Basilar artery migraine

Case report

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The case of a 40-year-old woman with a 24-year history of basilar artery migraine is described. She was admitted to the hospital after suffering a large cerebellar infarction causing hydrocephalus and deep stupor during an attack of migraine. She recovered after ventricular shunting and removal of the infarct.

KEY WORDS • basilar artery • migraine • cerebellar infarction

While migraine is a common ailment, afflicting perhaps 5% of children and 15% or more of adults, it is rarely associated with permanent neurological deficits, even when focal symptoms such as hemiparesis or hemianopsia accompany an attack. Migraine in the distribution of the basilar artery is one of its less common forms, and it was this variety that afflicted the patient who is the subject of this report.

Case Report

This 40-year-old woman was admitted to the hospital on March 3, 1982, because of severe occipital headache and deep stupor.

Examination and Course. It was difficult to awaken her but, with stimulation, she was able to say that she had severe pain in the back of her neck and back of her head. She could not cooperate in an examination. Her deep-tendon reflexes were hyperactive but there were no Babinski signs. Computerized tomography (CT) demonstrated a large area of decreased density in the left cerebellar hemisphere near its upper surface. The fourth ventricle was deviated to the right side and there was partial enlargement of the third and lateral ventricles. There was no evidence of hemorrhage (Fig. 1), and no neck stiffness.

A right ventriculoperitoneal shunt was placed. During the next 72 hours, the patient’s condition improved and she was able to give some medical history, although she remained stuporous. She said she had experienced headaches since she was 16 years old. These invariably began at the base of her skull posteriorly and radiated upward to the vertex. Sometimes, she had a sensation of numbness about the lips and of clumsiness in the tongue before the headache began. The area at the base of her skull was tender when the headache was at its height. The pain had often required a visit to the local emergency room where an injection of meperidine might be given. The headache usually lasted several hours and made her “want to get away from anything that might hurt” so that she would usually avoid contact with people and go to sleep. When she awakened, the headache would be gone. She usually slept 10 or more hours.

Fig. 1. Computerized tomography scan showing left cerebellar infarct and dilated ventricles.
She had one such headache at least every month after the age of 16 until the present episode. Often the headaches preceded or accompanied her menstrual period. She had taken birth control pills for about 15 years but had stopped their use 4 years prior to the present episode. After discontinuing that medication, the headaches had become and remained worse. She had experienced a very severe episode 3 weeks prior to her admission to the hospital, and she had also had one about 5 days before the episode that precipitated her admission.

As she became more alert and could cooperate in examinations, nystagmus on looking to the left was found and dysmetria was present in the left upper extremity, most marked in the hand, with a lesser degree of incoordination in the left leg and foot. A repeat CT scan showed that, while the shunted ventricles had decreased in size, the large hypodense area in the cerebellum persisted and there was still a shift of the fourth ventricle.

**Operation.** On March 8, a left posterior fossa craniectomy was performed. When the cerebellum was exposed, it looked normal inferiorly and laterally, but superiorly and medially there was a loss of normal architecture. Here, the tissue was very soft and sagged out of the wound. It appeared infarcted. A pyramidal area in the superior and medial part of the cerebellum corresponding to this soft area was excised and sent for pathological examination. Some of this tissue extended up to the tentorial notch and, when it had been excised, one could see up along the brain stem. There was obviously no compression of the brain stem after this had been done and the cerebellum pulsed well. The superior cerebellar artery, which appeared to contain no blood, was excised. When it was cut it did not bleed; the proximal stump was coagulated. Microscopic examination of the artery showed no evidence of acute or chronic inflammation. The arterial wall had some slight focal medial degeneration.

**Pathological Examination.** The specimens of the cerebellar parenchyma, which included portions of the molecular layer, Purkinje cell layer, granular layer, and adjacent white matter showed scattered to focally plentiful foamy macrophages and occasional heterophilic infiltration as well as local fresh hemorrhage. Capillary and small-vessel proliferation was occasionally seen within the leptomeninges and the molecular and granular cell layers. There was no evidence of vasculitis. The white matter was markedly hypocellular and had a pale, spongiform appearance. Several other fragments contained moderate gemistocytic astrocytosis in the white matter and microglial and astrocytic proliferation in the molecular layer. There were rare collections of macrophages in the leptomeninges. All of these changes were considered consistent with infarction of less than 2 weeks’ duration.

**Postoperative Course.** Following surgery, the patient became much more alert and her cerebellar deficits were more obvious. She had slurred speech and showed marked incoordination and dysmetria in her left upper extremity. There was very mild decomposition of movement in her left lower extremity. It was moderately difficult for her to manipulate her tongue when she spoke or ate. Within 48 hours after surgery, she was able to get up and walk. There was still some ataxia on the left side, especially in the hand and arm, but it was not severe and decreased daily. There was no headache. She went home on the 9th postoperative day, by which time she was able to care for herself.

