CRANIAL ULTRASONIC ASSESSMENT OF INFANTS WITH ACUTE BACTERIAL MENINGITIS

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ABSTRACT

A prospective study was performed on 44 patients (newborn to one year old) with acute bacterial meningitis. Sonograms were obtained within 72 hours of diagnosis, and repeated on 7th, 14th or at an early date, if required. The spectrum of sonographic features of meningitis included normal scan (35.4%), echogenic sulci (63.7%) and parenchyma (29.5%), ventriculomegaly (59%), ventriculitis (35%), pseudo-porencephalic cyst (4.4%), extra axial fluid collection (4.4%), encephalomalacia (2.2%) and cerebral abscess (2.2%) in patients.

Key words: *Bacterial-meningitis, Ultrasonography.*

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Acute bacterial meningitis still remains as one of the causes of infant mortality and morbidity even in the present antimicrobial era. Early diagnosis and appropriate medical and surgical intervention of the complications can reduce the morbidity and mortality to a considerable extent.

The use of sonography to examine these infants has rapidly increased in the last few years and the relative simplicity of the procedure, makes it an ideal method for early detection of complications and follow-up of these patients(l). Moorthy *et al.* have concluded that Cranial Ultrasonography (CR-USG) is a very effective diagnostic tool in infants with pyogenic meningitis particularly in the diagnosis of its complications. It is also useful for monitoring ventriculomegaly and cortical mentle thickness in these "children on follow up(2). Reader *et al.* have observed that sonography has a definite edge over computed tomography in demonstrating intra-ventricular septae(3).

The present study was undertaken to determine the sonographic findings in the patients with pyogenic meningitis within 72 hours of diagnosis and on follow-up screening.

Subjects and Methods

A prospective study was carried out in forty four infants of acute bacterial meningitis. Of them thirty one were males and thirteen were females. Ages ranged from newborn to one year, ten of them were under twenty eight days of age.

The criteria of diagnosis 6f pyogenic meningitis was—cerebrospinal fluid showing presence of polymorphonuclear leucocytes, more than 2 per cu mm in neonates and 1 or more per cu mm after the neonatal period; protein more than

120 mg/dl in neonates and more than 40 mg/dl after the neonatal period and sugar content less than 60% of the blood glucose(4). Cerebrospinal fluid was also examined microbiologically by Gram's staining, culture and sensitivity.

All infants with proven meningitis were subjected to sonography within seventy two hours of diagnosis and repeated in all on 7th or 14th day or at an early date if *(a)* new symptoms appeared, *(b)* repeat cerebrospinal fluid did not clear with antibiotic therapy, or (c) sudden deterioration of the infant's condition.

The scan were performed on 2D real time SIM 3000 OTC Biomedier with a 5 MHz transducer. Axial, coronal and sagittal scans were obtained through open sutures and fontanelles(5,6). A water bag over anterior fontanelle enabled sonologist to detect smaller anterior subdural effusion at the earliest. The sonograms were reviewed for presence of echogenic sulci, any abnormal increase or decrease in parenchymal echogenecity, ventricular size, ventriculitis (echogenic ependyma, ventricular strands and echoes within the ventricles), extra-axial fluid collection and for any evidence of abscess, pseudo-porencephaly or encephalomalacia.

CT scan was done in three patients-one each of sub and epidural effusion and encephalomalacia.

Results

A wide spectrum of sonographic abnormalities were observed in the present series of 44 infants of pyogenic meninigitis. Of them, 16 had normal sonogram despite clinical and biochemical profile of pyogenic meningitis.

The qualitative increase in brightness and widening of sucli was the most common sonographic finding seen in 63.6% patients *(Table I).* The findings persisted (with treatment) in 5 (11%) patients, who developed other complications *(Table II).*

There were 12 cases having focal areas of increased echogenecity of brain parenchyma *(Table I).* Of these, two developed pseudo-porencephaly and one each developed brain abscess and encephalomalacia *(Table III).*

Ventriculomegaly was observed in 59% of infants and remained as such till the last sonography. Fifteen of them had evidence of ventriculitis. Ventriculitis was seen in the

