

# Cerebral hydatid disease: CT and MR imaging findings

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## Summary

**Objective:** Cerebral hydatid disease is very rare, representing only 2% of all cerebral space occupying lesions even in the countries where the disease is endemic. The aim of this paper is to describe the characteristic features of cerebral hydatid disease in computed tomography (CT) and magnetic resonance imaging (MRI).

**Methods:** We retrospectively reviewed the CT and MR imaging findings of 18 patients with pathologically confirmed cerebral hydatid disease over a period of 13 years (1990–2002).

**Results:** The study group consisted of 17 cases of *Echinococcus granulosus* and 1 case of *Echinococcus multilocularis (alveolaris)*. They were 12 male (66.7%), and 6 female patients (33.3%), ages ranging from 7 to 50 years with an average age of 20.3 years. Headache, vomiting and seizures were the predominant symptoms. Papilloedema was present in 14 patients (77.7%). Common CT and MR imaging findings of *E. granulosus* lesions were well-defined, smooth thin-walled, spherical, homoge-

neous cystic lesions with no contrast enhancement, no calcification, and no surrounding oedema. The lesion seen with *E. multilocularis* was a well-defined multiseptated mass consisting of solid and cystic components with calcification in the solid portion. Cystic lesions with surrounding hyperintensity of perifocal oedema with complete or incomplete rim enhancement were seen in two patients, and were labeled as complicated and infected cysts.

**Conclusion:** Although cystic cerebral hydatid disease is well demonstrated by CT and MR examinations, CT is superior in detecting calcification in the cyst, when present, MR is better in demonstrating cyst capsule, detecting multiplicity and defining the anatomic relationship of the lesion with the adjacent structures, and it is more helpful in surgical planning.

**Key words:** cerebral hydatid disease; echinococcosis; computer tomography (CT); magnetic resonance imaging (MRI)

## Introduction

Hydatid disease (echinococcosis) is a worldwide zoonosis produced by the larval stage of the *Echinococcus* tapeworm. In humans, the two main types of hydatid disease are caused by *E. granulosus* and *E. multilocularis*. The disease is endemic in many parts of the world, particularly in the Middle East, Australia, New Zealand, South America and central and south Europe, as well as in Turkey. These regions are all noted for the raising of sheep and cattle. It is important to be aware of the condition even in nonendemic parts of the world, where only occasional cases are encountered, because of the rapid movement of large human groups from endemic to nonendemic areas [1]. The diagnosis of hydatid cyst relies on serologic tests and imaging techniques. The growth of hydatid cysts is usually slow and asymptomatic, and clinical manifestations are caused by compression of the involved organ [2, 3]. Cysts may be single or multiple, uni- or multiloculated, and thin- or thick-

walled. More specific signs include visualisation of a calcified wall, presence of daughter cysts, and membrane detachment. Cerebral hydatid cysts are extremely rare, forming only 2% of all intracranial space-occupying lesions. Most cysts are supratentorial [4–6]. They can occur anywhere within the brain, but are especially located in the middle cerebral artery territory. The parietal lobe is the most frequently involved region. The hydatid cyst of the brain is more common in children than in adults. Several diagnostic methods have been employed, but CT has been providing definitive results up to recent years. The exact location, size and number of hydatid cysts in the brain can be determined with a CT scan. However, MR is becoming more and more widely used as a diagnostic tool, as it can show some details that are not be seen on CT [7]. We present the features of CT and MR findings of our cases with cerebral hydatid disease.

## Methods

Over the 13-year period (1990–2002), a total of 18 patients with cerebral hydatid disease were imaged at our institution. In seven cases the lesions were only examined by CT and in three others only by MR. In eight cases the lesions were examined by both CT and MR. The findings of CT and MR were retrospectively evaluated. The CT examinations were performed on two CT scanners: Toshiba-600S and Toshiba-Xvision. MR examinations were performed on three MR imagers: Siemens Magnetom Vision Plus 1.5T, Siemens Magnetom Expert 1.0T, Philips Gyrosan NT 0.5T.

CT examinations were performed with contrast enhancement in 7 patients, without contrast enhancement in 8 patients, and with and without contrast enhancement in 7 patients. All MR examinations were performed with and

without contrast enhancement in 11 patients. On CT and MR, the number, location, internal structure and contour of the lesions, the presence of contrast enhancement, calcification, and surrounding oedema were detected. Histopathological confirmation was done pathologically with specimens obtained by neurosurgery in all patients. Our accepted gold standard for the diagnosis of cerebral hydatid cyst was histopathological examination results. As a follow-up protocol, our patients were called to periodical CT and/or MRI examinations postoperatively, then at sixth months and one year.

