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Triology of neurosurgical complications after radiotherapy for nasopharyngeal carcinoma

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Abstract

Background: Radiotherapy is an effective treatment for NPC. With improved survival, radiation-induced neurological complications are being diagnosed more commonly. We presented a patient with a trilogy of radiation-induced pathologies after radiotherapy for NPC. The diagnostic and surgical implications are discussed.

Case Description: A 57-year-old man, previously irradiated for NPC, presented with mental confusion and was found to have radiation-induced carotid stenosis and bitemporal lobe necrosis on MR imaging. His condition deteriorated suddenly a year later, and a gliosarcoma was found to have developed within the area of right temporal lobe necrosis.Tumor removal was complicated by injury to the MCA branches, causing basal ganglion infarction. This was likely because of a combination of technical error and arterial insufficiency secondary to radiation-induced arterial stenosis.

Conclusions: In patients with known temporal lobe radiation-induced necrosis, alternative diagnosis such as gliosarcoma should be considered when there is sudden clinical deterioration. Radiation-induced carotid stenosis may reduce the safety margin during surgery. Preoperative carotid screening may be indicated.

Keywords: Carotid stenosis; Temporal lobe necrosis; Gliosarcoma; Radiotherapy; Nasopharyngeal carcinoma

1. Introduction

External radiation therapy is an effective treatment for NPC. Neurological complications secondary to radiotherapy for NPC have been reported, including carotid artery stenosis and pseudoaneurysm, radiation-induced dementia, temporal lobe necrosis, and brain tumors [3,6,9,11,12,14,19]. We report a patient who had previously received radiotherapy for NPC and who later presented with symptoms of cognitive dysfunction and triple pathologies of radiation-induced carotid stenosis, temporal lobe necrosis, and gliosarcoma. The clinical and pathologic findings and their clinical implications are discussed.

2. Case report

2.1. Clinical presentation

The patient was a 57-year-old Chinese man who had external irradiation for NPC 30 years ago. The initial treatment plan (radiation dose and field) was unfortunately not available for review. He presented in 2004 with mental confusion. There were no symptoms suggestive of raised intracranial pressure or vascular insufficiency. On examination, he was found to have depressive mood and poor short-term memory. There were bilateral carotid bruits but no focal neurological deficit. Carotid Doppler ultrasound revealed stenosis of bilateral CCA and ICA.

Abbreviations: ACA, anterior cerebral artery; CAM 5.2, anticytokeratin monoclonal antibodies clone CAM 5.2 (Becton Dickinson, San Jose, Calif); CCA, common carotid arteries; DWI, diffusion weighted images; EBER, Epstein-Barr virus-encoded early small RNA; EBV, Epstein-Barr virus; FLAIR, fluid-attenuated inversion-recovery; GFAP, glial fibrillary acidic protein; ICA, internal carotid arteries; MCA, middle cerebral artery; MEP, motor-evoked potential; MFH, malignant fibrous histiocytoma; MIB-1, monoclonal antibodies for Ki-67 labeling index; MNF116, anti-cytokeratin monoclonal antibodies clone MNF116 (DAKO, Carpinteria, Calif); MR, magnetic resonance; NAA, *N*-acetyl aspartate; NPC, nasopharyngeal carcinoma; T2W, T_2-weighted.

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2.2. Magnetic resonance features

Magnetic resonance angiography showed long segments of stenosis and poststenotic dilatations of bilateral CCA and proximal ICA (Fig. 1). Mild stenosis was also found in the cavernous portions of the right ICA, MCA, and ACA. The basilar artery was tortuous with mild stenosis. The vertebral arteries were normal. The overall picture was compatible with radiation-induced carotid artery stenosis.

Initial MR imaging of the brain performed in 2004 revealed bilateral temporal lobe lesions that were hyperintense on FLAIR and T2W sequences. There was no pressure effect or contrast enhancement. No abnormal signal change was seen on DWI. The features were suggestive of bilateral postirradiation temporal lobe necrosis (Fig. 2).

