Basilar Artery Narrowing and Hyperparathyroidism: Illustrative Case

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SUMMARY A patient presenting with a pontine infarction caused by mid-basilar artery narrowing associated with hyperparathyroidism is described. The narrowing reversed with surgical removal of his parathyroid adenoma and normalization of his serum calcium. This patient's illness lends evidence to the role of calcium in cerebral vasoconstriction.

THERE ARE MANY causes of irregular focal narrowing of the major intracranial arteries. Arteriosclerosis is the most common disease process. Focal stenosis can also be due to embolic-filling defects, aneurysmal subarachnoid hemorrhage or head injury induced spasm, tumor encasement, dissection or the various forms of arteritis.1-3 Many of these disorders are reversible spontaneously or with treatment.

The role of calcium in the genesis of cerebral vasoconstriction is under investigation but remains speculative.4 The following case report illustrates basilar artery focal constriction associated with a hyperparathyroid hypercalcemic state.

Case Report

A previously healthy, 42-year-old, right-handed, Caucasian construction worker became vertiginous with severe vomiting during January of 1984. After several hours he developed right-sided numbness and weakness, dysarthria and dysphagia.

His past history was negative except for hypertension noted on an enlistment physical at age eighteen. This was not treated and he later infrequently saw physicians.

Admission examination revealed moderate right hemiparesis. Right finger movements were clumsy and he moved his right leg less than his left. A right central facial paresis, inconstant right gaze preference, and severe dysarthria were present. He was initially treated with steroids which were tapered over the next five days.

Immediate CT scan was normal and arch and left carotid angiography the following day was also normal. Chest x-ray showed an enlarged cardiac silhouette. Admission laboratory results were within normal limits except for an elevated calcium to 11.9 mg/dl (normal being less than 10.5 mg/dl) and a phosphorus of 1.6 mg/dl (normal being greater than 2.3 mg/dl). A lumbar puncture on day four of his illness was also normal. Blood and CSF serology tests for lues were normal. Admission laboratory results were within normal limits except for an elevated calcium to 11.9 mg/dl (normal being less than 10.5 mg/dl) and a phosphorus of 1.6 mg/dl (normal being greater than 2.3 mg/dl). A lumbar puncture on day four of his illness was also normal. Blood and CSF serology tests for lues were normal. Clinical cardiac evaluation and laboratory studies including serial electrocardiography and an echocardiogram were normal. Vasculitis evaluation in terms of serial sedimentation rates and antinuclear antibody factors also was normal.

Only intermittent hypertension (diastolic blood pressures elevated to 110 torr, but stabilizing between 80 and 90 torr, with mainly normal systolic blood pressures) was recorded during his hospitalization. His blood pressure stabilized without treatment. He had taken aspirin regularly at home and coagulation studies were consistent with aspirin use.

On the sixth hospital day he had a transient increase in right-sided numbness. On the eighth day he had an increased right hemiparesis accompanied by left gaze paresis, conjugate left beating nystagmus and trunk ataxia. Left vertebral angiography via femoral catheterization showed an irregular constriction of the middle portion of the basilar artery beginning distal to the anterior cerebellar artery origin and extending to below the superior cerebellar artery origin (fig. 1). There was no intraluminal defects nor evidence of distal branch occlusion.

He was retreated with steroids without significant change in his spastic hemiparesis. Brain stem auditory-evoked response testing after his clinical deterioration was abnormal with prolongation and attenuation of wave 5 and increased 1–5 and 3–5 interpeak intervals on stimulating the right ear. The following day a CT scan now showed a left pontine infarct.

On day twenty of his illness, repeat left vertebral angiography and abdominal aortography were performed. The mid-basilar irregular stenosis was persistent. There was no evidence of aortic or renal artery disease. The patient was transferred to a rehabilitation center where multiple gastrointestinal complaints were noted including nausea, vomiting, indigestion, and intermittent hiccups. Serum calcium values were consistently elevated. Parathyroid hormone assays were also abnormally high.

In March 1984, an isolated parathyroid adenoma was removed surgically. During surgery he had a surge of hypertension and transient atrial fibrillation, each resolving spontaneously in the immediate postoperative period. Subsequent extensive evaluation for multiple endocrine tumor syndrome was negative. There were no abnormalities of thyroid, adrenal or catecholamine metabolism. No cause for his intermittent hypertension could be found despite an aggressive search for renal and endocrine disease. Postoperative calcium levels were normal.

