Neuroradiology (1981) 21: 93–96

Neuroradiology

Benign Communicating Hydrocephalus in Children

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Summary. A review was performed of the computed tomograms (CTs) of 500 children which had been reported as showing widening of the supratentorial subarachnoid spaces with normal cerebral substance. On the basis of this a radiological diagnosis of cerebral atrophy had been made in all but five, who were said to have megalencephaly. From these, the children with large or abnormally enlarging heads, but normal or only slightly enlarged ventricles, were selected; there were 40 such cases (8%). The clinical condition either improved or remained stable over a period of 2 years; in the majority the scan abnormality regressed (22.5%) or remained static (67.5%). In three cases there was slight progression of the CT changes before stabilisation, but only one case developed classical communicating hydrocephalus necessitating a shunt procedure. This condition is a generally benign and mild form of communicating hydrocephalus, for which an aetiological factor was apparent in about two-thirds of the cases studied.

Key words: Computed tomograms, cranial – Benign paediatric communicating hydrocephalus – Benign subdural effusion – Prognosis

The increase in the cerebrospinal fluid (CSF) pressure which follows occlusion of the subarachnoid space near the superior sagittal sinus is transmitted to the skull and brain. In a young child, the yield pressure of the sutures may be low enough to result in widening of the intracranial subarachnoid space and enlargement of the vault of the skull without distension of the ventricles. In such cases, communicating hydrocephalus may present with a large or too rapidly enlarging head and wide subarachnoid spaces, but slight or absent ventricular enlargement [1].

Raised cerebral venous pressure of any aetiology tends to increase the cerebral interstitial and CSF pressures, whilst absorption of fluid remains normal. When the sutures are fused, this may cause a pseudotumour syndrome, but when the skull can expand, mild hydrocephalus, sometimes associated with macrocephaly, may result [2].

The widening of sulci and fissures with little or no ventricular enlargement may simulate cerebral atrophy or the reversible cerebral shrinkage associated with dehydration, malnutrition, hypercorticosteroidism, whether iatrogenic or due to Cushing's syndrome, or hypocarbia which may be induced by controlled ventilation.

The CT diagnoses of generalised widening of CSF spaces, and of megalencephaly, are merely descriptive, and imply that the clinician should seek an aetiological factor, which may be readily evident from the history.

We considered that it would be of interest to review our cases with widening of the supratentorial subarachnoid spaces and select those patients with features of past or present raised intracranial pressure or of an enlarged or enlarging head in order to establish the aetiology and the clinical and CT progress on a follow-up study.

Material and Methods

A review was made of the notes and CT scans of 500 consecutive children, presenting under 4 years of age, who had been diagnosed, on the basis of widening of the supratentorial subarachnoid spaces, with normal cerebral substance shown on CT, as having cortical atrophy, or in five cases as megalencephaly. Those

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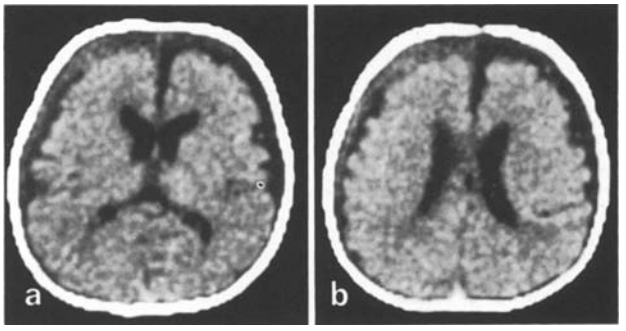

Fig. 1 a and b. Boy aged 15 months. Minor head trauma 3 months previously followed by abnormal increase of head size. There is widening of convexity subarachnoid spaces. A shallow subdural effusion isodense with brain is shown over the left frontal convexity and a minimal one anteriorly on the right

with clinical evidence of a head which was either above the 98th percentile or abnormally enlarging, or who had features of past or present raised intracranial pressure, were separated for further study. In particular, indications of the aetiology were sought and examinations were made over a minimum period of two years to elucidate the prognosis.

