Child's Nerv Syst (2000) 16:402–405

BRIEF COMMUNICATION

Liana Beni-Adani
Vitaly Siomin
Yoram Segev
Sara Beni
Shlomi Constantini

Increasing chronic subdural hematoma after endoscopic III ventriculostomy

Received: 28 December 1999

L. Beni-Adani (✉) · V. Siomin
S. Constantini
Division of Pediatric Neurosurgery,
Dana Children's Hospital,
Tel-Aviv Sourasky Medical Center,
6 Weizman Street,
Tel-Aviv, Israel
e-mail: lianaadani@hotmail.com
Tel.: +972-3-6974686
Fax: +972-3-6973451

Y. Segev
Department of Radiology,
Tel-Aviv Sourasky Medical Center,
Tel-Aviv, Israel

S. Beni
Department of Pediatrics,
Sheba Medical Center,
Tel-Hashomer, Tel-Aviv, Israel

Abstract *Object:* Endoscopic III ventriculostomy (ETV) is an effective and a rather safe treatment for noncommunicating hydrocephalus secondary to aqueductal stenosis and other obstructive pathologies. Though not devoid of risk, ETV is increasingly replacing shunt operations, and it prevents related complications, including overdrainage. *Methods:* We report a rare case of a large chronic subdural hematoma (ChSDH) after ETV in a patient with aqueductal stenosis. Three weeks after he was shunted elsewhere, he presented to us with clinical symptoms of intracranial hypotension and overdrainage. ETV was performed and the shunt removed uneventfully. On routine postoperative MRI a few weeks later, a large ChSDH was noted, the patient being totally asymptomatic. Since the ChSDH grew significantly, causing a mass effect on the follow-up MRI, it was finally drained. Large and increasing ChSDHs have previously been reported secondary to overdrainage after shunt placement, but not after ETV. *Conclusions:* We conclude that though rare, a ChSDH may evolve even after ETV, if there is a substantial decrease in previously elevated intracranial pressure.

Keywords Endoscopic III ventriculostomy · Overdrainage · Chronic SDH · Shunt · Complications

Introduction

Overdrainage is a well-known complication of ventriculoperitoneal shunt operations [1, 5, 11, 14, 16]. Its clinical manifestations include nausea, vomiting, headache, and decrease in the level of consciousness [11, 14]. These symptoms are often aggravated when patients change position from supine to upright, thus distinguishing them from symptoms of elevated intracranial pressure (ICP). Radiological characteristics may include dural enhancement on MRI, slit ventricles, and formation of a chronic subdural hematoma (ChSDH) or effusion, which can be either asymptomatic or symptomatic [5, 6, 14–16]. Chronic subdural hematomas may occur within days to years after a shunt operation; they may require surgical treatment, and are more common in adults [5, 11, 14, 15].

In recent years, endoscopic III ventriculostomy (ETV) has become an accepted method of treatment for noncommunicating hydrocephalus, replacing shunt procedures in many cases. It has been successfully used in aqueductal stenosis, tectal plate tumors, posterior fossa tumors, and meningomyelocele-associated hydrocephalus [2, 4, 8–10, 17]. Some patients who developed slit ventricle syndrome after shunt insertion, being dependent on CSF drainage, have also successfully undergone ETV and had their shunts removed [1, 2, 8]. Most series report success rates of more than 70% in adults and 30–70% in infants for ETV [2–4, 8, 9, 17], depending on the pathology that originally caused the obstructive hydrocephalus.

While complications after ETV are rare in experienced hands, they may be fatal. Possible complications include CSF leak, meningitis, postoperative memory def-

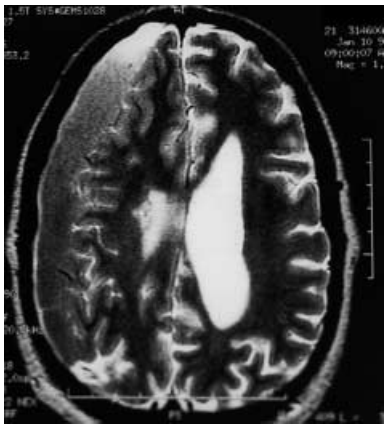

Fig. 1 An axial FSE-T2 MR image of the brain shows a mixed-intensity right subdural collection. A mass effect is noted with impression on the ipsilateral sulci and ventricle, and some midline shift

icit, hemiparesis, midbrain damage, hypothalamic dysfunction, ventriculitis, basilar artery injury including aneurysm formation, massive subarachnoid bleeding, and arrhythmia with cardiac arrest [2, 4, 6–9, 12, 16–18]. A rare case of *acute* contralateral massive subdural collection that developed within hours after ETV was also reported [13]. However, we could not find any reports in the literature of significant *chronic* subdural hematomas as a complication after ETV that developed and grew weeks after the operation and ultimately needed surgical drainage. This was also the only case observed in our series of more than 100 ETVs.

