Cerebral abscess in the under 6 month age group

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SUMMARY Cerebral abscess is rare in children under 6 months of age but is associated with high mortality and morbidity. We report the 12 cases admitted to this hospital in the past 20 years and review the published reports on cerebral abscess in infancy and childhood. Although mortality associated with cerebral abscess in infancy has improved, morbidity is still high. We recommend that all infants aged under 6 months presenting with bacterial meningitis have a computed tomogram or ultrasound examination to exclude cerebral abscess and hydrocephalus.

Patients

The study included all infants aged less than 6 months admitted with cerebral abscess in the years 1961-82, except those with any predisposing congenital anomaly such as meningomyelocele.

Results

Clinical details of the infants are listed in Table 1. Eleven of the babies were 3 months of age or less.

Three were preterm. Two preterm babies and one other had a documented preceding septicaemia. Seven infants presented with fits (6 generalised, one focal), 9 with fever, 8 with meningitis, and 7 with raised intracranial pressure, evidenced by a bulging fontanelle or abnormally increasing head circumference. Only one had a focal neurological sign (a divergent strabismus).

The method of diagnosis of cerebral abscess varied according to the technology available. Of the three patients early in the series (1961-2) one

Table 1 *Clinical features of infants with cerebral abscess*

Case (year)	Sex	Age	Predisposing conditions and symptoms	Signs
1 (1961)	M	10 dys	Preterm, 35 wks: fitting <24 hrs.	Fever, no focal signs.
2 (1961)	M	5 mths	Fever, irritability, poor feeding for 7 dys.	No focal signs, fever.
3 (1962)	F	3 wks	Fitting <24 hrs.	Divergent squint, rapid rise in head circumference (OFC).
4 (1969)*	M	6 wks	Preterm, 32 wks: severe HMD, apnoea, anaemia, proteus and streptococcus septicaemia wk 1 of life.	Rapid increase in OFC over 1 wk before transfer.
5 (1972)	M	5 wks	Febrile, irritable, drowsy for 3 dys.	Full fontanelle, OFC >97th percentile.
6 (1972)	F	6 wks	Febrile, fitting for 1 wk.	Febrile, head and eyes deviated to right.
7 (1978)*	M	6 wks	Fitting, lethargic for 3 dys.	Rapidly increasing OFC.
8 (1978)*	M	6 wks	Fitting and vomiting for 3 dys.	Fever, hypertonia, meningism.
9 (1981)*	F	3 mths	Preterm, 28 wks: severe HMD, serratia sepsis in neonatal period, fever.	Febrile, full fontanelle 6 dys before diagnosis.
10 (1982)*	M	4 wks	Small for gestational age term infant: proteus meningitis at 3 wks.	Fitting, febrile, full fontanelle.
11 (1982)*	M	5 wks	Suppurative bilateral otitis media, proteus meningitis at 4 wks.	Fever, irritable, full fontanelle, head to right, eyes to left: ultrasound suggested abscess.
12 (1982)*	F	6 wks	Citrobacter septicaemia on dy 3, citrobacter meningitis at 3 wks.	Fever, fitting, full fontanelle, hyper-reflexia.

*Transferred from another hospital
HMD=hyaline membrane disease.

Fig. 1 CT scan case 10 showing abscess and cerebritis.

was diagnosed at necropsy, one by ventricular and cerebral puncture, and one by ventriculogram (Table 2). The three infants presenting between 1969 and 1972 were diagnosed by radionuclide (RN) scans and computed tomography (CT) was available for the 6 most recent cases (1978–82). Five of these 9 infants had multiple abscesses. A clinical diagnosis of cerebral abscess was suspected in only two infants.

Fig. 2 CT scan case 9 showing frontal abscess.

