Entrapment of the temporal horn: a form of focal obstructive hydrocephalus

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SUMMARY Three cases of a form of focal hydrocephalus are described which the authors term "entrapment of the temporal horn". Obstruction of one lateral ventricle in the region of the trigone isolates the temporal horn. Continued secretion of cerebrospinal fluid within the temporal horn causes it to behave as a mass lesion. In the cases described the causes of the condition were recurrent glioma, previous tuberculous meningitis and surgical excision of an arteriovenous malformation which extended into the trigone. Shunting of the trapped temporal horn provides satisfactory treatment.

We describe three cases of a syndrome in which obstruction of the trigone of the lateral ventricle seals off the temporal horn from the rest of the ventricular system. Continued secretion of cerebro-spinal fluid by the choroid plexus within the temporal horn leads it to expand into a cyst which behaves as a mass lesion. This entity which we have termed "entrapment of the temporal horn" is a form of focal hydrocephalus, which has previously attracted little notice. It is distinct from two other rare but well recognised forms of partial hydrocephalus: unilateral hydrocephalus caused by obstruction of one foramen of Monro and septation of the ventricular system after infantile meningitis.

Case reports

Case 1

This 23-year-old woman had undergone resection of a right temporal malignant glioma 2½ years previously followed by a course of radiotherapy. Eighteen months later a local recurrence of her tumour had been treated by further surgical resection and chemotherapy. At neither operation had the ventricular system been entered and she had been left with no neurological defect. One year later she developed severe progressive headache and drowsiness over a period of three days. On admission she was found to be confused and irritable with a marked left facio-brachial weakness and a tense sub-temporal decompression. On re-exploration there was found to be very little recurrent tumour within the temporal lobe but there was a cystic expansion of the right temporal

horn which contained 60 ml of clear colourless fluid resembling cerebro-spinal fluid. The communication of the temporal horn and the trigone was severely narrowed by fibrous tissue. This stenosis was opened up so that the temporal horn communicated freely with the trigone. After operation she made a rapid recovery although she was left with a left lower facial weakness and a homonymous lower quadrantanopia. She remained well until six months later when she died from a recurrence of her tumour.

Fig 1 Case 2. CT scan showing expanded right temporal horn displacing the lateral ventricles to the left.

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Case 2

This 35-year-old Asian housewife, had been treated for two months for tuberculous meningitis but had persistent head-

aches and had had two epileptic fits. A CT scan showed an area of low attenuation around the trigone of the right lateral ventricle. The headache improved spontaneously but a month later it recurred and she developed a slight left sided weakness. Over the next few days she became increasingly drowsy and was referred to the neurosurgical department. On examination she had a temperature of 36°C and no signs of meningeal irritation. She was well orientated but drowsy with a left homonymous hemianopia and a left hemiparesis, more marked in the arm than the leg. Haemoglobin was 14.1 gm, white cell count 7000/mm³ and sedimentation rate 25 mm in the first hour. CT scan (fig 1) showed expansion of the temporal horn of the right lateral ventricle with low attenuation of the surrounding white matter and a shift of the midline structures to the left. The choroid plexus of the right lateral ventricle was prominent. A right temporal craniotomy was carried out and the right middle temporal gyrus was incised to enter an expanded temporal horn which was 4 cm across. Communication with the trigone was obstructed by an engorged choroid plexus. The choroid plexus was largely destroyed with diathermy and it was hoped that the encysted temporal horn would drain through the cortical incision into the subarachnoid space. However, after operation she became progressively drowsy and the left hemiparesis worsened. A repeat CT scan showed that the cyst had re-formed and that there was considerable oedema of the surrounding white matter. The cyst was shunted into the right atrium through a Pudenz valve system. Thereafter the patient made an uneventful recovery. CT scan three weeks after insertion of the shunt showed that the cyst had resolved and six months later the patient remained symptom-free and had no neurological deficit.

Fig 2 (a) Case 3. CT scan: expanded left temporal horn compressing the remainder of the left lateral ventricle and causing midline shift. (b) Case 3. CT scan showing appearance after shunting of trapped temporal horn.