All patients underwent a chest x-ray and abdominal ultrasound to reveal any hydatid disease in the lung and/or the liver.

## Results

This study included 12 male and 6 female patients ranging in age from 7 to 50 years with an average age of 20.3 years. The average age of patients with cystic echinococcosis was 18.7 years. The unique case of alveolar echinococcosis was observed in a 48 year old patient. Most of the patients were children, 13 patients out of 18 (72.2%) were between 7 and 15 years of age (table 1). There was a substantial prevalence of male patients 12 cases (66.7%). Among our patients, there were 17 cases of cystic echinococcosis (*E. granulosus*) and 1 case of alveolar echinococcosis (*E. multilocularis*). The most common clinical findings were headache, vomiting, seizures and visual disturbances (table 2). Papilloedema was seen in 14 patients (77.7%), hemiparesis was seen in 7 patients, other neurologic findings are also shown in table 2. There were extracerebral organ involvement in 6 cases.

CT scan and MRI was done for all cases for follow up and showed recurrence of the cysts in 4 patients, severe cerebral hemisphere atrophy in

one patient, mild atrophy in 3 patients, post-operative encephalomalacia in 4 patients, mild hydrocephally in 2 patients and normal post-operative CT/MRI findings in 7 patients (table 1).

In our series most of the cases were single lesions, i.e. 16 patients out of 18 (88.9%), two of them were complicated or infected cysts. Another two cases (2 patients out of 18, 11.1%) had multiple lesions.

In the single-lesion cystic echinococcosis cases, lesions were located as follows: 5 in the parietooccipital region, 3 in the temporoparietal region, 4 in the frontotemporoparietal region, 2 in the temporoparietooccipital region, 2 in the occipital lobe, 1 in the frontotemporal region and 1 in the posterior fossa, (in the fourth ventricle).

In the single-lesion cystic echinococcosis cases, except for the complicated or infected cysts, CT demonstrated large, smooth, thin-walled, spherical well-defined, nonassociated oedema and nonenhancing homogeneous lesions, which have

**Figure 1**

An 11-year-old boy with headache, nausea and vomiting.

A Axial unenhanced CT scan demonstrates well-defined, smooth, thin walled round cyst in the left parieto-occipital region causing mass effect and midline shift to the right. There is no surrounding oedema and no mural nodule to suggest a neoplastic cause.

B Axial contrast-enhanced CT scan of the same patient. No calcification, surrounding oedema, or contrast enhancement is seen.

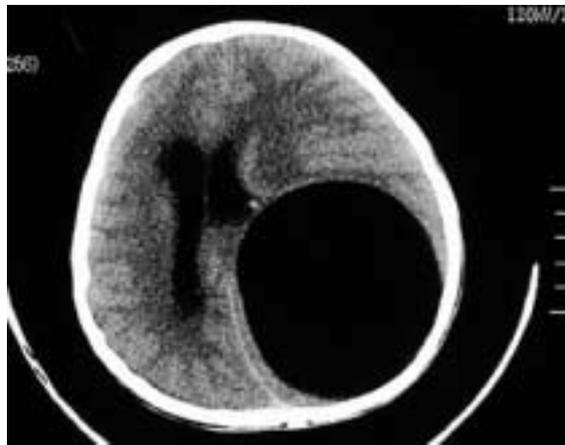


Fig. 1A



Fig. 1B

**Table 1**

Clinical features and CT/MRI follow up of 18 patients with intracranial hydatid cysts.

