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CT of cerebral hydatid disease

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Summary. Six cases of cerebral hydatid disease (CHD) were seen in Kuwait over a period of 8 years. The typical CT appearance of a large well-defined spherical nonenhanced unilocular cyst was seen in four cases. Two unusual but characteristic types of calcification were seen, one in each of the remaining two cases.

Key words: Cerebral hydatid - Echinococcus - CT

Cerebral hydatid disease (CHD) is very rare. Only about 2% of cases of hydatid disease have cerebral involvement. However, it is important to be aware of the condition, even in nonendemic parts of the world where only the occasional case is encountered. CT has been extremely useful for the diagnosis of CHD and for the planning of appropriate surgical management [1-4].

Patients and methods

Six patients with surgical and histological confirmation of CHD due to *Echinococcus granulosus* were seen in Kuwait between 1978 and 1986. There were five males and one female, ranging in age from 5-45 years (mean, 26 years). The clinical presentations and CT findings are summarized in Table 1. Five patients were Beduins and one was Pakistani. CT scans were performed with an EMI 1010, GE 8800 or GE 9800 CT scanner. Intravenous contrast medium was administered before rescanning in some of the cases.

Results

In four cases (cases 1-4) CT showed the characteristically-large, well-defined smooth, thin-walled spherical unilocular cyst with no calcification or surrounding oedema (Fig. 1). No enhancement was noted when intravenous contrast medium was given. In all four cases, the cyst was removed intact without spilling any of the scolex-bearing cyst fluid by the standard method of forcing saline around the cyst capsule. Three patients had uneventful recovery. In case 1, however, surgery was delayed unnecessarily by the patient's parents, and the child died in the immediate postoperative period.

In the other two cases (cases 5, 6) the CT appearances were rather unusual. A multilocular cyst with rings of calcification was seen in case 5 with adjacent localized brain atrophy (Fig. 2). The patient had undergone a right parietal craniotomy abroad 6 years previously; no information could be obtained about the surgical or histological findings at that time. At reexploration the cyst was found to be adherent to the lateral ventricle, and in spite of careful dissection, the patient developed postoperative intracerebral, intraventricular and extracerebral haematomata. The haematomata were evacuated, but the patient deteriorated, developed progressive hydrocephalus and died seven months later.

In case 6 the skull radiograph showed a fairly

Table 1. Clinical summary and CT findings of CHD

Case no. Age/sex	Site	Clinical summary	CT findings
1/5/F	L. fronto-parietal	1 month headache and vomiting. Neck stiffness. Parkinsonian tremors R. side. Bilateral papilloedema.	Large well-defined smooth-walled unilocular cyst with no surrounding oedema and no enhancement.
2/21/M	L. parieto-occipital	2 months headache and vomiting. Bilateral papilloedema.	Large well-defined smooth-walled unilocular cyst with no surrounding oedema and no enhancement.
3/25/M	L. parieto-occipital	6 months headache and 3 days vomiting. Bilateral papilloedema.	Large well-defined smooth-walled unilocular cyst with no surrounding oedema and no enhancement.
4/45/M	L. parieto-occipital	2 weeks amnesia. Papilloedema R. eye (Leukoma L. eye). Confused. Right lower limb reflexes slightly exaggerated.	Large well-defined smooth-walled unilocular cyst with no surrounding oedema and no enhancement (Fig. 1).
5/21/M	R. parietal	11 years. L. hemiplegia. Had R. parietal craniotomy 6 years ago. 2 days headache, vomiting, fever and diminution of vision. Bilateral optic atrophy. Neck rigidity.	Multilocular cyst with rim calcification and adjacent atrophy (Fig. 2).
6/37/M	R. parietal	2 years headache, diminution of vision and grand mal seizures. Bilateral optic atrophy.	Dense round calcification with internal calcified bands and adjacent atrophy (Fig. 3).