As overdrainage causing ChSDH is usually related to shunt operations and is uncommon in the absence of a drainage device, this paper discusses the possible etiology and clinical consequences of this rare complication after ETV.

Case report

A 20-year-old soldier presented at another hospital with severe headaches, acute agitated state, and confusion. Upon clinical examination, he was found to have papilledema, to be slightly confused but cooperative, and to have no other focal neurological deficits. A CT of the head showed obstructive hydrocephalus, with a ballooned III ventricle and a rather small IV ventricle. He was immediately taken to surgery, and a medium-pressure ventriculoperitoneal shunt was inserted on the right side. The patient improved immediately after the operation. The postoperative CT scan showed the ventricular catheter in place, with drained but not very small ventricles. However, a few days after surgery the patient complained of a severe headache, nausea, sleepiness, and vertigo. He was unable to rise from a supine to a sitting position. After 3 weeks of conservative treatment his symptoms remained very severe.

The patient presented to us with the classic clinical picture of intracranial hypotension. At this point an MRI was performed, which showed aqueductal stenosis and well-drained ventricles. No subdural collection was present. We decided to remove the shunt and perform an ETV procedure. The shunt was first externalized and closed to enable a preliminary ballooning of the ventricles, in order to facilitate insertion of the endoscope. An uneventful ETV was performed through a right frontal burr hole, and the shunt was removed. The patient recovered uneventfully and left the hospital 2 days after the procedure. On a routine postoperative MRI performed 2 weeks after ETV, a right ChSDH was noted. Since the patient was asymptomatic, we decided simply to observe him and to repeat the MRI 3 weeks later. The patient remained asymptomatic, but the second MRI showed a significant increase in the volume of the ChSDH compared with the first postoperative MRI, this time also causing a marked mass effect (Fig. 1). Since the images unequivocally showed an evolving process, the patient was operated on, and the SDH was drained out through a frontal burr hole leaving a subdural drain for 2 days. This procedure resolved the problem, and the patient has remained perfectly well. Follow-up scans several weeks later did not show reaccumulation of the fluid.

Discussion

Chronic subdural hematoma is a well-known complication after ventriculoperitoneal shunt insertion, and is considered to be an overdrainage-related phenomenon that may or may not cause symptoms [5, 11, 14, 15]. Subdural effusions, which are not genuine subdural hematomas, are often seen after craniotomies and other neurosurgical manipulations, but most of them are absorbed postoperatively, do not show any evolution, unless infection or bleeding complications are present, and do not, usually, need surgical intervention. To complicate the picture further, what seems on CT scan to be a subdural collection may actually be either a simple effusion or a ChSDH. Even in the context of a post-shunt phenomenon, effusions usually have a more benign course, and most of them will not require surgical treatment. However, a true ChSDH will usually require surgical intervention and drainage owing to the enlargement in volume, existing mass effect, or evolving symptoms [11, 14, 15].

In recent years endoscopic III ventriculostomy has replaced shunting for many noncommunicating hydrocephalus patients [1–4, 8, 10, 17]. Thus, complications were avoided, especially those related to foreign body placement and overdrainage. Yet this procedure is not without risk, and significant complications have been described [2, 6, 7, 12, 13, 16], some of them fatal. However, neither intracranial hypotension nor evolving ChSDHs are recognized as common complications of ETV.

Even though brain collapse and subdural collection may be caused immediately after removal of the endoscope from the ventricle, unless a strict surgical technique is applied to prevent it, the collection will usually disappear within days and significant ChSDHs needing drainage will not occur. Mohanty described an unusual case of *acute* massive subdural collection formation after ETV. The patient deteriorated in the recovery room immediately after the ETV procedure. He went into cardiorespiratory arrest, but was resuscitated in time and the fluid was

successfully drained [13]. In that case, however, the collection probably developed during the operation, and became manifest within a very short time period.

The mechanism of ChSDH formation and growth within weeks after ETV, as appeared in our case, is not clear. No draining device was present as the shunt was removed in the same session as the ETV was performed. One possible explanation for the phenomenon is that abrupt drainage of CSF during ETV may create a large space between the dura and the brain. This space may gradually enable development of subdural or epidural fluid collections. This may happen even though we usually flush continuously with Ringer during ETVs, and close the wound promptly as soon as the peal-away – through which the endoscope has been introduced – is removed. In a series of 103 patients who underwent III ventriculostomy for noncommunicating hydrocephalus, Jones described 2 patients with subdural effusions [8]. This, does not, however, explain well enough why the ChSDH had grown on follow-up several weeks later, as revealed by comparison of the first and second post-ETV MR images.

The second hypothesis may give a more physiological than a technical explanation. In patients suffering from chronic long-standing intracranial pressure, a decrease in ICP may change the regulation of CSF formation. This regulation may be more vulnerable in a patient who has sustained several changes in ICP within a rather short time.

If we analyze the events in the present case, we find that abrupt changes in ICP may indeed have occurred several times: high ICP was present on the patient's first admission; a shunt was then inserted, probably causing a dramatic decrease in ICP; later, in preparation for ETV, the shunt was externalized and closed to enlarge the ventricles, again causing a rise in ICP. Finally, ETV was performed, with an immediate intraoperative drop in ICP.

