Spontaneous Hypertensive-Arteriosclerotic Intracerebral Hemorrhage

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DR. JOSEPH A. TRONCALE (*Associate Professor and Director, Family Practice Residency*): This case introduces discussion of a patient whose presentation represents one of the more important neurologic syndromes frequently seen by family physicians.

CASE PRESENTATION

DR. DAVID CLOSE (*Second-Year Family Practice Resident):* Mrs. P. is an obese 47-year-old black woman with a 25-year history of hypertension. By history, her hypertension has been poorly controlled. She had first been seen at the University of Alabama at Birmingham Family Medicine Clinic in late June of 1987. At that time her blood pressure was 248/134 mmHg. She was noted on that visit to have run out of her medications. Her medications had been adjusted to 0.2 mg of clonidine three times a day, 50 mg of atenolol every day, and 40 mg of furosemide three times a day. Because of continued poor control over several visits and a history of poor compliance with her medications, the patient was referred to the Hypertension Clinic at this institution in October 1987. Her blood pressure regimen was adjusted to 0.3 mg of clonidine three times a day, 80 mg of long-acting propranolol every day, 10 mg of enalapril twice a day, and 40 mg of furosemide twice a day. The patient made no further visits to either clinic until the day of admission.

The patient came to the Family Medicine Clinic on February 4, 1988, with the chief complaint of waking up from a nap earlier in the day with several distinct neurological complaints. She stated that she had noted a slurring of her speech, drooping and numbness of the left side of her face, and a sensation that her mouth was "pulled" to the right. She also noted that she was unable to walk without a stagger. Her daughter noted that the patient had complained of a frontal headache before napping. The patient's daily medication regimen at the time of this visit included 0.3 mg of clonidine three times a day, 40 mg of furosemide twice a day, 10 mg of enalapril twice a day, 80 mg of propranolol, and 20 mEq of potassium chloride elixir.

Her medical history revealed her long-term hypertension as previously noted. There was no history of diabetes, stroke, heart disease, or other significant problems except for obesity. Obstetric and gynecologic history revealed the patient to be para 2012. She was not menopausal, but was using no contraceptives. The patient had no previous surgeries and was not allergic to any medicines.

Family history was remarkable for hypertension in her father, who died of a cerebrovascular accident at the age of 44 years. She has a sister who also has hypertension and who has required coronary artery bypass graft surgery. The patient's mother died of uterine cancer at the age of 55 years, and her 41-year-old brother has cancer of the central nervous system.

Social history revealed the patient to be married. She worked as a salesperson. She had no history of cigarette, alcohol, or other drug abuse.

A review of systems revealed only that the patient had a history of bilateral hand tremors.

The physical examination showed an initial blood pressure of 242/148 mmHg in the right arm sitting. She was immediately given nifedipine sublingually, for a total dose of 20 mg. Her blood pressure lowered slightly, to 212/112 mmHg.

The patient was alert throughout the examination and in no distress. Her pulse was 120 beats per minute and regular, her respirations were 20/min, and her temperature was 98.6°F. Her pupils were equal, round, and reactive to light. The discs were sharp. There was no evidence of subretinal hemorrhages. There was some mild A-V nicking noted. Findings on examination of the ears, nose, and throat were normal. The neck was supple, without thyromegaly or adenopathy. There was no jugular venous distension or ca-

Submitted, revised, January 24, 1989.

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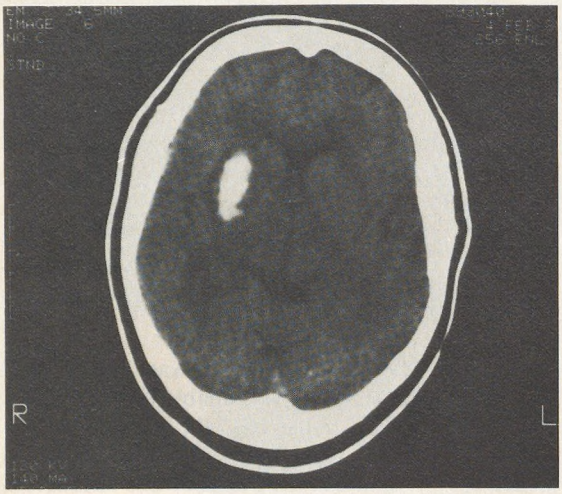

Figure 1. Computed tomographic scan of patient after admission revealing right putamenal hemorrhage

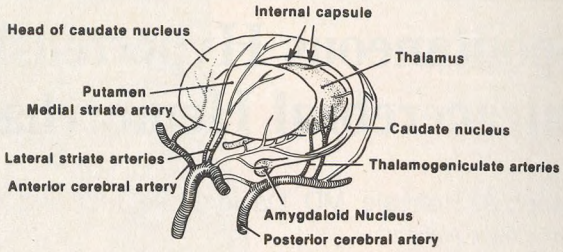

Figure 2. Blood supply of the basal ganglia (after Carpenter[3])

rotid bruits. The lungs were clear to auscultation. She had tachycardia without murmurs, gallops, or rubs. The abdomen was obese, nontender, and without masses.

