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Postmeningitic Hydrocephalus in Infancy

Ventriculography with Special Reference to Ventricular Septa

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Summary. Postmeningitic hydrocephalus in infancy accounts for at least 30% (maybe even 40%) of all new paediatric hydrocephalics seen in the Cape Province. The causes of the marked racial preponderance are considered. Meningitis is more prone to cause a CSF pathway block at any site in infancy than later in life. On ventriculography ventricular septa in 23%, foramen of Monro obstruction in 13%, multiple CSF pathway block occasionally and intraventricular detritus causing filling defects and irregularities of the ventricular walls are the salient features. Birth trauma with intraventricular bleeding may on occasion cause similar changes.

Introduction

The high incidence of hydrocephalus in infancy and childhood encountered at our hospital has prompted a review of the literature and an analysis of cases seen over a 6-year period. It became apparent that many of our cases (almost 40%) were the result of meningitis, often with a blockage in the basal cisterns or over surface pathways. Also there was a massive preponderance of coloured (mulatto) and black (Bantu) infants in the postmeningitic group which was not apparent in the congenital and tumour groups [5].

Reports published in Europe indicate that the incidence of postmeningitic hydrocephalus is of the order of 20% [7, 8, 12]. Very little has been published on this topic from less advanced countries. A Johannesburg study [2] concluded that 'hydrocephalus is a common disease in Bantu infants' but was not able to give any reasons. Almost half (51) of their 110 cases were of the communicating variety and in only four was meningitis held responsible. In a more recent Rhodesian series of 133 cases, it also emerged that only four of their cases were postinfective [14]. In both papers the accent was on an analysis of the results of various shunting operations. A short report from Khartoum indicates that the incidence of hydrocephalus amongst infants and children in the Sudan is high [1]. This is accounted for, in part at least, by the correspondingly high incidence of 'cerebrospinal meningitis' because of their similar geographic distribution and seasonal fluctuations.

It emerges from these reports from underdeveloped and developing countries that there are considerable problems in early referral, aetiology, and after-care following shunting [14]. As in other centres mentioned, we also attest to these difficulties.

Series

From amongst 364 new hydrocephalic infants and children seen over a 6-year period (1971-1976) there were 105 who definitely had had meningitis (28.8%). The breakdown by race is: coloured 73 (69.5%); Bantu 27 (25.7%); white 5 (4.8%). All cases with myelomeningocoele have been excluded from the study. All had an air study, usually ventriculography. In a few this was supplemented by dimer installation. (During the period of investigation CT was not available.) This group of 105 cases has been further studied in an effort to determine whether there are any features peculiar to the postinfective state which would enable us to make an aetiological diagnosis on ventriculography. As in other studies from Europe and especially Southern Africa, it is sometimes difficult to place a particular patient into one or other aetiological category because of the paucity of clinical information from rural clinics. For instance, difficulty was often experienced in placing a case into the postmeningitis or birth trauma category. The initial 122 cases of childhood hydrocephalus (1971-1972) revealed the following incidence: congenital, 22%; tumour, 20%; postmeningitic, 38%; birth trauma, 5%; indefinite, 15% [5].

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Findings

1. CSF Pathway Obstruction

The site of blockage to cerebrospinal fluid (CSF) flow may be anywhere within the system including the lateral ventricles (Fig. 3). In about two-thirds there is a communicating hydrocephalus, the site of the obstruction being in the basal cisterns or beyond at the level of the tentorial hiatus or even higher over the surface of the cortical sulci. The other sites –foramen of Monro, aqueduct, and fourth ventricle exit foramina – all share the remaining one-third in virtually equal proportions (Table 1). Of the 14 with obstruction at the level of the foramen of Monro, 12 also had septa and adhesions within the lateral ventricles.