TABLE I–*Sonographic Spectrum in Pyogenic Meningitis (n=44)*

Finding	Number	%
Normal	16	35.4
Echogenic sulci	28	63.6
Parenchymal opacities	12	29.5
Ventriculomegaly	24	59.0
Ventriculitis		
Ependymitis	15	33.0
Choroid plexitis	10	22.0
Septae	8	17.6
Ventricular exudate	5	11.0
Pseudo-porencephaly	2	4.4
Subdural effusion	1	2.2
Epidural effusion	1	2.2
Encephalomalcia	1	2.2
Abscess	1	2.2
Dilated 3rd, 4th ventricle and cisterna magna	15	33.0

TABLE II–*Sonographic Follow up of Infants with Pyogenic Meningitis*

Day of Diagnosis	Echogenic sulci	Parenchymal opacities	Ventriculo-megaly	Ependymitis	Ventriculitis		
					Ch. Plexitis	V. exudate	Septae
1-3	28	12	24	8	-	5	-
7th	5	4	24	15	10	5	-
14th	6*	4**	24	7	10	5	8
21st	5	4	24	7	8	5	8

* Extra axial fluid collection, Porencephaly 2, abscess 1 and encephalomalacia 1.

** Porencephaly 2, abscess 1 and encephalomalacia 1.

TABLE III–*Correlation Between Complications and CSF Profile*

CSF	Normal USG (n=16)	Abnormal USG$ (n=24 + 4)*						
		Ventriculo-megaly	Ventri-culitis	Porence-phaly	Sub-dural effusion	Epidural effusion	Abscess	Encephalo-malacia
Cell count per cu mm								
<1000	10	3	1	-	-	-	-	-
> 1000 (uncountable)	6	21	14	2	1	1	1	1
Protein (mg/dl)								
40-150	10	2	-	-	-	-	-	-
>150	6	22	15	2	1	1	1	1

* Had only increased echogenicity of sulci.

$ CSF sugar ranged between 0 to 6 mg/dl.

form of echogenic irregular ependyma (33%), choroid plexitis (22%), septae (17.6%) and ventricular exudates (11%) *(Table I)*.

Pseudo-porencephaly (4.4%), sub-dural effusion (2.2%), epidural effusion (2.2%), abscess (2.2%) and encephalomalacia (2.2%) were observed in patients with evidence of increased echogenicity of brain parenchyma and ventriculitis in their early sonogram.

The infants with uncountable pus cells and protein of > 150 mg/dl in CSF had various types of sonographic abnormalities as compared to the infants with pus cells <1000/cumm and protein of <150 mg/dl *(Table III)*. The CSF sugar ranged between 0 to 6 mg/dl in all infants.

In this study, 8 absconded and 4 expired, because of complications. One patient required ventriculoperitoneal shunt because of progressive abnormal increase in head size and ventricles on sequential sonographic evaluation.

Discussion

Pyogenic meningitis is a common cause of morbidity and mortality in the pediatric age group. Mortality is high during the acute stage while sequelae are often seen among survivors. An early recognition of structural changes is imperative for timely medical and surgical intervention. In this study, sonogram was normal in 35.4% infants, while 16%(7) and 37%(8) infants had normal sonogram in earlier studies.

The earliest and commonest abnormal sonographic finding in such infants is widening and increased echogenicty of sulci due to intense inflammatory exudate which accumulates in the depth of the Fissures and sulci, particularly around pia and subarachnoid vessels(2,3). In this study it was observed in 63.6% infants while it was earlier reported in 82%(7) and Han et al.(8) infants. On subsequent sonography it persisted in 5 cases.

In the present study diffuse or patchy alteration in the parenchyma echogcnicity due to cerebritis *(Fig. 1)* was seen in 29.5% cases. Chowdhary *et al*.(7) and Han *et al.*(8) reported this in 65% and 12% of cases, respectively.

Ventriculomegaly may be an early or late sonographic feature. An early ventriculomegaly represents non-obstructive normal pressure hydrocephalus and is usually reversible(9). However, late ventriculomegaly is because of obstruction secondary to accumulation of purulent exudate (acute) or due to chronic inflammatory changes in the subarachnoid space or within ventricles(l0). Ventriculomegaly can also be because of brain atrophy.

We observed ventriculomegaly in 59% infants, while others reported it in 15-45% cases(l,8,ll). Sequential sonography revealed their persistence in all cases till discharge.

