6. Ternberg LJ, Keathing JP. Acute acalculous cholecystitis complication of other illnesses in childhood. Arch Surg 1975, 110: 543-547.

7. Barth RA, Brasch RC, Fill RA. Abdominal pseudotumor in childhood. AJR 1981, 136: 341-343.

8. Kumari S, Lee WJ, Baron MG. Hydrops of the gall bladder in a child-diagnosis by ultrasonography. Pediatrics 1979, 63: 295-297.

Acanthameba Meningoencephalitis Complicating Pyogenic Meningitis

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Two genera of free-living amebae, *Naegleria* and *Acanthameba* are known to cause primary amebic meningoencephalitis (PAM) in man. Fowler and Carter in 1965(1) were the first to report 4 fatal cases of PAM. Despite their widespread distribution in soil, water, sewage and even air, PAM is a very rare disease. Less than 120 cases have been reported in world literature, of which less than 30 are due to *Acanthameba*(2). From India, Pan *et al.*(3) and Gogate *et al.*(4) have reported 2 cases each, all 4 due to Acanthameba. The extreme rarity and unusual clinical presentation in our case, has prompted us to report it.

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Case Report

On November 11, 1989, a 7 week-old-male infant was referred, from Baroda, for treatment of resistant ventriculitis. The child had developed pyogenic meningitis on the sixth day of life, following a normal vaginal hospital delivery. He responded well to 3 weeks treatment with intravenous antibiotics and other supportive measures. Four days later, the child was readmitted with complaints of poor sucking and convulsions. Although full details of treatment given were not available, the mother stated that 4 lumbar punctures and 2 ventricular taps were done during the two hospital admissions at Baroda.

At the time of admission with us, the baby was having recurrent generalized tonic spasms with partial loss of consciousness. Physical examination revealed a head circumference of 39 cm, with a tense bulging anterior fontanelle, absent menace reflex and exaggerated deep tendon reflexes. Investigations done revealed a normal total leucocyte count and *X*-ray chest. Ventricular fluid was suggestive of pyogenic meningitis, but no organisms were detected on Gram stain and routine culture (*Table I*). CT showed evidence of moderate hydrocephalus, with a hyperdense area in the right frontal region (*Fig.*). This mass lesion was thought to be a hematoma resulting from the earlier ventricular taps done at Baroda.

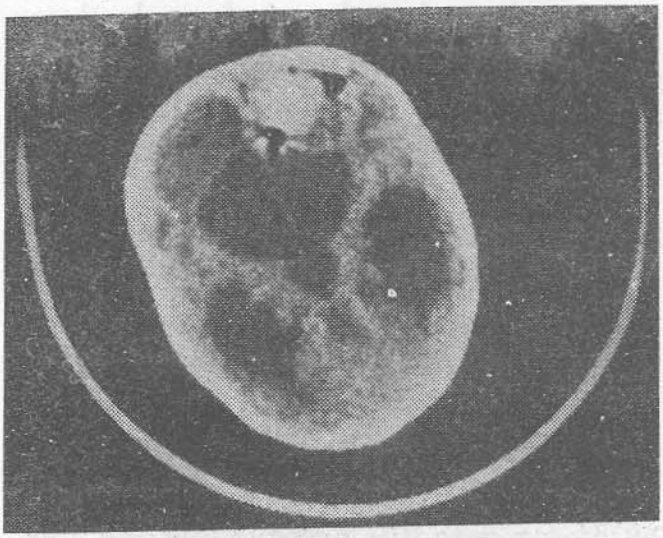

Fig. CT head on admission showing hyperdense area in right frontal region (black dots) with moderate hydrocephalus.

With a diagnosis of resistant pyogenic ventriculitis, 3 weeks treatment with cefotaxime and amikacin was given. Alternate right and left taps were done whenever there was clinical evidence of raised intracranial pressure. Each time, 3 mg of amikacin was instilled intraventricularly. At the end of 3 weeks, as ventricular fluid still showed evidence of infection, it was sent to rule out a parasitic, anaerobic or fungal etiology (*Table I*).

To our surprise, CSF wet mount examined within 20 min of collection, showed presence of *Acanthameba* trophozoites, and this was reconfirmed thrice subsequently by daily taps. All simultaneously done parasitic cultures by monoaxenic cultivation(5) grew *Acanthameba* trophozoites. Serum Ig, T-cell count and serum complement were normal. With a diagnosis of Acanthameba meningoencephalitis, intravenaus cotrimoxazole (7.5 mg/kg/day, 12 hourly) was given for 3 weeks in which time the interval between therapeutic tapping progressively increased, indicating a response to treatment. Subsequently, cotrimoxazole was given orally for 6 days. Ventricular fluid now showed improvement with fewer trophozoites (*Table I*).

TABLE I—*Serial Ventricular Fluid Examination Findings*

Date	11-11-1989	1-12-1989	28-12-1989	10-1-1990	18-1-1990
Appearance	yellow turbid	Yellowish hazy	Yellowish clear	Clear colorless	Clear colorless
Total cells/mm^3	340	280	21	13	5
Polymorphs/mm^3	300	220	17	2	0
Lymphocytes/mm^3	40	60	4	11	5
Protein (g/dl)	0.75	0.66	0.8	0.8	0.7
Sugar (mg/dl)	16	16	36	36	38
Gram stain	NOS	NOS	NOS	NOS	NOS
Routine culture	NOG	NOG	NOG	NOG	NOG
Wet mount	ND	At seen	Fewer At seen	No At seen	No At seen
Parasitic culture	ND	At grown	At grown	No At grown	No At grown

NOS - No organisms seen, NOG - No organisms grown,
ND - Not done, At - Acanthameba trophozoites.

