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SONOGRAPHY OF MENINGOENCEPHALITIS AND VENTRICULITIS IN INFANCY

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On the basis of eleven own cases sonographic features of infant meningoencephalitis and ventriculitis are discussed. The characteristic findings are as follows: abnormal parenchymal echogenicity of brain, changes of the size and wall echogenicity of cerebral ventricles and that of cerebrospinal fluid echogenicity.

INTRODUCTION

It is well known that infant intracranial anatomy can be clearly shown with transfontanelle real-time sonography /1,10/. Several authors have reported abnormal sonographic findings in infants with bacterial meningitis, meningoencephalitis /4,5,6,9,17/ and ventriculitis /10,12,13,16,22/.

Inflammation of the meninges, the brain and the ventricles may result from infection by bacterial, viral, fungal, rickettsial and protozoal agents. The site and extent of disease vary from organism to organism and from case to case.

The diverse inflammatory laesions can be demonstrated successfully by ultrasound as abnormal parenchymal echogenicity, consistent with calcific deposits, cystic development of the brain and as changes of the ventricular size, ventricular wall regularity and echogenicity of cerebrospinal fluid, and ventricular septations.

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SUBJECTS AND METHODS

The cranial ultrasonography was performed with 5Mhz transducer using Picker LS 7 000 equipment, through the anterior fontanelle as an acustic window. Every patient was examined on both coronal and parasagittal planes. In the last two years we examined 11 patients (newborns and infants) with acquired intracranial infections. Six of them had uncomplicated bacterial meningitis, two of them meningoencephalitis, one newborn meningitis and ventriculitis and two infants had shunt ventriculitis.

All infants underwent sonography on admission. If the initial study showed parenchymal or ventricular abnormalities; if new symptoms appeared; if the cerebrospinal fluid did not clear with antibiotic therapy; or if the infant's condition deteriorated acutely, sequential studies were performed.

The scans were evaluated as to the presence of ventricular size, ventricular wall regularity and echogenity, intraventricular debris and septations, increased or decreased diffuse or focal parenchymal echoes, evidence of encephalomalacia or abscess and unusually bright convolutions. Two patients died and the sonographic findings were correlated with post-mortem examination.

RESULTS

A wide variety of sonographic abnormalities were observed in all patients except the six uncomplicated bacterial meningitis cases, where no abnormalities were found. In one meningoencephalitis case we saw diffuse increased parenchymal echogenicity with compressed cerebral ventricles, suggesting cerebral oedema (Fig. 1). Some days later focal areas of increased and decreased echogenicity developed in the brain parenchyma and very prominent convolutional marking appeared indicating inflammation and exudate over the convexities, respectively (Fig. 2). After three weeks of coma the brain lost its usual structural appearance on sonography. The patient died and the post-mortem examination showed purulent exudate over the convexities and complete decomposition of the brain. In the other meningoencephalitis case we found increased periventricular echogenity, which resolved in three weeks time.

In cases of shunt ventriculitis markedly echogenic ependyma was visible with dilation of ventricles and with echogenic

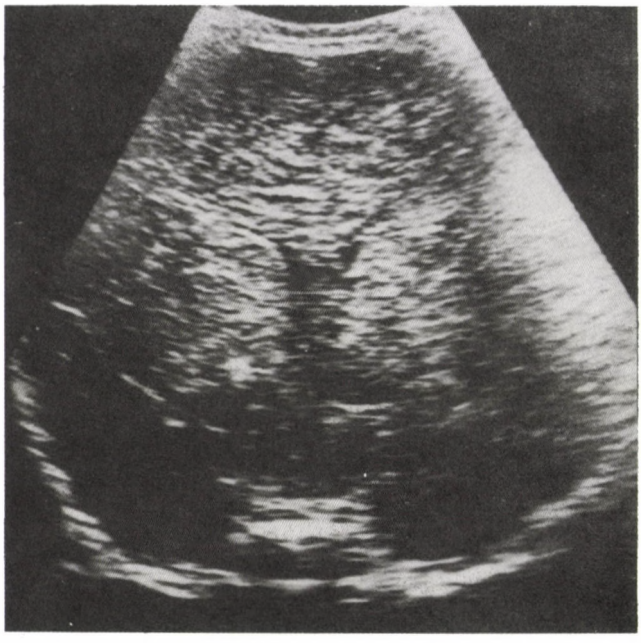

Fig. 1. Diffuse increased parenchymal echogenicity with compressed lateral ventricles

