Antithrombin-III Deficiency in Ischemic Stroke

To the Editor:

Hereditary antithrombin III (AT-III) deficiency is associated with an increased risk for venous thrombosis and pulmonary embolism and possibly also for arterial embolism, including cerebral thromboembolism. Acquired AT-III deficiency is more common, but its pathogenetic and clinical relevance is unclear. We present here three patients with acquired AT-III deficiency as a possible risk factor for ischemic stroke.

Blood samples were drawn within 24 hours after stroke onset in 45 consecutive patients (24 men and 21 women) with cerebral infarction (n=38) or transient ischemic attack (TIA) (n=7). Mean age was 62±10 (range 36–83) years. Antithrombin III activity was measured with Coatest Antithrombin (Kabi, Stockholm, Sweden) in the automated coagulation laboratory instrument. Reference values were established in 84 healthy blood donors (85–120%).

The mean AT-III level was not significantly decreased in patients with cerebral infarction (98±19%) or TIA (97±12%) as compared with the control group (102±9%) (mean±SD). However, three patients with cerebral infarction had AT-III levels lower than 3 SDs from the mean of the control group.

The first patient (AT-III 57%), a 43-year-old previously healthy woman, developed a cerebral infarction in the territory of the right median cerebral artery and, simultaneously, an arterial embolus in her right brachial artery. She smoked 30 cigarettes daily and was on temporary medication of Gestagen (norethisteron acet., 10 mg/day). The second patient (AT-III 60%) was a 68-year-old woman with type II diabetes mellitus who had iterated venous thromboses for 12 years and three earlier brain infarctions despite medication with 250 mg acetylsalicylic acid daily. The third patient (AT-III 62%) was a previously healthy 65-year-old man who had a cerebral infarction in the territory of the left median carotid artery 3 days after operation for a prostatic neoplasm. None of the patients had a family history of vascular disease or thrombosis, and blood tests, electrocardiogram, and ultrasonic duplex scanning of the carotid arteries were normal in all three patients. Antithrombin III levels were normal in all patients after 6–18 months.

The mean AT-III levels of all 45 patients were not significantly lower as compared with the control group, which agrees with previous studies. However, earlier studies did not report the number or characteristics of patients with low AT-III levels. Our three patients had acquired AT-III deficiency since their levels were normalized during follow-up. Also, all three had low antithrombin activity associated with some other risk factor.

Hypercoagulability has previously been described in contraceptive users, after surgery, and in diabetes mellitus. Thus, a combination of low AT-III activity and other risk factors may predispose for ischemic complications. We conclude that the possibility of substitution therapy should be considered in patients with ischemic infarction in combination with low AT-III activity.

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References

Transient Hemiballism and Striatal Infarct

To the Editor:

We report an unusual case of lacunar striatal infarction that produced a transient hemiballism. At 1 PM, a 70-year-old hypertensive right-handed man suddenly developed abnormal and involuntary flinging movements principally affecting the left arm. The movements were continuous and were increased by emotion and decreased during relaxation. The only abnormality disclosed on neurological examination was a slight hypotonia of the left side. His blood pressure was 160/100 mm Hg. Auscultation of the heart and carotid arteries was normal. Hemiballism reached a peak in 6 hours and then progressively decreased. In 20 hours, it had disappeared. Computed tomography (CT) scans (Figure 1a) at 4 and 12 days disclosed a small, rounded, hypodense area involving the right anterior part of the external capsule. This topography was confirmed by T2-weighted magnetic resonance imaging (MRI) (Figure 1b) obtained 7 days after onset. Neither the two CT scans nor the MRI showed an abscess or tumor.

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Generally, hemiballism is due to a hemorrhagic or ischemic lesion of the subthalamic nucleus (corpus luysii) and, more rarely, to an inflammatory process, such as multiple sclerosis, or from tumor. Under these circumstances, hemiballism is explained by the absence of the regulatory activity of the subthalamic nucleus on the pallidum. Occasionally, the lesion is located in the striatum, and the abnormal involuntary movements are explained by the suppression of the regulatory activity of the caudate nucleus on the pallidum. Hemiballism may then be associated with
Antithrombin-III deficiency in ischemic stroke.
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