# Running–induced headaches in aqueductal stenosis patient

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## ABSTRACT

This is a report on a 16-year-old boy with lateral and third ventriculomegaly who complained of episodic headaches after a long distance running. Following third ventriculostomy, he ceased to have such headaches. The authors conclude that his headaches were caused by increased intracranial pressure (ICP) during a strenuous running. The preoperative condition was considered to be an equivalent to the formal term "aqueductal stensosis.".

**Key Words:** aqueduct stenosis, third ventriculostomy, headaches, hydrocephalus; intracranial pressure;

## INTRIODUTION

Common signs and symptoms of the idiopathic aqueductal stenosis (AS) are headaches, gait disturbance, cognitive dysfunction, urinary incontinence, and seizures (1, 2). Approximately 50% of adult AS patients (> 16 years old) are noted to have headaches (2, 3), which are caused by increased pressure in the lateral and third ventricles, secondary to inadequate flow from the third ventricles into the fourth ventricle through the narrow aqueduct of Sylvius (3). In the presence of papilledema, increased cerebrospinal fluid (CSF) pressure must be controlled medically or surgically without delay. In contrast, surgical treatment of patients with intermittent headaches and lateral and third ventriculomegaly is debatable.

## CASE PRESENTATION

A 16-year-old boy presented with a 6-month history of intermittent headaches that always followed 3 km running. Three years earlier, incidental ventriculomegaly was found by CT scan after minor head trauma. He remained asymptomatic, and annual MRI studies showed unchanged ventricular size and shape. However, while participating in the running training at the age of 15, he experienced an occipital headache when he barely reached the 3-km goal. After passing the goal, he developed severe headache, extending to the entire head, and lost his ability to concentrate. He rested for 30 minutes before the headache subsided. There were no overt signs of increased ICP. Non-steroidal anti-inflammatory drugs (NSAIDs) and Triptans (tryptophan) failed to control such headaches. We postulated that the headaches reflected the sub-threshold ICP during his sedentary work, which changed to abnormally high level after the running stress. The MRI finding suggested narrow aqueduct stenosis (Fig.1A white arrow head: upper entrance of aqueduct). The patency of the basal cistern and cisterna ambiens by MRI (Fig. 1A) favored third ventriculstomy. Three months after surgery, the patient returned to running, and achieved a 5-Km goal without developing headaches.

## SURGICAL AND POST-SURGICAL FINDINGS

The endoscope was inserted into the right lateral ventricle, and then into the third ventricle through the dilated foramen of Monro. Posteriorly, the rostral end of the aqueduct was noted to be narrow (Fig. 2a). A very thin third ventricle floor (Fig. 2b) was penetrated by cautery. Through a 5 mm-long opening (Fig. 2c), CSF flowed readily into the basal and chiasmatic cisterns (Fig. 2d arrow). Two months after surgery, MRI showed significant decrease in size of the lateral and third ventricles, and clearly identified all the sulci (Fig. 3). Cine-MRI showed CSF flow from the third ventricle into the chiasmatic cistern, and the interpeduncular and prepontine cistern (Fig. 3 white arrows).

