Calcified Intracranial Hydatid Cyst: Case Report
Kalsifiye İntrakraniyal Kist Hidatik: Olgu Sunumu

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ABSTRACT
We present a 26-year-old patient who did farming related work had an asymptomatic intracranial hydatid cyst. A calcified intracranial cystic mass was found on radiological investigations and the patient underwent surgery. The mass was totally excised. The histopathology result reported a hydatid cyst. There were no postoperative complications. Calcification is quite rare in cerebral hydatid cyst. The computed tomography and magnetic resonance imaging provide information that is especially useful for preoperative diagnosis. Observing membrane detachment and daughter cysts during these investigations is pathognomonic. The presence of calcification in the cyst wall and intracystic membranes indicated hydatid cyst
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Key Words: Calcification, Hydatid cyst, Intracranial cyst

INTRODUCTION
Hydatid disease is caused by the larval stage of the Echinococcus granulosus and is endemic in various regions of the world. The prevalence is higher in North Africa and Mediterranean countries such as Turkey, Greece and some regions of Spain and Italy and it ranges between 1-150/100.000 (1, 2). The most common localization of the disease is the liver and lung. The intracranial involvement incidence of the cyst if 1-4%, while 1-5% of these cases show calcification (2-5). When intracranial involvement occurs, it is most commonly seen in the parietal and frontal areas, respectively. The diagnosis is usually made with clinical findings, imaging techniques and serologic tests (2, 6). We present a case of calcified hydatid cyst which was discovered incidentally.
CASE REPORT

A 26-year-old male patient who did farming-related work presented at our clinic with headache following a minor head trauma. The physical and neurological examinations were normal. The x-ray showed a left frontal calcified lesion (Figure 1). There was a 6x5 cm mass with homogeneous peripheral calcification in the left frontal region on cranial computed tomography (CT) (Figure 2). Magnetic resonance imaging (MRI) showed the mass to contain membranous structures and to be hyperintense. There was no perilesional edema area and no contrast enhancement (Figure 3a, b). The patient was operated on via a left frontotemporal craniotomy. The mass was strongly adherent to the dura and it was therefore opened circularly, leaving this part on the mass. The mass ruptured while being dissected from the surrounding cerebral tissue and a large amount of yellow-brown material that was viscous in character drained out. The ventricle was opened while the posterior section was being dissected and cerebrospinal fluid (CSF) was seen to come out. The mass was totally excised. There were no postoperative complications. The pathology result was hydatid cyst (Figure 4). Serological results including enzyme-linked immunoabsorbant assay (ELISA) test and indirect hemagglutination (IHA) test were negative and no other lesion was found on abdominal and thorax investigations. Culture of the cyst content sent during surgery did not grow anything. The patient was discharged on the 14th postoperative day.

DISCUSSION

Hydatid cyst involves the liver in 75% of the patients, the lung in 15% and other organs in 10% (6, 7). Central nervous system involvement is seen in 1% of the cases and is usually diagnosed in childhood. It can be located in any area of the brain but is most commonly seen at the area supplied by the middle cerebral artery in both hemispheres (most commonly in the parietal region, followed by the frontal region) (7-9). They are not symptomatic until they reach a certain size. There is usually no other focus, as in our case. Hepatic or pulmonary localization has been shown in only 10-20 % of cerebral hydatid cysts (9, 10). The epidemiologic and clinical findings, imaging techniques and serologic tests are used to make a diagnosis (9, 11). The serologic diagnosis of hydatid cyst usually is more of a challenge in the brain than in the other organs because the antibody response is usually absent or very low in the case of intracranial hydatid cyst, especially if calcified (12). In the presented case, the serologic tests including ELISA and IHA were negative but the imaging tests were specific for an intracranial calcified hydatid cyst.

CT shows cranial hydatid cysts as intraparenchymal, homogenous cystic lesions with distinct borders. The cyst fluid is isodense with the CSF and the cyst itself is usually circular. Noncontrast CT shows an isodense or hyperdense cyst wall compared to the brain tissue. The perilesional edema and contrast enhancement seen with abscesses or cystic tumors are unusual for hydatid cysts (7). MRI produces a similar radiological appearance. T1- and T2-weighted images show a low signal intensity rim of the cyst wall, while the cyst content is isointense with the CSF. There is mild or moderate perilesional edema in infected hydatid cysts and there may be limited contrast enhancement in the capsule region (13, 14). Membrane detachment and daughter cyst are pathognomonic findings on CT and MRI. The presence of calcification in the cyst wall and intracystic membranes indicate a hydatid cyst (13, 14). We found calcification both on
direct cranial X-ray and CT in our case. MRI showed membrane detachment and intracystic membranes.

The incidence of calcification in hydatid cyst is 20-30% for liver localization and 1-5% for lung or liver localization (6, 7). The calcification is usually ring-like with a curvilinear pattern and develops in the pericyst consisting of modified host cells with a fibrous protective zone several mm thick. The hydatid cyst capsule consists of 3 layers with the pericysts outside, laminated membrane in the middle and germinal layer inside (15). The laminated membrane is a structure that lets nutrients pass through but stops bacteria. Damage to this membrane predisposes to infection. Calcification develops in all components of the cyst during the natural healing stage once the pericyst is calcified. The clear fluid that fills the cyst is replaced by a thick and viscous substance rich in cholesterol as in our case. Finding calcification in the pericysts does not indicate death of the parasite. Although an association has been found between pericyst calcification and cyst inactivation for hydatid cysts localized in the liver, such a classification is not available for cerebral cysts due to the low number of cases (6, 16). Complete calcification indicates death of the parasite (15).

The primarily treatment of intracranial hydatid cysts is surgical. One of the aims of treatment for hydatid cysts located in the liver is calcification of the cyst. Surgical or medical treatment is therefore accepted to be contraindicated in the case of partial or total calcification in the cyst (6). A ‘wait and see’ approach is recommended before any surgical intervention for hydatid cysts with homogeneous calcification in the walls and those localized in the liver (6). There is no data on whether the same approach can be used for intracranial cysts. Cases of calcified hydatid cyst in the literature have almost all presented with epileptic seizures and this approach may therefore not be appropriate for intracranial hydatid cysts (4, 5, 17-20).

In conclusion, a hydatid cyst should be considered in patients living in or coming from areas with endemic hydatid cyst disease, especially when calcification is found during radiological investigations and a membrane or detached membrane is seen inside the cyst.

**Conflict of Interest**
No conflict of interest was declared by the authors.

**REFERENCES**
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