Results

Only 40 cases fitted our criteria and these could be subdivided into 5 groups by the possibly significant aetiological factors:

1. Postnatal trauma	6 cases
2. Infection	9 cases
3. Premature birth/traumatic delivery	7 cases
4. Raised venous pressure	4 cases
5. No cause evident	14 cases

There was a definite history of mild head injury related to the onset in 6 cases. Three presented with enlarged heads and the other three with abnormally increasing head size. In three, small bilateral subdural effusions were visible outside the distended convexity subarachnoid spaces (Fig. 1); in all six there was slight ventricular enlargement.

CSF flow studies using metrizamide were performed in two cases; one showed ventricular retention but the other was examined at a later stage, when the CT abnormality was resolving, and the flow was normal.

In one case the head size reverted to normal and the degree of hydrocephalus decreased; in five the head remained about the 98th percentile and the CT appearance was unchanged.

Nine cases had a history of meningitis occurring between 8 months and 2 years previously; bacterial

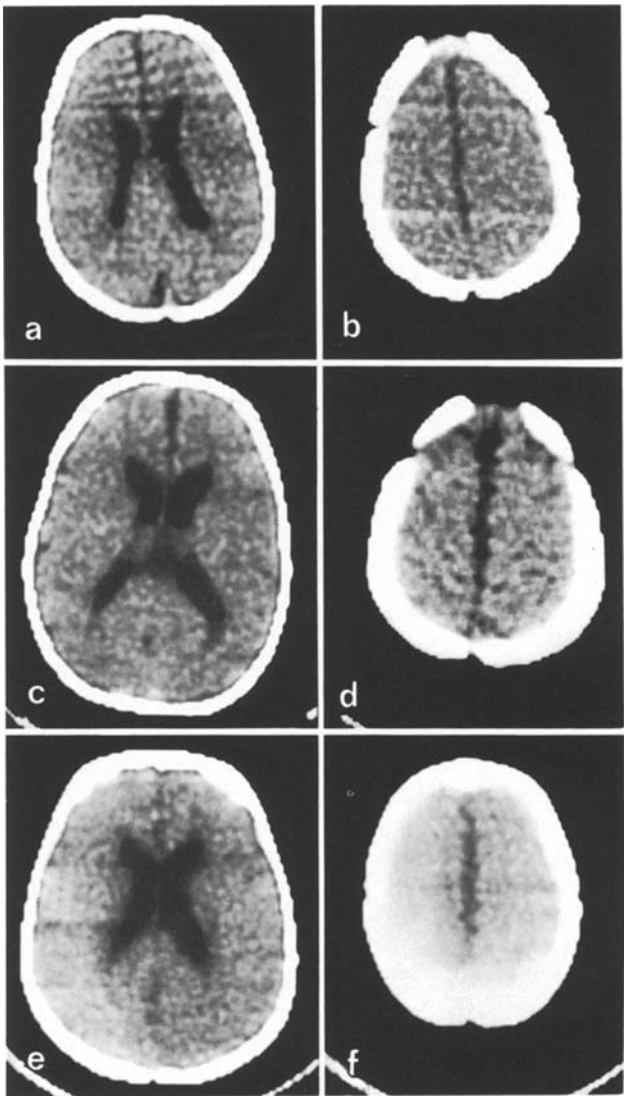

Fig. 2 a–f. Boy, aged 4 months with nephrotic syndrome, developed abnormal increase in head size following superior vena caval drip. **a, b** Initial scans show minimal ventricular enlargement and widening of interhemispheric fissure. **c, d** Scans after interval of 3 months showed enlargement of ventricles and subarachnoid space. A metrizamide scan was considered to be indicated but was not performed until 2 months later, by which time head growth had slowed. There was no entry of contrast medium into the lateral ventricles and normal passage through the subarachnoid space. **e, f** Taken 20 h after lumbar injection of metrizamide show only slight residual opacification of cortical sulci and interhemispheric fissure. Lateral ventricles and interhemispheric fissure have decreased in size

meningitis had been proven and treated in four (H. influenza 3; B. haemolytic streptococcus 1) and lymphocytic meningitis, presumed to be of viral origin, had occurred in five. In all these patients the head was large or enlarging and the convexity subarachnoid spaces were wide. On clinical follow-up all the

