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Causes and treatment of intracranial haemorrhage complicating shunting for paediatric hydrocephalus

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Abstract Intracranial haematomas are a well-known complication of shunting procedures for hydrocephalic patients. Most are subdural haematomas, and epidural haematomas are much less common in this setting. Their aetiology is thought to be due to an overdrainage of cerebrospinal fluid and a rapid lowering of intracranial pressure, leading to the development of these haematomas. Since the advent of modern neuroimaging techniques, prompt diagnosis of postshunting intracranial haematoma has been possible even in asymptomatic patients. The choice between surgical and nonsurgical management of postshunting intracranial haematoma is a difficult and controversial issue, especially in asymptomatic patients. Several therapeutic options have been proposed for the treatment of postshunting intracranial haematoma. Evacuation of the haematoma by conventional neurosurgical methods with the implantation of a higher pressure valve system is the most common option adopted. Intraventricular haemorrhage is occasionally reported, chiefly in children with hydrocephalus associated with vein of Galen malformation.

Key words Shunt complications · Subdural haematomas · Epidural haematoma · Intraventricular haemorrhage · Hydrocephalus · Vein of Galen malformation

Introduction

Since Ames [1] developed a peritoneal catheter with a slit valve at its tip in 1967, there has been an increase in the frequency of ventriculoperitoneal (VP) shunting, which has become the procedure of choice in the treatment of hydrocephalus. VP shunting gives better results than other methods used to decompress the cerebral ventricles [22]. However, this procedure is also beset with a considerable number of complications, which may be related to shunt obstruction, infection [6] and excessive drainage of cerebrospinal fluid (CSF), such as slit ventricle syndrome, formation of intracranial haematomas (subdural, epidural and intraventricular haematomas), and the development of craniosynostosis and microcephaly [11, 13].

In this article, causes, incidence, management, and prevention of intracranial haematomas following VP shunting are discussed.

Subdural haematomas

Subdural haematoma (SDH) is a well-known complication after shunt operations for hydrocephalus, and it was described by Anderson in 1952 [2] and by Davidoff in 1963 [7]. Since then, its occurrence has been reported by several authors [4, 18, 26, 32, 35]. It is usually encountered in the setting of a large head with little brain parenchyma, as usually occurs in children with macrocrania and large ventricles on initial evaluation [10, 13, 23]. It can also follow shunting in elderly patients who have se-

vere brain atrophy [13]. Its incidence seems to be higher in adults (4%-23%) [18, 26, 32, 35] than in children (2.8%–5.4%) [4, 17, 18, 32] and in normal-pressure hydrocephalus (20–46%) [26, 31, 32, 35] than in hypertensive hydrocephalus (0.4%–5%) [4, 12, 18, 32]. In children with hydrocephalus associated with vein of Galen malformations, the incidence of postshunting subdural haematomas may reach about 10% [36].

The cause of postshunting SDH is thought to be overdrainage of CSF. According to Puca's review [32], an excessive CSF loss at the time of shunt insertion could contribute to the opening of the subdural space; a markedly negative ventricular fluid pressure could be facilitated by the upright position. In addition, it has been demonstrated that the opening pressure of the valves could change postoperatively, resulting in an increased risk of overdrainage [23, 32]. Therefore, the brain tends to collapse, with tearing of bridging veins forming the haematoma extending into the subdural space. Minor traumatic events could increase the risk of rupture of bridging veins [35]. As the cause of postshunting SDH is considered to be overdrainage of CSF, it is possible to find an association with slit ventricle (Fig. 1).

With the advent of CT and MRI prompt diagnosis of postshunting SDH became possible even in asymptomatic patients [11, 33]. In a paediatric series, 58% of postshunt SDHs were asymptomatic [17]. The clinical picture of postshunt SDH varies from absence of symptoms to symptoms of shunt malfunction or a mass lesion [8, 11, 28, 33].