Table 2 Mode of diagnosis, EEG result, and site of abscess

Case (year)	Mode of diagnosis. EEG findings	Site of abscess
1 (1961)	Necropsy: no EEG.	Frontal and occipital—R and L.
2 (1961)	Ventricular and cerebral punctures on dy of admission: no EEG.	R parietal.
3 (1962)	Ventriculogram 3 wks after admission: EEG, frontotemporal abnormality.	L parietal.
4 (1969)	Ventriculogram, nuclear brain scan dy of admission: EEG, low voltage, no focal anomaly.	R frontal, L temperofrontal.
5 (1972)	Nuclear brain scan 7 dys after admission: EEG, R frontal slow wave focus.	R frontal, L posterior parietal.
6 (1972)	Nuclear brain scan 5 dys after admission: no EEG.	L posterior, R parietal.
7 (1978)	Nuclear brain scan and CT scans on dy of admission: EEG, multifocal epileptic process.	L and R frontal and occipital.
8 (1978)	CT scan dy of admission: EEG, multifocal bilateral epileptic activity.	R frontal.
9 (1981)	CT scan 3 dys after admission: no EEG.	Large L and midline frontal.
10 (1982)	Ultrasound, CT scans done serially before and during abscess formation. EEG, scattered epileptic activity and abnormal bilateral low voltage activity.	Roof of R lateral ventricle.
11 (1982)	Ultrasound, CT scan dy of admission: no EEG.	Bilateral frontal.
12 (1982)	CT scan dy of admission RAHC (6 dys after onset of fever, irritability), no EEG.	R frontal.

R = right; L = left; EEG = electroencephalogram; CT = computed tomography.

Table 3 Laboratory findings in infants with cerebral abscess

Case (year)	Laboratory findings
1 (1961)	Lumbar CSF—polymorphs $1 \times 10^9/l$; protein 4 g/l; Gram negative rods; coliform bacillus. Culture of abscess contents— <i>Escherichia coli</i> .
2 (1961)	Haemoglobin 12.6 g/dl; white cell count $26 \times 10^9/l$ (mostly neutrophils). Culture of abscess contents— <i>Staphylococcus aureus</i> .
3 (1962)	Lumbar CSF—polymorphs $4.5 \times 10^9/l$; protein 6 g/l; culture, <i>E. coli</i> . Culture of abscess contents— <i>E. coli</i> . Haemoglobin 16.6 g/dl; white cell count $30 \times 10^9/l$ (65% polymorphs).
4 (1969)	Blood culture— <i>Proteus vulgaris</i> . Haemoglobin 11.8 g/dl; white cell count, normal total count with left shift. Ventricular tap culture— <i>Pr. vulgaris</i> . Culture of abscess contents— <i>Pr. vulgaris</i> .
5 (1972)	Haemoglobin 11 g/dl; white cell count $11 \times 10^9/l$ (39% polymorphs). Lumbar CSF—white cell count $0.512 \times 10^9/l$ (20% polymorphs); protein 4 g/dl; no culture growth. Culture of abscess contents—coliform bacillus.
6 (1972)	Lumbar CSF—white cell count $2.25 \times 10^9/l$; protein 1 g/l; culture, <i>E. coli</i> . Culture of abscess— <i>E. coli</i> .
7 (1978)	Haemoglobin 14.7 g/dl; white cell count $22 \times 10^9/l$ (55% polymorphs). Lumbar CSF—monocytes $.059 \times 10^9/l$, polymorphs $.009 \times 10^9/l$; protein 1.39 g/l; no culture growth. Culture of abscess contents— <i>Pr. vulgaris</i> .
8 (1978)	Lumbar CSF—white cell count $22 \times 10^9/l$ (95% polymorphs); protein 5 g/l; culture, <i>Pr. vulgaris</i> . Culture of abscess contents— <i>Pr. vulgaris</i> .
9 (1981)	Lumbar CSF—polymorphs $5.75 \times 10^9/l$; protein 1.2 g/l; culture, <i>Serratia marcescens</i> . Blood culture; no growth. Culture of abscess contents—no growth.
10 (1982)	Lumbar CSF—polymorphs $2 \times 10^9/l$; culture, <i>Pr. vulgaris</i> . Haemoglobin 12.7 g/dl; normal blood film. Culture of abscess contents—no growth.
11 (1982)	Lumbar CSF—polymorphs; culture, <i>Pr. vulgaris</i> . Haemoglobin 8.8 g/dl; normochromic; white cell count, normal. Culture of abscess contents— <i>Pr. vulgaris</i> .
12 (1982)	Lumbar CSF—polymorphs $2.3 \times 10^9/l$; protein 3 g/l; culture, <i>Citrobacter diversus</i> . Culture of abscess contents— <i>Citrobacter diversus</i> .

CSF=cerebrospinal fluid.

Electroencephalograms (EEG) were performed in 6 patients—three of them showed epileptic activity and the remainder abnormal low voltage activity. EEG's proved inaccurate for diagnosis and localisation. The commonest sites for abscesses were the frontal and parietal lobes.