The process of ventriculitis mainly originates from the choroid plexus. Ventriculitis is seen in the form of echogenic irregular ependyma, irregular hyperechoic choroid plexus and ventricular exudates. If disease process progresses, at 2-3 weeks sonography reveals septa formation, compartmentalization of ventricles, break in ependymal lying and in severe cases, the picture may be of multicystic encephalomalacia(5,12). In the present study, ventriculitis was observed in 15 cases in the form of ependymitis, choroid plexitis,

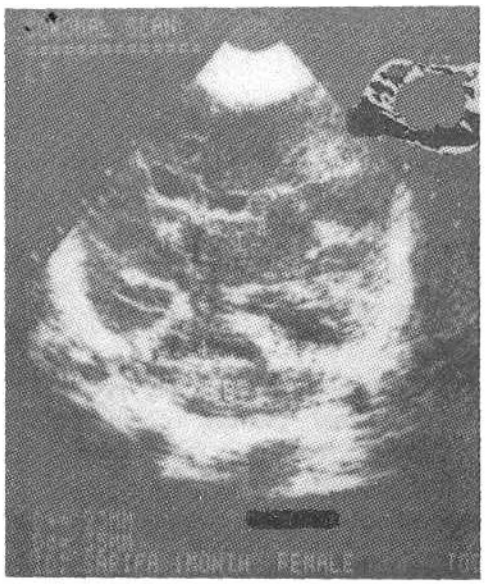

Fig. 1. Mid-coronal scan shows increased echogenecity of right cerebral hemisphere and in left thalamus.

ventricular exudate and septae *(Fig. 2)* by sonography in 33, 22, 11 and 17.6% cases, respectively. Chowdhury *et al.*(7) demonstrated ventriculitis in the form of echogenic irregular ependyma, choroid plexitis, ventricular exudate and septae in 55, 20, 1.6 and 10% cases, respectively.

Abscess is seen as sharply circumscribed lesion with thick rim of intense echogenicity surrounding the hypoechoic centre. The absecess is usually found in an area that previously exhibit increased echogenicity and poor margination, representing a focus of cerebritis, vasculitis or parenchymal infarction that becomes infected(13). In the present study, a neonate with increased opacity of brain parenchyma, on sequential sonography revealed it on the seventh day of therapy. Fisher *et al.* reported it in 3% infants with pyogenic meningitis(l). Chowdhury *et al.* from their two studies(7,ll), reported it in 8% cases.

The infections may manifest as cerebral edema characterized by diffuse effacement of the normal ventricles, sulci and gyri(10). The edema may cause a decreased cerebral blood flow and subsequent infarction. This is seen as an area of increased echogenecity which may later on break down to irregular echolucencies and pseudo—porencephalic cyst(14). It may communicate with ventricles or sub-aracahnoid space depending upon the site and extent by breaking ependymal living(15).

In the present study, two patients with evidence of increased parenchymal echogenicity and ventriculitis revealed pseudo-porencephalic cyst communicating with lateral ventricle, on sonography *(Fig. 3).* One of them expired and the other absconded.

Extra axial fluid collection (EAFC) displaces the brain away from the vault so sonogram reveals visible crown of the gyri,

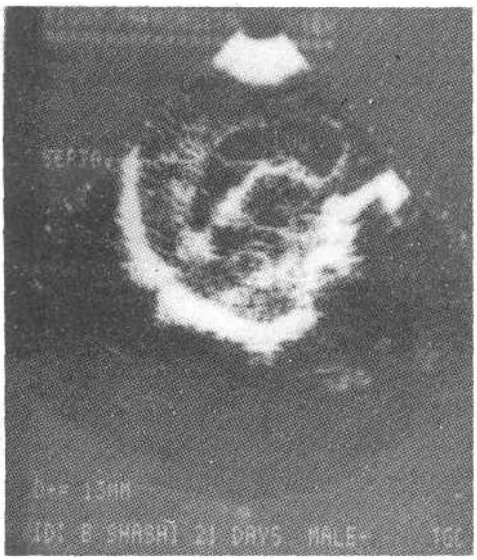

Fig. 2. Right para-sagittal scan revealing thick septa in lateral ventricle.

