Radiation injury of the brain

Introduction

Radiation therapy is the most common and often the most effective adjuvant treatment not only for malignant brain tumors but also for benign tumors such as pituitary adenoma. However, it may also cause clinically significant damage to normal nervous system structures with serious long-term neurological deficits, or may induce secondary tumors at the irradiated areas. For this review, we selected 11 papers concerning the various aspects of radiation-induced brain injury.

[1] Long-term evaluation of radiation-induced brain damage by serial magnetic resonance imaging


Information. Eight patients (range 7–57 years) with radiation-induced brain damage were followed with serial MR imaging for 27 to 96 months (mean 45 months). Three patients had received conventional radiotherapy (50–65 Gy), and five patients were treated with slit-beam moving therapy (40–60 Gy).

The radiation damage to the brain appeared as an enhanced lesion on T1-weighted MR images with Gd-DTPA from 3 to 30 months after undergoing radiotherapy (mean 12.8 months). In all patients a high signal intensity area on T2-weighted images preceded the emergence of the enhanced lesion. The volume and number of enhanced lesions continued to increase for 3–23 months (mean 10.3 months), and were accompanied by a mass effect in seven patients. The enhanced lesions subsequently regressed after a mean period of 19.3 months of neuroimaging aggravation (range 12–35 months). Two patients showed a relapse of the enhanced lesions with latent periods of 8 and 9 months, respectively. The nature of these relapsed lesions, however, was not confirmed histologically.

Analysis. The authors demonstrated the time course of radiation-induced brain damage by serial MR imaging, and also emphasized the importance of a prominent high signal intensity on the T2-weighted image preceding the emergence of the enhanced lesion, in order to accurately diagnose radiation damage in its early phase. Although the possibility of a relapse of delayed radiation brain damage was suggested, the true nature of such a “relapse”, which was not examined histologically, still remains unknown.

[2] Necrotizing brainstem leukoencephalopathy six weeks following radiotherapy


Information. A 71-year-old man underwent a transsphenoidal resection of a nonfunctioning adenoma, and also received postoperative radiotherapy of 4346 cGy in 24 fractions. Approximately 6 weeks following the
completion of radiotherapy, he developed blurred vision, nystagmus, diplopia, 6th and 7th cranial nerve palsies, and ataxia. He further developed severe respiratory distress and somnolence, and an MRI scan showed lesions in the thalamus, midbrain, pons, and medulla. There was no improvement in his neurological status and the patient eventually died approximately 12 months following the onset of his symptoms.

At autopsy, various-sized yellowish-white necrotic foci were found throughout the brain stem, thalamus, basal ganglia, and dentate nucleus. Microscopically, these multiple lesions showed demyelination with central necrosis, vacuolization, dystrophic calcification, and a surrounding myelin breakdown with macrophage influx. The vasculature was normal, with no vasculitis, thrombosis, fibrinoid necrosis or telangiectasia. The optic nerves and chiasm and the 6th, 7th, and 8th cranial nerves also showed severe myelin loss.

**Analysis.** This unique case best fits chronologically and pathologically into the “early delayed” category of post-radiation brain injury. Unlike the damage seen in the far more common late delayed radiation brain injury, this lesion is characterized by demyelination without vascular alterations. The primary lesions of this case involved the brain stem, which may be prone to early delayed postradiation injury.

**[3] Progressive late delayed postirradiation encephalopathy with Klüver-Bucy syndrome.**
**Serial MRI and clinico-pathological studies**

**Information.** A 33-year-old man received irradiation of 60 Gy/30 fractions with portals at bilateral temporal areas after transphenoidal surgery for a pituitary adenoma. About 3.5 years later he developed dysarthria and tonic clonic convulsions. He further developed several episodes of intermittent visual hallucination, left homonymous hemianopsia, intellectual dysfunction, unrestrained outbursts of rage, aphasia, hyperphagia, and bulimia. MRI revealed a pontine infarct and enlarged bilateral temporal lobes with a hyperintense signal at the cortical gyri on T2-weighted images.

A right temporal lobectomy was performed 6 years after undergoing radiation therapy. Histologically, the lesion was composed of brain softening with a honeycomb appearance and fibrinoid thrombi formation.

He later showed hypermetamorphosis, a strong oral tendency, and inappropriate behavior such as showing his genital organs in public. Seven years after the completion of radiation therapy his decline in intellectual functions had caused him to become totally dependent on others.

**Analysis.** Except for the uncertainty regarding the presence of psychic blindness, the present case fulfilled all features of the Klüver-Bucy syndrome. Involved lesions were found in both temporal lobes, and they are the anatomical structures involved in this syndrome. The clinical course of this case was unique in that the Klüver-Bucy syndrome developed with repeated stroke-like episodes following radiation therapy.

**[4] Progressive cerebral occlusive disease after radiation therapy**

**Information.** A 7-year-old girl with a craniopharyngioma underwent partial removal of the tumor and subsequently received cranial irradiation with opposed beams of $^{60}$Co. The total dose of irradiation was 50 Gy, with single fractions varying between 0.5 Gy and 2.0 Gy.

Thereafter, she did well until 13 years of age, when rapidly progressing deterioration of her memory began. One year later, recurrent sensorimotor transient ischemic attacks occurred, and CT showed an infarction in the left lentiform nucleus. At the age of 19 years, cerebral angiography showed severe changes in the internal carotid arteries distal to the ophthalmic artery, with an occlusion on the right and a severe stenosis on the left. An extensive network of collaterals consisting of transdural anastomoses linking the middle meningeal artery, the external occipital artery and the middle cerebral artery, and a transdural rete mirabile on the frontal basis were found. In addition, a basal cerebral rete mirabile was found within the basal ganglia on the left.

**Analysis.** This report describes the time course of the clinical and neuroradiological findings in progressive occlusive cerebral vasculopathy, with an abnormal network of collaterals resembling moyamoya disease, several years after radiation therapy in a young woman. Radiation-induced cerebral vasculopathy occurs most-