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 heparin-dependent antiplatelet antibodies that induce platelet thrombosis by removing the pathogenic immunoglobulin. 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.

L. Manzano, MD
J.A. Vargas, MD
L. Barbolla, MD, PhD
Service of Internal Medicine I and Service of Hematology
Clínica Puerta de Hierro
Universidad Autónoma de Madrid

M. Alvarez-Mon, MD, PhD
Service of Internal Medicine I and Department of Medicine
Hospital Universitario de Alcalá de Henares
Universidad de Alcalá de Henares
Madrid, Spain

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 orbitalis oculli and soon spread over the left side of her face, occurring intermittently.

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.

Masato Kawakami, MD
Toyoeaki Sato, MD
Shoichiro Tochigl, MD
Toru Ito, MD
The Third Department of Internal Medicine
St. Marianna University School of Medicine
Kawasaki, Japan

Reference

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.

Masato Kawakami, MD
Toyoeaki Sato, MD
Shoichiro Tochigl, MD
Toru Ito, MD
The Third Department of Internal Medicine
St. Marianna University School of Medicine
Kawasaki, Japan

Reference
The following is in response:

To the Editor:

We believe that vocalization does in fact influence the recordings presented by Droste et al. We agree that vocalization may interfere with the recordings, but we believe its effect occurs more by changes of intrathoracic pressure than by conduction of the voice frequency spectrum to the probe. This influence, together with fluctuation of the measured values due to vegetative, respiratory, and cardiovascular factors, accounts for the substantial standard deviation. Voice artifacts caused by direct conduction have a low frequency and merely alter the envelope curve.

Figure 1 in the report by Diehl et al gave us the impression that the initial peak during reading approximately 8 seconds after starting the task was consistent with our findings. Those authors also found an initial peak during rest phases.

In the study conducted by Diehl et al, mean rest values were higher than the mean task values, which agrees with observations in some of our subjects. Lower values while performing a task have also been recorded in individual persons with the xenon-133 inhalation technique, but most of these studies, like ours, showed an increase in values while performing a task.

There are two main differences limiting the comparability of our two studies. The first concerns the reference baseline, that is, the last 9 seconds of the 42-second rest phase in our study and the mean of all the rest and task values in the study of Diehl et al. We are not sure that the last 9 seconds of the 42-second rest phase really represent a rest steady state; longer monitoring under rest conditions has to be done to confirm this hypothesis. Secondly, the rest phase in the report by Diehl et al preceded the task phase, possibly reflecting increased attention, which might be the reason for the increase of MCA blood flow velocity in their rest phases.

In our study, the rest phase followed the task phase, and the decrease in values probably reflects relaxation. Furthermore, the difference in blood flow velocity in the middle cerebral artery before and after vocalization could be due to vegetative, respiratory (e.g., arterial PCO2), and cardiovascular factors. 

A.G. Harders, MD
Department of Neurosurgery
University of Freiburg
Freiburg, FRG

References

The following is in response:

To the Editor:

We believe that voice artifacts have influenced the recordings presented by Droste et al.1,2 shifting the CBFV curve during the reading phase above its real value. At first glance, their results fit with the response one would expect from investigations of regional cerebral blood flow (rCBF) measurement with the xenon-133 inhalation technique,2 showing that cerebral circulation is increased during a verbal task in selected brain areas. On the other hand, Lassen et al3 showed that rCBF values increase in considerably larger parts of the frontal and prefrontal cortex during a rest state and decrease during mental activity. Since most of these cortices are supplied by the MCA, enhanced frontal circulation during the rest phase may lead to a mean increased CBFV as measured from this vessel. Based on this average, smaller parts of the MCA territory related to verbal tasks may only be detected during the abrupt onset of activity. The dynamics and mechanisms of this obvious rapid and transitory shift of cerebral circulation are to be investigated further.

Rolf R. Diehl, PhD
Matthias Sitzer
Michael Hennerici, MD
Department of Neurology
Klinikum Mannheim
University of Heidelberg, FRG
Changes of cerebral blood flow velocity during cognitive activity.
R R Diehl, M Sitzer and M Hennerici

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http://stroke.ahajournals.org/content/21/8/1236.3.citation

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