Case Report

Neurocysticercosis as a Single Lesion Mimicking Glial Tumor

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Abstract

Neurocysticercosis is a well known central nervous system parasitosis around the world. The tapeworm Taenia solium is responsible for this infection. However, a single brain lesion is rarely reported. The differential diagnosis of neurocysticercosis is very important because of its misdiagnosis with many different brain lesions. We have reported a patient whose neurocysticercosis is appeared as a single lesion mimicking glial tumor. A 44-year-old female patient admitted to our hospital with seizure and headache. A 7-8 mm single cystic lesion which has an enhanced periferic gadolinium and surrounded by perifocal edema in the right parietal lobe was determined at magnetic resonance imaging. The lesion resection was done through a right parietal craniotomy. The diagnosis of cysticercosis was confirmed by histological examinations. Neurocysticercosis should be considered in the differential diagnosis of the solitary brain lesions.

Keywords: Neurocysticercosis, glial tumor, single lesion, taenia solium

Glial Tümörü Taklit Eden İzole Soliter Nörosistiserkoz

Özet


Anahtar Kelimeler: Nörosistiserkoz, glial tümör, tek lezyon, taenia solium

INTRODUCTION

Neurocysticercosis (NCC) is the most common parasitic disease of the CNS around the world (1). Disease is caused by the larval stage of Taenia solium (2). It usually presents with seizures, headaches, and hydrocephalus. This varies by the stage of the parasite and the host’s immune response. Other signs and symptoms are syncope, dementia, vision changes, focal neurologic deficits,
meningitis, and stroke. Neurocysticercosis is endemic in Eastern Europe, Africa, Asia, and Central and South America (3). Seizures and hydrocephalus are the most frequent symptoms in adults living in these regions. According to the World Health Organization (WHO), T. solium infection is considered to be the most common preventable cause of epilepsy in the developing countries (4). Hereby, we aimed to report a case with neurocysticercosis mimicking glioma.

CASE PRESENTATION

A 44-year-old female was complaining of headache and seizure. The patient has suffered from the headache for 6 months worsening in the last 2 weeks and had an epileptic seizure 2 weeks ago. She has not travelled to any foreign countries. There was no evidence of any neurological examination. MRI disclosed an approximately 7-8 mm solitary cystic lesion with enhanced gadolinium in the peripheral and surrounded by a perifocal edema in the right parietal lobe (Figure 1). Hematologic and biochemical tests were normal. Although intense microbiological examinations were conducted, no infectious agents could be detected. Both thorax and abdominal computed tomography (CT) scans were normal. A massive, circular shaped lesion with a light-yellow-colored, bright surface was observed during surgery operation. The patient has showed an uncomplicated post-operative recovery. During histological examination, pathologist diagnosed a scolex of a Taenia solium surrounded by inflammatory chronic granulomatous infiltrates (Figure 2-3). NCC was diagnosed with no extraneural involvement. The patient received an antiparasitic treatment with albendazole 800 mg/day in combination with dexamethasone 4 mg, 4 times a day for 2 weeks, and she had seizure-free under anticonvulsant therapy with levetiracetam 1000 mg/day.

Fig 1: A: Pre-op CT showed a 7-8mm lesion with cyst in the central B: T2W MRI showed cystic lesion with perifocal edema C: T1 contrast MRI showed solitary cystic lesion with gadolinium enhancement in the peripheral and in the right parietal lobe surrounded by a perifocal edema D: Post op CT showed the total excision of the lesion.
DISCUSSION

Neurocysticercosis is a parasitic infection which is caused by the larval stage of Taenia solium (5). Humans become infected with the metacestode stage (cysticercus) of T. solium after eating uncooked pig meat. Humans are not only definitive but also intermediate hosts for this parasite. During the intermediate stage in humans, the parasite is not infective and not transmitted. But it can be said that T. solium is transmitted from pig to human, human to pig, pig to pig, and human to human. The larval stage of the parasite can infect brain tissue and stimulate inflammation. The initial clinical presentation can include seizures, ischemic
stroke, intracranial hypertension, and motor and sensorial deficits (2). It is most often associated with seizures and then headache. These symptoms are the result of the perilesional edema and the mass effect of the lesion. Generally, the parasite is eliminated by the patient’s immune system by provoking a granulomatous reaction with calcification at the posterior and then resolution is completed in 3-24 months (6). Glioblastoma multiforme (GBM), which is a malignant glioma frequently seen in young adults, might have similar symptoms and radiological images as NCC. Both diseases could be presented with parenchymal cysts.

NCC could be seen with cerebral lesions such as ischemic stroke, glioma, tuberculoma, and Japanese encephalitis. Calcifications are the most common imaging finding in NCC, accounting for > 30% of symptomatic cases and ≥ 95% of the general population screened by CT in endemic regions (7). "Cyst with dot inside" is the best diagnostic clue for diagnosis on CT scans (8). Magnetic resonance imaging plays an important role in the diagnosis of NCC (5,9). According to MRI findings, the larval phase, the early degenerative phase, the late degenerative phase, and the calcified phase are the four phases of NCC (5).

The larval phase, known as the vesicular stage, identified on the MRI T1 signals as cystic lesions isointense to CSF (Cerebrospinal Fluid). The early degenerative phase, known as the colloidal vesicular stage, shows on the T1 signal as a cyst that is mildly hyperintense to CSF. The late degenerative phase, known as granular nodular stage, on the T1 signal has a thickened retracted cyst wall and a decrease in edema. The calcified phase, known as the nodular calcified stage, has a shrunken Ca++ lesion (8). Our patient was in the colloidal vesicular stage according to the classification. Bouillot et al. reported 2 cases of parenchymal NCC, which could be misdiagnosed as glioma (10).

Moreover, Zammarchi L et al. noticed that one-third of neurosurgical operations reported in the literature were avoidable surgery because the indications for surgery among those were not commonly accepted by treatment guidelines (11). In domestic and imported cases of cysticercosis, 41.4% and 32.6% of operations, respectively, were performed for suspected CNS neoplasm (11). Inadequate preoperative diagnostic tools and low experience and awareness cause the increase of this high rate of neurosurgical operations (12). Uncontrolled epilepsy, a single lesion, lesions that compress important functional areas, and multiple lesions with diameter greater than 3 cm are the indications for surgery in patient with NCC.

Enzyme-linked immunosorbent assay (ELISA) and enzyme electroimmune transfer blot (EITB) are the most useful techniques for the diagnosis of this infection (7,13). Once the diagnosis is confirmed as neurocysticercosis, drugs like albendazole, praziquantel, and topiramate-silicone can be used for treatment. Nash et al. suggested that corticosteroids could reduce the rate of epileptic seizure and accelerate resolution of this lesion (6). We have used steroids for the treatment of our patient for two weeks. and seizure-free condition was remained with anticonvulsant drugs. In conclusion, neurocysticercosis should be considered in the differential diagnosis of the single brain lesion.

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