Case/ Age/Sex	Site/Size	Clinical symptoms	Clinical signs	Type	Clinical outcome	CT/MRI follow up
1/48/M	right temporoparietal 11 × 8.5 cm	headache, vomiting, fever, blurring of vision	left hemiparesis, bil. papilloedema	simple (infected)	grecovery; no deficit	normal
2/11/M	left temporo- parietooccipital 9.5 cm	headache and vomiting, blurring of vision	right hemiparesis, decreased vision of the right eye, bil. papilloedema	simple	alive with headache	recurrent multipl cysts, T <sub>2</sub> hyperintense area in the left occipital region
3/15/M	left parietooccipital, the biggest cyst 5 cm	headache and vomiting	no neurological deficit	multiple	good recovery	normal
4/12/M	left temporoparietal, extending in temporal fossa 7.5 cm	headache, vomiting, fever, blurring of vision	no neurological deficit, bil. papilloedema	simple ruptured (infected)	good recovery	mild left cerebral hemisphere atrophy
5/11/F	left frontotemporo- parietal 9 cm	6 years epilepsy	right hemiparesis, bil. papilloedema	simple	reoperation, alive with epilepsy	recurrent one cyst, mild hydrocephaly left subdural effusion.
6/14/M	right frontotemporo- parietal 8 cm	headache, vomiting	left hemiparesis, bil. papilloedema	simple	reoperation alive good; no deficit	recurrent one cyst, severe right cerebral hemisphere atrophy
7/7/F	post. fossa, 4th ventricle 4 cm	cerebellar deficit, ataxia	ataxia and cerebellar dysfunction, 3rd cranial nerve palsy	simple	good recovery	right temporoparietal encephalomalacia
8/33/F	left parietooccipital 7 cm	headache and vomiting, seizures	6th cranial nerve palsy, bil. papilloedema	simple	alive good; no deficit	mild hydrocephaly
9/50/M	right parietooccipital, 5 cm	headache, vomiting	paresis of the left lower site, seizures	simple	good recovery	normal
10/10/F	Right occipital 4.5 cm	headache, seizures; before 6 months cerebral infarct	central facial palsy, right hemiparesis, speech dis- turbances, bil. papilloedema	simple	reoperation alive good; no deficit	recurrent multiple cysts
11/8/M	left temporo-parieto- occipital 9.5 × 9 cm (20 to 40 mm)	headache, vomiting; difficulity in walking; blurring of vision	ataxia, speech disturbances, bil. papilloedema	multiple	good recovery	left temporo- parietooccipital encephalomalacia
12/13/M	left parietooccipital with xtracalvarial extension 6.5 × 5.5 cm	headache, vomiting, seizures	no neurological deficit, bil. papilloedema	simple multilocular	alive good; no deficit	normal
13/15/M	right frontotemporo- parietal 7 cm	4 yrs epilepsy, blurring of vision	bil. papilloedema	simple	alive with epilepsy	normal
14/9/F	left occipital 8 × 7 cm	headache and vomiting, seizures	no neurological deficit, bil. papilloedema	multiple	good recovery	mild left occipital atrophy
15/14/M	left frontotemporo- parietal 5.5 cm	headache, blurring of vision	no neurological deficit, bil. papilloedema	simple	good recovery	normal
16/39/F	right parietooccipital, 8 × 9 cm	headache, vomiting	no neurological deficit	simple	good recovery	right occipital atrophy
17/46/M	right frontal 6 × 5 cm	headache, seizures	no neurological deficit, bil. papilloedema	simple	alive good; no deficit	normal
18/12/M	left temporoparietal 7 × 9 cm	headache, seizures, vomiting	right hemiparesis, bil. papilloedema	simple	alive good; no deficit	T <sub>2</sub> hyperintense area in the left temporopari- etal region

**Table 2**Presenting symptoms  
and signs.

Symptoms and signs	no. of pat.	%
Headache	15	83.3%
Vomiting	12	66.6%
Seizures	6	33.3%
Visual disturbances	5	27.7%
Papilloedema	14	77.7%
Hemiparesis	7	38.8%
Dysphasia	2	11.1%
Cranial nerve palsy	2	11.1%
Facial palsy	1	5.5%
Ataxia	2	11.1%

an inner density similar to CSF (fig. 1). MR demonstrated well-defined round (spherical) hypointense lesions on T<sub>1</sub>-weighted images (fig. 2A) and hyperintense lesions on T<sub>2</sub>-weighted images (fig. 2B). The walls of the cysts were hypointense on T<sub>1</sub>- and T<sub>2</sub>-weighted images (fig. 2B). No calcification, no surrounding oedema, and no contrast enhancement were seen on CT and MR in non-complicated cystic echinococcosis lesions (figs. 1 and 2).

One of these single lesions demonstrated multilocular cerebral hydatid cyst with extracalvarial extension in the left parietooccipital region. In this

**Figure 2**

A 12-year-old boy with headache, vomiting and seizures.

A Non contrast axial T<sub>1</sub>-weighted MR image of the brain shows well defined, spherical, hypointense cystic lesions.

B Axial T<sub>2</sub>-weighted MR image shows well defined homogeneously hyperintense cysts with hypointense rims (arrows).

C Coronal T<sub>2</sub>-weighted MR image reveals multiple hyperintense cysts.

D Post contrast coronal T<sub>1</sub>-weighted MR image of the same patient shows multiple hypointense cysts, no rim enhancement at all.