cerebrospinal fluid in the first days (Figs.3 and 4). Some days later the cerebrospinal fluid echogenity became normal (anechoic) and echogenic debris was present in the ventricles followed by fibrin strands and intraventricular septations (Figs. 4 and 5). In one case two, anechoic regular shaped formations developed as the inflammation extended into the brain parenchyma, possibly encephalomalacic cysts due to inflammatory vascular obstruction and necrosis (Fig. 6).

In the neonatal meningitis-ventriculitis case, mild diffuse increased echogenicity was found with mildly dilated ventricles, with echogenic ependyma and echogenic cerebrospinal fluid. The patient died and the post-mortem examination revealed brain oedema, inflamed meninges and ventricles and purulent cerebrospinal fluid.

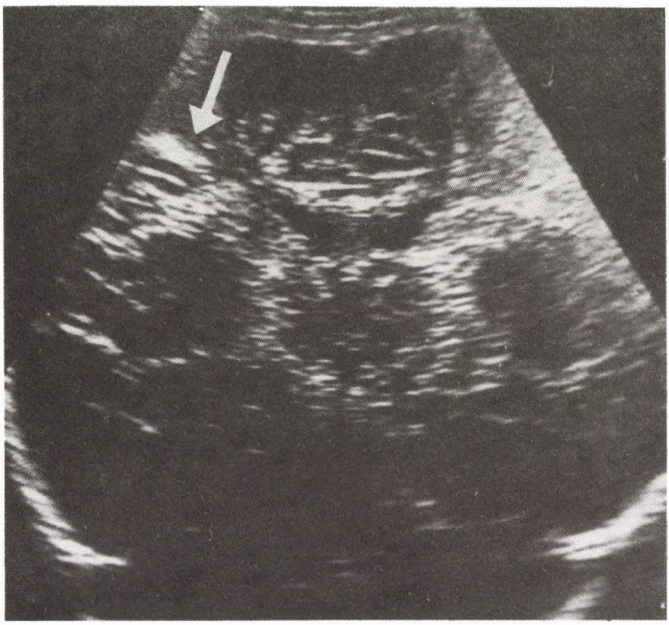

Fig.2. Focal areas of increased and decreased echogenicity in the brain parenchyma. Prominent convolutional markings (arrow)

DISCUSSION

The ultrasound findings in infants with complicated meningitis first was reported by Edwards et al in 1982 /5/. They found the earliest abnormality to be a focal increase in the echodensity of the cerebral cortex. It was thought to be due to meningoencephalitis. Echodensity was predominantly seen in the cortical grey matter and caused accentuation of the grey/white matter interface. Levene /14/ found the most extensive parenchymal abnormalities in the periventricular white matter as did we in both cases (Fig. 2). These areas resolved with antibiotic therapy. Infants with meningoencephalitis may develop more widespread echodensity throughout the brain (Fig. 1) obscuring anatomical landmarks /9,16/. This diffuse change is thought to represent oedema. Sometimes other focal abnormalities can be found such as gyral infarction /2/, brain abscess /7/, or necrotic areas as a

