



Disseminated Alveolar Echinococcosis Presenting with Cerebral Involvement

Serebral Tutulumla Seyreden Dissemine Alveolar Ekinokokkoz

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ABSTRACT

Alveolar echinococcosis (AE) is a rare but potentially fatal parasitic disease caused by *Echinococcus multilocularis* and is characterized by infiltrative growth and malignant-like behavior. Although the liver is the primary site of involvement, extrahepatic dissemination, particularly to the central nervous system, is uncommon and presents diagnostic challenges. We present a 35-year-old man admitted with progressive headache, gait disturbance, and seizures. Brain magnetic resonance imaging revealed multiple intracranial lesions with peripheral contrast enhancement. Because of progressive neurological deterioration and mass effect, urgent surgical excision was performed. Laboratory findings showed markedly elevated inflammatory and cholestatic markers without eosinophilia. Thoracoabdominal computed tomography demonstrated an infiltrative, calcified hepatic mass and a pulmonary lesion, whereas spinal magnetic resonance imaging revealed involvement of the conus medullaris and dura mater, indicating central nervous system dissemination. Serology for hydatid disease was strongly positive, and the diagnosis of AE was confirmed by histopathological examination. Because of extensive multiorgan involvement, including the liver, lung, brain, and spinal cord, the disease was considered inoperable, and albendazole therapy (15 mg/kg/day, continuous regimen) was initiated. The patient showed clinical improvement and was discharged with a recommendation for outpatient follow-up. This case underscores the aggressive nature of AE and the importance of considering this disease in the differential diagnosis of cystic or tumor-like central nervous system lesions in endemic regions.

Key Words: Alveolar echinococcosis; Cerebral involvement; Multiorgan dissemination; *Echinococcus multilocularis*

ÖZ

Alveolar ekinokokoz (AE), *Echinococcus multilocularis* tarafından oluşturulan, infiltratif büyüme paterni ve maligniteyi taklit eden davranışı ile seyreden nadir ancak potansiyel olarak ölümcül bir paraziter hastalıktır. Hastalık öncelikle karaciğeri tutmakla birlikte, santral sinir sistemi gibi ekstrahepatik organlara yayılım da görülebilmektedir; ancak bu durum oldukça nadirdir ve tanılabilir güçlüklerle karşılaşmaktadır. Bu olgu sunumunda, ilerleyici baş ağrısı, yürüme bozukluğu ve nöbet şikayetleri ile başvuran 35 yaşındaki erkek hasta sunulmaktadır. Beyin manyetik rezonans görüntülemesinde periferik kontrast tutulumu gösteren çoklu intrakraniyal lezyonlar saptanmış,

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klirik k t le  me ve kitle etkisi nedeniyle acil cerrahi e ksiyon uygulanmı tır. Laboratuvar incelemelerinde eozinofil olmaksızın inflamatuar ve kolestatik belirte lerde belirgin y kseklik izlenmi tir. Torakoabdominal bilgisayarlı tomografide kalsifikasyon i eren infiltratif hepatik kitle ve pulmoner lezyon saptanmı ; spinal manyetik rezonans g r nt lemede konus medullaris ve dura mater tutulumu g sterilerek santral sinir sistemi yayılımı ortaya konmu tur. Serolojik testlerin pozitifli i ve histopatolojik inceleme ile alveolar ekinokokoz tanısı do rulanmı tır. Karaci er, akci er, beyin ve spinal kord tutulumu ile seyreden yaygın hastalık nedeniyle olgu inoperabl kabul edilmi  ve albendazol tedavisi (15 mg/kg/g n, s rekli rejim) ba lanmı tır. Hasta klinik olarak d zelme g stermi  ve ayaktan takip  nerilerek taburcu edilmi tir. Bu olgu, alveolar ekinokokkozun agresif seyrini vurgulamakta ve endemik b lgelerde santral sinir sistemi yerle imli kistik veya t m r benzeri lezyonların ayırıcı tanısında mutlaka aklıda tutulması gerekti ini g stermektedir.