Table 1. Ventriculography

Total	105	
Site of block:		
Basal cisterns	63	60%
Foramen of Monro	14	13%
Ventricle IV	12	
Aqueduct	12	
Surface pathways	3	
Septa or loculi	24	23%
Detritus	5	
Dimer adheres to walls	Occasionally	
Diverticula	Occasionally	

2. Ventricular Septa and Loculi

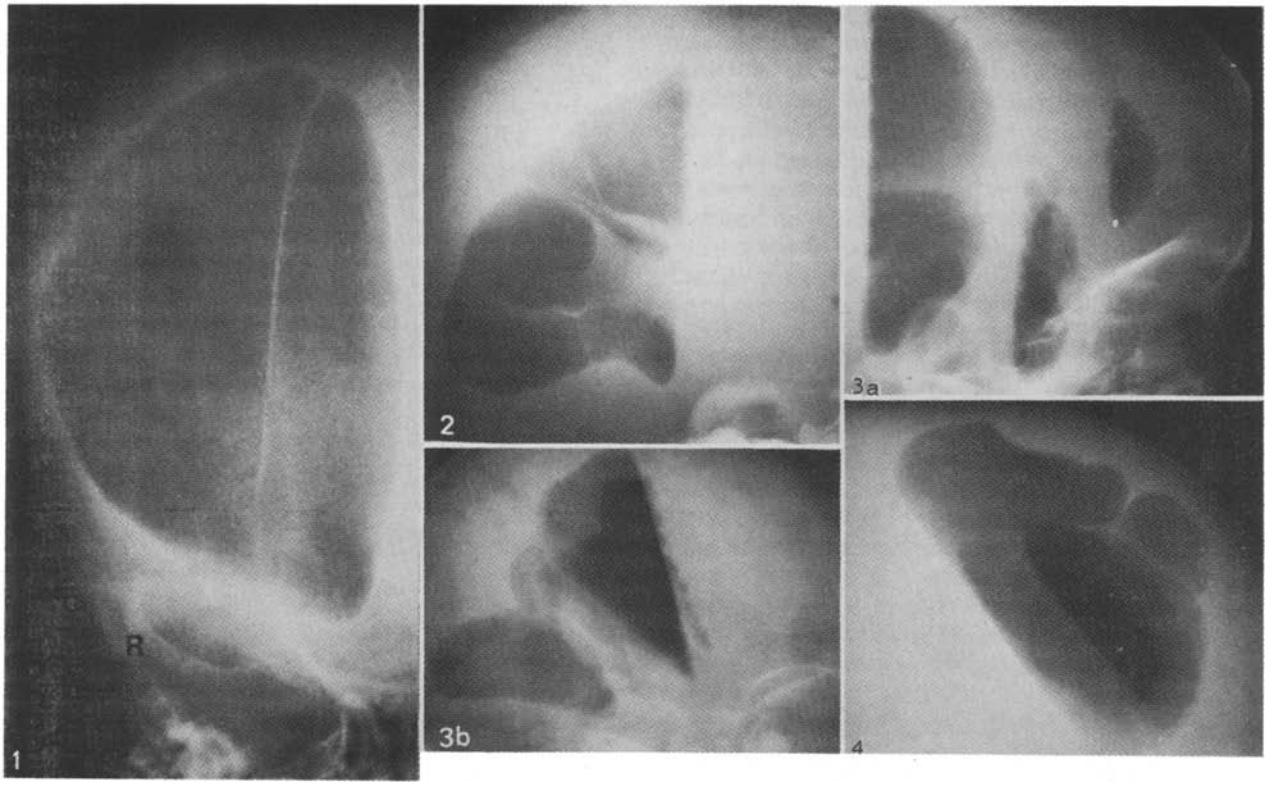
During the 6-year period all patients with ventricular septa and loculi were carefully scrutinised because initially it was assumed that all were the result of meningitis. From amongst the 364 new cases, 34 (coloured 19, black 12, white 3) were found to have septa most frequently in the lateral ventricles. On occasion they were also found within the third and fourth ventricles. These fibrinous strands vary greatly in thickness from that of gossamer (Figs. 1 and 2) on the one hand to the thick adhesions and septa which divided the cavity of a dilated lateral ventricle into separate loculi, sometimes preventing the passage of air from one loculus to another (Fig. 3). They occurred with equal frequency in the anterior and posterior parts of the lateral ventricles. These 34 cases were analysed by cause and found to be due to meningitis in 24, birth trauma in four, tumour in two and unknown in four. It is likely that the four undetermined cases may well have had meningitis and ventriculitis during the neonatal period, but clinical information was entirely lacking.