On examination 7 weeks later, the patient had mild nystagmus on left lateral gaze and marked tremor of the left hand when she used it purposefully. Past pointing was present with the left hand. There was a very mild disturbance of heel-to-shin testing on the left side. Her gait was normal including compass gait. She had no difficulty with turning quickly and could walk in tandem stride. Her deep-tendon reflexes were still hyperactive. A CT scan made 1 month after she left the hospital showed that the ventricles were symmetrical and normal in size and the fourth ventricle was in the midline.

Six months after discharge, her condition continued to improve. She had had no more headaches. Scanning speech had improved greatly and her chief problem remained the intention tremor in the left arm which occurred when she got nervous. A little difficulty in swallowing still persisted. Sometimes she had a vague tingling sensation in the left side of her face. She was back at work, training horses.

Seven years after her operation she had no scanning speech. When she is angry it does get “a little slurred.” She still has clumsiness in the use of her left arm, and tremor may occur in that hand when she is very tired or angry or occasionally when she is excited. The incoordination in her tongue and numbness in her face noted after surgery have disappeared. She rides horseback a good deal and continues to train horses. Interestingly enough, since her operation, she has not had another severe headache and only rarely, again when angry, has a generalized mild attack.

**Discussion**

Although migraine was known in antiquity, it was not until 1961 that Bickerstaff2 really delineated a variety associated with abnormalities in the distribution of the basilar artery. While it may commence at any age, this type usually begins in adolescent girls. The onset is often associated with some loss of vision followed by flashes of light which is ascribed to constriction of the posterior cerebral arteries, the terminal branches of the basilar artery. Then the patient may have numbness in a limb or in the perioral area and there may be unsteadiness, dysarthria, and vertigo. The headaches are always occipital, although they may extend upward to the top of the head and down into the back of the neck.
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In reviewing the subject in 1987, Bickerstaff noted that permanent defects were quite exceptional in basilar artery migraine, although he described two cases in which there was prolonged cortical blindness and ataxia followed by a typical attack. The CT scans in those patients showed bilateral occipital and cerebellar hemisphere lesions. He also stated that CT scans repeated later showed infarcts of the medial occipital lobe, presumably from vasoconstriction of the posterior cerebral arteries. In a small proportion of his cases, oral contraceptives changed generalized migraine to attacks with focal features. In 10 such patients, cerebral infarction had followed within weeks. Headaches had been made worse in some patients who used those agents although, in a very few, headaches had actually improved during their use. In the patient described here, oral contraceptives had been discontinued 4 years before the episode of infarction, and during that time the headaches had become worse.

Fortunately, infarction is a rare accompaniment of migraine and, when strokes occur, they commonly involve the distribution of the anterior or middle cerebral arteries. Rarely have they been life-threatening. There are associated risk factors that increase the possibility of stroke in migraineurs, such as prolapse of the mitral valve, hypertension, arteritis, abnormalities in the clotting mechanism, or the effect of contraceptive medications and vasoconstrictive agents. None of these factors were present in this patient.

Rothrock and associates described a group of 22 patients who had suffered a stroke in association with migraine, and in all but one the headache had clearly occurred at the time of the vascular insult. Sixteen of them had some permanent neurological deficit. One patient became comatose after having an arteriogram in which severe spasm of the vertebral artery was seen. While most of the patients whose symptoms, signs, or radiological findings were tabulated had unilateral cerebral lesions, two had some evidence of posterior cerebral artery changes bilaterally such as cortical blindness or bioccipital infarction.

Shortly after his first paper, Bickerstaff did describe three adolescent girls who became unconscious while having migraines. He postulated that this was probably from vascular insufficiency of the lower brain stem reticular formation. Later he mentioned that a third of his patients with basilar artery migraine had at least a brief loss of consciousness without epilepsy. Still another case of migraine associated with loss of consciousness in a 16-year-old girl was recorded by LaWall and Oommen. This girl had begun having migraine headaches at the age of 13 years, and 3 years later became unconscious during one of these episodes.

While it is obvious that complex migraine may occasionally be associated with infarction and residual neurological deficits, the risk of death in such episodes is extremely rare. In the present case, there was massive infarction of the cerebellum in the distribution of the left superior cerebellar artery sufficient to compress the fourth ventricle, causing hydrocephalus and a real threat to life. The patient has done well following resection of the infarcted area within the cerebellum and has not subsequently had a migraine headache such as she had suffered for over 20 years prior to this event.

References

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