Delayed Posttraumatic Hydrocephalus Secondary To An Aqueductal Web Treated With Endoscopic Third Ventriculostomy: A Case Report.

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Citation

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Abstract

BackgroundDelayed posttraumatic hydrocephalus is typically communicating secondary to arachnoid villi insufficiency, due to a reactive inflammatory response to blood products. Acute obstructive hydrocephalus secondary to an aqueductal clot or midbrain contusion has been published; however, no previous publications of delayed obstructive posttraumatic hydrocephalus have been found. Case descriptionWe present a case report of delayed obstructive hydrocephalus secondary to an aqueductal web. The patient was successfully treated by an endoscopic third ventriculostomy. ConclusionTo our knowledge, this is the first description of delayed obstructive posttraumatic hydrocephalus secondary to an aqueductal web, and we discuss the pathophysiological cause, and relevant treatment options.

INTRODUCTION

In the vast majority of instances post-hemorrhagic hydrocephalus is the result of extraventricular obstruction of CSF absorption, hence communicating hydrocephalus. The treatment options available in this setting are often limited and placement of a ventriculoperitoneal shunt is often required [1-4]. In rare instances, post-hemorrhagic hydrocephalus is due to obstruction within the ventricular system. The cerebral aqueduct, being the smallest caliber conduit for CSF flow, is particularly vulnerable to obstruction by intraventricular blood, or by compression from expansive lesions within the tectum or tegmentum [5].

A wider array of treatment options is available for hydrocephalus caused by obstruction of the cerebral aqueduct. Increasingly, endoscopic techniques, including third ventriculostomy, and aqueductoplasty with or without stent placement, have been used [6-9]. We describe a case of acquired aqueductal obstruction due to a severe closed injury with a focal hemorrhagic contusion of the midbrain tegmentum and tectum. Performance of an endoscopic third ventriculostomy (ETV) allowed for durable radiographic and clinical improvement without the need for shunt placement.

CASE REPORT

HISTORY

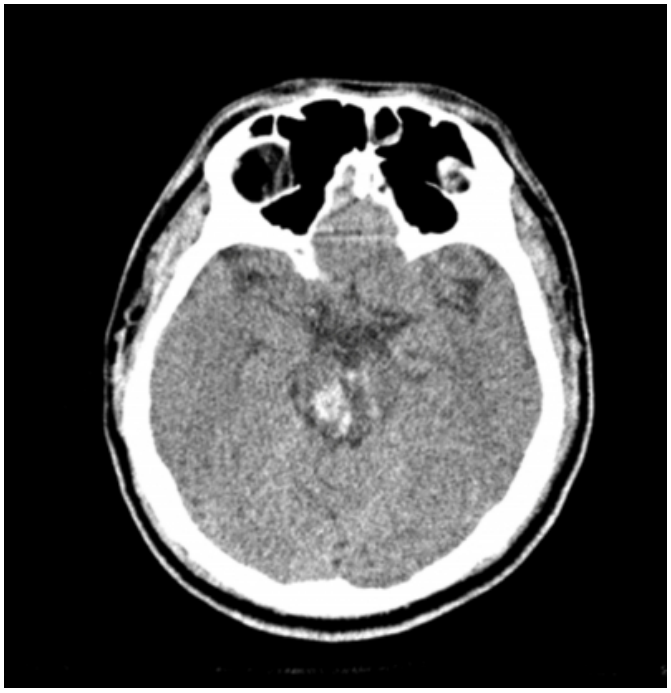
A 23-year-old man presented in extremis following a high speed motor vehicle accident. He was the unrestrained driver in a single vehicle, high speed accident, wherein the car struck a utility pole. Endotracheal intubation was performed at the scene. Upon arrival to the emergency department, neurological examination revealed bilateral dilation of the pupils, which were minimally reactive. Motor responses were extensor. Glasgow coma scale was 4T.

COMPUTED TOMOGRAPHY

CT scan of the brain demonstrated diffuse shearing injuries involving the pons, mid-brain, splenium of the corpus callosum and bilateral frontal and left parietal subcortical regions. A small hemorrhage was seen in the occipital horn of the right lateral ventricle, and along the posterior interhemispheric fissure.