a. Postmeningitis Group. Of the 24 (coloured 14, Bantu 8, white 2) with proven meningitis, 18 (75%) were under the age of 3 months at the time of the infective illness, whilst 12 had meningitis in the first month of life. These ventricular septa are clearly the sequela of ventriculitis in the neonatal period or shortly thereafter. Repeat ventriculography was undertaken in only one after an interval of 2 months and the septum had disappeared (*E. coli* neonatal meningitis). Information on the CSF in the acute phase was available in only nine. The protein level was 5 g/l or more in eight of these and 1.40 g/l in the other. The organism was identified in six. Apart from two cases of meningococcal meningitis, the others were all due to enterobacteriaceae (two: *E. coli*, one: *Acinetobacter*, and one: *Enterobacter cloaca*). In one child of 2 years, a diagnosis of tuberculous meningitis was entertained because of the CSF findings, but the disease had been contracted at age 10 months, at which time no growth was obtained from purulent CSF presumably following an inadequate course of antibiotics. Although TBM is common especially in infants over the age of 6 months, we have not seen a case with intraventricular bands and adhesions. The interval between meningitis and ventriculography varied between 0 and 15 months. In 18 (75%) the latent period was 2 months or less, in four others, all of whom had neonatal meningitis, the interval before investigation was 4, 6, and 10 months. In a preponderance of this group the site of CSF pathway obstruction was in what is generally considered a very unusual location: twelve were obstructed at the foramen of Monro. The other sites of CSF pathway narrowing, i.e., aqueduct and fourth ventricle exit foramina, accounted for six and the remainder had a communicating hydrocephalus (basal cistern block).

b. Birth Trauma Group. In three of the four, cephalopelvic disproportion was a factor, whilst in the last case the baby was born 8 weeks prematurely after prolonged period of ruptured membranes (Fig. 4). Blood in the CSF had been obtained in all cases. The septa tended to be more substantial.

c. Tumour Group. There were two cases both with posterior fossa masses and both had a single diaphanous septum. A medulloblastoma had been removed from a 9-year-old about 6 years previously. The other, a 6-year-old child, had a juvenile pilocystic astrocytoma removed after initial ventriculography (ventricular CSF protein 0.6 g/l). At surgery there was no evidence of haemorrhage from the astrocytoma, which incidentally had evoked a local meningeal response. As far as can be gauged in this case, the ventricular septum had developed whilst the ventricular CSF protein was only slightly elevated.

d. Unknown Group. The four cases in this group had no clinical feature pointing to a cause. Age range between 5 months and 3 years with a mean of almost 2 years is much higher than that of the infected group. It is possible that one or more children may have had neonatal meningitis, but the clinical information had been lost in the interim.



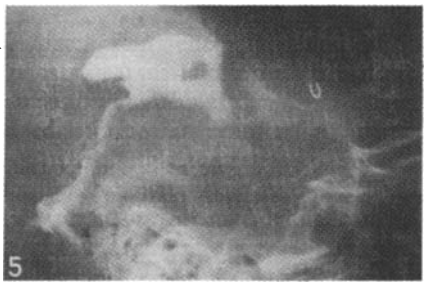


Fig. 1. Single diaphanous septum in right anterior horn. Obstruction in basal cisterns. Post-neonatal meningitis (J.M. aet. four months). **Fig. 2.** Numerous septa in right trigone and occipital horn. Obstructions at foramen of Monro and aqueduct. Neonatal *Acinetobacter* meningitis (G.M. aet. 2 months). **Fig. 3.** Distortion and non-filling of portions of lateral ventricles by septa. **a.** Left anterior horn contains small amount of air but right anterior horn is obliterated. Obstructions at foramen of Monro and aqueduct. Post-neonatal meningitis (M.K. aet. 16 months). **b.** Distortion of left trigone and non-filling of right occipital horn in another case. Obstruction at foramen of Monro. Neonatal *Enterobacter cloaca* meningitis. CSF protein 5.0 g/l (M.P. aet. 6 weeks). **Fig. 4.** Adhesions in left anterior horn from intraventricular bleeding (birth trauma). Period of gestation 32 weeks. Heavily bloodstained CSF with protein 7.85 g/l on day 9 (T.M. aet. 2 months)

