Short report

Radionecrosis of the brain presenting as a mass lesion: a case report

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SUMMARY A case of radionecrosis of the brain in a patient previously treated for squamous cell carcinoma of the scalp is presented. Cerebral angiography revealed a mass effect primarily in the right frontal area. Computed tomography showed extensive hemispheric oedema. A frontal lobectomy was performed, and the recovery was excellent. The lower limit for brain tolerance for irradiation is about 5000 rads given in five weeks. This dose should not be exceeded when extracranial lesions are treated. Although computed tomography cannot diagnose brain necrosis, specifically, it should, along with the clinical history, be very helpful.

Radionecrosis of the brain has been reported a number of times in the literature, and has been well reviewed by Kramer (1968). The doses per treatment and the total dose delivered have varied considerably. Radionecrosis occurs most often in patients who had been treated for intracranial neoplasms. However, there have been cases reported for patients treated for extracranial lesions (Pendergrass *et al.*, 1940; Kramer, 1968; Takeuchi *et al.*, 1976). Three of these patients presented with space occupying masses (Lowenberg-Scharenberg and Basset, 1950; Dugger *et al.*, 1954; Takeuchi *et al.*, 1976). We are describing an additional case of brain necrosis which presented as a mass lesion after a patient had been treated for squamous cell carcinoma of the scalp. A unique aspect of this case report of radiation necrosis of the brain is the preoperative demonstration by computed tomography of the area of cerebral oedema and necrosis. The availability of detailed information about this patient, computed tomographic demonstration of the lesion, and the infrequent initial presentation of radionecrosis as a mass lesion are significant and deserve attention.

Case report

This 49 year old white male had an excision and grafting of a squamous cell carcinoma of the right forehead. Six months later there was recurrence at the inferior margin. On presentation to the Department of Radiation Therapy a lesion 40 mm in diameter with thickness of about 15 mm was noted. Therapy was initiated on a 100 kV unit using a half value layer of 2 mm of aluminium. The patient received two doses of 400 rads to the surface using a direct 55×75 mm field. He was then switched to the ⁶⁰Cobalt unit and treatment was continued at 467 rads per day for an additional 5604 rads. A direct 60×70 mm field was used with a 5 mm of bolus.

One year later there was evidence of recurrent disease and the area was excised. Repeated attempts at grafting were unsuccessful. Eighteen months after the course of radiation therapy, the patient developed a change of personality, left sided weakness, headaches, nausea, and vomiting. On admission, the patient showed a 65 mm scalp defect with exposed bone in the right frontal area. Routine blood studies were normal except for a raised sedimentation rate of 52 mm/h. Skull films revealed decreased density in the right frontal bone. The EEG showed a pronounced disturbance in the right frontal area with slow and interposed sharp waves. A right carotid arteriogram indicated an avascular mass effect in the right frontal area

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with a pronounced shift of the anterior cerebral artery. Computed tomography showed a mass effect in the same area with extensive oedema in the right cerebral hemisphere and a shift of the midline to the left (Figure).

Surgery was performed for diagnosis, decompression of the scalp defect, and removal of the diseased bone. A prefrontal lobectomy was elected for internal decompression and permanent diagnosis. A frozen section was not diagnostic. The immediate postoperative course was excellent, but an attempt to withdraw dexamethasone therapy resulted in increased lethargy and weakness of the left side. Dexamethasone was continued and these findings promptly disappeared. Three months later the patient stopped dexamethasone; his mental functioning and neurological examination were essentially normal.

The permanent sections showed extensive areas of parenchymal necrosis involving both grey and white matter with focal central calcifications, a focal fibrinoid change of the fibres, nuclear pyknosis, and a radiating border of relatively healthy parenchyma. Adjacent to the necrotic areas were extensive chronic white matter oedema, reactive astrocytic proliferation, and a peculiar pattern of oligodendroglial proliferation.

Figure Computed tomogram. Large area of decreased density is present in the right frontal-parietal region causing compression of the right lateral ventricle, and marked shift of both ventricles from right to left.

There was evidence of an extensive vascular destructive process, with severe fibrinoid necrosis of the vessel walls, enlarged occasionally vacuolated endothelial cells, and plump adventitial fibroblasts. The lumina were often completely occluded with fibrin thrombi; occasionally there were recanalisations. A few binucleated neurones were noted. We felt that the microscopic changes agreed with the diagnosis of radiation necrosis of the brain parenchyma.

Discussion

The reported tolerance of the brain to irradiation has varied from 5000 to 7000 rads given at doses of approximately 200 rads per treatment (Dugger *et al.*, 1954; Verity, 1968). However, less than 100 cases of radiation necrosis from a wide range of daily and total doses have been reported in the literature. Using the methods of Orten and Ellis (1973) and Goitein (1974) to convert this patient's treatment to an equivalent of 200 rads per day, and calculating the dose at surface, 20 and 40 mm depth, it was determined that this patient received an equivalent of 10 400, 7600, and 6000 rads respectively.

The symptomatology of radiation necrosis can appear as early as nine months or not until many years later. Although the patient may exhibit a slow, progressive central nervous system deterioration, the condition can also develop more rapidly. There are three cases in the literature which presented with mass lesions. Although radiation necrosis was considered in the differential diagnosis, surgery was indicated for a definitive diagnosis. This patient's response to frontal lobectomy was excellent.

Computed tomography (CT) demonstrated a focal area of decreased attenuation values consistent with oedema. This finding corresponded with the radiation necrosis and its adjacent chronic white matter oedema which were noted histologically. The CT findings alone cannot diagnose radiation necrosis specifically. Yet, when considered in conjunction with clinical observations, they should indicate this possibility.

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