Multiple Cerebral Infarcts Caused by Emboli from Carotid Atheroma

—A Case Confirmed by Indium-111 Platelet Scintigraphy—

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Abstract

A 47-year-old male presented with frequent transient ischemic attacks (TIAs) of weakness of the left upper extremity. Computed tomography demonstrated multiple infarcts in the right cerebral hemisphere. Cerebral angiography showed marked atheromatous changes at the siphon of the right internal carotid artery, characterized by wall irregularities and heterogeneous filling. Indium-111-labeled platelet scintigraphy demonstrated abnormal tracer accumulation at the right carotid siphon. Following right carotid artery ligation, this abnormal platelet deposition was resolved and no further TIA was experienced. This suggests that the release of microthrombi from thrombogenic carotid atheroma contributes to embolic stroke.

Key words: indium-111 platelets, transient ischemic attack, carotid atherosclerosis, cerebral infarction

Introduction

Carotid atherosclerosis is a common cause of cerebral ischemia.⁵⁾ Platelets aggregate and adhere at the exposed connective tissue of injured endothelium.¹⁾ Atherosclerosis and thrombus formation are thus triggered by interaction between platelets and the vessel wall,³⁾ and microemboli released from the atherosclerotic lesions cause cerebral transient ischemic attacks (TIAs).⁴⁾ Clinically, conventional computed tomography (CT) and cerebral angiography only demonstrate anatomical alterations in the brain or vessel wall and do not assess the dynamics of the thrombotic process *in vivo*. The relationship between the thrombogenicity of the lesion responsible for the embolic stroke and the symptoms has not been thoroughly investigated.

Indium-111 platelets can be used as a reliable marker of endogenous platelet distribution *in vivo*.⁶⁾

We report a case of embolic stroke in which the potential source of microemboli was visualized well by indium-111 platelet scintigraphy.

Case Report

A 47-year-old male experienced frequent right retro-orbital pain in February, 1988. CT scans obtained at another hospital on March 7 showed a low-density area in the right frontal lobe (Fig. 1 *left*). Since then, he suffered several episodes of TIA characterized by left upper extremity weakness for a few hours, and gradually developed visual disturbance. Repeat CT scans taken on March 24 demonstrated a new low-density area in the right temporoparietal region (Fig. 1 *right*). Embolic stroke in the right cerebral hemisphere was suspected, so he was referred to The Center for Adult Diseases, Osaka on April 4.

The neurological examination on admission showed left upper extremity weakness and left lower

Fig. 1 *left*: Initial CT scans, demonstrating a low-density area in the right frontal lobe (*arrow*). *right*: Repeat CT scans 17 days later, showing a newly developed cerebral infarction in the ipsilateral temporoparietal region (*arrow*).

quadrant homonymous hemianopsia. Electro- and echocardiograms were normal. A right carotid angiogram showed marked atheromatous changes at the siphon of the internal carotid artery, characterized by arterial wall irregularities and heterogeneous filling (Fig. 2). Four-vessel angiography revealed no other abnormal lesions. The blood supply to the right anterior cerebral artery (ACA) and middle cerebral artery (MCA) territories was from the left ACA through the anterior communicating artery during compression of the right common carotid artery for 60 minutes (Matas test). Cerebral perfusion images with iodoisopropylamphetamine (I-123 IMP) demonstrated decreased activity in the right MCA territory. Electroencephalograms demonstrated no abnormal change during the Matas test. On April 10, indium-111 platelet scintigraphy of the head, neck, and upper thorax was performed by the method of Heaton *et al.*²⁾ Forty-eight hours after injection of 500 μ Ci of labeled platelets, the anterior and bilateral lateral views showed abnormal platelet deposition at the right carotid siphon (Fig. 3 *upper*). Clinically, we suspected embolic stroke due to the release of microthrombi from the carotid atheroma.

Antiplatelet drugs were not administered to prevent the risk of uncontrollable bleeding during

Fig. 2 Selective right carotid angiogram, showing irregular narrowing and multiple intraluminal filling defects (*arrow*) at the carotid siphon.

Fig. 3 Unprocessed anterior and right lateral views (200,000 counts/view) of indium-111 platelet scintigrams obtained 48 hours after injection. *upper*: Preoperative scintigrams, clearly demonstrating increased activity at the right carotid siphon (*arrow*). *lower*: Postoperative scintigrams, showing no abnormal activity at the carotid siphon.

surgery. On April 29, 1988, an extracranial-intracranial bypass followed by right carotid artery ligation at the bifurcation was performed. The postoperative course was uneventful, and no TIA has

since occurred. A Doppler sonogram of the right ophthalmic artery 3 months after surgery demonstrated the same antegrade flow as seen preoperatively. Indium-111 platelet scintigrams taken on July 9, 1988 revealed no abnormality in the bilateral extra- and intracranial regions (Fig. 3 lower). Cerebral perfusion images with I-123 IMP taken on July 16 showed no improvement. Selective external carotid angiograms demonstrated the patent anastomosis but insufficient blood flow through the donor artery. The right MCA territory was supplied mainly by the left ACA through the anterior communicating artery.

Discussion

Microthrombi released from carotid atheroma are important in the induction of TIA.⁴⁾ Embolic sources are difficult to identify by arteriography, as cerebral angiograms do not provide information about thrombogenicity on vessel walls. In the present case, indium-111 platelet scintigraphy visualized the active thrombi in the cerebral vessels easily, and the symptoms correlated well with the results of platelet scintigraphy. Comparison of the cerebral angiograms and indium-111 platelet scintigrams achieved a more precise evaluation of thrombogenicity.

In the present case, the right carotid siphon was identified as the embolic source of the multiple cerebral infarcts for two reasons. Firstly, when frequent TIAs occurred there was considerable platelet deposition at the carotid siphon, which ceased postoperatively when TIA no longer occurred. Secondly, there was an atheromatous lesion characterized by wall irregularities and heterogeneous filling at the same site of platelet deposition. The TIAs were not considered to be hemodynamic, because the postoperative cerebral perfusion images with I-123 IMP revealed no improvement, and the preoperative Matas test induced no TIA or electroencephalographic change. Moreover, cross circulation through the anterior communicating artery was demonstrated on both the pre- and postoperative angiograms.

We preferred surgical intervention to conservative therapy with antiplatelet drugs such as aspirin.⁷⁾ The

marked platelet deposition and atypical angiographic findings at the carotid siphon suggested severe atheromatous changes, thrombus formation, and a high risk of microthrombi release. Severe neurological deficits would occur due to newly developed and the previous multiple cerebral infarcts if antiplatelet therapy failed to prevent thrombus formation and ischemic attacks. Moreover, the surgical risk was considered to be low.

This is the first report of indium-111 platelet scintigraphy visualizing the intracranial microemboli as a potential source of TIA. In addition to detecting the atherosclerotic lesion, the indium-111 platelet scintigraphy detected postoperative changes in platelet behavior.

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