Fig. 5. Detritus in hypophyseal recess and on floor of aqueduct, third and fourth ventricles. Dimer pencils floor of right anterior horn after instillation through ventricular catheter with clip in blunt tip. Meningococcal meningitis CSF protein 7.5 g/l (F.M. aet. 4 years)

3. Ventricular Detritus

In the same 6-year period, six cases with detritus within the ventricular system (Fig. 5) have been identified, all in older children varying between 3 and 8 years with a mean of 5 years. Two also had ventricular septa. Meningitis was present in four and probably in the fifth as well. The sixth case resulted not from infection but from a traumatic subarachnoid (intraventricular) haemorrhage. Where available, the CSF protein was markedly elevated and this was associated with an obstruction of the basal cisterns in four cases. Detritus was found at 11 sites in these six children, all but two being within the midline ventricles or aqueduct.

4. Dimer Pencilling

On occasion dimer installation (via a right frontal catheter) has been used in the investigation of hydrocephalus in infancy, as is our usual practice whenever the lateral ventricles are only moderately dilated. On occasion, we have identified a certain adherence to, or pencilling of, the ependymal lining of floor of the right anterior horn and third ventricle by dimer, presumably due to ependymitis which when severe caused a recognisable irregularity of the wall (Fig. 5). This irregularity was perhaps better demonstrated in the early cases in which myodil was used as contrast agent.

Discussion

Features of Hydrocephalus

In two-thirds of the total group of 105 cases, the obstruction to CSF flow was in the basal cisterns or beyond (communicating hydrocephalus). We suspect that

many of the cases assigned to the unknown group with a block in the basal cisterns are the result of infection or birth trauma. Similarly there are some cases of Dandy-Walker syndrome which would have been transferred from the congenital to postmeningitic hydrocephalus group but for lack of more clinical information: for example, there are three such cases of fourth ventricle exit foraminal obstruction in which the clinical history of meningitis and ventricular septa make allocation easy. Foramen of Monro obstruction is rare and is virtually always the result of infection whenever there is no obvious tumour to account for it. It has been reported in six of 19 cases (30%) of postmeningitic hydrocephalus in the newborn [9]. In this series it occurs about half as frequently, probably because ours is not limited to a study of the effects of neonatal meningitis. As reported previously [6] multiple obstructions were also found in this series especially in association with ventricular adhesions.

The ventricular adhesions and septa, which on occasion cause actual loculation of the lateral ventricles, follow on meningitis within the first 3 months of life, most often the neonatal period. Schultz and Leeds [13] who first described the ventriculography speculated that the raised CSF protein in all seven of their cases was a causative factor. We think this is so because the CSF protein in the acute phase was in excess of 5 g/l in eight of nine. They indicated that the septations varied in extent but their illustrations are gross examples. In this series there is a continuous gradation from a single gossamer-thin septum (Figs. 1 and 2) to thick bands preventing air filling (Fig. 3).

The neuroradiologic appearances of these septa are specific, but on occasion the filling of an isolated loculus may cause confusion with a subdural fluid collection or porencephalic cyst. Similarly non-filling of portion of a lateral ventricle may cause difficulty in interpretation (Fig. 3). Superficially, the bubbles on the surface of a rather frothy CSF resemble diaphanous adhesions. With a poorly filled occipital horn the indentation caused by the calcar avis may suggest loculation. CT has been done in one subsequent case but did not demonstrate the obvious septa shown by ventriculography (EMI 180 matrix). For this and other reasons we prefer to bypass CT in the investigation of infantile cases with open fontanelles.

Detritus has been identified infrequently in the midline ventricles in young children whose ages varied between 3 and 8 years. Where available the CSF protein levels were very high. One case with a debris nodule on the floor of the fourth ventricle came to autopsy and it was reported that this was fibrinous exudate and the ependyma had been destroyed.