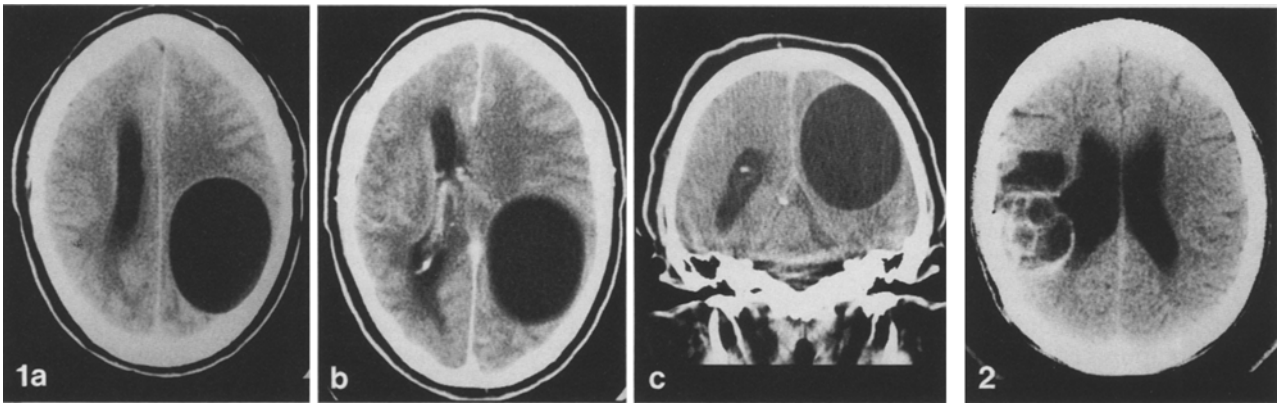

Fig. 1 a–c. Case 4: CT scan, plain (**a**), after contrast (**b**) and a coronal scan after contrast (**c**) show a well-defined smooth-walled unilocular cyst with marked shift of midline structures to the right. No calcification, no surrounding oedema and no enhancement are seen

Fig. 2. Case 5: Plain CT of a multilocular right parietal cyst with rim calcification and adjacent atrophy. Dilated lateral ventricles with no shift of the midline structures. No enhancement seen on the contrast scan

round, calcified lesion with an almost homogeneous ground glass density (Fig. 3 a). With proper windowing, however, the CT showed dense linear strands within the lesion, most likely representing a calcified collapsed cyst membrane (Fig. 3 c). Dilated lateral ventricles and adjacent localized atrophy were noted also in this case (Fig. 3 b). At operation many adhesions were found between dura, arachnoid and overlying cortex. When the cyst capsule was incised, mucoid gelatinous material was evacuated. The deep part of the lesion lay within the lateral ventricle and was adherent to the choroid plexus. This was freed and the lesion completely removed. The patient made an uneventful recovery.

Discussion

The adult worm of *Echinococcus granulosus* lives in the small intestine of the dog; the definitive host. The intermediate host, usually sheep, and only occasionally man, is infected by swallowing the ova

