Positive Platelet Scintigram of a Vertebral Aneurysm Presenting Thromboembolic Transient Ischemic Attacks

Michiyuki Maruyama, MD, Tsutomu Asai, MD, Yoshihiro Kuriyama, MD, Tohru Sawada, MD, Jun Ogata, MD, Tsunehiko Nishimura, MD, and Teruo Omae, MD

We describe a patient with transient ischemic attacks secondary to a giant aneurysm who showed increased activity on platelet scintigrams at the origin of the left vertebral artery. This is assumed to be the first report of a presumed embolizing aneurysm with positive activity of labeled platelets. Platelet scintigraphy is useful for diagnosing transient ischemic attacks as being secondary to an aneurysm and was proved to provide direct evidence of a thromboembolic source in vivo. (Stroke 1989;20:687-690)

Intracranial or extracranial unruptured aneurysms as a possible source of emboli have been reported.'-4 Unruptured aneurysms are regarded as a proper clinical model to explain the mechanism of thromboembolic transient ischemic attacks (TIAs). We describe a rare patient with TIAs and a giant aneurysm of the left vertebral artery who showed increased [111] oxine autologous platelet deposition and we emphasize the importance of platelet scintigraphy, which provides evidence of in vivo platelet activation as the presumed mechanism of thromboembolic TIA.

Case Report

A 40-year-old man experienced several episodes of neurologic deficits in the last 6 months. Except for an attack that gave fixed left quadrantanopsia, all other transient symptoms such as double vision, sensory impairment, and weakness of either side of the extremities appeared abruptly and resolved completely in 1-24 hours. These attacks occurred while the patient was moving. The last TIA, consisting of numbness of the left extremities, occurred 3 weeks before admission. According to the temporal profile, a clinical diagnosis was made of thromboembolic TIAs and a completed stroke. There was no history or symptoms of a subarachnoid hemorrhage.

On admission, physical examination was normal except for left quadrantanopsia. Hypertension or cardiac arrhythmia were not present. Complete blood count, serum cholesterol, total protein and fibrinogen concentrations, prothrombin and partial thromboplastin times, chest x-ray film, electrocardiogram, and ultrasonic cardiogram were normal. Computed tomography (CT) of the brain revealed a low-density area in the right occipital lobe, and cranial magnetic resonance imaging (MRI) revealed small infarcts of the thalamus bilaterally and of the right cerebellar hemisphere in addition to the CT abnormalities. Aortic arch and four-vessel cerebral angiography revealed a giant saccular aneurysm at the origin of the left vertebral artery. There were no other abnormalities such as atherosclerotic changes, other aneurysms, or vasospasm (Figure 1). B-mode ultrasound imaging and MRI of the aneurysm showed a structure suggestive of mural thrombus within it. These clinical findings suggested that the TIAs were brought about by thromboemboli originating from the giant aneurysm of the left vertebral artery.

Platelet scintigraphy of the neck was performed to detect an active site of platelet deposition that might indicate the embolic source. Platelets were isolated from 43 ml peripheral whole blood, washed, and labeled with [111] oxine according to the method described by Heaton et al.5 The anterior images of the neck, obtained 28 and 50 hours after the injection of 200 µCi labeled autologous platelets, revealed a well-defined focus of abnormal activity in the giant aneurysm (Figure 2). Because technetium-99m-labeled human serum albumin injected simultaneously did not show any abnormal activity in the aneurysm, the increased activity revealed by platelet scintigraphy was interpreted to be not a blood
pooling image but active platelet deposition within the aneurysm.

From these radiologic examinations, we regarded the etiology of the recurrent ischemic episodes of the brain to be thromboembolism caused by microemboli breaking off from the thrombus in the giant aneurysm of the left vertebral artery, so we took preventive measures against embolism.

Transient attacks of sensory impairment of either side of the extremities persisted even 2 weeks after administration of 330 mg/day aspirin, when platelet scintigraphy performed with 150 μCi labeled platelets showed no increased activity within the giant aneurysm. Because antiplatelet therapy could not prevent recurrence of the TIAs, an aneurysmectomy was performed. A giant saccular aneurysm (2.8x2.8x1.7 cm) was removed (Figure 3), and an end-to-end anastomosis of the left vertebral artery was performed using an artery graft from the left radial artery. The wall of the removed aneurysm was composed of degenerated elastic and muscle layers and intimal fibrosis. The aneurysm contained a brown thrombus firmly attached to the wall. The thrombus consisted of an aggregation of fibrin. Enmeshed in this aggregation were entrapped erythrocytes, and granular structures staining lightly eosinophilic with hematoxylin and eosin but not staining with phosphotungstic acid hematoxylin and Masson's trichrome stain. This material was interpreted as representing aggregates of platelets. Most of the luminal surface of the thrombus was covered with endothelium (Figure 4).

The patient had no subsequent TIAs after the aneurysmal removal.

Discussion

The mechanisms of TIA are very complex, and many reports have focused on thromboembolic TIAs caused by microemboli from several embolic sources. Recently, embolizing aneurysm as an embolic source of TIAs has been reported. Cohen et al proposed four criteria based on clinical findings to determine whether a TIA is caused by an embolizing aneurysm: 1) clinical TIA or completed stroke, 2) arteriographic, surgical, and/or autopsy verification of the aneurysm, 3) no other lesions that could produce a TIA or stroke, and 4) no clinical or radiographic evidence of recent subarachnoid hemorrhage or vasospasm that could provide an alternative etiology. Among others, a TIA secondary to an aneurysm can be thus regarded as the most proper clinical model of the thromboembolic TIA because focal circulatory disturbance due to atherosclerotic occlusive changes of the arteries can be excluded.

FIGURE 1. Left: Digital subtraction aortogram of 40-year-old man. Giant saccular aneurysm (short arrows) originating from left vertebral artery at C7 level is demonstrated. Right: Oblique view of aneurysm demonstrated by left subclavian digital subtraction angiogram.
FIGURE 2. Indium-111 platelet scintigram of neck of 40-year-old man obtained 50 hours after injecting labeled autologous platelets. Well-defined focus of abnormal activity at aneurysm of left vertebral artery (short arrows) is shown.

On the other hand, [111mIn] oxine platelet scintigraphy may be useful in detecting sources of platelet activation that may lead to embolization. Although platelet scintigrams of the neck have been obtained from patients with ischemic cerebrovascular disease, the results did not always correlate significantly with the clinical findings. There is no previous report of scintigraphy in patients with cerebral ischemia secondary to embolizing aneurysms.

FIGURE 3. Photograph of aneurysm of left vertebral artery. Longitudinal sections of aneurysm through orifice (arrowhead) and exit (short arrow) show mural thrombus (*) in aneurysmal sac. Bar=5 mm.
In the patient reported here, we successfully demonstrated an increased activity of labeled platelets in the aneurysm, which was diagnosed to be the embolic source of the TIA's according to clinical criteria and pathologic findings. Thus, we obtained direct evidence of the embolic source of the TIA on the platelet scintigram. Our success in demonstrating the embolizing aneurysm distinctly by platelet scintigraphy can be attributed to the fibrin-platelet thrombus, which was as yet unorganized, within the aneurysmal sac in the absence of atherosclerotic changes in the arteries.

References


Key Words • cerebral aneurysm • radionuclide imaging • vertebral artery
Positive platelet scintigram of a vertebral aneurysm presenting thromboembolic transient ischemic attacks.

M Maruyama, T Asai, Y Kuriyama, T Sawada, J Ogata, T Nishimura and T Omae