

M. Tunaci
A. Tunaci
G. Engin
B. Özkorkmaz
B. Ahishali
I. Rozanes

MRI of cerebral alveolar echinococcosis

I.Ü. Kütüphane ve Dok. D. Bşk.

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M. Tunaci (✉)
Department of Radiology,
Istanbul Medical Faculty,
PK 112 Acibadem, Kadıköy,
Istanbul, Turkey

Abstract Cerebral alveolar echinococcosis is rare. We report a case with multiple intracranial masses which show cauliflower-like contrast enhancement pattern on MRI. The lesions originated from hepatic involvement with invasion of the inferior vena cava.

Key words Brain · Echinococcosis alveolar · Magnetic resonance imaging

Introduction

Alveolar echinococcosis (AE), a rare parasitic disease due to the larva of *Echinococcus multilocularis*, is endemic in central Europe, the former Soviet Union, northern Iran, northern Afghanistan, eastern Turkey, Japan and central and northern North America [1–3]. Their primary site of involvement is the liver; haematogenous dissemination to other organs such as the lungs, brain or bone occurs in only 5% of cases [4]. The AE lesion in any site behaves like a malignant neoplasm, growing by exogenous proliferation, with invasive and destructive change; distant metastases may also occur [2].

We report multiple cerebral AE lesions from a primary hepatic focus, with and secondary pulmonary involvement revealed by CT.

Case report

A 28-year-old woman presented with headache and vomiting. She had undergone laparotomy for abdominal masses 3 years previously, when hepatic AE was diagnosed. Examination disclosed a left homonymous hemianopia, neurogenic hypoacusis, hemiparesis and hemihypoalgesia. The EEG showed right frontoparietal slow waves; the blood eosinophil count was 25%, gamma globulin was elevated, the indirect haemagglutination titre was more than 1:2048 for echinococcus protein and the Casoni skin test was strongly positive.

MRI, performed after resection of a lesion in the right parietal lobe, demonstrated four intraparenchymal masses: one in the left frontal, one in the left parietal and one in the right occipital lobe the fourth being in the right frontoparietal operculum. All lesions were isointense with white matter but contained nodular low-signal areas on T1-weighted images. T2-weighted images revealed heterogeneous low-intensity masses with high signal consistent with oedema around them. Diffuse, heterogeneous contrast enhancement of the lesions resembled a cauliflower (Fig. 1). Dural contrast enhancement adjacent to the left parietal lesion was also seen (Fig. 2). Biopsy of the right parietal lesion confirmed AE histologically. The patient was treated with albendazole but died during the second month of treatment.

Discussion

The adult echinococcus worm lives attached to the intestinal villi of the primary hosts, red and white foxes, field mice and voles. The larval stage occurs in a variety of animals, including humans, accidentally infected by direct contact with rodents or ingestion of contaminated vegetables or water. After being ingested, embryos hatch in the duodenum, and the embryos penetrate the intestinal wall and enter the portal circulation. The main metastatic pathway of AE extends from the liver to the inferior vena cava and right atrium. Afterwards, the embryos may implant in lung parenchyma, forming a source of metastases to the systemic arterial circulation.