Anahtar Kelimeler: Alveolar echinococcosis; Cerebral involvement; Multiorgan dissemination; Echinococcus multilocularis

INTRODUCTION

Alveolar echinococcosis (AE) is a chronic, tumor-like parasitic infection caused by *Echinococcus multilocularis*^[1]. AE is primarily associated with environmental and occupational exposure in endemic regions. Major risk factors include rural residence, close contact with definitive hosts such as foxes and dogs, consumption of contaminated food or water, and agricultural activities. Although the liver is the primary site of infection, hematogenous spread may result in lung, bone, and brain involvement^[2]. Cerebral AE is rare and often mimics malignancy radiologically^[3]. Radiologic imaging, laboratory analyses, and histopathologic confirmation all contribute to the diagnosis of AE^[4]. Long-term albendazole therapy is crucial for disease control when radical resection is not feasible^[1]. This case illustrates the malignant-like, infiltrative nature of AE and the difficulty of achieving a complete cure in advanced, disseminated disease. Early detection and preventive measures remain essential to reduce the disease burden in endemic regions.

Case

A 35-year-old man presented with progressively worsening headache, gait disturbance, and seizures. Neurological examination revealed gait instability without clear evidence of paraparesis, a sensory level, or sphincter dysfunction. No specific exposure history could be identified; however, the patient resided in an endemic region, which represented a significant epidemiological risk factor. Brain magnetic resonance imaging (MRI) demonstrated multiple space-occupying lesions that were hypointense on T2-weighted sequences and showed marked peripheral contrast enhancement (Figure 1). Based on these imaging findings, cystic lesions were considered in the

preliminary diagnosis, and the intracranial lesions were surgically excised.

Initial laboratory evaluation at admission revealed elevated alkaline phosphatase (ALP), gamma-glutamyl transferase (GGT), and C-reactive protein (CRP) levels of 399 U/L, 1.352 U/L, and 418 mg/L, respectively, whereas all other biochemical and hematological parameters were within normal limits. Given the initial suspicion of an infectious intracranial process, empirical intravenous ceftriaxone therapy was initiated at a dose of 4 g/day (2 x 2 g) to cover potential bacterial central nervous system infections.

Concurrent thoracoabdominal computed tomography revealed an infiltrative liver mass characterized by irregular margins, a lack of contrast enhancement, and coarse calcifications. In addition, a microlobulated lesion with irregular borders was identified in the right lung (Figure 1). Contrast-enhanced thoracolumbar spine MRI demonstrated enhancement involving the distal spinal cord and dura mater at the level of the conus medullaris, findings suggestive of central nervous system dissemination (Figure 2).

Serological testing showed a positive indirect hemagglutination assay for hydatid disease, with a titer of 1:1,280. Histopathological examination of the brain lesion confirmed the diagnosis of alveolar echinococcosis (Figure 3). Given the presence of widespread disease involving the liver, lung, brain, and spinal cord, the condition was deemed inoperable, and albendazole therapy was initiated at a dose of 15 mg/kg/day in a continuous regimen.

On the ninth day of hospitalization, the patient's Glasgow Coma Scale (GCS) score improved to 15, accompanied by marked clinical recovery