Eleven babies had cerebrospinal fluid (CSF) specimens taken (10 by lumbar puncture, and one ventricular). Eight of the specimens grew the causative organisms, which were confirmed by aspiration of the abscess. These were *Escherichia coli* (4 infants), *Proteus vulgaris* (5 infants), *Staphylococcus aureus* (1 infant), *Citrobacter diversus* (1 infant), and *Serratia marcescens* (1 infant) (Table 3). The abscess cavity was aspirated and antibiotic instilled in 9 babies. All had parenteral antibiotics, the duration varying from 3–7 weeks, and two infants subsequently had a prolonged course of oral antibiotics. Ten babies were given anticonvulsants (8 for fitting) but only three continued them for more than a year. Steroids were used twice (Table 4).

In this series mortality was 33%. Three deaths occurred in infants treated before 1970 and one in 1982. At the latest follow up four of the surviving children were neurologically and developmentally normal, one was in a class for mildly retarded

children and had a hemiparesis (case 5), four had hydrocephalus requiring shunting (cases 3, 5, 11, and 12) and two had fits after recovery from the initial illness (cases 5 and 7). One of the recent patients (case 11) has not been followed long enough to assess his eventual neurological outcome and the other (case 12) is blind and hypertonic.

Discussion

There are more infants aged less than 6 months in the present series than in previous studies,¹ most of which have not confined themselves to this age group. Several causes of cerebral abscess in infancy have been cited. In 1898, Holt² found that trauma and otitis media were the most common predisposing factors. Other causes suggested have been skin and umbilical sepsis,³ osteomyelitis,³ and meningitis.^{4–6} In most series⁷ no predisposing causes were found in approximately 25% of the cases (77% in the present study).

In 1973, Samson and Clark⁸ emphasised the importance of culturing for anaerobes in cerebral abscess pus. It is clear that both anaerobes and Gram negative organisms have replaced *Staphylococcus* and *Streptococcus* as the most common

Table 4 Management and outcome

Case (year)	Management	Outcome
1 (1961)	IM streptomycin, IV chloramphenicol, oral sulphadimidine, IM phenobarbitone.	Death after 24 hours.
2 (1961)	IM streptomycin, IM methicillin, IM chloramphenicol, oral erythromycin, oral sulphadimide (4–5 weeks).	Death 1 month after admission.
3 (1962)	IM streptomycin, chloramphenicol (3 wks), oral sulphadimidine (2 wks), oral phenobarbitone (5 wks), oral prednisone.	Hydrocephalus. Lost to follow up.
4 (1969)	IV chloramphenicol, IM kanamycin, IM ampicillin, oral sulphadimidine (5 wks). Daily aspiration of abscess and instillation of kanamycin (1 mth), oral phenytoin, methylphenobarbitone.	Died at 6 months of aspiration.
5 (1972)	IM gentamicin, penicillin (1 mth), oral co-trimoxazole (7 wks). Daily aspiration abscess and instillation of gentamicin (2 wks), methylphenobarbitone.	Age 10 yrs—hydrocephalus (VP shunt), right hemiparesis, mild retardation, no fits.
6 (1972)	IV chloramphenicol, aspiration abscess and instillation gentamicin into ventricular reservoir (1 mth), phenobarbitone (temporarily).	Age 3 yrs—normal, no fits, lost to follow up
7 (1978)	IV chloramphenicol, IM gentamicin (7 wks). Aspiration of abscess and instillation penicillin, dexamethasone, phenytoin, phenobarbitone long term.	Age 4 yrs—normal neurologically and developmentally, slight increase in ventricular size, 1 fit only with fever. 7 mths after abscess.
8 (1978)	IV chloramphenicol, ampicillin (6 wks). Daily aspiration abscess and instillation of chloramphenicol (15 dys), phenobarbitone (1 yr).	Age 4 yrs—normal neurologically and developmentally, no fits.
9 (1981)	IV chloramphenicol, gentamicin (3 wks), oral chloramphenicol, co-trimoxazole (5 wks). Drainage of abscess ($\times 1$), carbamazepine long term.	Age 2 yrs—normal neurologically and developmentally, no fits.
10 (1982)	IV chloramphenicol (2 wks), IV ampicillin (1 mth), IV gentamicin (6 dys), IV co-trimoxazole (2 wks). Rickham ventricular reservoir, phenytoin, phenobarbitone.	Hydrocephalus, grossly retarded, died 6 wks after admission.
11 (1982)	IV chloramphenicol, ampicillin (6 wks). Aspiration of abscess ($\times 6$), Rickham reservoir to tap ventricles and relieve hydrocephalus.	Hydrocephalus, VP shunt, porencephaly right frontal lobe. Neurologically normal at discharge.
12 (1982)	IV chloramphenicol (1 wk), IV gentamicin cefotaxime—(6 wks). Drainage of abscess ($\times 2$), Rickham reservoir to relieve hydrocephalus till CSF clear, phenobarbitone, phenytoin.	Hydrocephalus, VP shunt, hypertonic, blind.