Significant neurologic findings included a drooping of the nasolabial fold on the left. Asking the patient to make facial movements revealed diminished tone of the left lower face, consistent with a central VII nerve abnormality. The cranial nerves were otherwise intact. There was slight but demonstrable left arm and left leg weakness with a positive pronator drift sign of the left arm. Muscle strength was judged to be 4/5 on the left and 5/5 on the right. There was no significant sensory impairment. Cerebellar examination revealed no significant abnormalities. Reflexes were 2+/4+ and equal bilaterally. No Babinski sign was elicited. Mental status examination revealed no abnormalities.

The patient was immediately transferred to the hospital, where a computed tomographic (CT) scan was performed. Dr. Pham will review the CT scan results.

DR. XUAN-DAO PHAM (*Second-Year Family Practice Resident*): The CT scan revealed a significant hemorrhage in the region of the right putamen (Figure 1). As you can see, the bleeding can be seen on several of the "cuts" and represents an area of approximately 3 to 4 cm^3.

CLINICAL FEATURES

DR. TRONCALE: Spontaneous hypertensive-arteriosclerotic intracerebral hemorrhage (SHIH) represents a subset of intracranial hemorrhage that also includes arterial aneurysms, cerebrovascular malformations, and hemorrhage with vasculopathies such as polyarteritis nodosa.[1] Our discussion today will concentrate on SHIH.

Hypertensive vascular changes are associated with intraparenchymal hemorrhages in about two thirds of adult cases. This case is representative of the hypertensive aspect of the syndrome, but unfortunately for our patient, these types of hemorrhages are usually seen in an older population, the average being around age 70 years.[2]

It is important to understand the pathophysiologic arterial changes of this syndrome, which are at the root of its hemorrhagic sequelae. Arteriolar damage occurs characteristically in the lenticulostriate arteries of the middle cerebral artery and the posterior cerebral artery, the penetrating branches of the basilar artery, and the central cerebellar artery (Figure 2).

These arteries are small (50 to 150 μm in diameter) as opposed to their parent arteries (measured in millimeters). The hypertension causes damage to these small arteries, and microaneurysms, called Charcot-Bouchard aneurysms, form. These aneurysms lose their lining epithelium, media, and elastic tissue. What is left is fibrous tissue. This process is called *fibrinoid necrosis*.

These arterioles are therefore weak and susceptible to rupture, especially with continued hypertension. Certainly the rupture of one of these tiny vessels could not cause such extensive damage as seen in our patient's CT scan. It is postulated, however, that in these patients what is termed the *cascade effect* occurs. It is certain that more bleeding occurs, and it is thought that after one of these smaller vessels ruptures, the other similarly weakened arterioles rapidly deteriorate under the pressure of expanding blood and tissue. This theory accounts for the rapid onset of symptoms and explains why these patients frequently have a rapid change in mental status, with many losing consciousness immediately.

Where in the brain do most of these hemorrhages occur? According to the data compiled from large case series, almost one half of SHIHs occur in the putamen, as seen in

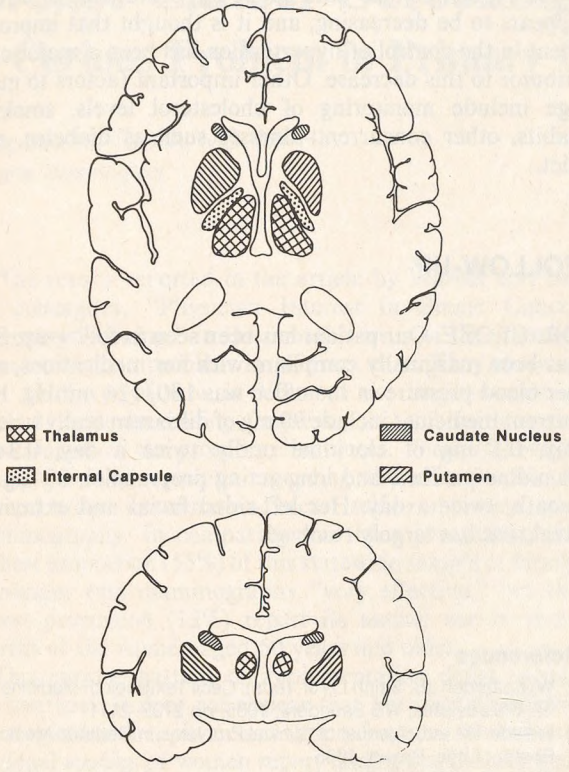

Figure 3. Anatomical locations of the basal ganglia in saggital and coronal sections

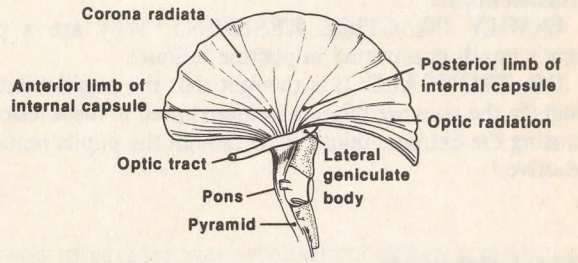

Figure 4. Anatomic relationships of the internal capsule and the optic tract (after Carpenter[3])

our patient. About 35% occur in the thalamic region. About 9% occur in the pons, 8% in the cerebellum, and 7% in the white matter or centrum ovale (Figure 3).

