2013

Transient obstructive hydrocephalus due to intraventricular hemorrhage: A case report and review of literature

Eriks A. Lusis
Washington University School of Medicine

Ananth K. Vellimana
Washington University School of Medicine

Wilson Z. Ray
Washington University School of Medicine

Michael R. Chicoine
Washington University School of Medicine

Sarah C. Jost
Swedish Medical Center

CASE REPORT
J Clin Neurol 2013;9:192-195

Print ISSN 1738-6586 / On-line ISSN 2005-5013
http://dx.doi.org/10.3988/jcn.2013.9.3.192

Open Access

Transient Obstructive Hydrocephalus due to Intraventricular Hemorrhage: A Case Report and Review of Literature

Eriks A. Lusis,[a] Ananth K. Vellimana,[a] Wilson Z. Ray,[a] Michael R. Chicoine,[a] Sarah C. Jost[b]

[a]Department of Neurological Surgery, Washington University School of Medicine, St. Louis, MO, USA
[b]Department of Neurosurgery, Swedish Medical Center, Seattle, WA, USA

Received February 8, 2012
Revised July 9, 2012
Accepted July 9, 2012

Correspondence
Ananth K. Vellimana, MD
Department of Neurological Surgery,
Washington University
School of Medicine,
660 S. Euclid Ave, Campus Box 8057,
Saint Louis, MO 63110, USA
Tel +1-314-362-3552
Fax +1-314-362-2107
E-mail vellimana@wustl.edu

Background Acute transient obstructive hydrocephalus is rare in adults. We describe a patient with intraventricular hemorrhage (IVH) who experienced the delayed development of acute transient hydrocephalus.

Case Report A 33-year-old man with a previously diagnosed Spetzler-Martin Grade 5 arteriovenous malformation presented with severe headache, which was found to be due to IVH. Forty hours after presentation he developed significant obstructive hydrocephalus due to the thrombus migrating to the cerebral aqueduct, and a ventriculostomy placement was planned. However, shortly thereafter his headache began to improve spontaneously. Within 4 hours after onset the headache had completely resolved, and an interval head CT scan revealed resolution of hydrocephalus.

Conclusions In patients with IVH, acute obstructive hydrocephalus can develop at any time after the ictus. Though a delayed presentation of acute but transient obstructive hydrocephalus is unusual, it is important to be aware of this scenario and ensure that deterioration secondary to thrombus migration and subsequent obstructive hydrocephalus do not occur.

J Clin Neurol 2013;9:192-195

Key Words acute hydrocephalus, intraventricular hemorrhage, arteriovenous malformation, transient hydrocephalus.

Introduction

While obstructive hydrocephalus is a relatively common and potentially life-threatening condition, transient obstructive hydrocephalus is a rare condition in adults. Transient obstruction of cerebrospinal fluid (CSF) flow through the ventricular system has been reported to result from systemic causes such as lead[1] and carbon monoxide poisoning[2] as well as CNS infections and meningitis.[3] Previous case reports have also described spontaneous resolution of obstructive hydrocephalus after intraventricular hemorrhage (IVH) in neonates[4] and adults.[5] Herein we report a 33-year-old male with a large arteriovenous malformation (AVM) who presented with a small IVH and subsequently developed transient acute hydrocephalus.

Case Report

The patient was a 33-year-old male with a previously diagnosed Spetzler-Martin Grade 5 AVM in the left parietal lobe that had been treated conservatively. On the night prior to presentation he developed a sudden-onset, diffuse, severe headache that radiated to the suboccipital region. There was no nausea, vomiting, or loss of consciousness.