CSF = cerebrospinal fluid; VP = ventriculoperitoneal.

causative organisms,^{5 9–13} and in a recent report¹⁴ *Candida albicans* was the pathogen. The most common organisms in the present series were Gram negative. It is of interest that the pathogen in one of the recent patients was *Serratia marcescens*, an organism appearing more frequently in neonatal intensive care units,¹⁵ but hitherto not reported as a cause of cerebral abscess in infancy.

The most frequent presenting symptoms of cerebral abscess in infancy are vomiting,¹¹ convulsions,^{9 11} and an enlarging head.⁷ In Sanford's series,⁹ 72% had ocular signs such as nystagmus, strabismus, and pupillary changes. All authors emphasise the difficulty in making an early diagnosis. In the earlier series most diagnoses were made at necropsy (100% of Holt's cases).

There is general agreement that parenteral penicillin and chloramphenicol are the initial antibiotics of choice to cover the most likely pathogens.^{7 8} Prolonged antibiotic treatment lasting 6 weeks is recommended.⁷

The operative management of cerebral abscess is controversial. Aspiration of the abscess cavity is considered sufficient for a total cure by some authors.^{7 11 17} Wright and Ballantine¹⁸ and Samson and Clark,⁸ however, found a lower mortality in those undergoing total excision of the abscess. In this series the experience with repeated aspiration was good. It is likely that unless there is a well defined abscess capsule (a rare occurrence) total excision of the abscess would destroy normal brain tissue.

Although mortality from cerebral abscess in infancy is clearly falling, morbidity is still high. From the studies with reliable follow up data^{5 19} it seems that about 60% of children may have neurological and developmental sequelae. The commonest problems reported^{16 19} are hemiparesis, seizures, visual field defects, and learning difficulties.

Legg, Gupta, and Scott²⁰ reviewed 70 patients who had cerebral abscess, none of whom was under 6 months old. The incidence of epilepsy in their

follow up of 3–30 years was 72%. The interval between diagnosis of cerebral abscess and onset of epilepsy was longest in the younger patients. Onset of fits was maximal 4–5 years after the abscess. Prospective studies are needed to assess the value of giving anticonvulsants for five years after a cerebral abscess.

Conclusions

The diagnosis of cerebral abscess in babies aged under 6 months remains difficult. It must be suspected in any infant with a CSF pleocytosis accompanied by an unusual organism (citrobacter, proteus, serratia) or no organism.^{4 6 20} Prolonged fitting or a rapidly enlarging head are other diagnostic pointers.

To improve outcome, brain abscess should preferably be diagnosed at the stage of cerebritis. To this end, RN and CT scans and ultrasound have simplified the investigative process.²¹ Ultrasound is a useful tool in neonates and preferable to CT where multiple studies need to be done. It is a mobile service and enables close monitoring of the development of cerebritis, cerebral abscess, and complications such as ventriculitis. The RN scan is positive in most inflammatory brain lesions, whereas CT has a lower detection rate unless a discrete abscess is present.²² If either of these scans is negative and the clinical diagnosis is cerebral abscess then the other study should be done.²² RN scan and CT also show hydrocephalus which, although usually temporary, is present in a high proportion of infants with meningitis.

It is the practice at this hospital to perform ultrasound or CT, or both in all infants aged under 6 months presenting with bacterial meningitis. As a result of this policy, three patients with unsuspected cerebral abscess (cases 8, 9, and 12) were diagnosed and two (cases 4 and 11) were discovered to have multiple abscesses. Because a cerebral abscess was anticipated in the infant in case 10, serial ultrasound studies were performed and the diagnosis was made early. Unfortunately this did not alter the outcome.

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