At eighteen-month follow-up, he had a slight residual right hemiparesis. Gait was slightly spastic but he was independent on all surfaces without aids or de-
vices. He could perform isolated hand movements and was independent in self-care. He complained that he still could not hammer so could not return to his former construction work. His speech was mildly slow and his balance was intact. His affect was flat and he showed little ambition. This was a change from his preillness aggressive nature. Calcium and thyroid function studies were normal but he was hypertensive to 180/110 torr. Repeat vertebral angiography via femoral catheterization now showed a completely normal basilar artery with no evidence of the previous constriction (fig. 2). He was started on antihypertensive medication.

Discussion

We have described a patient with a parathyroid adenoma who developed a pontine infarct caused by vasoconstriction of the mid-basilar artery confirmed on two angiograms twelve days apart. After surgical correction of hyperparathyroidism and hypercalcemia, the basilar artery narrowing disappeared. In this patient no other cause of reversible stenosis was found despite vigorous searches and prolonged observation. Infectious, granulomatous, collagen vascular, rheumatic, or chemical-pharmacologic-induced arteritides were not present. There was no evidence of subarachnoid bleeding, head trauma, embolization or tumor. Follow-up during the eighteen months from presentation to angiographic demonstration of basilar artery normalization documented only the cured hyperparathyroidism and labile hypertension.

The relationship between stroke and hyperparathyroidism has been analyzed by Bostrom and Alveryd.3 Nine of 170 hypercalcemic patients referred for parathyroid exploration had definite strokes. One of the four autopsied stroke patients had vertebral basilar territory occlusion. A single case report described reversible cerebral arterial spasm after correction of iatrogenic hypercalcemia.6 A patient described by Gorelick and Caplan7 with hypercalcemia due to probable hyperparathyroidism had angiographically demonstrable constriction in large intracranial arteries and distal branches that explained bilateral hemispheric neurologic signs. Gorelick and Caplan also noted improvement in neurologic signs and symptoms in three of six patients following correction of hypercalcemia. Hyperparathyroidism is also known to be associated with hypertension.8

A treatable although perhaps unusual cause of focal vasoconstriction and stroke is documented. This case
Subarachnoid Hemorrhage and Granulomatous Angiitis of the Basilar Artery: Demonstration of the Varicella-Zoster-Virus in the Basilar Artery Lesions

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SUMMARY A 70-year-old man, with regional herpes zoster (C2) of 10 weeks duration, died following subarachnoid hemorrhage caused by the rupture of an aneurysm in the basilar artery. Granulomatous angiitis, with multinucleated giant cells, was found at autopsy in the wall of the aneurysm. Electron microscopy of the basilar artery disclosed intracytoplasmic viral particles with an envelope which measured 150-220 nm in diameter. Immunohistochemistry studies revealed varicella-zoster-virus-related antigen in the cytoplasm and/or in the nucleus of histiocytes in the vessel wall. These findings suggest that varicella-zoster virus may be linked to the development of granulomatous angiitis.

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Received May 27, 1985; revision #1 accepted January 13, 1986.

CASE REPORT

In May 1983, a 70-year-old Japanese man consulted a dermatologist because of pain and cutaneous lesions in the occipital region following symptoms of a common cold. He was admitted to Shin-Nittetsu Hospital and was treated with analgesics, vitamins, anti-inflammatory drugs, gamma-globulin and occasional stellate ganglion blocks. He was then transferred to the Fukuoka Anesthetic Center for control of the pain, in June 1983. Intensive stellate ganglion block, continuous peridural anesthetic block using 1% me pivacaine, and gamma-globulin therapy without corticosteroid therapy were prescribed, but fever and pain persisted. About 70 days after the onset of this illness, he fainted and cardiac arrest occurred. He was soon resuscitated and transferred to the intensive care unit in Kyushu University Hospital. Massive inoperable subarachnoid hemorrhage was diagnosed following brain computer assisted tomography. The patient died several hours later.

Laboratory Data

Blood pressure, complete blood count, chest X-ray, blood-glucose, serum-enzymes and -electrolytes were within normal limits for the full period of this episode. Mild proteinuria was present, but renal dysfunction and inflammation were mild. Left axis deviation, left ventricular hypertrophy and coronary ischemic changes were detected on the electrocardiograms. Serum antibody titers to varicella-zoster virus were 32X, 32X, and 16X (normal; below 4X), 40, 50 and 60 days after the onset of the common cold-like symptoms, respectively.
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Stroke. 1986;17:1022-1024
doi: 10.1161/01.STR.17.5.1022

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