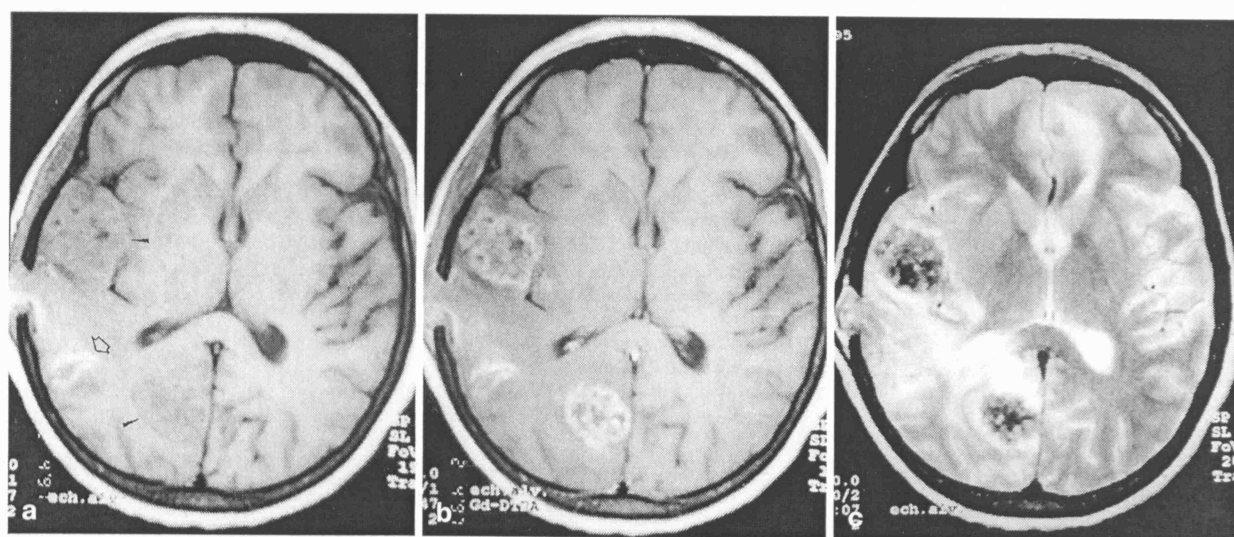


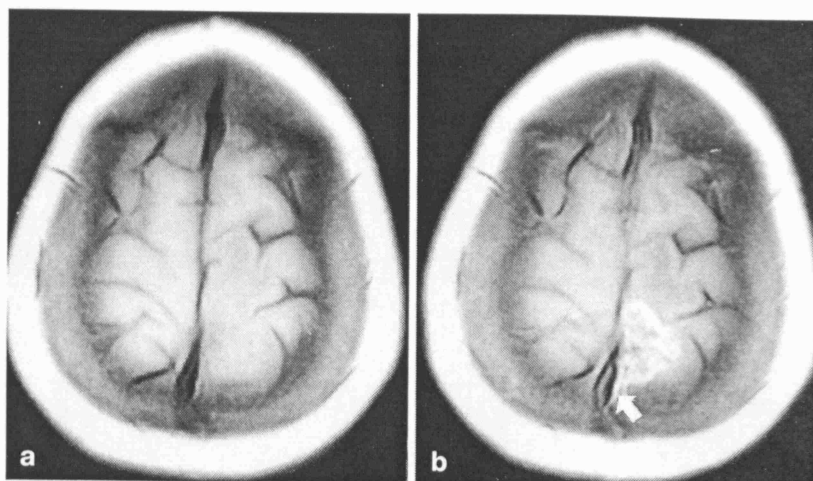
Fig. 1 **a** On T1-weighted images two masses, isointense with white matter, are seen, containing nodular low-signal areas (*arrowheads*). A craniotomy defect and gyrus high signal are seen in the right parietal lobe (*open arrow*). **b** There is diffuse, heterogeneous contrast enhancement, showing a (cauliflower-like appearance). **c** T2-weighted image demonstrates heterogeneous low-intensity masses with surrounding oedema

Cerebral AE lesions are usually supratentorial, mainly in the territory of the middle cerebral artery [5]; they may be multifocal or solitary. In our case, the multiplicity of the intracranial lesions and primary focus in the liver suggest that haematogeneous spread was responsible for the cranial metastases.

MRI and CT appearances of cerebral AE lesions have been reported [3, 7–8]. They are generally mul-

tilocular cystic masses with sharp margins. Calcification and surrounding oedema are common. Contrast enhancement occurs around the cysts in the inflammatory zone. A few cases have shown enhancement patterns including heterogeneous, ring, peripheral and nodular enhancement [3, 7, 8]. Another report described AE lesions without enhancement; lack of enhancement may be a useful clue in differentiating these lesions from cerebral abscesses and tuberculomas [3, 6]. In our case, the cranial lesions showed cauliflower-like contrast enhancement. This pattern may help to distinguish these lesions from tumours, particularly oligodendrogliomas, tuberculomas and brain abscesses. The dural contrast enhancement probably corresponded to the dural involvement reported previously [3].

Fig. 2a,b T1-weighted images before and after contrast medium show a heterogeneously enhancing lesion, and dural enhancement (*arrow*)



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