On the initial assessment the patient was awake, alert, and fully oriented. There were no cranial nerve, motor, or sensory deficits. A fundoscopic examination revealed no papilledema or retinal hemorrhage. A CT scan at admission revealed IVH within the left frontal horn, both occipital horns, both temporal horns, and the third ventricle. There was no ventriculomegaly (Fig. 1). Comparison of this scan with previous CT scans did not suggest the early hydrocephalus. We were concerned about the possibility of recurrent hemorrhage or the development of hydrocephalus, and hence the patient was ad-

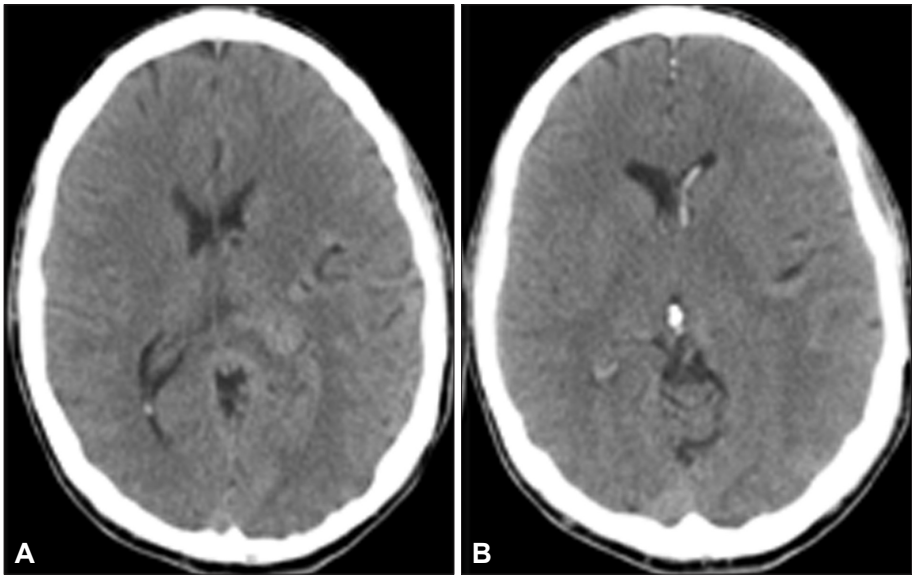

Fig. 1. A and B: Axial CT scan without contrast obtained at presentation demonstrates intraventricular hemorrhage within the left frontal horn, right occipital horn, and the third ventricle without evidence of ventriculomegaly.