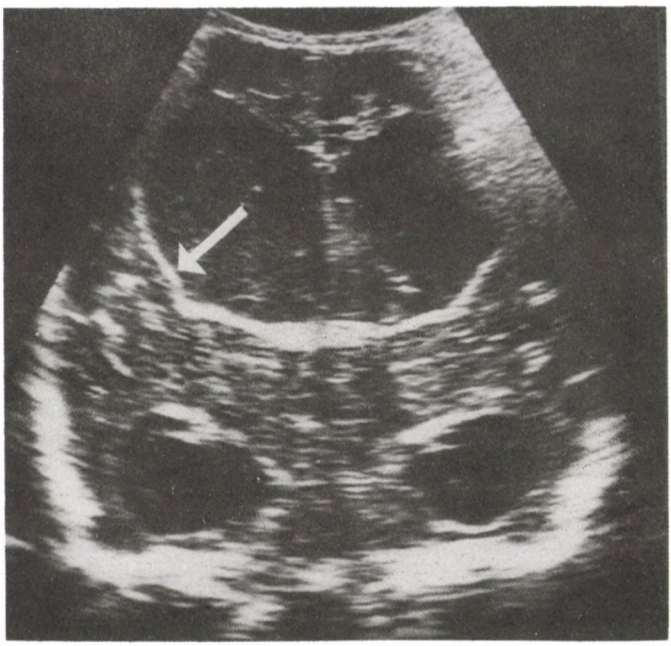

Fig. 3. Echogen ependyma of lateral ventricles (arrow), increased echogenicity of cerebrospinal fluid

result of cerebral vasculitis, vascular thrombosis and infarction /15, 21/. Cystic degenerations and calcium deposits in the degenerated periventricular white matter are most frequently seen in CMV and Herpes simplex typ. 2 infections /15/.

Ventriculomegaly is considered to be the most common complication of meningoencephalitis. In the acute stage it probably is a result of obstruction of cerebrospinal flow over the surface of the brain. Ventriculomegaly appearing later may represent the loss of brain parenchyma /20/.

Extraaxial fluid accumulated in the subarachnoid space is an exudate that comes from the cellular inflammatory response of the meninges and which widens the interhaemispheric fissure. Fluid collection over the convexity can be well seen by 7.5 MHz transducer with a short focus; this is the so called "near filed technique". Inflammatory exudate increases the brightness

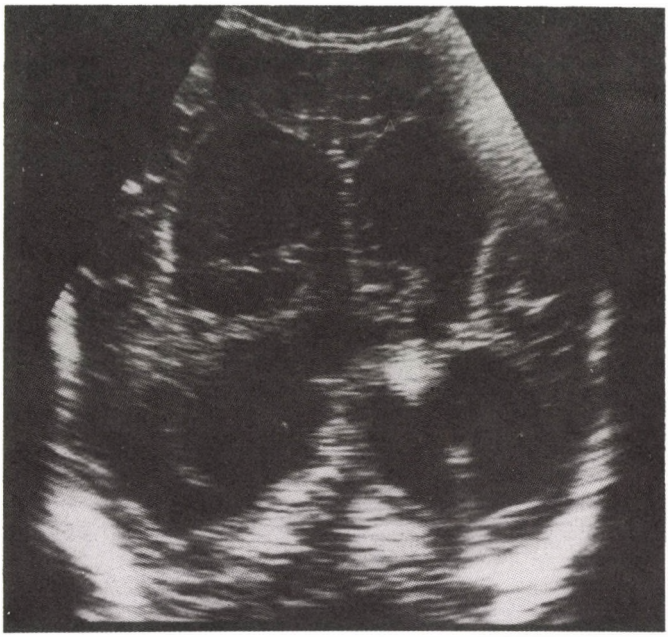

Fig. 4. Next stage of ventriculitis: heterogenous echogenicity of cerebrospinal fluid

of the sulci and widens the sulcal echoes as well (Fig. 2., arrow) /9/.

Ventriculitis cases are of interest, as 65-90% of neonatal meningitis is associated with ventriculitis. Bacteria in neonatal meningitis enter first into the lateral ventricles through the choroid plexus, then are taken into the subarachnoid space by cerebrospinal fluid. Sometimes ventriculitis occurs as a complication of ventriculo-peritoneal shunt placement. Establishing the diagnosis of ventriculitis is important as it indicates commencing local antibiotic treatment, given the fact that systemic antibiotics may not reach therapeutic levels within the ventricles /8/. The stages of ventriculitis can be followed by sonography /22/. In the first week of the disease ependymitis and choroid plexus inflammation result in purulent exudation in the dilated ventricles. Prominent echogenic ependyma can be seen with