It is not clear whether a previously shunted patient has a greater chance of developing ChSDH after ETV than one in whom ETV is performed as a first procedure. Baskin recently described 16 previously shunted patients who have undergone ETV [1]. This series included 22 patients who developed slit-ventricle syndrome after an initial shunt insertion and several subsequent revisions. Sixteen of these patients, who were dependent on CSF drainage, underwent ETV to have the shunt removed. In this series, complications were limited to transient short-term memory loss in 2 patients, which resolved within 6 months. Of these 16 patients, 62.5% experienced resolution or improvement in symptoms, and successfully remained shunt-free after a median follow-up period of 18.8 months [1].

Even in shunted patients, it is not clear which patients will develop overdrainage clinically or radiologically. However, we may speculate that a patient who developed such symptoms after a shunt operation would be more prone to develop a similar picture after an ETV.

Finally, we believe that our present patient had a rare complication of ChSDH formation and growth, which was related to and probably a complication of the ETV performed several weeks earlier. Since ETV is becoming the treatment of choice for many pathologies causing noncommunicating hydrocephalus, all possible complications should be acknowledged when this option is presented to the patient. Routine postoperative MR films may be an important diagnostic tool even in asymptomatic patients.

References

1. Baskin JJ, Manwaring KH, Rekate HL (1998) Ventricular shunt removal: the ultimate treatment of the slit ventricle syndrome. J Neurosurg 88:478–484
2. Brockmeyer D, Abtin K, Carey L, Walker ML (1998) Endoscopic third ventriculostomy: an outcome analysis. Pediatr Neurosurg 28:236–240
3. Buxton N, Macarthur D, Mallucci C, Punt J, Vloeberghs M (1998) Neuroendoscopic third ventriculostomy in patients less than 1 year old. Pediatr Neurosurg 29:73–76
4. Cinalli G, Sainte-Rose C, Chumas P, Zerah M, Brunelle F, Lot G, Pierre-Kahn A, Renier D (1999) Failure of third ventriculostomy in the treatment of aqueductal stenosis in children. J Neurosurg 90:448–454
5. Decq P, Barat JL, Duplessis E, Leguerinel C, Gendrault P, Keravel Y (1995) Shunt failure in adult hydrocephalus: flow-controlled shunt versus differential pressure shunts – a cooperative study in 289 patients. Surg Neurol 43:333–339
6. Grant JA, McLone DG (1997) Third ventriculostomy: a review. Surg Neurol 47:210–212
7. Handler MH, Abbott R, Lee M (1994) A near-fatal complication of endoscopic third ventriculostomy: case report. Neurosurgery 35:525–527
8. Jones RF, Kwok BC, Stening WA, Vonau M (1994) Neuroendoscopic third ventriculostomy. A practical alternative to extracranial shunts in noncommunicating hydrocephalus. Acta Neurochir Suppl (Wien) 61:79–83
9. Jones RF, Stening WA, Brydon M (1990) Endoscopic third ventriculostomy. Neurosurgery 26:86–91
10. Jones RFC, Kwok BTC, Stening WA, Vonau M (1994) The current status of endoscopic third ventriculostomy in the management of non-communicating hydrocephalus. Minim Invasive Neurosurg 37:28–36
11. Major O, Fedorcsak I, Sipos L, Hantos P, Konya E, Dobronyi I, Paraicz E (1994) Slit ventricle syndrome in shunt operated children. Acta Neurochir (Wien) 127:69–72
12. McLaughlin MR, Wahlig JB, Kaufmann AM, Albright AL (1997) Traumatic basilar aneurysm after endoscopic third ventriculostomy: case report. Neurosurgery 41:1400–1403

13. Mohanty A, Anandh B, Reddy MS, Sastry KV (1997) Contralateral massive acute subdural collection after endoscopic third ventriculostomy – a case report. Minim Invasive Neurosurg 40:59–61
14. Pudenz RH, Foltz EL (1991) Hydrocephalus. Overdrainage by ventricular shunts. A review and recommendations. Surg Neurol 53:200–212
15. Raftopoulos C, Massager N, Baleriaux D, Deleval J, Clarysse S, Brotchi J (1996) Prospective analysis by computed tomography and long-term outcome of 23 adult patients with chronic idiopathic hydrocephalus. Neurosurgery 38:51–59
16. Schroeder HWS, Warzoc RW, Assaf JA, et al (1999) Fatal subarachnoid hemorrhage after endoscopic third ventriculostomy – case report. J Neurosurg 90:153–155
17. Teo C, Jones R (1996) Management of hydrocephalus by endoscopic third ventriculostomy in patients with myelomeningocele. Pediatr Neurosurg 25:57–63
18. Teo C, Rahman S, Boop FA, Cherny B (1996) Complications of endoscopic neurosurgery. Child's Nerv Syst 12:248–253