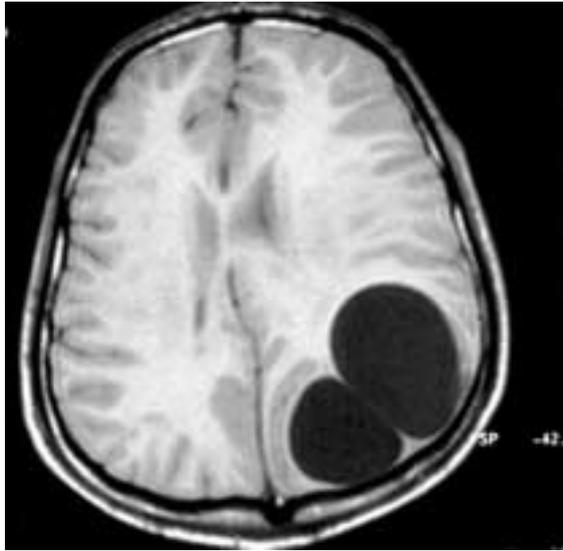


Fig. 2A



Fig. 2B

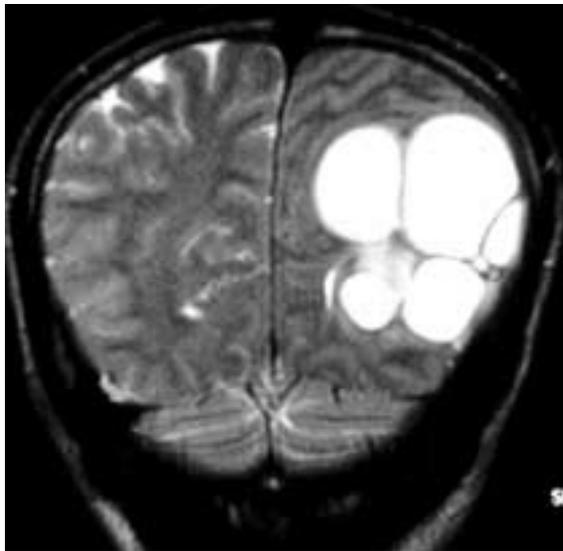


Fig. 2C

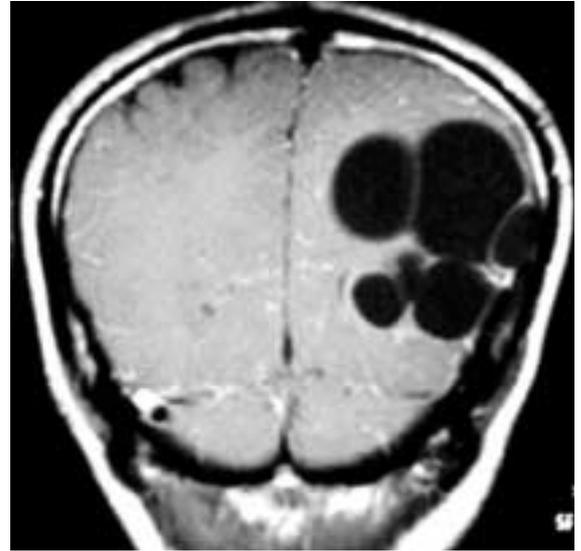


Fig. 2D

case CT examination revealed destruction and thinning in the adjacent left parietal bone [8]. Another patient with single lesion presented a well-rounded cystic lesion in the right occipital region. The patient had been treated 6 months previously for a cerebral infarct in the left parietooccipital region and a cystic lesion had not been present. Echocardiography identified a mass lesion on the left ventricular surface of the anterior mitral valve. Following cardiac surgery, the diagnosis was confirmed as cardiac hydatidosis. It is presumed that the cardiac echinococcosis caused the cerebral infarct as a result of thromboembolism and the parasitic embolism grew into the opposite hemisphere. The cyst was obviously growing at a very fast rate: the growth rate was 4.5 cm in 6 months [9].

Two of our single-lesion cystic echinococcosis cases demonstrated a surrounding hypodense areas of perifocal oedema and hyperdense complete or incomplete rim of contrast enhancements on CT scans (fig. 3A). With MR, lesions were hyperintense on T<sub>2</sub>-weighted images and hypointense on T<sub>1</sub>-weighted images (figs. 3B–D).

These lesions were labeled as complicated or infected cysts (cysts with superadded pyogenic infection). One of them presented an aggressive hydatid disease that was attributed to cyst rupture during surgery. The cyst had germinal membrane detachment that herniated through weakness in the pericyst and extended into the temporal fossa (fig. 3A–D).