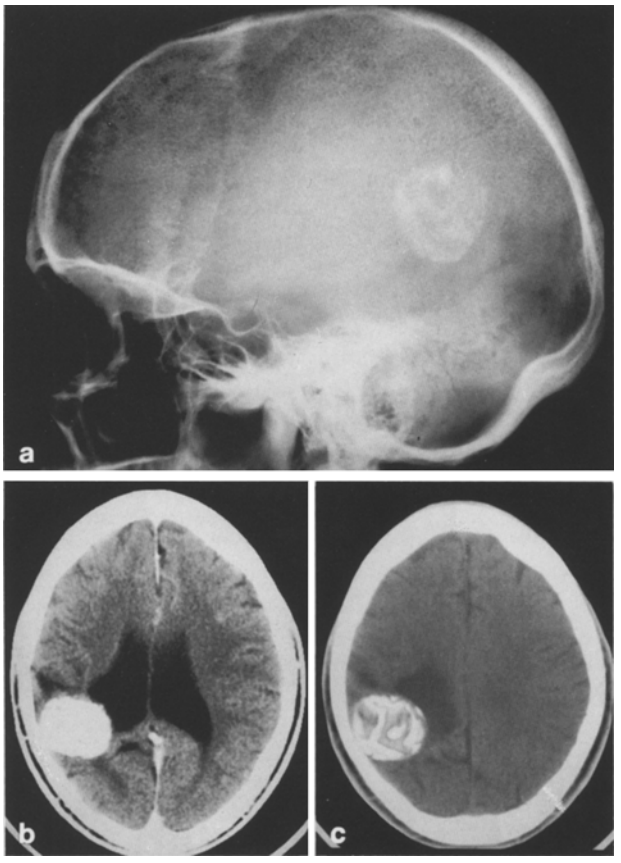

Fig. 3 a–c. Case 6: Skull radiograph (**a**), contrast enhanced CT scan with standard (**b**) and plain CT with high window setting (**c**), show fairly round dense calcification with internal calcified bands and adjacent atrophy. Dilated lateral ventricles particularly the right which is also drawn towards the lesion. No enhancement seen on the contrast scan

either from close contact with dogs or by ingesting food contaminated by dog's faeces.

Human cystic hydatid disease is caused by the larval form of Echinococcus granulosus. The embryo penetrates the intestinal mucosa and reaches the liver via the portal circulation. Only embryos which succeed in passing through the hepatic and pulmonary filtering systems reach the brain by the systemic circulation [5]. Although hydatid cysts can occur practically anywhere in the body, the commonest site is the liver. Only about 2% occur in the brain.

Hydatid cysts are usually acquired in childhood and grow in size slowly but progressively. The common presentation is that of a child or young adult with signs and symptoms of raised intracranial pressure, slowly progressing over a period of 1-6 months. The patients remain in remarkably good condition with relatively little neurological deficit [2], despite the large size of the cyst, the considerable mass effect and bilateral papilloedema. This is due to the slow growth of the cyst with no oedema and no reaction of the surrounding brain tissue. The parietal lobe is by far the commonest part of the brain to be involved [6], as occurred in all six cases in the present series. Less commonly, intraventricular or infratentorial sites have been reported [3, 7], including one in the pons [8] and one in the aqueduct of Sylvius [9].

Fortunately in the majority of cases the CT appearances are typical, as in four of our six cases. This is also true in the larger series reported from countries where the disease is common [2, 3]. Other lesions to be differentiated from CHD are arachnoid cyst, porencephalic cyst and cystic tumours. The arachnoid cyst is not so round and is not surrounded by brain tissue as in CHD and the porencephalic cyst usually communicates with the ventricle. Cystic tumours usually have a soft tissue part which enhances after intravenous contrast.

In the rare case where the cyst is multilocular this is usually due to rupture of the primary cyst. When Sharma et al., 1982 [10] reviewed the literature there were only 9 such cases reported and he reported the first multiple primary hydatid cyst of the brain. In case 5 of our series (Fig. 2) presumably the cyst ruptured during previous surgery. Cerebral hydatid cysts are well protected and vascularised and rarely undergo degeneration. This explains why calcification which is not uncommon in liver hydatid cysts is extremely rare and occurs in less than 1% of all cerebral hydatid cysts [7]. Dense calcification as seen in case 6 (Fig. 3) is even rarer than rim calcification. Only one similar case was reported where a densely calcified mass was shown both by plain skull radiography and CT [11]. In our case, however, with proper windowing it was possible to see the internal structure suggestive of a calcified collapsed membrane, almost identical to the cross sectional appearance of a specimen of a hydatid cyst of the liver [5]. If seen, therefore, it should be diagnostic of CHD. Calcified tuberculomata are usually irregular in outline and density and can easily be differentiated from CHD.

Hydatid disease is still prevalent in some countries and with increased travel isolated cases can be seen anywhere in the World [12, 13]. CT scanning, which is more widely available now than in previous years, is extremely useful for diagnosis and hence for proper surgical management of the condition.

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