Various therapeutic options can be considered for the treatment of postshunting SDH; besides the usual methods of evacuating the SDH, implantation of a higher pressure valve system [4, 9] or closure of the shunt [9, 18] is commonly used. Recently some cases were managed with a programmable pressure valve, which may be useful in avoiding multiple surgical intervention [21]. Subdural drainage, subduroperitoneal shunt or craniotomy with membrane excision is often necessary [3, 17, 18, 35]. Refilling of the ventricular system with isotonic solution [9, 24, 39], removal of the bone flap that overlies the haematoma [9], and surgical reduction of the calvarium have occasionally been performed [30].

In order to prevent the formation of SDH after ventricular shunting, the usual precautions of minimal CSF loss at the time of ventricular catheter insertion, use of higher pressure valves, use of antisiphon valves to stop the overshunting that can be caused by siphoning [26, 31, 34, 41] and slow return to the full upright position in the immediate postoperative period [20] are necessary. III Ventriculostomy has been indicated in cases of noncommunicating hydrocephalus, decreasing the risk of postoperative complications [25].

The choice between surgical and nonsurgical management of postshunting SDH is a difficult and a controversial issue and requires the evaluation of various factors, such as the size and stage of the haematoma, its mass effect, the amount of fresh blood present, the age of the patient, and the clinical picture. Some authors feel that huge acute or subacute haematomas in children with closed fontanels usually require surgical treatment [9, 23, 24, 26, 32, 35]. The presence of clinical symptoms seems to be the most important factor for many authors [18, 28, 32, 38]. In the experience of Puca et al. [32] a nonsurgical policy should be followed in patients with long-standing hydrocephalus and macrocrania if they develop large haemorrhagic SDH and remain asymptomatic after shunting. On the other hand, in a paediatric series it was stressed that asymptomatic pericerebral collections could become symptomatic later and it would therefore be safer to treat all postshunt pericerebral collections, whether symptomatic or not [17].

Epidural haematomas

Epidural haematomas (EDHs) are an unusual complication of ventriculoperitoneal shunt in the treatment of chronic hydrocephalus (Fig. 2) and are much less common than postshunting SDHs [15, 20, 28]. To our knowledge, only six cases of EDHs after valve-regulated shunt placement have been reported in the English literature[14, 19, 20, 40, 42, 43]. Reviewing the cases of EDH attributable to external ventricular drainage, air or contrast ventriculography, or posterior fossa craniectomy allowed us to identify common factors, such as age less than 20 years, evidence of chronic hydrocephalus (macrocephaly or radiographic findings) and operation in the prone position [16, 20]. Massive bifrontal haemorrhage occurred in these cases with a mortality of up to 54% [16, 37, 42].

The mechanism of EDH formation is related to the rapid reduction in intracranial pressure as a result of overdrainage of the CSF, resulting in separation of the dura mater from the skull and tearing of small dural vessels [11, 14, 15, 28, 37, 40, 42]. According to Kalia [20], in some patients the skull-to-dura mater adhesion may be less strong than the dura mater-to-arachnoid adhesion, so that an EDH forms instead of the more common SDH. It is known that the dura mater in children and young adults is less adherent to the skull than in older adults, explaining why most reported cases of postventriculostomy EDH occur in children and young adults. In addition, arachnoidal scarring from previous trauma or infection might result in an anatomical obliteration of part of the subarachnoid space, causing adhesion of the brain to the dura mater, which would facilitate the formation of postshunting EDH.

At the time of diagnosis, as in cases of postshunting SDH, patients may be asymptomatic or present with symptoms of shunt malfunction or a mass lesion [11, 20, 28].