We had two patients with multiple cystic echinococcosis. In one of them there were 25 cystic lesions in the left temporoparietooccipital region and was labeled as a gigantic intracranial hydatid cyst (95 × 90 × 75 mm). CT and MR scans showed a large mass of multiple cystic space-occupying lesions in the left temporoparietooccipital region, with midline shift to the right hemisphere. No peripheral oedema was noted around the cysts [10]. In the other patient with multiple cystic echinococcosis, there were 9 lesions in the left parietooccipital region.

The size of the cystic echinococcosis lesions ranged from 1 to 11 cm. Ten cystic echinococcosis cases were located in the left cerebral hemi-

**Figure 3**

A 23-year-old woman with headache, nausea, vomiting and seizures.

A Contrast-enhanced CT scan after direct rupture during surgery shows a hypodense cystic lesion with hyperdense rim enhancement and surrounding oedema with germinal membrane detachment (arrows) in the left parietal region. Note marked shift of midline structures to the right.

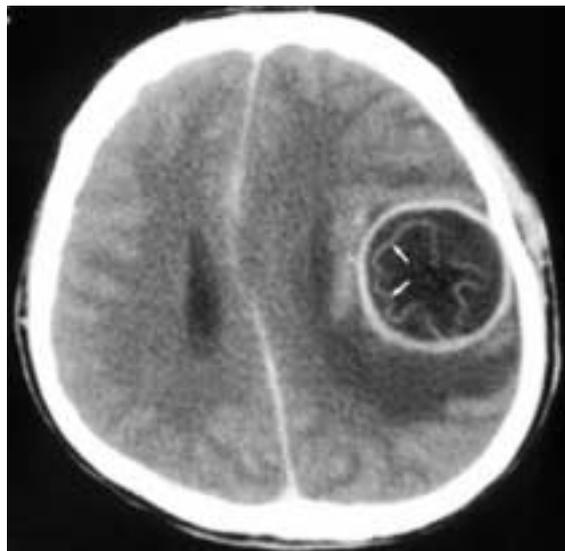


Fig. 3A

B Axial T<sub>2</sub>-weighted MR image shows hyperintense cysts and pericystic oedema. Detached germinal membrane is clearly seen (arrow).

C Axial T<sub>2</sub>-weighted MR image of the same patient shows a hyperintense cystic mass with irregular margin in the temporal fossa, which contains a detached membrane (arrow).



Fig. 3C

D Sagittal T<sub>1</sub>-weighted MR image of the same patient demonstrates a hypointense cystic mass herniating through a weakness in the pericyst (arrows) and extends into the temporal fossa.

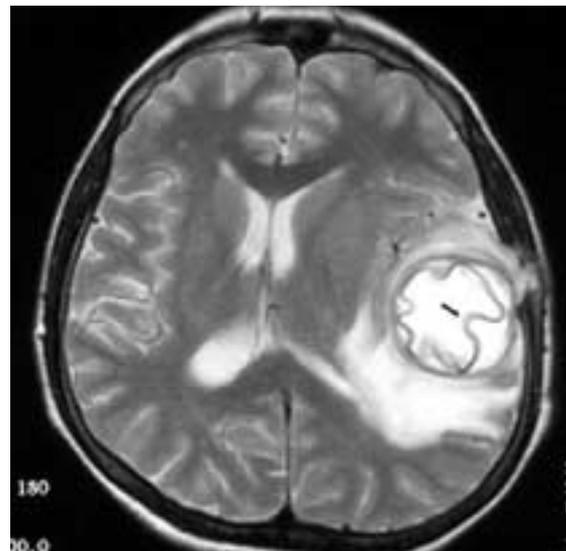


Fig. 3B



Fig. 3D

sphere, while 6 cystic echinococcosis cases were located in the right cerebral hemisphere. The parietal lobe was the commonest lobe affected. In cerebral alveolar echinococcosis case was located in the right parietooccipital region and was 11 × 8.5 cm in diameter.

In the cerebral alveolar echinococcosis case, CT showed a well-defined, multiseptated mass consisting of hyperdense solid, hypodense cystic components and hyperdense calcifications (fig. 4A). On MR of the same patient, both on T<sub>1</sub>- and

T<sub>2</sub>-weighted images, the mass showed a multicystic appearance with a central solid component and punctate calcifications with contrast enhancement of the wall, septatae and solid components (fig. 4B–D). The solid components were iso/hyperintense in both T<sub>1</sub>- and T<sub>2</sub>-weighted images. The cystic components were hypointense in T<sub>1</sub>-weighted images and hyperintense in T<sub>2</sub>-weighted images. On T<sub>2</sub>-weighted images, the solid portion of the lesion showed markedly small low signals corresponding to calcification (fig. 4B).