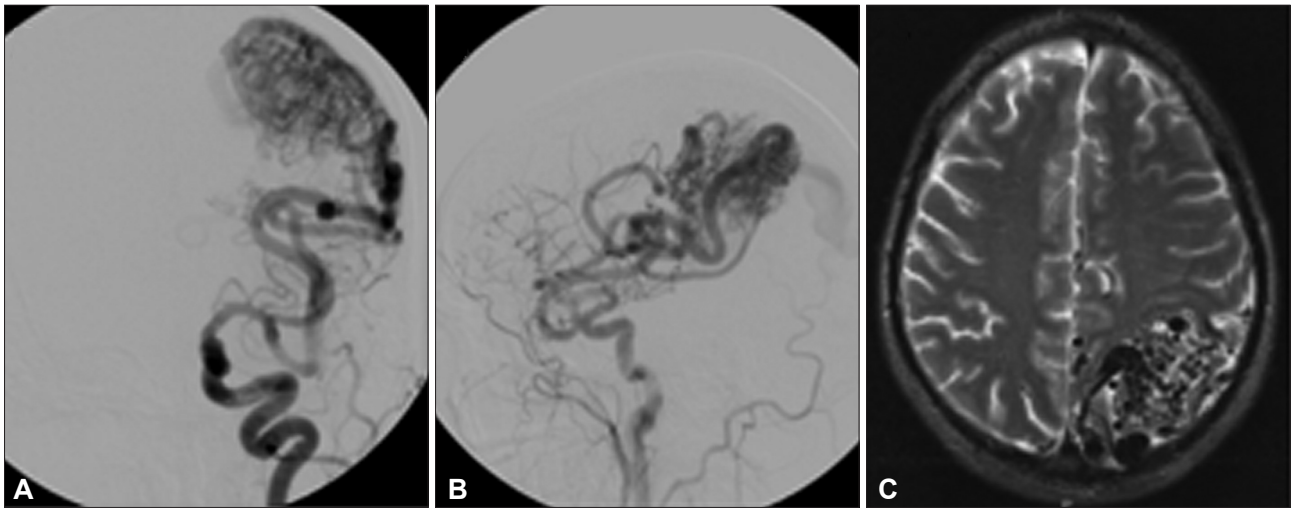

Fig. 2. A and B: Anteroposterior (A) and lateral (B) cerebral angiograms demonstrate the patient's large left parietal AVM with primary blood supply from branches of the left middle cerebral artery and also the anterior and posterior cerebral arteries. C: Axial T2-weighted MRI scan demonstrating multiple flow voids within the AVM. AVM: arteriovenous malformation.

mitted to the neurosurgical intensive care unit (ICU) for close observation. A catheter angiogram and MRI scan obtained on the following morning revealed no change in the AVM (Fig. 2). A repeat head CT scan on the second day of hospitalization demonstrated stable IVH without ventriculomegaly. The patient was then transferred from the ICU to the neurosurgical ward for observation.

Approximately 40 hours after initial presentation, the patient experienced severe headache with associated nausea. An urgent CT scan revealed the interval development of hydrocephalus, with the suggestion that a portion of the intraventricular thrombus had migrated from the left lateral ventricle to the junction of the third ventricle and the cerebral aqueduct (Fig. 3). There was no recurrence of IVH or subarachnoid hemorrhage. The patient was immediately transferred back to the ICU with anticipation of a need for ventriculostomy placement.

Shortly thereafter his headache began to improve. On examination the patient appeared more comfortable and was not lethargic. He remained without focal neurological deficits. The headache resolved within 4 hours of onset. A ventricular drain was not placed, and the patient was monitored. Head CT performed 6 hours after the onset of headache and the first CT revealing hydrocephalus, showed an interval decrease in the ventricular size and further clot migration through the cerebral aqueduct (Fig. 4). There were no further clinical events during the hospital stay and the patient was discharged without further sequelae from his transient hydrocephalus.

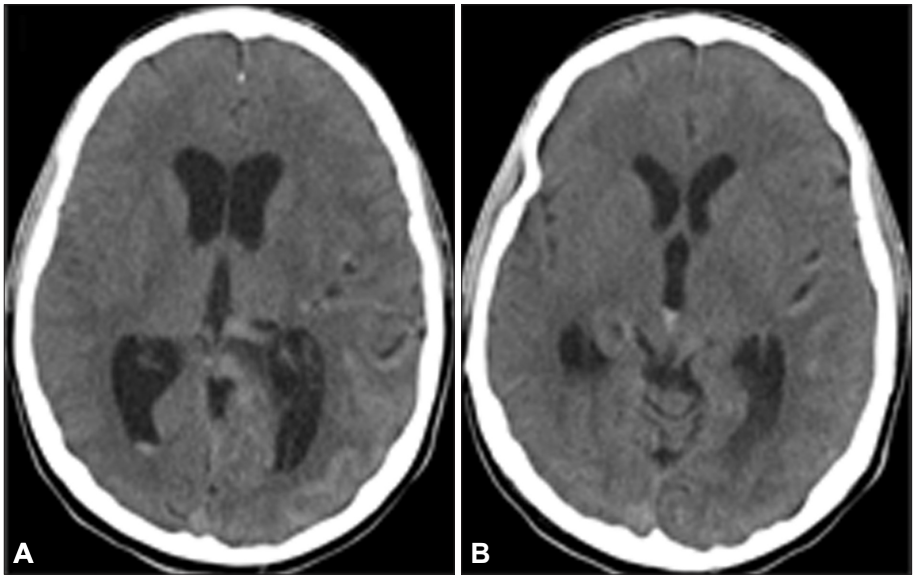

Fig. 3. A and B: Axial CT scan obtained 40 hours after presentation reveals the interval development of hydrocephalus with suggestion that a portion of the intraventricular thrombus had migrated from the left lateral ventricle to the junction of the third ventricle and cerebral aqueduct.