Meningitis

The incidence of meningitis in the neonatal period and infancy is unknown because our catchment population is dispersed over a very large area and is served by smaller hospitals and rural clinics. The number developing hydrocephalus cannot be compared with other studies [4, 9] but it must be high. This is obvious from the fact that 30-40% of all infantile hydrocephalus seen at the hospital are the result of meningitis. Although we are in the unfortunate position of seeing so many preventable cases, we have not come to any firm conclusions about the causative factors:

a. Neonatal Meningitis. The cause of most postmeningitic hydrocephalus is notoriously difficult to recognise because of the vague, ambiguous, and nonspecific clinical manifestations [3, 4]. The younger and smaller the child, the more nonspecific are the features. It has been suggested that the birth history is of more help in diagnosis. Error in diagnosis is of the order of 60% and, therefore, it is suggested that lumbar puncture be freely undertaken to exclude the disease whenever it is considered [4]. It is possible that cases referred to us from rural centres have previously received inadequate antibiotic therapy for mistaken diagnoses, e.g., diarrhoea, jaundice, or respiratory distress. Had a timely lumbar puncture been performed at the local health centre or hospital, neonatal meningitis might have been diagnosed and adequately treated, thereby maybe preventing excessive accumulation of fibrinous exudate.

B. Inadequate Defence Mechanisms. These have been demonstrated pathologically in the neonate [3]; positive cord blood culture, poor chemotaxis, and phagocytic response coupled with an increased permeability of the blood-CSF barrier in the neonate, especially the premature infant, have been implicated [4, 11]. This poor host reaction may be further impaired in the coloured and Bantu communities. These two population groups account for 66% of the hospital's overall admissions. As one would expect, 65% of all tumour hydrocephalics and 70% of all congenital infantile hydrocephalics come from these two population groups in the same approximate proportions as occur in the general population [5]. Similarly the racial distribution of certain paediatric diseases (teratoma, nephroblastoma, and Hirschsprung's disease) mirrors the ratio of the three population groups. Yet 95% of the postmeningitic hydrocephalics are coloured and black infants.

c. Hydrocephalus. Hydrocephalus has been shown to develop in 30% of those surviving neonatal meningitis especially if *ineffective, delayed antibiotic therapy* has been administered [9, 11]. Smouldering active ventriculitis, as shown by ventricular CSF, may continue in spite of a clear lumbar fluid beyond a block [8]. Unlike meningitis in the older age groups, the neonatal variety has a special propensity to cause ventriculitis [6]. Of 25 cases examined at autopsy, 23 had inflammatory disease within the lateral ventricles [3] which may amount to a pyocephalus [6]. It is well known that the African seeks medical attention late in the natural history of a disease – maybe we are seeing more late and perhaps inadequately treated cases than other centres.

In spite of adequate morbid anatomic studies on series with neonatal meningitis there is little description of the ventricular adhesions [3, 6, 9, 10, 15]. Lorber and Pickering [9] describe multiple loculi which precluded any possibility of surgical treatment but failed to describe the radiology and pathology. Milhorat [10] refers to their frequency. Schultz and Leeds [13] were able to perform autopsies on three of their seven patients who had had postmeningitic hydrocephalus with ventricular adhesions. They found that the ependyma had been destroyed by the inflammatory process and that the septa consisted of fibrinous strands containing a few fibroglial elements with a few round cells and polymorph infiltrations. Because of the nature of the disease and the huge drainage area our autopsy material is limited.

Conclusions

The high incidence of hydrocephalus in the Cape Province is due to meningitis either in the neonatal period

or shortly thereafter. It accounts for 30-40% of all new hydrocephalic cases that we study in infancy and childhood. CSF pathway obstruction is most frequent at the level of the basal cisterns but may occur anywhere within the ventricular system. The combination of ventricular septa with foramen of Monro obstruction is diagnostic of postmeningitic hydrocephalus. Intraventricular septa and loculi are almost always the result of infection and occur in about 25% of the postmeningitic cases. On occasion they may be the result of birth trauma or very rarely be found in association with a posterior fossa mass. Detritus in the ventricular system is sometimes found in older children with severe meningitis and ventriculitis.

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