

Embollic Stroke Following Carotid Radiation Angiopathy Demonstrated with I-123 IMP Brain SPECT

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Radiation angiopathy was developed by the process of accelerated atherosclerosis at the site of irradiation. The case of a 44-year-old man with right hemiparesis showing a high signal intensity in the left semioval center on MRI and a defect in the left temporo-parietal area with subsequent filling-in with I-123 IMP brain SPECT is reported. Digital subtraction angiography showed typical radiation angiopathy with ulceration in the left common carotid artery. Twenty-four years previously, he underwent curative irradiation of a neck mass that revealed Hodgkin's disease by biopsy. The emboli formed at the site of radiation-induced angiopathy and caused cerebral infarction. The perfusion abnormality in the territory of the embolic artery was detected by I-123 IMP SPECT.

Long-term survivors of neck irradiation are at high risk for the development of carotid arterial disease and should be watched carefully.

SUCCESSFUL RADIATION THERAPY for neck cancer has increased the survival time of afflicted patients but has also put them at risk for radiation-induced carotid arteriosclerosis (1,2). The stroke rate in the neck irradiation population is reported to be higher than that of the control population (3). Several decades usually pass between radiation therapy and the onset of neurologic symptoms (4). I-123 IMP SPECT has been applied to visualize regional cerebral blood flow (rCBF) in the early image. Filling-in of I-123 IMP in the delayed image may reflect a viable area (5,6). We describe a case of cerebral infarction where the perfusion abnormality was detected by I-123 IMP SPECT due to emboli that arose at the site of radiation-induced atherosclerosis.

Method

Brain SPECT was performed with a rotating gamma camera (STARCAM, 400AC/T, General Electric) from 64 projections into a 64 × 64 matrix, using a general all-purpose collimator with a sampling time of 20 seconds. For 15 minutes before and after injection, 4.5 mCi (166.5 MBq) of I-123 IMP was administered

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intravenously in supine patients with their eyes closed by a mask. The early brain SPECT was started at 30 minutes and the delayed brain SPECT was done 4 hours after the tracer was injected. All data were corrected for an attenuation of 0.1 cm^{-1} , and tomographic data were reconstructed using a filtered back-projection algorithm. Each section was 6 mm thick.

Case Report

A 44-year-old man was admitted because of a heavy sensation in his right hand when he used his electric shaver in the morning. He gradually developed right hemiparesis but did not show dysarthria or loss of consciousness. Twenty-four years previously he had a left neck mass, which was excised for biopsy and diagnosed as Hodgkin's disease. The patient received 40 Gy (4000 rad) of 200 kVp to the left neck area. Fourteen years previously, he was noted to be hypertensive with a blood pressure of 150/100 mmHg but denied having had episodes of transient cerebral ischemia.

On physical examination, the skin overlying the larynx and adjacent muscles showed typical postirradiation pallor and atrophy. A bruit was audible in the left neck area. His blood pressure was controlled at 110/70 mmHg. Digital subtraction angiography (DSA) (Angiotron, Siemens Co.) showed an irregularity of the anterior wall in the long segment of the left common carotid artery and normal intracerebral arteries including the right internal carotid artery. The transaxial section of the T2 weighted image on MRI showed a high intensity area in the left semioval center (Fig. 1). I-123 IMP SPECT showed a defect in the left temporo-parietal area of the early image, with subsequent filling-in of the delayed image (Fig. 2).

Discussion

Radiotherapy causing selective injury to the internal elastic membrane is followed by the development of intimal

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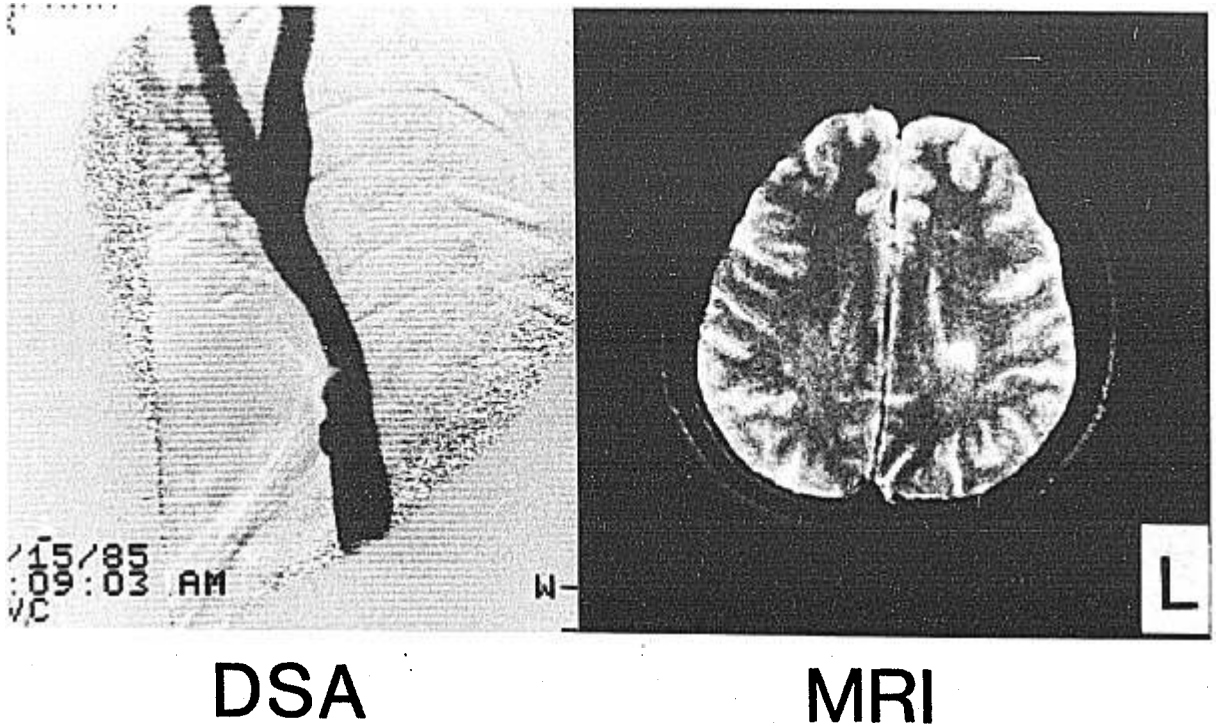