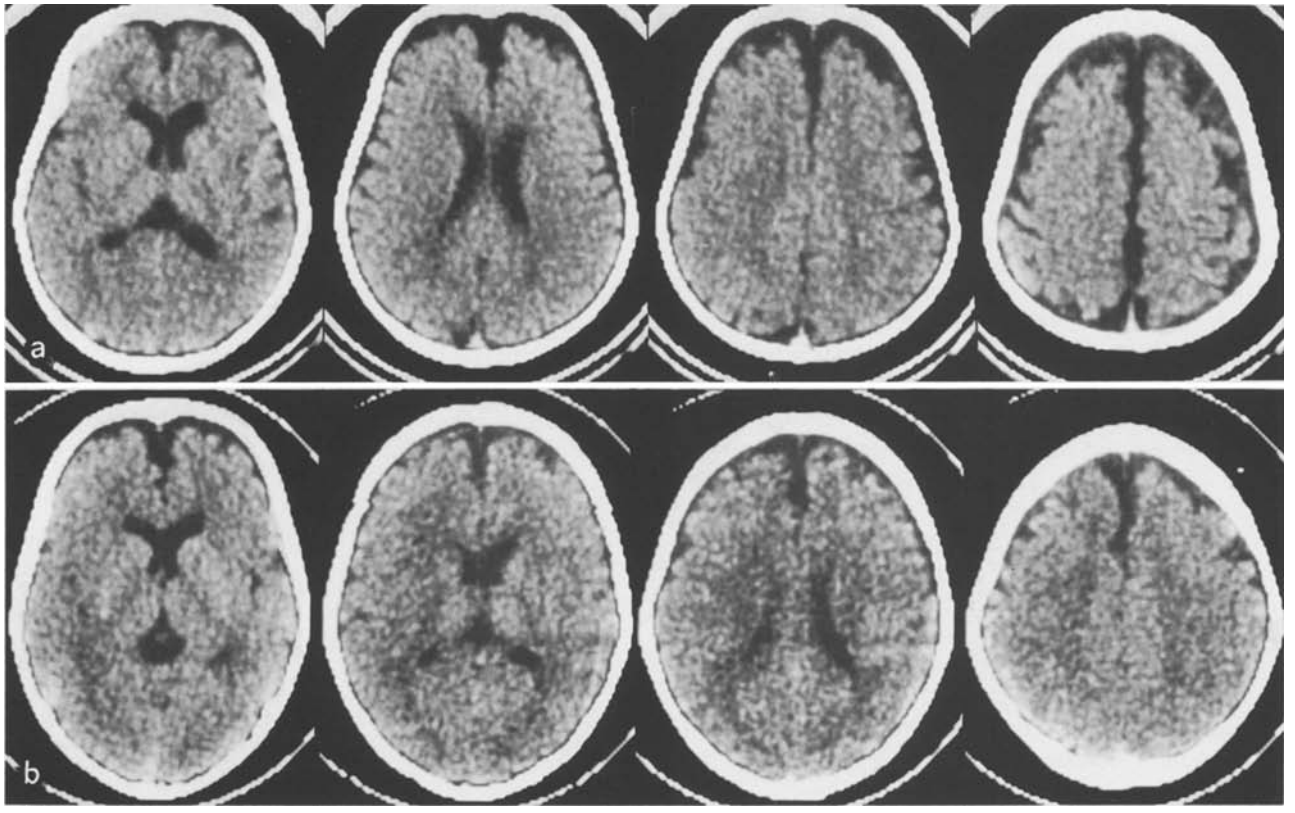

Fig. 3a and b. Young boy, clinically normal apart from enlarged head. **a** Initial scan at 15 months shows slight ventricular enlargement but considerable widening of interhemispheric fissure and subarachnoid spaces over cerebral convexities. **b** At 2 years there is slight decrease in ventricular size but marked reduction in width of subarachnoid spaces

children were asymptomatic and CT showed complete resolution in two, but no change in six. In the other one the hydrocephalus slightly increased and then regressed to its former appearance.