Postshunting EDHs may be treated by evacuation of the haematoma by craniotomy. In addition, placement of

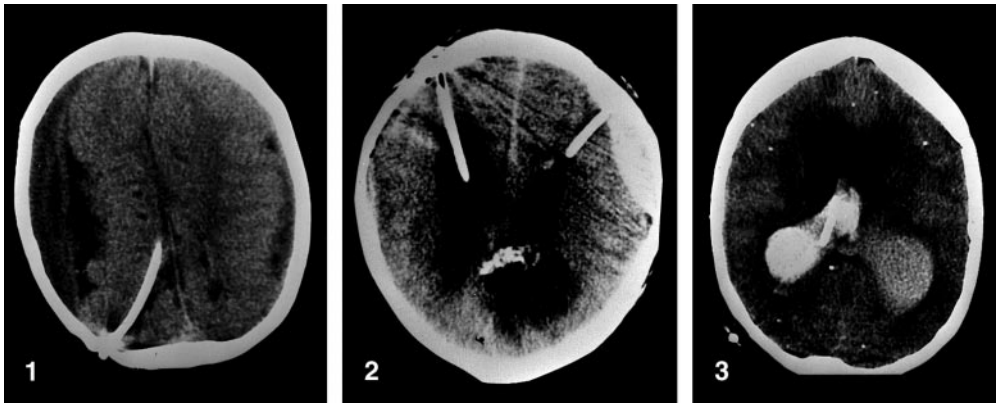

Fig. 1 Axial brain CT demonstrating a chronic subdural collection and slit ventricles

Fig. 2 Postoperative control axial brain CT demonstrating a small right epidural haemorrhage with a slight expansive effect

Fig. 3 Axial brain CT demonstrating an intraventricular haemorrhage attributable to a direct lesion from the catheter

a higher pressure valve system or closure of the shunt may be necessary [9]. Nonsurgical treatment of postshunting EDHs may be adopted in selected cases, according to rigid clinical and radiological parameters as discussed for traumatic EDHs [5, 27, 29].

Intraventricular haemorrhage

Intraventricular haemorrhage may occasionally complicate any shunt operation. It can occur after withdrawal of a ventricular catheter following bleeding from the choroid plexus veins and arteries or ependymal veins, or after insertion of a ventricular catheter (Fig. 3), as seen more commonly in children with hydrocephalus associated with vein of Galen malformation. In Schneider's series [36] of children with hydrocephalus associated with vein of Galen malformation, VP shunting was associated with a high incidence of complications that included intraventricular haemorrhage in 7 cases out of the 20 operated on. Haemorrhage was mild in 3 patients and moderate in 4. Despite angioembolization before VP shunt placement, 2 of the 7 patients with intraventricular haemorrhage suffered subsequent haemorrhage. All these cases of intraventricular haemorrhage occurred with ventricular catheters inserted from an occipital approach with concurrent use of low-pressure valves. The authors called attention to the high incidence of intraventricular haemorrhage, which may be due to disruption of engorged ependymal veins along the posterior wall of the ventricles. The presence of the engorged veins in the region of the occipital horn and the trigone in children with hydrocephalus associated with vein of Galen malformation may be seen on angiography. Therefore, in light of all this information, the authors suggest the use of medium-pressure valves and frontal approaches for ventricular catheter insertion in such children.

Conclusion

Intracranial haematomas, especially subdural haematomas, are a well-known complication of hydrocephalus shunting procedures. With the routine use of modern neuroimaging techniques, asymptomatic intracranial haematomas have been diagnosed after shunting, raising the problem of choosing the most appropriate management for such conditions. Reviewing the literature and considering the pathophysiology of these haematomas' formation, we conclude that the choice between surgical and nonsurgical management of postshunting subdural haematomas should be based on each patient's clinical presentation. These haematomas should only be treated when they are symptomatic. If they are not symptomatic they will merely be filling the subdural space that results from collapse of the brain after the hunting procedure, since there is a discrepancy in volume between the size of the brain and of the cranial cavity, as in the case of long-standing hydrocephalus with macrocrania.

Epidural haematomas and intraventricular haemorrhage can occasionally complicate any shunt operation.

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