## Discussion

Hydatid disease, although rare, is usually seen in endemic areas of sheep-raising countries. Humans acquire the disease mostly during childhood. The liver (50–77%) and the lung (8.5–43%) are the organs most commonly involved. The remaining lesions may involve any organ in the body, including cerebral involvement in approximately 2% of

patients infected with the parasite [3]. They may reach a considerable size before the patient becomes symptomatic.

In cystic echinococcosis, the parasitic cyst consists of an inner germinal layer (endocyst) and an outer laminated layer (ectocyst). The host reacts to the cyst by forming a fibrous capsule (pericyst),

**Figure 4**

A 15-year-old boy with headache, nausea and vomiting.

A Axial unenhanced CT scan shows a cystic lesion with septated solid component and calcifications (black arrows). Septation is clearly seen (white arrows).

B Axial T<sub>2</sub>-weighted MR image of the same patient demonstrates a large hyperintense multiseptated cyst with solid portion and punctate calcifications (arrows).

C Axial T<sub>1</sub>-weighted MR image of the same patient shows a large multiseptated cystic mass with solid portion.

D Post contrast axial T<sub>1</sub>-weighted MR image shows the incomplete rim enhancement and fairly contrasted enhancement of the solid portion. Note shift of mid-line structures to the left



Fig. 4A



Fig. 4B

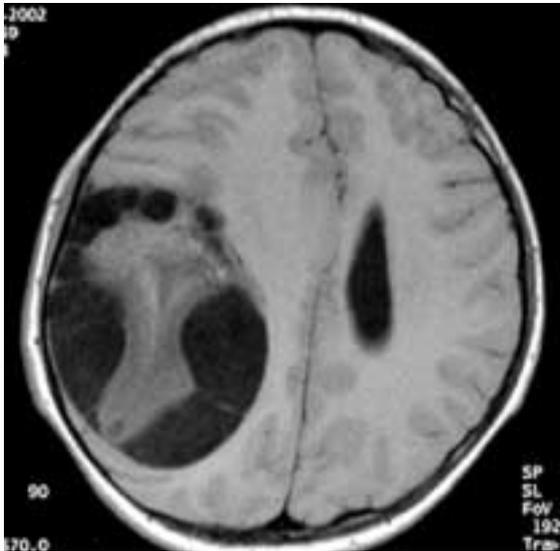


Fig. 4C



Fig. 4D

which contains blood vessels that provide nutrients for the parasite. From the germinal layer, scolices, brood capsules, and daughter cysts are formed by endoproliferation (internal budding) [11–13].

The cyst of alveolar echinococcosis differs from that of cystic echinococcosis in that it grows by external budding of the germinal membrane with progressive infiltration of the surrounding tissue [13].

Cerebral cystic echinococcosis is most commonly seen in children and young adults (approximately 50–70%) [14]. Lunardi et al. [15] and El-Shamam et al. [7], reported that cerebral hydatid cysts are commonly seen in children especially in males and young adults. We have similar results with 13 out of 18 patients (72.2%), and a male prevalence in the series (66.7%). Beskonaklı et al. [16], reported a reasonable explanation for this as young male children were more closely occupied with animals than girls or adults and were not as aware of the importance of hygienic principles.

The average age of patients with cerebral alveolar echinococcosis is significantly higher than in

cystic echinococcosis [11]. In our cases the average age of patients with cystic echinococcosis was 18.7 years.

Cerebral cystic echinococcosis lesions are usually single. Multiple cerebral cystic echinococcosis is very rare [17]. In our study there were 16 cases of single cystic echinococcosis and 2 cases of multiple cystic echinococcosis. Cerebral alveolar echinococcosis lesions may be single or multiple [18]. Our unique case of alveolar echinococcosis was single and multiseptated.

Cerebral hydatid cysts are benign and slowly growing cysts. The lesions may remain asymptomatic until they are quite large. Headache and vomiting were the most commonly reported symptoms in other series as well as in our patients (table 2). Other symptoms such as hemiparesis, seizures, visual field alteration and gait disorders, may vary with the location of the cyst [7]. Papilloedema is usually present in patients with intracranial hydatid cysts at the time of diagnosis [15]. In the series of Ciurea et al. [19], papilloedema was present in 24 out of 27 patients

(88.8%). We have similar results: bilateral papilloedema was seen in 14 of our patients (77.7%) (table 2).

Lesions of cerebral hydatid disease are usually distributed in the territory of the middle cerebral artery, especially in the parietal lobe. Most of the cysts are located in the supratentorial regions [14, 18, 20], and very rarely in the posterior cranial fossa, or ventricles [8]. In our series, 17 cysts were supratentorial and one cyst in the fourth ventricle. Distribution of the cysts in the cerebral hemispheres were 10 at left and 7 at right.