In seven cases, there was prematurity (Case 3), birth trauma (Case 2) or perinatal respiratory distress (Case 2), all presenting with enlarged or abnormally enlarging heads. Six developed normally and were clinically well but only two of these had follow-up scans; in one the abnormality had regressed but the other showed small subdural effusions. One case was retarded and subsequent CT showed progressive hydrocephalus which was relieved by a shunt.

Of the four patients with raised venous pressure, one presented with a large head and two with abnormally rapid increase in head size as the only neurological sign. The other developed transient hemiparesis after correction of Fallot's tetralogy, the enlarged head being noted incidentally on neurological assessment. Two of the cases with Fallot's tetralogy and patent ductus arteriosus respectively had suffered from cardiac failure. The other two had had superior vena caval occlusion at the time of an infective illness and in association with nephrotic syndrome. All made satisfactory clinical progress; one CT returned to normal, in one the ventricles increased slightly in size and then regressed (Fig. 2). Two remained unchanged.

In 14 other patients presenting with enlarged heads, no aetiology was evident; a similar group has been discussed by Sahar [3]. Five of our cases were of normal intelligence and nine had a minor to moderate degree of retardation. There was no change in the clinical condition on follow-up. CT appearances were static in 11 cases, two showed decrease in the hydrocephalus (Fig. 3), and the other one had slight increase in the hydrocephalus followed by resolution.

Discussion

It may appear from the description that these patients should be recognised easily and distinguished from those with loss of brain volume. This may indeed be so when a detailed and accurate history is forthcoming and measurements of the skull are indicated to the radiologist. In practice, the relevant clinical data often have to be requested and

should always be sought when unexplained wide subarachnoid spaces over the cerebral convexities are associated with normal brain substance, without evident microcephaly. Some relatively uncommon storage disorders, such as the mucopolysaccharidoses, and metabolic disturbances such as metachromatic leukodystrophy, which can cause macrocephaly and progress to atrophy, may give similar appearances. These conditions can generally be recognised clinically and distinguished by biochemical tests. In many such cases, the cerebral substance is abnormal on CT.

In three of our patients small higher attenuation subdural effusions were evident outside the widened subarachnoid spaces. Subdural effusions of CSF density could not have been excluded in some of the other children also and somewhat similar appearances have been described as benign subdural effusion in infants [4, 5]. Subdural effusions may be associated with communicating hydrocephalus, possibly due to compression of the convexity subarachnoid spaces [6], or to simultaneous onset of both conditions after trauma or inflammation in which both spaces may be involved. In our cases, the fissures and sulci, and usually the basal cisterns, were wide. Visible subdural effusions were small and, in the few metrizamide studies, there was no evidence of a bicompartmental distribution. Therapeutic aspiration of the fluid was not considered necessary and diagnostic aspiration was therefore not justified.

Our study suggests that regardless of its actiology this condition is generally self-limiting and frequently regresses spontaneously. Although many of the children were somewhat retarded, possibly as the result of the original insult, deterioration, which frequently occurs with ongoing degenerative, inflammatory and abnormal metabolic processes associated with atrophy, was not a feature of the condition.

In our series, three cases had slight transient increase in the hydrocephalus but only one case progressed to significant dilatation of the ventricles to give the classical picture of communicating hydrocephalus. Such progression occurred, however, in several cases recorded by Robertson and Gomez [1]. Management is conservative, but with clinical follow-up to ensure satisfactory neurological progress and normal rate of head growth, together with CT monitoring, so that progression of the hydrocephalus can be noted and treated at an early stage if necessary.

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Received: 28 October 1980

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