As one would expect, the location of the hemorrhage creates distinct stroke syndromes. Before discussing the various syndromes, it should be noted that the prognosis of SHIH depends on the size and location of the hemorrhage. Our patient in this case has an excellent prognosis, provided no further bleeding occurs, since the size of the hemorrhage was so limited. Larger hemorrhages, as one would expect, cause a patient to have a much graver prognosis because of both the amount of brain tissue damaged and the tremendous increases in intracranial pressure caused by an extensive hemorrhage.

The putamenal or external capsular hemorrhage causes a syndrome that one would expect from pressure applied to the internal capsule fibers that traverse medially to the putamen (Figure 4). In a classic putamenal hemorrhage the patient complains of a headache, the face begins to sag, and speech becomes slurred or aphasic. Hemiparesis then gradually ensues over about a half-hour period. Depending on the size of the hemorrhage, either the patient stabilizes, as with our patient, or the paralysis and mental status changes worsen, leading to stupor or coma.

The effects of a thalamic hemorrhage are similar to those of a putamenal hemorrhage because of their proximity within the internal capsule. In thalamic hemorrhages, the hemiplegia or hemiparesis is accompanied by more sensory loss because of the functional nature of the thalamus. Also, because the optic radiation is contiguous to the thalamus, a transient homonymous visual field defect can occur. Classic eye findings in thalamic hemorrhage include the deviation of eyes into a downward gaze and palsies of vertical and lateral gaze, as well as a number of other findings.[3]

Because of its critical location, a hemorrhage into the pons is devastating. The resulting syndrome is characterized by deep coma, paralysis, decerebrate posturing, and pinpoint pupils. This lesion carries a high mortality, and those who survive the initial insult are frequently left with severe neurological sequelae because of the frequent bilateral involvement.

An important syndrome to recognize because of the grave consequences if not quickly diagnosed is that caused by a cerebellar hemorrhage. Patients who experience cerebellar hemorrhage are frequently among those who take coumarin-type anticoagulants regularly, although cerebellar hemorrhage is not limited to this group. Symptoms in cerebellar hemorrhage patients are somewhat different from those caused by the other hemorrhagic syndromes described previously. Unlike the other syndromes, loss of consciousness is unusual. As one might expect from a cerebellar lesion, vertigo and truncal ataxia are prominent features. As one might not expect, however, nystagmus is not a common finding. There are a number of other prominent eye findings including an inability to look laterally to the side of the lesion or an ipsilateral sixth nerve palsy. The importance of this lesion is that it is a neurosurgical emergency. A cerebellar hemorrhage can be quickly evacuated,

and the patient's life may be spared before brainstem compression occurs.

FAMILY PRACTICE RESIDENT: Why are a patient's pupils constricted in pontine lesions?

DR. TRONCALE: It is thought that the pupillodilator fibers in the pons are bilaterally interrupted in these lesions causing the extreme miosis even though the pupils remain reactive.[4]

MANAGEMENT

Except for the neurosurgical emergencies noted above, treatment of SHIH is largely one of supportive care. Depending on the size and location of the hemorrhage, seizures may occur and should be controlled with appropriate anticonvulsant therapy. As with all strokes, there are alterations that must be made by the patient in terms of activities, movement, speech, vision, or other impairments that the stroke syndromes provoke. It is important that the physician be aware of the family support system, the resources available to care for these patients both emotionally and physically, and a treatment plan that can be undertaken by both the patient and the patient's family.

The best treatment for the various intracranial hemorrhage syndromes is largely one of prevention. This is most appropriately accomplished by the practitioner in the everyday management of hypertension, especially in elderly patients and in patients, such as the one today, who have a strong family history of hypertensive disease and stroke. As recent epidemiologic data show, the prevalence of stroke appears to be decreasing, and it is thought that improvement in the control of hypertension has been a major contributor to this decrease. Other important factors to manage include monitoring of cholesterol levels, smoking habits, other concurrent illnesses such as diabetes, and diet.

FOLLOW-UP

DR. CLOSE: Our patient has been seen in follow-up. She has been marginally compliant with her medications, and her blood pressure in the office was 180/114 mmHg. Her current medicines include 90 mg of diltiazem orally twice a day, 0.1 mg of clonidine orally twice a day, 0.3-mg clonidine patches, and long-acting propranolol, 80 mg by mouth, twice a day. Her left-sided facial and extremity weakness has largely resolved.

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