Letters to the Editor

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Radiation-Induced Cerebral Vasculitis Revisited

To the Editor:

In 1971, Kagan et al described a case of foam cell arteritis of the brain in a patient with Hodgkin’s disease. They suggested that the arteritis was a sequel of previous irradiation. Fifteen years have passed since this case was reported, and I have been unable to find a similar case in the literature. However, the patient reported by Kagan et al did have another potential etiology for the arteritis: herpes zoster.

The 20-year-old woman had developed a disseminated zoster rash approximately 10 weeks before her death, but she had been treated with prednisone. The first symptoms of arteritis developed 9 weeks later, on March 2, 1966, records the presence of a healing zoster rash, which was most severe on her face (including the ophthalmic divisions of the trigeminal nerve) and trunk. Electron microscopy of the cutaneous lesions was consistent with herpes zoster. We report an unusual case of lacunar pontine infarction producing hemifacial spasm as an isolated clinical finding.

A 54-year-old hypertensive man awoke one morning with malaise, dizziness, nuchal headache, numbness, and tightness of the left side of his face. Within a few hours, he noted rapid, involuntary muscular twitches in the left inferior orbicularis oculi that spread downward to the lower facial muscles that afternoon. Neurologic examination revealed only rapid, irregular, and painless contractions on the left side of his face. Computed tomography showed a small lacunar infarct in the left pons (Figure 1). In 2 weeks, the hemifacial spasm disappeared and only a sensation of tightness in the left periorbital area remained.

In retrospect, the cerebral arteritis in this patient was most likely a result of herpes zoster, not radiation.

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References


The following is in reply:

To the Editor:

There is no histopathologic microscopic picture that defines radiation injury. The marked proliferation of foam cell or so-called foam cell arteritis has been associated with radiation injury. These vascular changes are nicely illustrated in a book by Luis Felipe Fajardo, MD, entitled Pathology of Radiation Injury, published by Masson in 1982.1 Herpes zoster has been reported in patients with leukemia and lymphomas, but this is usually associated with giant cell granulomatous arteritis in which many inflammatory cells are present at the level of the internal elastic lamina. An inflammatory change of some kind is usually associated with infectious angitis.

There is a thrombotic, "noninflammatory" arteriopathy that occurs with herpes zoster, but foamy histiocytes are not present. We did not look for virus-like particles in our arterioles as we did in the cutaneous lesions, so I suppose a case could be made for a viral angitis. Today, immunoperoxidase stains for virus-particle-associated problems are available. The absence of inflammatory cells, thrombi and atheromatous plaques, diabetes, hypercholesterolemia, and hyperlipemia encouraged us to propose radiation as the etiologic agent.

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References


Lacunar Pontine Infarction Presenting as Isolated Facial Spasm

To the Editor:

We report an unusual case of lacunar pontine infarction producing hemifacial spasm as an isolated clinical finding.

Lacunar pontine infarction with isolated hemifacial spasm as the only clinical sign has not been previously reported. A 53-year-old hypertensive man awoke one morning with malaise, dizziness, nuchal headache, numbness, and tightness of the left side of his face. Within a few hours, he noted rapid, involuntary muscular twitches in the left inferior orbicularis oculi that spread downward to the lower facial muscles that afternoon. Neurologic examination revealed only rapid, irregular, and painless contractions on the left side of his face. Computed tomography showed a small lacunar infarct in the left pons (Figure 1). In 2 weeks, the hemifacial spasm disappeared and only a sensation of tightness in the left periorbital area remained.

In our case, a possible pathogenetic explanation could be that the perifocal ischemic edema compressed the intrapontine roots of the facial nerve, producing hemifacial spasm with the well-known mechanism of ectopic excitation and ephaptic transmission.2 Resolving edema could have removed the compression and abolished the ectopic/ephaptic excitation phenomenon, thus producing recovery from the hemifacial spasm.

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References


Lacunar pontine infarction presenting as isolated facial spasm.
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