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CORRESPONDENCE

Successful thrombolytic therapy in a patient with infective endocarditis-related stroke

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Stroke is common in patients with infective endocarditis (IE). While thrombolysis has been the standard therapy for acute ischemic stroke within 3 hours of onset, the guidelines vary regarding the management of IE-related stroke.[1] The urgency of treatment may preclude the accurate diagnosis of IE.

A 54-year-old woman presented 26 minutes after sudden right hemiparesis. She had suffered an intermittent fever, polyarthralgia, and body weight loss for 1 year. On physical examination, her vital signs were as follows: temperature 38.1°C, pulse 139, blood pressure 169/85. A systolic murmur was audible at the apex. Her skin appeared normal. Her initial National Institutes of Health Stroke Scale score was 9. White blood cell count was 15.55×10^9/L. Electrocardiography showed sinus tachycardia. Baseline head computed tomography was not remarkable. A transthoracic echocardiography showed a trivial mitral regurgitation without evidence of valvular vegetation.

She was treated with intravenous tissue plasminogen activator (tPA) 108 minutes after stroke onset. At 24 hours, she regained normal muscle strength with an improvement of National Institutes of Health Stroke Scale score from 9 to 0. A repeat head computed tomography demonstrated an infarction in the left basal ganglia, two intracerebral hemorrhages in the occipital lobes, and subarachnoid hemorrhage (Fig. 1). Her rheumatoid factor was 244 kIU/L (normal: <20 kIU/L). Six days later, two sets of blood culture yielded *Streptococcus viridans*, so a diagnosis of infective endocarditis was entertained. She received intravenous penicillin G 3 million units every 6 hours for 4 weeks. The results of repeated blood cultures were all negative. Five weeks after admission, she was discharged in good health status.

Because vegetations from IE consist of platelets, fibrin, microorganisms, and inflammatory cells,[2] tPA may be helpful in thrombus resolution. However, IE patients are prone to intracranial hemorrhage. The findings of intracerebral hemorrhages remote from the presumed site of acute cerebral infarction and subarachnoid hemorrhage in the present case support the notion that intracranial hemorrhage in IE arises from multiple mechanisms, such as pyogenic arteritis and mycotic aneurysms, in addition to hemorrhagic transformation.[3] The use of tPA may potentiate some of these mechanisms.

Although fever is the most common manifestation of IE, it may be caused by other infections. Moreover, the

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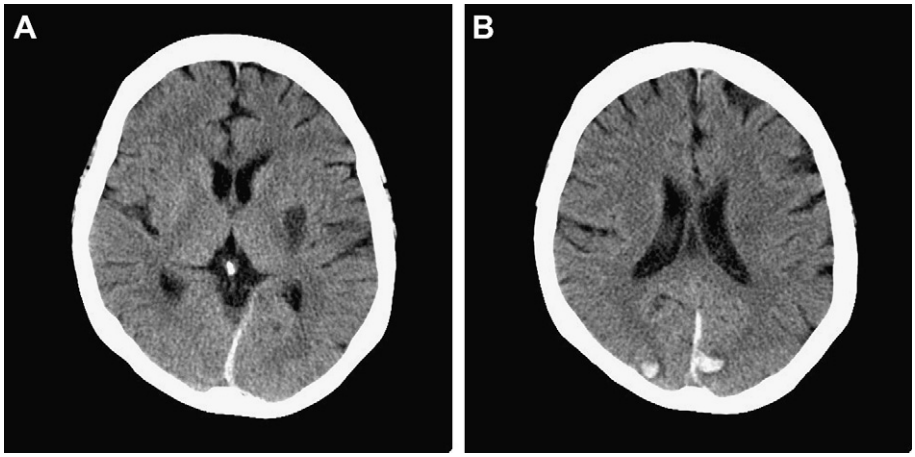

Figure 1 (A) An infarction in the left basal ganglia and subarachnoid hemorrhage in the posterior interhemispheric fissure. (B) Two small intracerebral hemorrhages in bilateral occipital lobes.

modified Duke criteria include positive blood cultures and endocardial involvement as the major diagnostic criteria.[4] Therefore, it is hardly practical to make a definite or possible diagnosis of IE within 3 hours, considering the lack of culture results and the low sensitivity of transthoracic echocardiography in detecting IE.

The application of magnetic resonance imaging (MRI) to assist in making clinical management plans in IE is evolving. Up to 82% of IE patients with or without neurological symptoms harbored at least one cerebral lesion, such as infarction, intraparenchymal hemorrhage, microbleed, subarachnoid hemorrhage, and unruptured aneurysm, and 66% had more than one lesion.[5] Hence, we propose that before thrombolysing stroke patients, MRI may be a promising method to identify patients at risk of developing intracranial hemorrhage. MRI findings of multiple acute ischemic lesions or cerebral microbleeds may raise the possibility of IE.

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