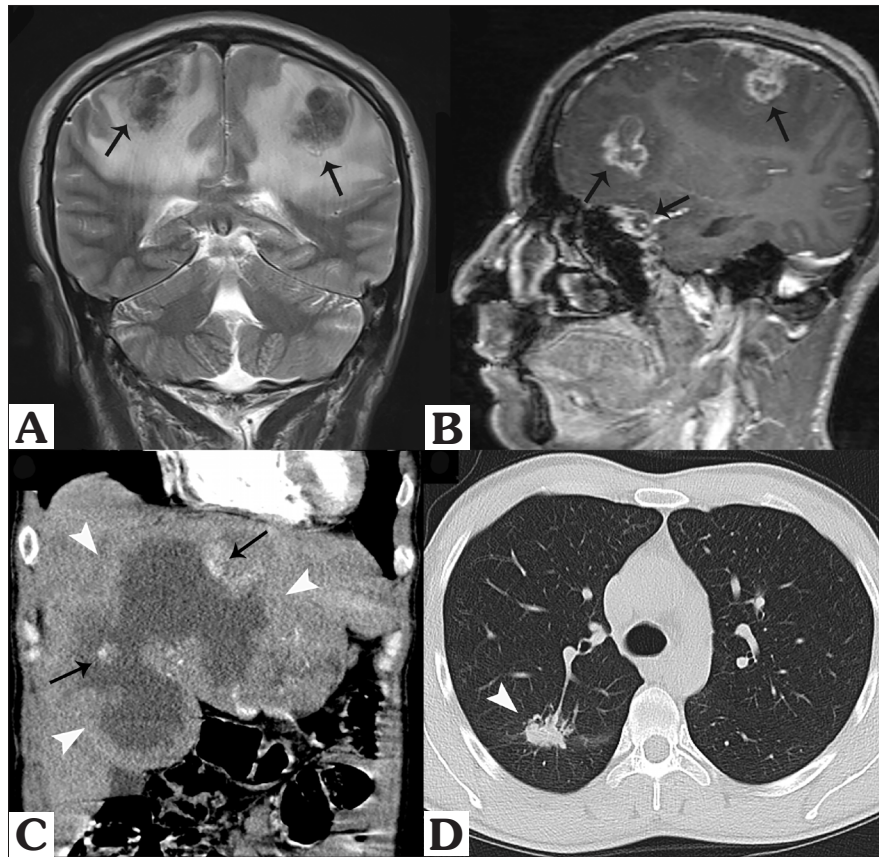


Figure 1. Brain magnetic resonance imaging revealed T2-hypointense space-occupying lesions (black arrows in Figure 1A) with marked peripheral contrast enhancement (black arrows in Figure 1B). The lesions were surgically removed because cystic lesions were suspected. Concurrent thoracoabdominal computed tomography demonstrated an ill-defined mass in the liver (arrowheads in Figure 1C) without contrast enhancement and with coarse calcifications (black arrows in Figure 1C). A microlobulated mass with irregular margins was also detected in the right lung (arrowhead in Figure 1D).



Figure 2. Contrast-enhanced thoracolumbar spine MRI showed dural enhancement at the level of the distal spinal cord and conus medullaris (arrows).

and improvement in the presenting neurological symptoms. Laboratory parameters that were elevated at admission improved significantly before discharge. The patient was discharged while receiving albendazole therapy and was advised to undergo close outpatient follow-up. Follow-up imaging was planned to evaluate treatment response and disease progression.

DISCUSSION

AE is a progressive parasitic disease caused by *E. multilocularis* that primarily affects the liver and exhibits an infiltrative growth pattern with metastatic potential, thereby mimicking malignant tumors^[5]. Although cystic echinococcosis is widespread throughout Türkiye, the alveolar form is rare and predominantly endemic in the Eastern

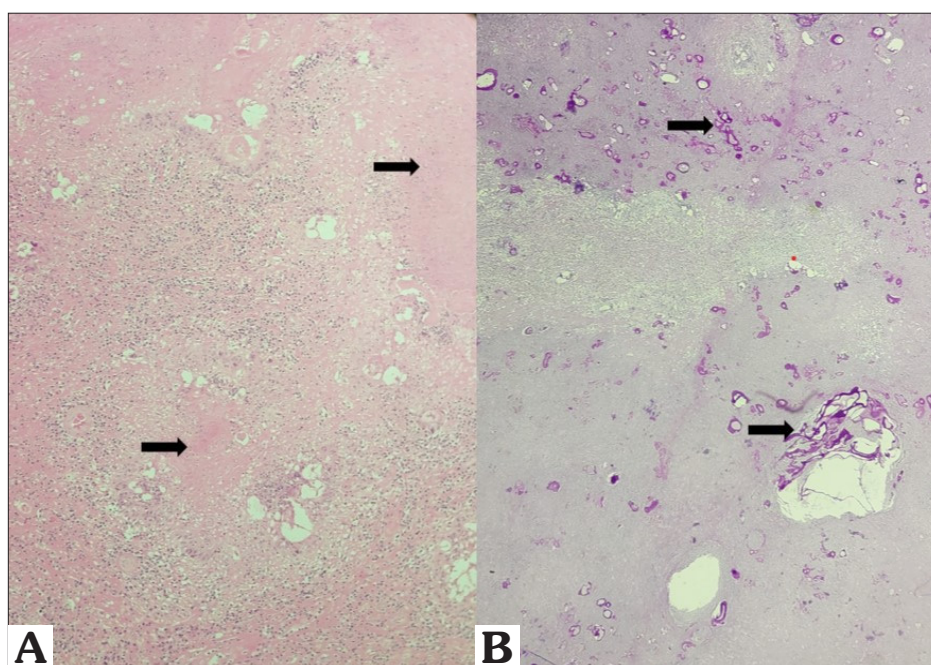


Figure 3. An area of necrosis surrounded by an acellular eosinophilic structure and palisading mononuclear inflammation, compatible with parasitic infestation (blue arrow in Figure 3A; hematoxylin and eosin stain, $\times 400$). PAS-positive acellular membrane structures are shown in Figure 3B (blue arrow).