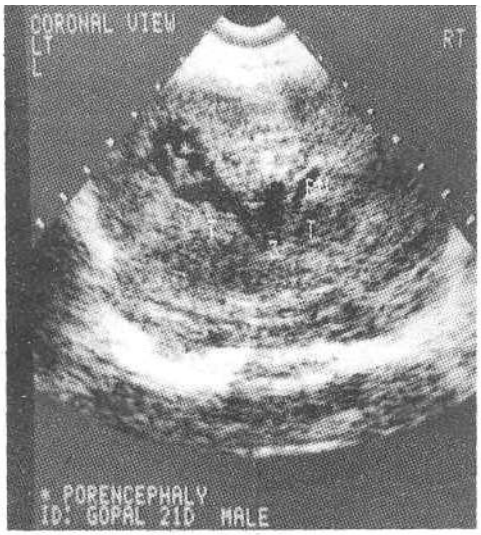

Fig. 3. Mid-coronal image showing pseudo-porencephalic cyst in left cerebral hemisphere communicating with left lateral ventricle.

shift of falx to opposite side (away from midline), compression of lateral ventricle on same side and dilatation of lateral ventricle on opposite side. EAFC will show lot of internal echoes in case of empyema, whereas EAFC is hypoechoic when fluid is sterile(l). In the present study, EAFC was seen in two cases. One of them had subdural effusion, CT confirmed *(Fig. 4)* which, improved following repeated aspiration. In this patient aspirated fluid was sterile on culture. Contrast CT scan of another patient of EAFC revealed epidural effusion *(Fig. 5)*. In this patient, aspiration revealed thick pus. He absconded following the diagnosis. Chowdhury *et al.*(7) and Fisher *et al.*(1) reported subdural effusion in 8 and 18% cases of pyogenic meningitis, respectively.

The present study revealed a good correlation between sonographic abnormalities and cerebrospinal cell (pus) count and protein levels but not with CSF sugar, as reported by Chowdhury *et al.*(7).

In conclusion, realtime ultrasonography is a rapid, safe, and effective method for evaluating complications of meningitis, even when signs and symptoms are vague or nonspecific. Ultrasound provides accurate information regarding change in normal neuroanatomy; therefore, adequate medical treatment can be monitored by sequential sonography and surgical intervention can be done when indicated.

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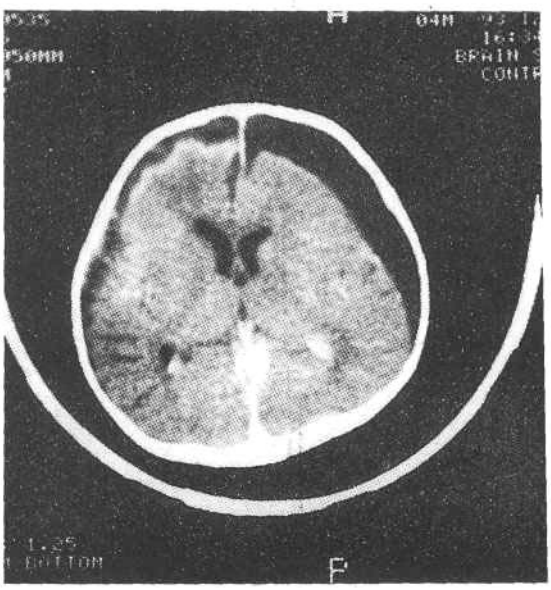

Fig. 4. CT scan shows bilateral subdural effusion.

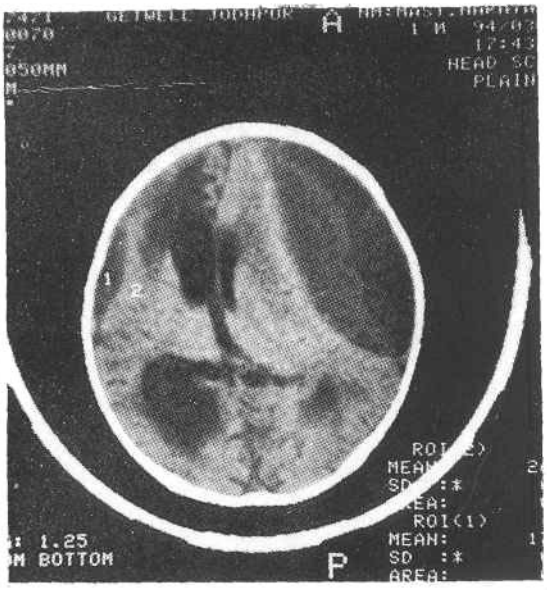

Fig. 5. Mid-coronal CR-USG (negative) and CT scan showing left sided epidural effusion.

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