Endovascular stenting for the carotid stenosis was planned but was delayed because of the patient's uncertainty about the procedure. His condition, however, deteriorated rapidly in mid-2005, with increasing drowsiness and mental confusion. Reassessment MR imaging, performed 20 months after the initial scan, revealed a contrast-enhancing lesion in the right temporal lobe measured 5.5 cm in maximum dimension. It was iso- to hypointense on T1W, and iso- and hyperintense on T2W sequences. Low signal intensity areas were seen on the postcontrast images suggestive of necrosis. There was marked surrounding vasogenic edema (Fig. 3). Magnetic resonance spectroscopy showed loss of NAA peak and elevated choline and lactate peaks. The features were compatible with a newly developed malignant tumor in the right temporal lobe. The left temporal necrosis remained unchanged. Endoscopic examination of the nasopharynx did not reveal tumor recurrence. There was no clinical evidence of metastasis from the NPC.

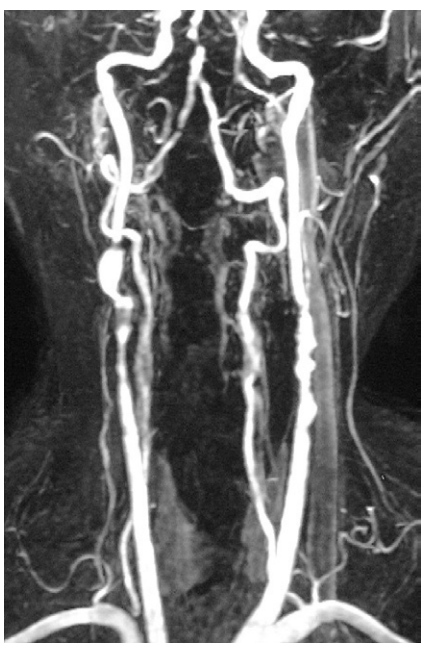

Fig. 1. Magnetic resonance angiography showing long segments of stenosis and poststenotic dilatations of bilateral CCA and proximal ICA.

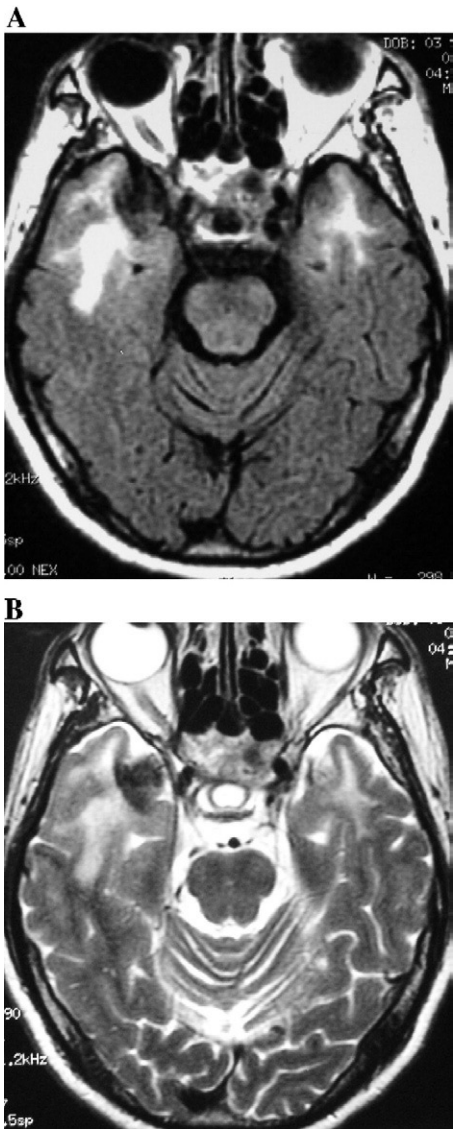

Fig. 2. Initial MR images showing bilateral temporal lobe radiation-induced necrosis that were hyperintense on FLAIR (A) and T2W (B) sequences.

2.3. Intervention

Endovascular stenting of the carotid arteries was withheld. The patient underwent excision of the right temporal lobe tumor. Intraoperatively, a firm, yellowish, infiltrative, and avascular intraparenchymal tumor was found. There was macroscopic invasion into the surrounding dura and skull base. During dissection of the superior aspect of the tumor, there was a sudden deterioration in the patient's MEP. Further tumor removal was abandoned. Postoperatively, the patient developed a left hemiplegia. Magnetic resonance DWI sequence confirmed recent infraction in the right internal capsule that was likely due to injuries to the MCA branches during surgery. The patient made a slow recovery with an unresolved left hemiplegia. In view of the poor functional and oncological prognosis, his family declined further treatment. He was transferred to a hospice for palliative care.