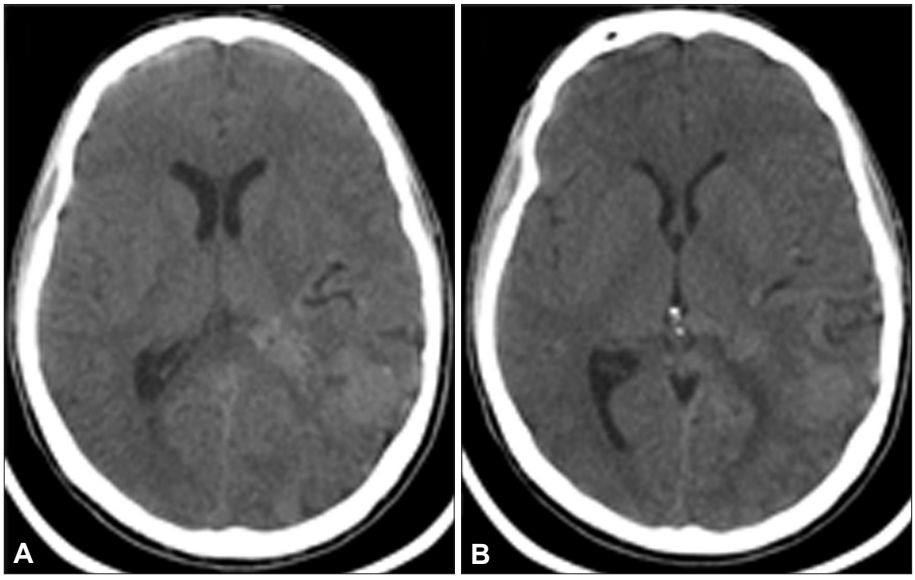

Fig. 4. A and B: Axial CT scan obtained approximately 6 hours after the development of hydrocephalus reveals resolution of the hydrocephalus and disappearance of the third-ventricle clot.

Discussion

Obstructive hydrocephalus is common after IVH due to obstruction of normal CSF flow and absorption by the thrombus. The presence of clinically significant ventricular dilatation in this condition typically requires external ventricular drainage for an extended period, and may ultimately necessitate ventricular shunting. A delayed development of acute but transient obstructive hydrocephalus is unusual in this patient population. Our patient experienced acute development of hydrocephalus approximately 48 hours after the onset of symptoms of IVH, and this resolved spontaneously over 6 hours.

From our case and a review of the literature, it appears that acute hydrocephalus subsequent to aqueductal obstruction by a migratory clot can present in two ways. In the first situation the hydrocephalus develops soon after the ictal event and the patient may actually present with the symptoms of acute hydrocephalus.[4-7] It this situation, it is possible that areas of the intraventricular clot may begin to break away before the clot becomes organized and hardens, with these fragments subsequently migrating along the direction of CSF flow leading to obstruction soon after the initial hemorrhage. In the second situation, of which our case is an example, there is a period of stability after the initial hemorrhage followed by sudden clinical deterioration and the development of hydrocephalus due to aqueductal obstruction by a migrating clot.[8,9] The delayed development of hydrocephalus in our patient may be explained by the natural time course of clot dissolution by the fibrinolytic activity of CSF, which is increased after IVH;[10,11] however, this increase is not sufficient for rapid and complete

resolution of a clot.[10,12] We speculate that the increased fibrinolytic activity in our case could have contributed to partial breakdown of the existing third-ventricle clot producing a mobile fragment, which migrated in the direction of CSF flow and was large enough to lodge at the aqueduct, resulting in acute obstructive hydrocephalus. Thus, it is important to consider this possibility when making observational and discharge decisions in patients with IVH, and especially when patients who are discharged after IVH return to the hospital with the sudden onset of symptoms associated with elevated intracranial pressure.