Figure 1

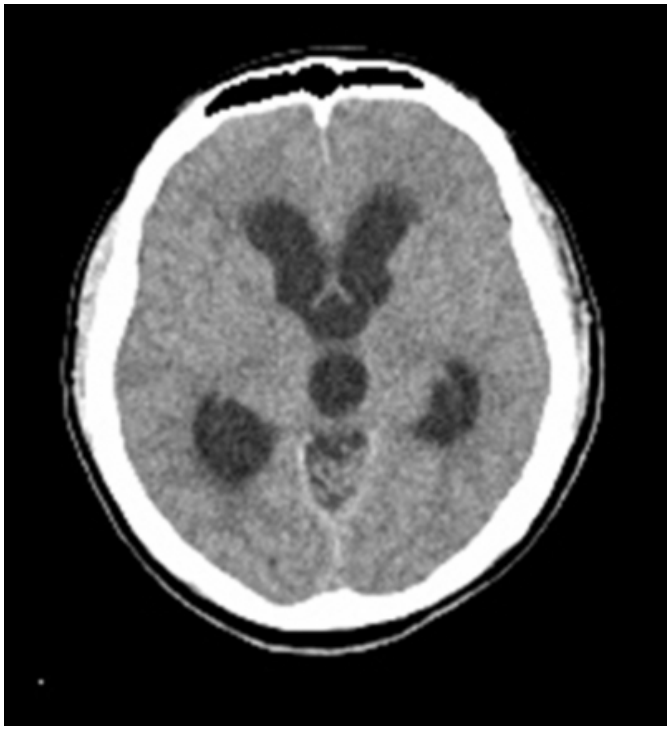
Figure 1. Admission CT scan. Focal contusion of the midbrain, including the tegmentum and tectum.



A right frontal external ventricular drain (EVD) was placed for intracranial pressure (ICP) monitoring. The ICP was intermittently elevated during the five days following admission, requiring CSF drainage and sedation. Subsequently the ICP normalized and the catheter was removed. The patient slowly improved over the next three weeks and was discharged to an inpatient rehabilitation facility. Eventually the patient became increasingly lethargic, thus CT and MRI were performed.

Figure 2

Figure 2. Follow-up CT scan. Extreme enlargement of the lateral and third ventricles.

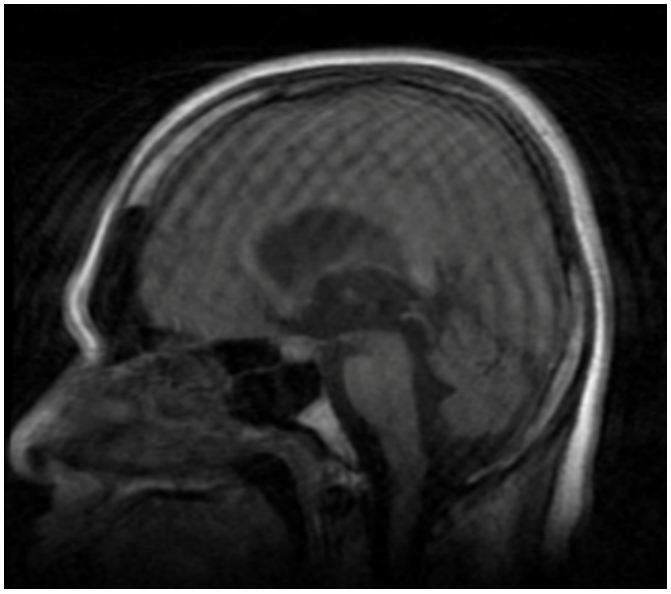


COMPUTED TOMOGRAPHY

The third and lateral ventricles have enlarged with effacement of the cerebral sulci. The region of brainstem hemorrhage has evolved unremarkably. Low-attenuation changes are present in the region of the hemorrhage. Some of these changes extend towards the area of the cerebral aqueduct.

Figure 3

Figure 3. MRI, sagittal T1-weighted image. There is a ballooning dilatation of the cerebral aqueduct, and enlargement of the third ventricle. There is no dilation of the forth ventricle



MAGNETIC RESONANCE IMAGING

Multiple well-defined focal areas of porencephaly/encephalomacia within the posterior pons and mid-brain, as well as areas of abnormal increased T2 signal intensity throughout the posterior mid-brain in a distribution of the periaqueductal gray, as well as within the parasagittal mid-brain extending towards the cerebral peduncles, consistent with areas of resolved hemorrhagic contusion with residual tissue loss, gliosis and healed ischemic. There is gross dilatation of the ventricular system at the level of the aqueduct of
Sylvius and proximally including the third ventricle, bodies of the lateral ventricles and temporoventricular horns.

