Radiation-induced Cerebrovasculopathy of the Distal Middle Cerebral Artery and Distal Posterior Cerebral Artery

—Case Report—

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Abstract

A 15-year-old girl underwent partial removal of a pituitary adenoma followed by local irradiation of the brain with a total of 70 Gy through two lateral opposing ports. Twenty years later, she experienced frequent transient ischemic attacks with left sensory disturbance. Cerebral angiography revealed stenoses of the right distal middle cerebral artery (MCA) and the right distal posterior cerebral artery without net-like vessels. There was a severe decrease of vasoreactivity in the right hemisphere. Right superficial temporal artery (STA)-MCA anastomosis was performed. Her neurological deficits were resolved and perfusion reserve capacity had markedly improved 6 months later. We recommend STA-MCA anastomosis in such cases.

Key words: radiation, cerebrovasculopathy, distal middle cerebral artery, distal posterior cerebral artery, treatment

Introduction

Recent advances in the treatment of brain tumors have allowed many patients to have a useful, long life. However, radiation-induced complications such as cerebrovasculopathy are an important problem in such long survivors, so the damage induced by radiation therapy must be considered carefully.

Many cases of radiation-induced cerebrovasculopathy have been reported, but none presented detailed findings of stenoses in the distal middle cerebral artery (MCA) (M2) and distal posterior cerebral artery (PCA) (P2). We treated a patient with radiation-induced M2 and P2 stenoses, using bypass surgery.

Case Report

A 35-year-old female presented with bitemporal hemianopsia and panhypopituitarism when she was 15 years old. She was admitted to our hospital, and pneumoencephalography revealed a suprasellar mass lesion. Right internal carotid angiography showed no stenosis or occlusion of the cerebral vessels, especially the M2 portion (Fig. 1 left). She underwent right frontotemporal craniotomy for partial removal of the tumor on November 25, 1975, and again on February 20, 1976. Histological examination showed the tumor was a chromophobe adenoma. She was treated 40 Gy and 30 Gy (total 70 Gy) local irradiation to the brain through two lateral opposing ports after these procedures. She received no further treatment, except for supplements to control hormonal levels in the normal range, during the next 20 years.

She complained of dysesthesia of the left hand and around the mouth in October 1995. Magnetic resonance (MR) imaging demonstrated multiple infarctions in the right hemispheric white matter and the right basal ganglia, and bitemporal radiation-induced encephalopathy and cystic change of the intrasuprasellar lesion (Fig. 2). Right internal carotid angiography and right vertebral angiography revealed stenoses at the M2 and P2, which were con-
under a diagnosis of transient ischemic attacks (TIAs). However, left hemiparesis appeared and reached an advanced stage in January 1996. General examination revealed moderate obesity but no signs of hypertension, diabetes mellitus, hyperlipidemia, or abnormal levels of lipoprotein(a), protein C, protein S, or anti-cardiolipin antibody. She had no history of meningitis or other diseases. Therefore, we suspected that her progressive TIAs were due to radiation-induced cerebrovasculopathy. Conservative therapy was not effective and there were no net-like vessels detected. Therefore, we decided to perform bypass surgery. The frontal branch of the right superficial temporal artery (STA) had been sacrificed when the tumor was removed, but single right STA-MCA anastomosis could be performed. The artery supplying the frontal lobe, which appeared normal, was selected as the recipient artery. Observation of the M1 during operation was too risky. Her postoperative course was uneventful and she was discharged without neurological deficits in July 1996. Six months after surgery, right external carotid angiography showed good filling of the MCA via the anastomotic artery (Fig. 4). Tc99m-I-IMP SPECT demonstrated marked improvement of the perfusion reserve capacity in the right hemisphere (Fig. 3 lower row). We plan to perform anastomotic surgery for stenosis of the P1 in the future.

**Discussion**

The etiology of cerebrovasculopathy in patients with brain tumors can be classified as follows: Compression by a slowly growing tumor, secondary arterial
changes induced by radiation therapy for a tumor, association with neurocutaneous syndrome, and coincidence.\textsuperscript{6} Occlusive cerebrovascularopathy might develop in one or a combination of these four ways. In our case, there was no contributing arteriosclerotic factor, tumor regrowth, neurocutaneous syndrome, or coincident moyamoya disease. Normal angiographic findings were obtained at the initial examination including the M\textsubscript{2} and P\textsubscript{2}, and there were no signs of inflammatory disease such as vasculitis. MR imaging indicated that both stenotic sites at the M\textsubscript{2} and P\textsubscript{2} were within the ports of the previous irradiation. Therefore, we concluded that the observed M\textsubscript{2} and P\textsubscript{2} stenoses were long-term complications of the radiation therapy administered to treat pituitary adenoma.

Many patients with intracranial tumors have develop radiation-induced cerebrovascularopathy.\textsuperscript{1-5,7-11} Patients below the age of 15 years who received about 50–70 Gy irradiation tend to develop cerebrovascularopathy. The most frequent locations are in the terminal parts of one or both carotid arteries or its branches: the ICA in 86% of cases, the MCA in 56%, the ACA in 53%, the PCA in 9.8%, and the basilar artery in 2.4%.\textsuperscript{11} Stenotic and occlusive changes are also frequent in the circle of Willis, the terminal parts of the ICA, the proximal portions of the MCA, and the ACA.\textsuperscript{11} The M\textsubscript{2} and P\textsubscript{2} were apparently not involved in any of the previously reported cases. In the present case, cerebrovascularopathy did not develop at the ICA or proximal MCA, and the stenoses were recognized only at the surgical site. Although the reason for this is unknown, we suggest that both exposure to irradiation and surgery may induce stenosis. For example, the contributing factors may be inflammatory vasculitis due to surgical insult, increased x-ray permeability due to skin and/or muscle atrophy, and the craniotomy procedure. However, none of these factors explain the development of the P\textsubscript{2} stenosis.

Vasorconstrictive surgery such as encephalo-duro-arterio-myo-synangiosis combined with STA-MCA anastomosis may be beneficial in pediatric patients with radiation-induced cerebrovascularopathy presenting with net-like vessels.\textsuperscript{12} Pediatric patients with net-like vessels were doing well without neurological deficits after encephalo-duro-arterio-synangiosis.\textsuperscript{8} These net-like vessels are more frequently observed in children than in adults\textsuperscript{2,3} and in the absence of these net-like vessels.
indirect vasoreconstructive surgery might not produce beneficial neovascularization in patients with radiation-induced cerebrovasculopathy. Our patient did not harbor these vessels, so we performed STA-MCA anastomosis. This treatment resulted in improvement of her neurological deficits and vasoreactivity.

We suggest that STA-MCA anastomosis is useful in adults with radiation-induced cerebrovascularopathy without net-like vessels. We also suggest that a skin incision that preserves the STA and/or occipital artery may be important at craniotomy in children who will receive more than 50 Gy irradiation after surgery for intracranial tumors because they tend to develop cerebrovascularopathy.

References


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