Multiple cerebral hydatid cysts are rather rare and result from spontaneous, traumatic or surgical rupture of a solitary primary cyst or as a consequence of a cyst rupture elsewhere and embolisation of hydatids to the brain [21, 22]. These secondary cysts which are infertile, do not have a thick capsule [21]. Al Zain et al. [23] reported 77 cases, containing his own 34, with multiple cerebral hydatidosis between 1940 to 2000.

Tüzün et al. [3] reported another 2 new cases of multiple cerebral hydatid cysts in an 11-patient series, and we reported 2 new cases with multiple cerebral hydatid cysts.

Recurrent or secondary cysts due to the rupture of the primary cyst during surgery were seen in 5 (27.7%) patients, three of them with recurrent solitary cysts, while two patients had multiple cysts. Lunardi et al. [15] reported 2 recurrent cases (10%) in their 12-children series. The series of Ersahin et al. [24] included 19 children who underwent surgery for intracranial hydatid cysts, recurrence was seen only in 2 patients due to cyst rupture during surgery. Ciurea et al. [19], in a series of 27 children with cerebral hydatid cysts had 11 cases of recurrence (40.7%), due to cyst rupture during surgery.

Patients with cerebral hydatid cysts may have hydatid cysts in other organs. In recent studies, hepatic, pulmonary and other locations were found in 10% of the cases [25]. In the series of Ciurea et al. [19], 8 patients, presented hydatid infestation in multiple organs. Tüzün et al. [3] reported 7 patients with extracerebral organ involvement out of 11 patients. El-Shamam et al. [7] reported only one patient with involvement of other organs out of 16 patients. In our series, 6 patients with cerebral hydatid cysts had cysts at different locations, four in the liver, one in both liver and lung and one in the heart.

Both CT and MRI demonstrate a spherical and well-defined, smooth, thin walled, homogeneous cystic lesion with fluid density similar to the cerebrospinal fluid, with or without septations or calcification. On unenhanced CT, the cyst wall was isodense or hyperdense to brain tissue. The cyst wall usually showed a rim of low signal intensity on both T<sub>1</sub>- and T<sub>2</sub>-weighted images. Calcification of the wall was rare, being less than 1%. The presence of daughter cysts is considered pathognomonic but has been rarely reported [14, 17]. Compression of the midline structures and

ventricles are seen in most of the cases, however surrounding oedema and rim enhancement are usually absent in untreated or uncomplicated cases [17, 26]. Obvious mass effect was demonstrated in all cases we examined. Incomplete rim enhancement and variable amounts of pericystic oedema were observed in one of our cases. Obvious rim enhancement and surrounding oedema was observed in one case with ruptured and infected primary cyst. On the other hand, the detachment of the germinal membrane following direct rupture during surgery was also present in this case. Lewal and McCorkell [27] classified rupture of echinococcal cysts into three types in thoracoabdominal hydatid disease: contained, communicating, and direct. Contained rupture occurs when only the endocyst ruptures and the cyst contents are confined within the host-derived pericyst. When cyst contents escape as a biliary or bronchial structure, the rupture is communicating. Direct rupture occurs when both the endocyst and the pericyst tear and cyst contents spill directly into other structures. Our case had a direct rupture of the cyst and the cyst extended into the temporal fossa (fig. 3). We have not found any reported case of cerebral hydatid cyst presenting a detachment of the germinative membrane following operative trauma in the English literature.

The CT and MR appearances of cerebral alveolar echinococcosis have rarely been reported. On CT and MR, cerebral alveolar echinococcosis lesion appears as a solid, semisolid, or multilocular cystic mass with definite margins. Calcification and surrounding oedema are common. Contrast enhancement occurs within the region of inflammatory reaction around the cysts [18, 28, 29]. In our unique case of cerebral alveolar echinococcosis; CT and MR showed a well-defined, multicystic appearance with a central solid component, and calcifications. Contrast enhancement of the wall, septates and solid components were seen.

The differential diagnosis of intracerebral hydatid cysts includes cystic lesions such as porencephalic cyst, arachnoid cyst, cystic tumor of the brain and pyogenic abscess. In contrast to hydatid cysts, porencephalic cyst and arachnoid cysts are not spherical in shape and not surrounded entirely by brain substance. Arachnoid cysts are extra-axial masses that may deform adjacent brain. Porencephalic cysts result from insults to normal brain tissue and are lined by gliotic white matter that could easily be demonstrated with MR imaging. Cystic tumours of the brain could be differentiated by the enhancement of the mural nodule, if any, and periphery of the tumour. When a pyogenic abscess shows a cyst-like central necrotic area; peripheral oedema is almost always present, the rim enhances intensely following contrast administration and satellite lesions are commonly present, all of which could be demonstrated on both CT and MR images [30-32]. Clinical and laboratory findings could also aid in the differential diagnosis of the patients with cerebral abscess formation.

