Short Communication

The Value of Magnetic Resonance Imaging in the Detection of Type II Hemorrhagic Lacunes

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Lacunar infarcts (lacunes) are small necrotic/cystic lesions of the brain associated with hypertension. Many are clinically silent, but some are associated with localizing symptoms and signs. Lacunes recently were classified into three types, namely, those resulting from small infarcts (type I), those arising as small hemorrhages (type II), and those due to dilatation of perivascular spaces (type III). The type II lacunes are thought to be much less common than those of type I. Type II lacunes have not previously been reported during life. We report a case of subacute type II lacune in the pontine tegmentum that was detected by magnetic resonance imaging and later verified by histopathologic examination. The patient was hypertensive and had numerous other type I lacunes. This case shows that the magnetic resonance imaging features of hemorrhagic lacunes are identical to those of small hemorrhages or small hemorrhagic infarcts and that magnetic resonance imaging is a useful tool in the detection of lacunes and their correlation with clinical neurologic symptoms and signs. (Stroke 1989;20:822-825)

Lacunar infarcts (lacunes) are small cystic brain lesions associated with hypertension. The prevailing opinion is that the vast majority arise as small infarcts and undergo cystic changes with organization. Poirier et al labeled these type I lacunes. Their recent classification also includes a type II lacune that is supposed to be the result of an organized small hemorrhage. The relative incidence of these two types of lacunes is not clear, although type I lacunes are seen more commonly in pathologic practice. We describe a patient with a type II lacune in the pons that was followed clinically and by magnetic resonance imaging (MRI) for several weeks before the patient's death.

Case Report

A 73-year-old black woman was found unconscious at her home by a friend and brought to the emergency room. Nothing was known of her medical history except that she was on a "water pill" and a "heart pill."

On admission she was responsive but not oriented to person, place, or time. A neurologic examination revealed an uvula deviated to the left and eyes fixed in the midline. Ophthalmoscopic examination revealed no abnormality. No carotid bruits were present on examination of her neck. There was decreased strength in all extremities (2-3 out of 5) but more on the right side (2 out of 5). Her blood pressure was 180/100 mm Hg. There were rales in the left lung fields. A 2+ pitting edema was noticed in her feet. A decubitus ulcer was present over her left hip. Laboratory findings included a hemoglobin of 11.4 g%, and a leukocyte (WBC) count of 25,300/mm with a polymorphonuclear leukocytosis. Serum electrolytes and results of urinalysis were within normal limits. A chest radiograph showed cardiomegaly, small bilateral pleural effusions, and left pulmonary infiltrates. MRI of her head showed two small lesions in the same vertical axis with signal characteristics of hemorrhages in the pontine tegmentum (Figure 1). The contour of the brainstem was not distorted or enlarged. Cerebrospinal fluid showed 1,010 erythrocytes and 1 leukocyte per mm.

The patient was treated with broad-spectrum antibiotics, diuretics, antihypertensive medications, and digoxin, after which her lung infection cleared. However, her neurologic status continued to be poor at the end of the second week of hospitalization. She was never fully alert to her surroundings and frequently lapsed into periods of semiconsciousness. Her serum electrolytes were...
normal during these periods, thus excluding electrolyte abnormalities as a cause of mental status changes. A repeat MRI of her head showed the same pontine lesions with no evidence of herniation.

The patient continued to require care (debridement) for the decubitus ulcer and for malnutrition and chronic anemia. Her general and neurologic condition had not improved by the end of the fifth week of hospitalization, during which a Dobhoff feeding tube was placed and supportive therapy continued. She became progressively lethargic, and respiratory distress developed during the seventh week. She died on Day 52 of hospitalization. Her blood pressure during hospitalization ranged from 145/90 to 190/120 mm Hg.