Another interesting aspect of our case is the spontaneous clearing of the clot from the aqueduct and resolution of hydrocephalus, which contrasts with most cases of acute hydrocephalus having to be relieved by CSF drainage. In our case it is likely that the ongoing fibrinolytic activity of the CSF made the obstructing clot fragment fragile, thus allowing the increased intraventricular pressure and hyperdynamic CSF flow resulting from acute obstruction[13] to break down the clot fragment further and permit its passage through the aqueduct, relieving the acute hydrocephalus. Also, proximal aqueductal dilatation, which has been observed in cases of acute hydrocephalus, may have further facilitated the passage of the clot. While spontaneous passage of a clot obstructing the aqueduct occurred in our patient and in four other cases,[4-6,14] it did not occur in other reported cases, thereby necessitating CSF drainage in those patients.[7-9] Therefore, when patients present with acute hydrocephalus due to aqueductal obstruction by a clot, CSF drainage should be performed immediately (as is done routinely) without waiting for spontaneous resolution.

In conclusion, patients with IVH may develop acute or delayed obstructive hydrocephalus. While most cases of clinically significant hydrocephalus require CSF drainage, in rare situations the hydrocephalus may resolve spontaneously due to fibrinolysis and clot migration, as observed in our patient.

Conflicts of Interest

The authors have no financial conflicts of interest.

REFERENCES

1. Sharma RR, Chandy MJ, Lad SD. Transient hydrocephalus and acute lead encephalopathy in neonates and infants. Report of two cases. *Br J Neurosurg* 1990;4:141-145.
2. Prabhu SS, Sharma RR, Gurusinghe NT, Parekh HC. Acute transient hydrocephalus in carbon monoxide poisoning: a case report. *J Neurol Neurosurg Psychiatry* 1993;56:567-568.
3. Dubey AK, Rao KL. Pathology of post meningitic hydrocephalus. *Indian J Pediatr* 1997;64(6 Suppl):30-33.
4. Abubacker M, Bosma JJ, Mallucci CL, May PL. Spontaneous resolution of acute obstructive hydrocephalus in the neonate. *Childs Nerv Syst* 2001;17:182-184.
5. Hagihara N, Abe T, Inoue K, Watanabe M, Tabuchi K. Rapid resolution of hydrocephalus due to simultaneous movements of hematoma in the trigono-occipital horn and the aqueduct. *Neurol India* 2009;57:357-358.
6. Inamura T, Kawamura T, Inoha S, Nakamizo A, Fukui M. Resolving obstructive hydrocephalus from AVM. *J Clin Neurosci* 2001;8:569-570.
7. Yoshimoto Y, Ochiai C, Kawamata K, Endo M, Nagai M. Aqueductal blood clot as a cause of acute hydrocephalus in subarachnoid hemorrhage. *AJNR Am J Neuroradiol* 1996;17:1183-1186.
8. Gupta SK, Sharma T. Acute post-traumatic hydrocephalus in an infant due to aqueductal obstruction by a blood clot: a case report. *Childs Nerv Syst* 2009;25:373-376.
9. Khan R, Mamourian AC, Radwan T. Utility of multislice computed tomography and reformatted images: identification of migratory intraventricular clot exacerbating obstructive hydrocephalus. *J Neurosurg* 2008;109:156-158.
10. Tsementzis SA, Honan WP, Nightingale S, Hitchcock ER, Meyer CH. Fibrinolytic activity after subarachnoid haemorrhage and the effect of tranexamic acid. *Acta Neurochir (Wien)* 1990;103:116-121.
11. Wu KK, Jacobsen CD, Hoak JC. Plasminogen in normal and abnormal human cerebrospinal fluid. *Arch Neurol* 1973;28:64-66.
12. Tovi D, Nilsson IM, Thulin CA. Fibrinolytic activity of the cerebrospinal fluid after subarachnoid haemorrhage. *Acta Neurol Scand* 1973;49:1-9.
13. Quencer RM. Intracranial CSF flow in pediatric hydrocephalus: evaluation with cine-MR imaging. *AJNR Am J Neuroradiol* 1992;13:601-608.
14. Nomura S, Orita T, Tsurutani T, Kajiwara K, Izumihara A. Transient hydrocephalus due to movement of a clot plugging the aqueduct. *Comput Med Imaging Graph* 1997;21:351-353.