The differential diagnosis of cerebral alveolar echinococcosis includes gliomas, metastases, tuberculomas, and fungal infections. The diagnosis should be suggested by evidence of a primary hepatic focus, appropriate clinical history, high prevalence of the infection in the host's geographic location, and laboratory findings [3, 18, 28, 33]. Cerebral cysticercosis should also be kept in mind in the differential diagnosis of cerebral echinococcosis. According to the literature, in a small number of cases diagnosis was established preoperatively on CT and MRI [34]. Serologic analysis is in most cases negative; pathohistologic analysis is the most reliable method of diagnosis. Therefore, cerebral cysticercosis or echinococcosis should be suspected especially when cystic lesions appear in the central nervous system [34].

Proton MR spectroscopy (PMRS) and diffusion-weighted (DWI) MR imaging have recently been used to distinguish between cerebral abscess and cystic or necrotic brain tumor. The differentiation between cystic or necrotic brain tumor and cerebral abscess has been well documented [35]. However, there are a few reports of its use in differentiation in other types of cystic lesions. Shukla-Dave et al. [36] reported three cases of hydatid cysts demonstrated by resonances from lactate, acetate and succinate, and in one patient with perifocal oedema choline resonance was reported. In three cases of arachnoid cysts only small lactate resonance was observed. Neoplasm of the brain characteristically demonstrates an increase in choline compounds and lactate with a decrease in NAA. On the other hand, DWI is widely used in neuroimaging, with the greatest impact on the detection of acute cerebral stroke [37]. DWI is considered useful in differentiating cystic gliomas from cerebral abscesses [38], however, its use is still required in the differentiation of other types of cystic lesions such as hydatid cysts.

In conclusion, CT and MR imaging, alone or in combination, are helpful in the diagnosis of cerebral hydatid disease. Although CT is superior in detecting calcification of the cyst wall or septa, when present, MR is better in detecting multiplicity and defining the anatomic relationship of the lesion with the adjacent structures and helps in surgical planing. MR provides additional information and details that can not be seen by CT. It provides additional information in the exact localisation of the cyst. When present, in complicated or recurrent disease, surrounding oedema can better be demonstrated with MR imaging owing to the inherent capability of the imaging modality in revealing subtle differences in the tissue content. When a well-defined spherical cystic intracerebral lesion with obvious mass effect, but no surrounding oedema and no contrast enhancement following contrast administration is detected on CT and MRI, hydatid disease should be taken into consideration in countries where the disease is endemic. If perilesional oedema and thin rim enhancement are observed either complicated hydatid cyst or other cystic lesions of the brain should be considered in the differential diagnosis. CT and MR findings in cerebral echinococcosis are more characteristic than in cerebral alveolar echinococcosis, but characteristic CT and MR findings of multiloculated cystic alveolar echinococcosis should also be remembered for the differential diagnosis.

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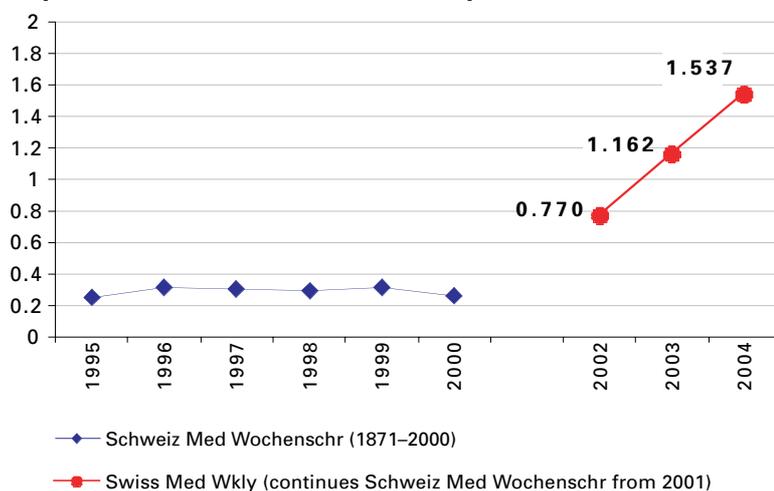
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