Fig. 1. Left: Left carotid arteriography with digital subtraction angiography (DSA) shows the irregularity, with ulceration, of the anterior wall of the left common carotid artery. Right: Transaxial slice of T2-weighted image with MRI reveals the high intensity area in the left semioval center.

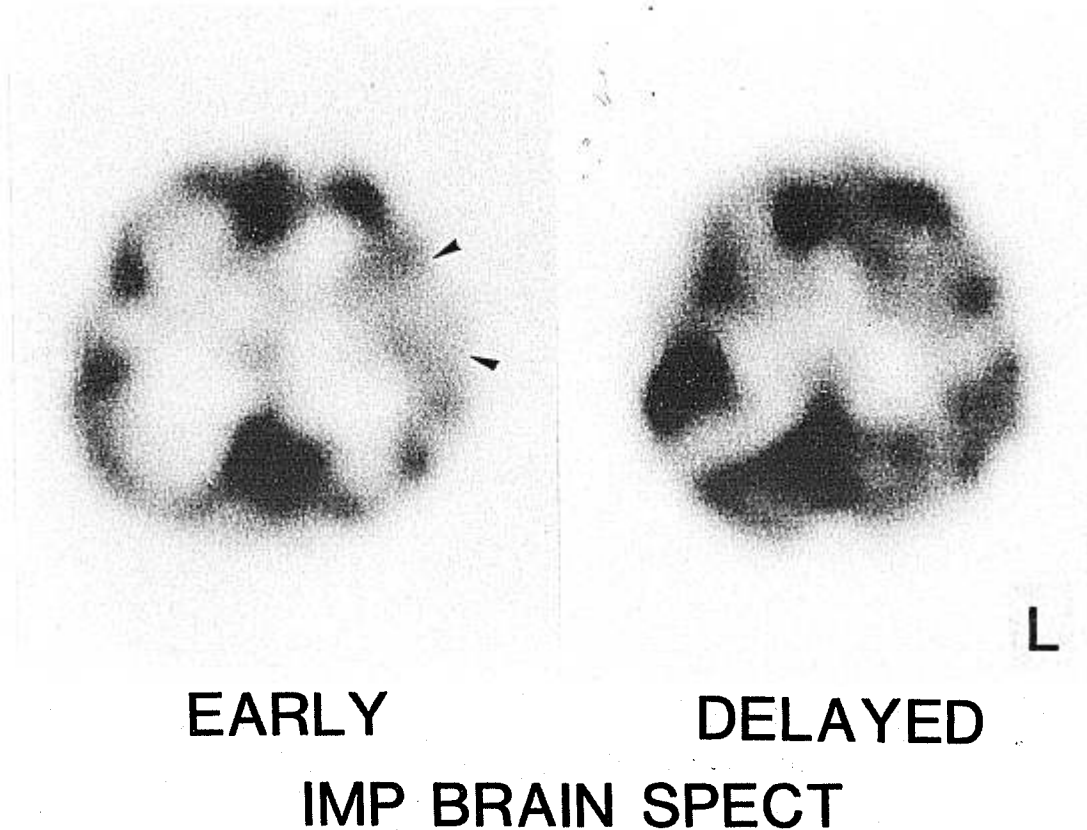


Fig. 2. I-123 IMP brain SPECT reveals the perfusion defect (arrowhead) in the left temporo-parietal area in the early image (Early) and the subsequent filling-in in the delayed image (Delayed).

fibrosis and plaque formation (7). The most common form of radiation-induced carotid damage is the late development of atheromatous occlusive carotid disease in the territory covered by the radiation port (4). Radiation angiopathy in neck irradiation is clinically important because it causes stroke. Many authors have noted unusual radiographic findings in the cases of radiation-induced carotid angiopathy, such as unilaterally disproportional involvement of the common carotid, unusually long lesions confined to the field of radiation, and those involving younger cancer patients (8). Cerebral infarction by the emboli from the radiation angiopathy was thought to occur in the same manner as that from the site of naturally occurring atherosclerosis. Atherosclerotic plaques tend to ulcerate, thus presenting a raw surface that becomes the site for adhesion of platelets. These collect on the surface, and thrombus formation begins. The thrombus, in turn, may break off and become an embolus, shutting off blood supply distal to that point (9). Since obstruction of intracerebral arteries was not observed angiographically, the embolus was suspected to have lysed after it had arisen from the common carotid artery, and traveled to the branch of left middle cerebral artery. I-123 IMP SPECT revealed a perfusion defect in the temporo-parietal area in the early image. This area showed filling-in of I-123 IMP on the delayed image, implying viability. Displacement of thyroid cartilage

when swallowing, with mild dysphagia, may have derived from radiation injury to the vagus nerve.

Even though they are unlikely to develop strokes, long-term survivors of neck irradiation should be watched carefully for the subsequent development of carotid arterial disease.

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