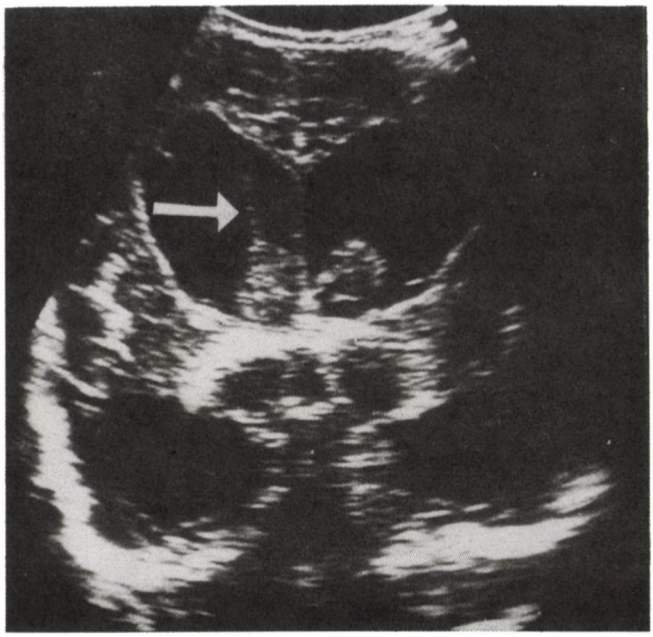

Fig. 5. Later stage of ventriculitis: echogen debris, fibrin strands in the strands in the lateral ventricles (arrow)

increased echogenicity of cerebrospinal fluid (Fig. 3). In the second week debris appears followed by clearing of exudate and developing fibrin strands (Fig. 4) and a week later by ventricular septations. The ventricular bands originate from the subependymal glial, node /3,12,13,16,18/. The septations cause compartmentalization of portions of ventricles. A particular danger of that is isolation of an area of ventricle which remains infected and may act as a source of reinfection even in adequate intravenous or intraventricular antibiotic therapy.

We recommend that an initial sonographic study be done at the time of diagnosis of bacterial meningitis. A follow-up study should be performed within one week if the initial scan demonstrates ventricular or parenchymal abnormalities. This will help to assess the development of further complications (hydrocephalus, encephalomalacia and/or abscess). In addition, a repeated scan is recommended if new symptoms appear; the

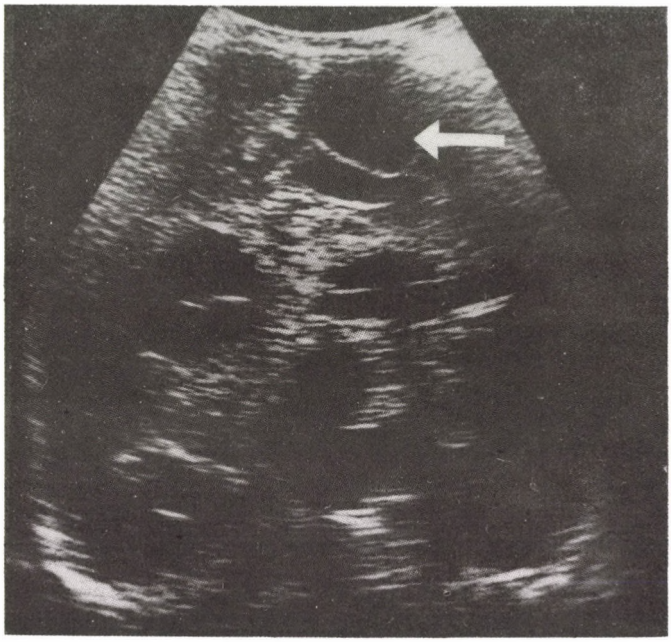

Fig. 6. End stage of ventriculitis: cavities of different size separated by septi, encephalomalacic cysts are also seen (arrow)

cerebrospinal fluid does not clear with antibiotic therapy; or if the infants' condition deteriorates acutely. Scanning is also advised after ventricular shunt placement.

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