and Southeastern Anatolia regions^[6]. Hepatic AE lesions may present as cystic, solid, or semisolid masses and frequently resemble malignant hepatic neoplasms. Histopathologically, necrosis and granulomatous inflammatory reactions are observed, whereas invasion of the bile ducts and vascular structures may lead to severe clinical complications^[7]. If left untreated, AE behaves aggressively, spreading to adjacent tissues and distant organs, with reported mortality rates of up to 90% within 10–15 years after diagnosis^[8,9]. Although hepatic involvement is present in most cases, multiorgan dissemination occurs in approximately 13%, most commonly involving the lungs, spleen, and brain^[10]. In the present case, simultaneous involvement of the liver, lung, brain, and spinal cord was identified, clearly illustrating the aggressive nature and capacity for dissemination of AE.

AE typically follows a long asymptomatic course and is often diagnosed at an advanced stage. Laboratory findings are nonspecific, and eosinophilia is detected in fewer than 25% of patients. Consistent with the literature, eosinophilia was not observed in our patient, emphasizing

that normal eosinophil levels do not exclude a diagnosis of AE^[11]. Notably, the markedly elevated CRP level observed in this case (418 mg/L) is uncommon in uncomplicated AE and may reflect an extensive inflammatory response associated with a high parasitic burden, significant hepatic tissue involvement or necrosis, or a concomitant secondary bacterial infection, although the latter could not be definitively demonstrated.

Radiologically, computed tomography is particularly valuable for demonstrating infiltrative features and characteristic calcification patterns, whereas MRI is superior for assessing central nervous system involvement^[11,12].

Early diagnosis is crucial to enable radical surgical resection in AE. In addition to surgery, long-term adjuvant therapy with albendazole (15 mg/kg/day) or mebendazole (50 mg/kg/day) has been shown to significantly improve survival^[13]. However, in cases with extensive multiorgan involvement, surgery is often not feasible, and prolonged or lifelong benzimidazole therapy constitutes the mainstay of treatment^[14]. In the present case, surgical intervention was deemed

inappropriate because of extensive multiorgan involvement; therefore, albendazole therapy was initiated in accordance with the recommendations of the WHO Informal Working Group on Echinococcosis (WHO-IWGE).

Central nervous system involvement, particularly in the form of multiple or intraventricular hydatid lesions, is rare but is associated with substantial morbidity. On MRI, thin-walled cystic lesions with cerebrospinal fluid-like signal intensity and perifocal edema are highly suggestive of the diagnosis^[12,15]. In endemic regions, alveolar echinococcosis should always be considered in the differential diagnosis of cystic brain lesions. In addition to metastatic tumors and brain abscesses, neurocysticercosis should be considered in the differential diagnosis of cystic intracranial lesions, particularly in endemic regions. Neurocysticercosis can be distinguished from alveolar echinococcosis by the presence of a scolex within the cyst, characteristic stage-dependent imaging features, and distinct epidemiological patterns.

This case contributes to the literature by demonstrating a rare and highly aggressive presentation of alveolar echinococcosis with extensive multiorgan dissemination, including concurrent brain and spinal cord involvement. The patient's initial presentation with neurological symptoms and the diagnosis established through combined surgical, radiological, and serological evaluation highlight the importance of considering AE in the differential diagnosis in endemic areas. Similar atypical presentations involving extrahepatic dissemination have also been reported in the literature^[16]. A case published in this journal describing primary muscular involvement further emphasizes the unusual sites of involvement and the broad clinical spectrum of echinococcosis^[16]. Overall, this case underscores the need for increased awareness of the diverse and severe clinical manifestations of alveolar echinococcosis to facilitate timely diagnosis and appropriate management.

INFORMED CONSENT

Informed consent was obtained from the patient for this study.

CONFLICT of INTEREST

The authors declare that they have no conflicts of interest.

AUTHORSHIP CONTRIBUTIONS

Concept and Design: All authors

Analysis/Interpretation: All authors

Data Collection or Processing: All authors

Writing: All authors

Review and Correction: All authors

Final Approval: All authors

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