Letters to the Editor

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 sequela 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 the onset of right-sided numbness and weakness. The admission physical examination from 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. One week after the onset of neurologic symptoms, left common carotid arteriography revealed beading of the proximal segments of the anterior and middle cerebral arteries and the pericallosal arteries. These angiographic findings are typical of herpes zoster arteritis. Thus, the clinical history, with a latency of 10 weeks between the rash and infarction, the angiogram, and the pathology in this case are all typical of zoster arteritis.

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 foamy 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.

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 angiitis. There is a thrombotic, "noninflammatory" arteriopathy that occurs approximately 10 weeks before the onset of right-sided numbness and weakness. The admission physical examination from 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. One week after the onset of neurologic symptoms, left common carotid arteriography revealed beading of the proximal segments of the anterior and middle cerebral arteries and the pericallosal arteries. These angiographic findings are typical of herpes zoster arteritis. Thus, the clinical history, with a latency of 10 weeks between the rash and infarction, the angiogram, and the pathology in this case are all typical of zoster arteritis.

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

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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.

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 peripheral area remained.

Lacunar pontine infarction with isolated hemifacial spasm as the only clinical sign has not been previously reported. It is now accepted that hemifacial spasm is commonly caused by compression of the root entry zone of the facial nerve, and it has been shown that this compression is caused by blood vessels in the great majority of cases. Exactly how such compression damages the nerve is not well understood. Some investigators have attributed the damage to ischemia, whereas others have found evidence that it is the pressure on the nerve that causes the damage. 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. 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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