Fig 3 Case 3. CT scan showing position and extent of encysted temporal horn in the sagittal plane.

Fig 4 Case 3. CT scan showing position of encysted temporal horn in the coronal plane.

Case 3

This 30-year-old housewife had a subarachnoid haemorrhage when 32 weeks pregnant. On admission to hospital she had hesitancy of speech, a mild right hemiparesis and sensory inattention of the right limbs. CT scan showed blood in the lateral and third ventricles and a small left parietal intracerebral haematoma. Angiography revealed a 5 cm diameter left parietal arterio-venous malformation fed by enlarged terminal branches of the middle cerebral artery and draining into the superior sagittal sinus. Seven days after the haemorrhage, craniotomy and complete excision of the malformation was carried out. A deep extension of the malformation into the trigone of the left lateral ventricle was found and in this region small patches of muslin were used to obtain haemostasis. Check angiography three days later showed no residual malformation. Seventeen days after operation she went into labour and a healthy child was delivered by Caesarean section. After excision of the malformation, there was some improvement in her pre-operative neurological deficit but this fluctuated and CT scan showed that the bed of the malformation was occupied by a cystic collection of fluid extending into the temporal lobe and causing displacement of the midline structures. This cyst persisted despite three aspirations of brownish muddy fluid through the craniotomy incision. The craniotomy was then reopened and 40 ml of slightly cloudy colourless fluid was aspirated from beneath the site of the malformation through the previous cortical incision. This further procedure produced some improvement but her condition then levelled out and she was left with a marked expressive dysphasia and a spastic weakness of the right arm. Subsequent scans showed a persistent and well defined expansion of the left temporal horn which was causing considerable cerebral displacement (figs 2, 3 and 4). It was thought that scarring within the trigone of the lateral ventricle, perhaps partly provoked by the muslin patches, had obstructed the egress of cerebro-spinal fluid from the temporal horn. It was now three months since removal of her malformation. A Holter medium pressure shunt system was inserted so as to drain the entrapped temporal horn into the peritoneal cavity. This led to a rapid and marked neurological recovery. A scan four days after the shunt showed that the cyst had resolved and that there was no longer any cerebral shift. One year later she was looking after her family without any functional disability. She had an occasional slight hesitancy of speech and a right homonymous hemianopia but otherwise no residual neurological deficit. CT scan at this time showed no re-formation of the cyst but there was a small dense nodule in the region of the left trigone which was thought to represent calcifying muslin.

Discussion

Hydrocephalus results when the flow of cerebro-spinal fluid is impeded. When the block lies within the ventricular system, it is termed "obstructive hydrocephalus". Both active secretion of cerebro-spinal fluid by the choroid plexus and the pulse waves from the plexus expand the trapped part of the ventricular system,^{1 2} for the brain parenchyma has only a limited capacity to absorb cerebro-spinal fluid.³ Obstructive hydrocephalus usually results from compression of, or

a block within, the fourth ventricle, the aqueduct of Sylvius, or the third ventricle, leading to symmetrical dilatation of the lateral ventricles.

If part of the ventricular system is closed off from the rest, and if the sealed off section contains choroid plexus, a partial or focal hydrocephalus may result. This is a rare event but may occur in two circumstances. First, obstruction of one foramen of Munro may cause hydrocephalus confined to one lateral ventricle. This so-called unilateral hydrocephalus leads to the symptoms of raised intracranial pressure accompanied by the features of dysfunction of the affected cerebral hemisphere. Unilateral hydrocephalus may be caused by a wide range of lesions in the region of the foramen of Munro, including colloid cysts of the third ventricle, tumours of the septum pellucidum and thalamus, cysticercosis, congenital gliotic atresia of the foramen of Munro and after ventriculitis or surgical procedures within the lateral ventricle.⁴⁻⁸ If the obstruction cannot be removed, the condition is treated by shunting the affected ventricle.

