to normal, and the ischemic alterations improved considerably.

The incidence of heparin-induced thrombocytopenia with thrombosis is approximately 1–2%, and it has a mortality of nearly 30%. It is of particular importance to neurologists in the treatment of cerebrovascular disease. An immune-mediated mechanism has been established as the cause of this complication, which is detected in the plasma of most affected patients by the presence of heparin-dependent antiplatelet antibodies that induce platelet aggregation. Withdrawal of heparin treatment is essential for recovery, but other therapeutic measures must be used as well to prevent progressive development of platelet thrombi. Plasmapheresis therapy, useful in other antibody-mediated diseases, can be very effective in severe heparin-induced thrombocytopenia with thrombosis by removing the pathogenic immunoglobulin.

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References

Lacunar Pontine Infarction With Hemifacial Spasm as the Initial Symptom

To the Editor:
Recently, Ambrosetto et al described an unusual case of lacunar pontine infarction producing hemifacial spasm as an isolated clinical finding. We report here our recent experience with a case of lacunar pontine infarction producing hemifacial spasm as the initial symptom.

Our patient was a 76-year-old hypertensive woman who noticed numbness and tightness on the left side of her face when she got up one morning. About 1 hour later, during her breakfast, rapid, involuntary muscular twitches occurred on the left inferior orbito-ocularis oculi and soon spread over the left side of her face, occurring intermittingly.

Two days later, she was admitted to our hospital. The neurologic examination on her admission revealed a clearly conscious woman who had a slight speech disturbance, a slight right hemiparesis, and intermittent hemifacial involuntary movements similar to the facial spasm that occurs as the common sequel of partial peripheral facial nerve lesions. Computed tomographic scans of her head on admission showed a small lacunar infarct in the left pons.

The hemifacial spasm occurred frequently for the first 4 or 5 days after onset, but thereafter gradually decreased until its disappearance about 2 weeks later.

Ambrosetto et al explain the mechanism of hemifacial spasm as the result of compression of the intrapontine roots of the facial nerve by perifocal ischemic edemas. Since our patient also had slight contralateral hemiparesis, the hemifacial spasm seemed to result from irritation of the nucleus or the facial nerve itself by ischemic edema. Though such a case is quite rare, we believe it important to pay attention to this symptom as a sign of lacunar pontine infarction.

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Changes of Cerebral Blood Flow Velocity During Cognitive Activity

To the Editor:
In the September issue of Stroke, Droste and colleagues measured the time course of cerebral blood flow velocity (CBFV) changes in the middle cerebral artery (MCA) to cognitive activity with transcranial Doppler sonography (TCD). Under their conditions of 90 seconds reading aloud followed by a 42-second rest-phase, the authors found an immediate increase of CBFV after onset with a maximum peak (15% increase relative to the steady-state at rest) after about 8 seconds. A steady-state of about 10% increase in CBFV appeared 24–42 seconds after onset. In the rest-phase, the enhanced CBFV continued until a small peak after 6 seconds when it decreased to a minimum and then increased.

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