**Figure 1: MRI before ETV**

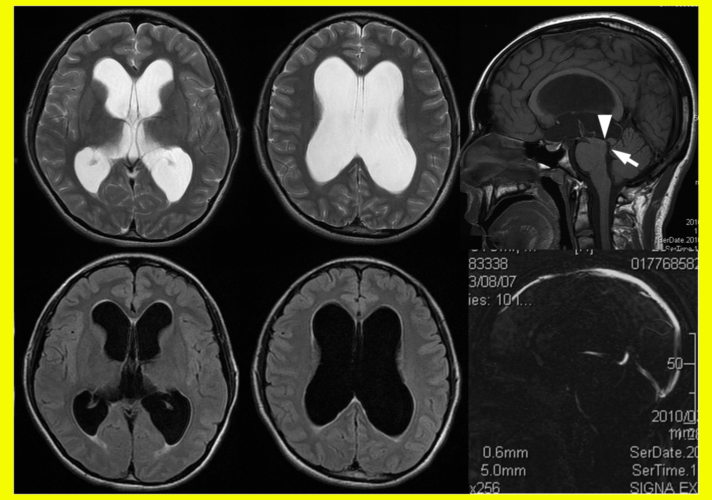


**Figure 2: at ETV**

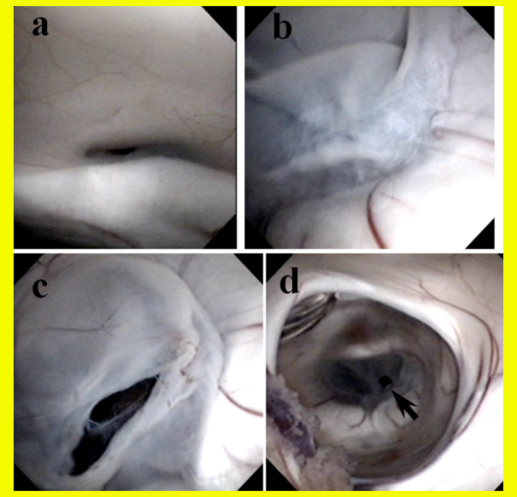
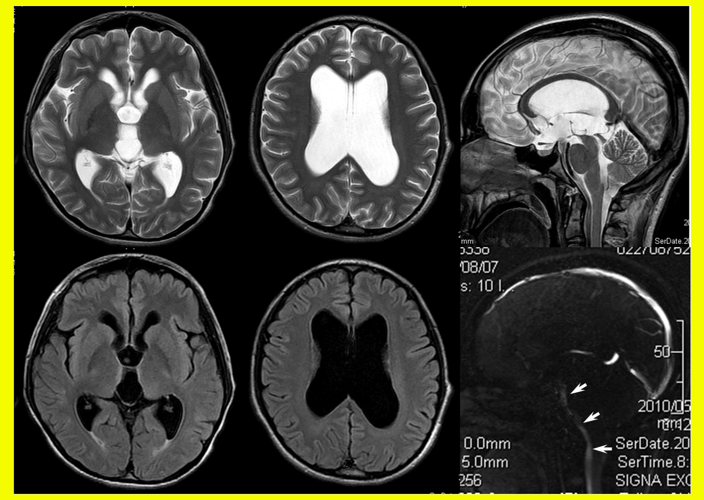


**Figure 3: MRI after ETV**



## DISCUSSION

In this section, the mechanism of running-induced headaches is analyzed. In the early human life, the balance between the ICP and volume is maintained by adaptive mechanisms. During infancy, the skull size increase is determined by the brain size, while the cranial sutures are slowly closing. It is postulated that the expansile force against the skull exerted by the growing brain and meninges accelerates skull expansion. This may be explained by activation of intra-cellular bone growth factor receptors in mesenchymal cells in response to increasing pressure (4). The skull expansion probably prevents ventricular enlargement, even under increased CSF pressure. After skull growth becomes slow and the suture closure is almost completed, compensation to increased CSF pressure is effected by suppression of CSF production and an increase in CSF absorption through the Pachionian granulations. However, as the stenosis of the aqueduct progresses, CSF circulation through the aqueduct further decreases, and the lateral and third ventricles become enlarged. The CSF pressure energy is then dissipated into the dilated ventricular walls, preventing development of increased ICP signs. In our patient, this compensatory mechanism was apparently offset during the long distance running due to the following process. The intra-abdominal pressure increased as the abdominal muscles continued to contract (5), and the intra-abdominal and intra-thoracic venous pressure steadily rose. Proportionately, the intraspinal venous pressure was elevated, and then was transmitted to the intracranial venous system (6,7). By the time the patient reached 3-Km goal, the intracranial pressure reached the level above the compensatory threshold, and headaches developed.

## CONCLUSION

The headaches that occur after long distance running in a patient with dilated lateral and third ventricle should be considered as hydrocephalic in nature. The third ventriculostomy was the procedure of choice in our case with aqueduct stenosis.

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