The second situation where partial hydrocephalus may occur is after meningitis in infancy. If the infection has involved the ventricles, and especially if it has been caused by a gram negative organism, the patient may develop a number of thin veils or septa within the ventricles separating them into a number of compartments.⁹⁻¹² These septa may progress after the infection has been overcome.¹³ They consist of thin translucent veils of tissue which are most often situated in the lateral ventricles just behind the foramina of Munro,¹⁰ though there may be multiple septa throughout the ventricular system.⁹ Their pathogenesis is uncertain but it has been suggested that they arise from tufts of glial tissue which grow out from areas of the ventricular wall which have been denuded of ependyma by the preceding ventriculitis.⁹ This so-called compartmentalised or multi-loculated hydrocephalus is difficult to treat. Ventricular shunts by themselves often fail and open ventriculostomy may be required to divide the septa.^{11 13} Cerebral endoscopy has also been used as a lesser procedure to achieve the same end.¹⁴ However, regardless of treatment, the condition is associated with a very poor prognosis. Most of the affected children die and a high proportion of the survivors are left with a severe psychomotor retardation.^{10 11}

These two examples of partial hydrocephalus should be distinguished from the situation where a cyst projects into the brain from the wall of one ventricle in association with hydrocephalus.¹⁵ This occurs most often when a generalised hydrocephalus is associated with a focal brain injury. This may happen in infantile hydrocephalus, after an injury in adult life, or from a cerebral haemorrhage which has caused hydrocephalus as well as a focal disruption of brain

tissue. Tearing of the ependyma at the point of focal injury permits the growth of a diverticulum from the ventricular system into the damaged white matter at that point. Such a porencephalic cyst communicates freely with the ventricle and it can be treated by a single shunt placed anywhere within the ventricular system.

The entity which we describe here is quite different from these syndromes. It consists of obstruction of the trigone of one lateral ventricle so that the trapped or isolated temporal horn containing choroid plexus expands into a cyst. This gives rise to the symptoms of raised intracranial pressure as well as appropriate features of focal cerebral dysfunction such as dysphasia or a contralateral hemiparesis. In normal individuals, the ventricles of the brain are narrow slit-like cavities rather than the full-bodied chambers which their diagrammatic representations often suggest. Indeed the opposing wall of the ventricles often adhere so that their extreme lateral angles may be pinched off.¹⁶ The trigonal part of the lateral ventricle is further narrowed by the bulk of the choroid plexus. It does not seem surprising that obstruction can occur at this point and many neurosurgeons must have experienced the phenomenon we describe, yet it does not seem to have been reported previously as a defined clinico-pathological entity other than by Cairns and colleagues in 1941.¹⁷ They described three patients with the syndrome. In two patients the temporal horn entrapment resulted from a penetrating wound of the brain which had entered the ventricle. In the remaining case, an infant, the ventricular obstruction was caused by a subependymal haemorrhage.

The aetiology of the condition was different in each of our three patients. In the first case the trigone was stenosed by a limited recurrence of a glioma around the ventricle. The expansion of the temporal horn mimicked a solid recurrence of the tumour. Drainage of the cyst into the main body of the ventricle sufficed to give a remission for several months until the tumour recurred. In case 2, the trigone was obstructed from within by a swollen choroid plexus after tuberculous meningitis. Ventriculostomy into the subarachnoid space failed and a shunt was required. In the third case, entrapment of the temporal horn followed excision of an arteriovenous malformation which extended into the trigone. Fibrosis around muslin squares placed in the trigone to obtain haemostasis may have contributed to the obstruction.

Entrapment of the temporal horn should be suspected in patients who develop symptoms of an expanding temporal lobe mass after a condition which has involved the region of the trigone of the lateral ventricle. Our first patient was treated before the development of the CT scan, an investigation which should make the diagnosis of this condition easy by

showing a large low attenuation cyst in the temporo-parietal region in the position of the normal temporal horn. Judging from our experience there seems little to be said for attempted aspiration or open exploration of such cysts and drainage through a shunt system seems a satisfactory method of treatment.

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