OPERATION

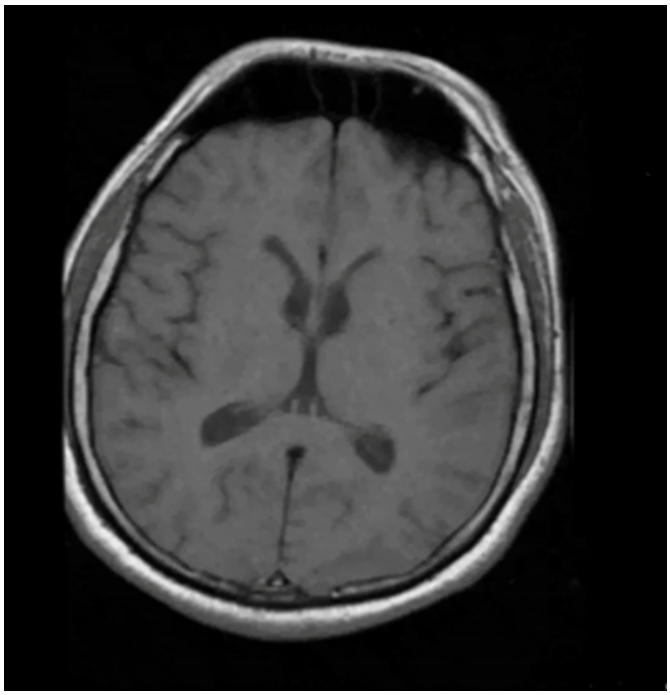
The patient underwent an endoscopic third ventriculostomy via a right frontal approach. A rigid, high resolution fiber optic endoscope (Channel ™ Neuroendoscope, Medtronic) was used to visualize the structures of the third ventricle. The floor of the third ventricle was thin, translucent and displaced inferiorly. Hemosiderin deposits were visible on the ependymal surface of the third ventricle and proximal portion of the cerebral aqueduct. Perforation of the third ventricular floor was performed with a 3-French Fogarty balloon. The stoma site was enlarged with dissecting forceps. The endoscope was removed.

POSTOPERATIVE COURSE

The patient made an uneventful recovery. Over the following two weeks, his cognition and attention improved. He was subsequently discharged home. Six months following the injury, he remained awake and alert, however wheelchair dependent due to lower extremity spasticity. Follow-up MRI demonstrated decrease in the size of the ventricles, and flow artifact at the site of the third ventriculostomy.

Figure 4

Figure 4. MRI, axial T1-weighted image. Three months post operatively, ventricular size is significantly reduced.



DISCUSSION

ETV has been established as the treatment of choice for obstructive hydrocephalus. Success rates (defined by improvement in symptoms and avoidance of a shunt) as high as 70-90% have been achieved for aqueductal stenosis, both primary and secondary to compressive lesions (e.g. tectal and pineal region tumors) [10, 11]. ETV is performed in various other etiologies with various success rates; however, it is still considered a treatment for obstructive hydrocephalus, with controversy regarding its use in communicating hydrocephalus [12, 13].

Post-traumatic hydrocephalus typically causes communicating hydrocephalus secondary to inflammatory response and scarring of the arachnoid granulations.

However, acute hydrocephalus may occur secondary to aqueductal obstruction by a blood clot, or periaqueductal contusions with secondary compression of the natural CSF pathways [5].

The presented patient demonstrates a unique mechanism of delayed post-traumatic hydrocephalus. An initial midbrain contusion (including the periaqueductal region) was demonstrated on imaging, and initial aqueductal obstruction (and elevated ICP) was managed with a temporary EVD. About three weeks later the patient deteriorated secondary to hydrocephalus. MRI demonstrated enlargement of the lateral and third ventricle, mild downward bowing of the third ventricular floor with ballooning of the aqueduct and absorption of the contusions. All these suggest a secondary web in the distal aqueduct causing local obstruction. This web most probably occurred as a local scarring secondary to the periaqueductal region trauma.

Previous reports of successful ETV's among patients with a history of intracranial bleed have been reported [14]. Yet, the current patient had a unique mechanism localized to the distal aqueduct. Miki et al published two cases with post-traumatic obstructive hydrocephalus that were treated endoscopically [15]. The first, a 16-year-old child suffered from chronic headaches years before the trauma, which had exacerbated following the trauma. MRI showed similar findings to our patient – a web in the distal end of the aqueduct. The patient underwent successful aqueductoplasty. The second case, a 35-year-old man had suffered a head trauma with a normal CT scan; however, 2 months later had a new triventricular hydrocephalus without a clear web in the aqueduct and without aqueductal dilation. He had a successful ETV.

Aqueductoplasty is a feasible alternative to ETV when the aqueduct is obstructed [6]. However, it necessitates utilizing a flexible endoscope, and risks periaqueductal region injury, leading to dysconjugate eye movements [16].

Performing an ETV in a posttraumatic hydrocephalus may have specific risks, especially regarding the anatomy of the floor of the third ventricle which may have hemosiderin staining obstructing usual anatomical landmarks. Thus, extreme caution should be applied, and if the procedure cannot be safely performed, a shunt should be placed.

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