**Results**

At autopsy the main findings were cardiac hypertrophy (500 g; left ventricular thickness 2.0 cm), renal arteriolosclerosis, bilateral pulmonary atelectasis, and generalized cachexia. The most significant findings were in the brain, which weighed 1,200 g. Sections of the pons showed a linear, cystic lacunar hemorrhagic infarct in the tegmentum of the right side of the pons (Figure 2) corresponding to the two lesions seen on the MRI (Figure 1, right). By serial sectioning of the pons, it was established that this single lesion extended for most of the vertical axis of the pons and across the midline at a lower level and that it accounted for both high-intensity lesions seen on the MRI. The age of this lesion corresponded to the duration of her hospital stay. Microscopically, both unabsorbed erythrocyte aggregates as well as clumps of hemosiderin (Figure 2, bottom) indicated the original hemorrhagic nature of this lesion. Nearby was a second lesion, a small fresh hemorrhage (Figure 2) that was thought to have occurred just before her death. Gross examination of the rest of the pons showed a total of five small lacunar infarcts in the basis pontis, involving corticospinal tracts bilaterally. Microscopic sections from the brain showed small, type I lacunar infarcts bilaterally in the basal ganglia and basis pontis and diffuse hypertensive arteriolosclerosis. Two partly thrombosed microaneurysms were seen on arterioles in the pons, one in the basis pontis on the left side (Figure 3) and the second at the origin of the left middle cerebellar peduncle. The multiple bilateral lacunar infarcts in the basis pontis corresponded to the bilateral paresis in the patient’s extremities during life.

**Discussion**

The small size of the lesion by MRI and by gross examination and the demonstration of a cystic cavity filled with hemosiderin-laden macrophages and unabsorbed blood lead us to conclude that the tegmental lesion should be called a lacune resulting from the organization of a small hemorrhage. If the lesion were a major hemorrhage, it would have caused distortion of the brainstem on MRI and would have resulted in death sooner. Lacunes,
literally meaning "small lakes," are small irregular or linear cystic lesions associated with hypertension. The basal ganglia, white matter of the centrum semiovale, and basis pontis are the usual sites of lacunes, although they can occur almost anywhere in the brain. Most lacunes are clinically silent, but patients with multiple lacunes can present with focal signs or a form of subcortical dementia. Fisher associated over 20 syndromes with these lesions. Many of the syndromes result in significant morbidity; severe sequelae including hemiballismus have been reported. We have seen a 3-mm lacunar infarct of the medullary pyramid cause a complete contralateral hemiplegia in an elderly man.

Although the clinical importance of lacunes is now well recognized, detailed pathologic studies of these lesions are few. Marie and others had proposed earlier that lacunes resulted from the resolution of small hemorrhages. Fisher made a painstaking histologic study of lacunes and showed that they were secondary to microvascular lesions such as narrowing and lipid deposition. Poirier et al recently proposed a classification of lacunes into three types. The first and by far the most common type is resolved small infarcts; type II lacunes are resolved small hemorrhages. Type III lacunes are, in reality, dilated perivascular spaces around penetrating blood vessels of the brain and not true infarcts or hemorrhages. The lacune we describe is of type II.

Microaneurysms have been shown to be one cause of Poirier type II lacunes although this must be very uncommon. Although two small microaneurysms were seen in the pons, the lacune in the tegmentum could not be ascribed to either one because the lacune was outside the territories of the vessels involved. Thus, the exact cause of the hemorrhage itself is unclear, although severe arteriolosclerosis with thinning or fibrinoid necrosis of a small arteriole could have resulted in the tegmental lesion.

The usefulness of MRI in detecting Poirier type II lacunes is documented by our study. Biller et al have shown its usefulness in the diagnosis of lacunar hemiballismus. It appears that small lacunes are
lesions in a geriatric Japanese population. Our case shows that type II lacunar infarcts can be added to this list of MRI-detectable lesions of the brain in hypertension.

Our patient's poor hospital course can be partly explained by the location of her hemorrhagic lacune. The pontine tegmentum is the site of reticular formation and of important ascending and descending reticular pathways that play a role in the maintenance of consciousness. Even a small lesion in this part of brainstem can cause serious effects. Although there are no controlled studies, it is the experience of most neuropathologists that the number of lacunes in the brain is proportional to the severity and duration of hypertension. It is therefore safe to presume that the vigilance of physicians in the diagnosis and management of hypertension plays a major role in the prevention of lacunar infarcts and their untoward effects, MRI appears to be a very useful tool in this crusade.

References


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