Radiation-induced bilateral cystic temporal lobe necrosis: reversal of memory deficit after fenestration and internal shunting

Case report

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The authors report a case in which bilateral cystic temporal lobe necrosis developed after treatment of nasopharyngeal lymphoepithelioma with 7000 cGy of external beam radiation. The patient presented with an isolated memory deficit that was documented by neuropsychological testing. After fenestration and internal shunting of both cysts, there was striking resolution of the lesions and of the memory deficit.

KEY WORDS: radiation necrosis • cyst • temporal lobe compression • memory deficit • nasopharyngeal carcinoma

The incidence of late bilateral temporal lobe necrosis after radical radiation therapy for nasopharyngeal carcinoma was 1% in a large series reported by Lee, et al. Administration of corticosteroids is the initial treatment of choice for symptomatic lesions, but progressive neurological deficits that persist despite corticosteroid treatment may require surgery. Unilateral surgical removal of necrotic and cystic tissue may reverse rapidly progressing symptoms, but long-term outcome is often poor. The case of a patient who developed bilateral temporal cystic radiation necrosis and an associated severe memory deficit is reported. The memory deficit resolved dramatically after successful fenestration and internal shunting of both cysts.

Case Report

This 55-year-old man underwent treatment of a nasopharyngeal lymphoepithelioma with 7000 cGy of external beam radiation therapy in 1977. He was irradiated with daily fractions of 150 cGy to the tumor, five times per week. He was well until 1986, when he first noted difficulty with memory. Neuropsychological testing found only subtle deficits without evidence of an organic pathology. Subjectively, the patient's memory continued to decline, and in January, 1987, a magnetic resonance image was obtained. Low-intensity bilateral signals in the inferior temporal lobe were consistent with initial signs of late-delayed radiation-induced injury. Three months before admission to our service, his memory deficit accelerated rapidly and he developed an agitated depression that did not respond to corticosteroids.

Examination. Routine neurological testing showed slowed, perseverative speech and very slow fine motor skills, but no focal deficit. He was evaluated with the Neurobehavioral Cognitive Status Examination, Rey and Taylor Complex Figure Test, logical memory from the Wechsler Memory Scale, Alberts' Famous Faces Test, and the Trail Making Test. The results showed a striking isolated memory deficit and a mild anomia. Memory was significantly impaired for verbal and nonverbal tests, but the remainder of cognitive functions were generally intact. A computerized tomography (CT) scan showed the presence of a 6 × 4.5 × 3.5-cm cyst within each temporal lobe (Fig. 1).

Operation. The slightly larger cyst on the right side was approached first. After the right temporal lobe was exposed by craniotomy, a transcortical sonogram was used to locate the cyst within the temporal lobe just anterior to the temporal horn of the lateral ventricle. A 2-cm cortical incision was made in the anterior aspect of the middle temporal gyrus. The cyst was opened, a
portion of the cyst wall was taken for histopathological analysis, and 60 cc of yellow proteinaceous fluid was drained from the cyst. A thin layer of gliotic ependyma separated the cyst from the ventricle. A 1 x 1-cm fenestration was made in this layer to establish communication between the cyst and the lateral ventricle, and an internal shunt was placed between the anterior part of the cyst and the atrium of the ventricle to maintain communication. The shunt was fashioned from two ventricular catheters joined by a straight connector and was fixed to the ependymal wall by a Weck clip that incorporated a suture over the straight connector. A postoperative CT scan showed that the cyst had collapsed (Fig. 2 left). Ten days later, a similar procedure was performed on the left side; a second CT scan showed that both cysts had collapsed (Fig. 2 right).

Postoperative Course. Microscopic examination of the biopsy specimens showed astrogliosis, hyalinization of vessels, and areas of focal necrosis; there was no evidence of tumor. During the 10 days between the first and second procedures, there was no change in the patient's symptoms; however, during the first several weeks after the second procedure, both the patient and his family noted progressive resolution of his memory deficit and improvement of his motor skills. The neuropsychological tests were repeated 4 months after surgery, and showed striking recovery of memory, particularly of verbal memory; his improvement stabilized by 6 months after surgery.

Discussion

Nasopharyngeal carcinoma is highly invasive, and extensive microscopic submucosal spread of tumor frequently occurs beyond the macroscopic tumor.\(^5\) The efficacy of radiation therapy for the treatment of nasopharyngeal tumors is well documented. Because the sphenoid sinus and middle cranial fossae are common sites of local extension, however, the anterior portions of both temporal lobes must be included within the irradiated field.\(^7\) While neither the optimum dose nor the best schedule for conventional fractionation therapy of nasopharyngeal carcinoma has been determined, it is believed that a mean tumor dose of 6000 cGy of radiation delivered in daily 200-cGy fractions is inadequate in most cases;\(^5,7\) patients with this disorder are frequently irradiated with total doses greater than those generally considered to be safe for the treatment of intracranial tumors.\(^5,8\)

It is necessary to distinguish between cerebral necrosis caused by radiation therapy of nasopharyngeal carcinoma and primary intracranial glial neoplasms. The presence of intracerebral gliomas and the frequent use of chemotherapy to treat them may accelerate and amplify the detrimental effects of radiation on the brain;\(^13,16,18\) these effects include cerebral edema, demyelination, hemorrhage, and late radiation necrosis.\(^3,8,11,13,18\) Radiation necrosis may be tumoral or peritumoral, and is usually microcystic,\(^2\) although macrocystic necrosis is known to occur. Cystic or solid necrosis can cause a mass effect that requires surgery.\(^2,16\) Delayed cyst formation after external beam radiation therapy of malignant gliomas is often associated with tumor recurrence, and surgical excision of these lesions usually produces only transient clinical improvement.\(^16\) In contrast, the late occurrence of cerebral cysts after radiation therapy for intracranial tumors is generally not associated with tumor progression or recurrence.\(^4,7\)

Since the first report in 1930 of late cerebral necrosis after radiation therapy of extracranial neoplasms,\(^3\) 180 cases have been reported, 115 of which occurred after treatment for nasopharyngeal carcinoma.\(^1\) In the series...
Radiation-induced cystic temporal lobe necrosis reported by Lee, et al., over 9600 patients with nasopharyngeal carcinoma were irradiated with daily fractions greater than 200 cGy. The incidence of late cerebral necrosis was 1.03% in this series. While the mean follow-up period was not reported, the mean latent interval between irradiation and diagnosis of necrosis was 4 years. Sixteen patients suffered bilateral cystic degeneration of the temporal lobes similar to the clinical picture reported here. Five patients had surgery for a single, unilateral lesion; after a brief remission of symptoms, all patients had a poor outcome. The use of corticosteroids as adjunctive treatment has been emphasized by these and other authors but, as in the case of the patient reported here, corticosteroid therapy is often of little or no value.

This patient presented with classic symptoms of bilateral temporal lobe injury with marked impairment of both verbal and nonverbal memory skills required for the acquisition and storage of new information, despite preservation of premorbid intellectual skills. This pattern is seen frequently in patients with bilateral traumatic temporal lobe injury caused by viral encephalitis or after bilateral temporal lobe removal for the treatment of epilepsy. In this patient, the dramatic recovery of function after decompression emphasizes the extent to which temporal lobe compression can reversibly reduce memory performance.

While the follow-up period for this patient is short, the low morbidity associated with the staged bilateral procedure and the excellent clinical recovery and lack of radiographic evidence of recurrence are encouraging. In the absence of evidence of tumor progression, cystic radiation necrosis can be treated as a mass lesion, and it may not be necessary to excise the cyst if fenestration and shunting into ventricles are possible.

References

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