To further the response, 5-Fluorocytosine (5-FC) and sulphadiazine were started orally (both, 150 mg/kg/day, 6 hourly). Total leucocyte count, platelet count, SGOT, SGPT and blood urea nitrogen monitored in view of possible 5-FC toxicity, remained normal. Within 2 weeks, the ventricular fluid showed absence of trophozoites (*Table I*) and further therapeutic ventricular taps were not indicated. At the end of 3 weeks, even polymorphs were absent (*Table I*). Only proteins remained elevated. Both drugs were omitted on completion of 4 weeks treatment. Three weeks later, on 17-2-1990, an elective ventriculo-peritoneal shunt was done as repeat CT showed progression of the hydrocephalus caused by post-meningitic aqueductal stenosis. Ventricular fluid collected at the time of the shunt reconfirmed the cure. The child was discharged on phenobarbitone prophylaxis.

At 8 months follow up the child is thriving well, without seizures, but has bilateral optic atrophy and a DQ of 50.

Discussion

PAM due to Acanthameba is an opportunistic CNS infection which may be associated with even systemic dissemination(6). It occurs in chronically ill, debilitated and immunocompromised patients. Martinez(7,8) has suggested the term granulomatous amebic encephalitis(GAE), which manifests as focal neurological deficits, raised intracranial pressure, and occasionally as an expanding intracranial mass(7), with death occurring within a few weeks. The precise portal of CNS entry is unknown, but is believed to be hematogenous, either from the lungs or skin(6). GAE has also occurred due to direct inoculation of the amebae into the CNS after trauma(9). Upto 1984, most cases had been diagnosed only on post-mortem, and some only a few days before death on biopsy of an intracranial mass.

Gogate *et al*,(4) were the first to demonstrate and even culture *Acanthameba* trophozoites from CSF collected antemortem in 2 cases with acute PAM. Acanthameba trophozoites can be seen in a fresh wet mount of CSF examined under low power. CSF should be unstained, and centrifuged at low speed to appreciate the sluggish *Acanthameba* trophozoites, which lack labopodia.

In our case it is possible that the amebae were inadvertently introduced during ventricular taps done in the neonatal period. All taps in our hospital were done in the Neurosurgery Theatre. The demonstration of a hyperdense space occupying lesion on CT in a patient with meningoencephalitis, especially when carotid angiography reveals an avascular mass, suggests the possibility of GAE(7). Brain biopsy has been diagnostic in 6 out of 8 such reported cases(8). When CSF proved to be diagnostic in our cases, biopsy of the frontal mass was not considered essential. As the CT at end of drug treatment revealed a markedly thinned out cerebral mantle, the effect of treatment on the hyperdense area could not be commented upon.

Ofori-Kwakye *et al*.(10) in 1986 have reported *Acanthamebic* CNS disease, presenting only as an intracranial mass, *i.e.*, without any associated meningoencephalitis. Diagnosis was made only after surgical excision and histopathological examination. As clinicians become more aware of the possibility of Acanthamebiasis in nonresponsive meningitis, it is possible that early diagnosis and institution of effective treatment, as in our case, will become more common. As yet, all chemotherapeu-

tic regimens remain empirical, although, sulfonamides, 5-FC and hydroxystibamide isethionate have been recommended(2).

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REFERENCES

1. Fowler M, Carter RF. Acute pyogenic meningitis probably due to Acanthameba sp: A preliminary report. Br Med J 1965, 2: 740-742.
2. Seidel J. Primary amebic meningoencephalitis. Pediatr Clin North Am 1985, 32: 881-892.
3. Pan NR, Ghosh TN. Primary amebic meningo-encephalitis in two Indian children. J Indian Med Assoc 1971, 56: 134-137.
4. Gogate A, Singh BN, Deodhar LP, Jhala HI. Primary amebic meningo-encephalitis caused by Acanthameba. Report of two cases. J Postgrad Med 1984, 30: 125-128.
5. Finegold SM, Baron EJ. Microorganisms encountered in the cerebrospinal fluid. *In:* Bailey and Scott's Diagnostic Microbiology, 7th edn. St Louis, CV Mosby Co, 1986, pp 225-236.
6. Gullett J, Mills J, Hadley K, *et al.* Disseminated granulomatous acanthameba infection presenting as an unusual skin lesion. Am J Med 1979, 67: 891-896.
7. Martinez AJ. Is acanthameba encephalitis an opportunistic infection? Neurology 1980, 30: 567-574.
8. Martinez AJ. Acanthamebiasis and immunosuppression: Case report. J Neuropathol Exp Neurol 1982, 41: 548-557.
9. Rinaldi I, Murphy D. Primary amebic meningoencephalitis with cerebral cerebellar abscesses: Case report. Neurosurgery 1979, 5: 607-610.
10. Ofori-Kwakye SK, Sidebottom DG, Herbert J, Fischer EG, Visvesvara GS. Granulomatous brain tumor caused by Acanthameba: Case report. J Neurosurg 1986, 64: 505-509.

Cholelithiasis Associated with a Variant of Annular Pancreas

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The association of annular pancreas with biliary tract anomalies is extremely rare. Only two cases of agenesis of gall bladder(1) and one case of biliary obstruction caused by annular pancreas(2) have been reported. We report a variant of annular pancreas associated with agenesis of common bile duct, ectasia of extrahepatic biliary tract and cholecystolithiasis.

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