Epileptic seizure as the first manifestation of tuberculosis in a young male

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Abstract:

Introduction: Central nervous system tuberculosis is a life-threatening condition that usually presents with seizures. It is most frequently seen in low-to-middle income countries where tuberculosis is endemic.

Case report: A 23-year-old male from Nepal presented to the emergency department after a generalized seizure that lasted for several minutes, according to his colleagues. He was previously healthy, except for the minor weight loss and nausea a few weeks prior to this event. A multi-slice computed tomography (MSCT) of the brain showed multiple ring-enhancing lesions with surrounding edema. Magnetic resonance imaging was performed to further characterize the lesions. The differential diagnosis included neoplastic process, cerebral metastases and various infectious diseases. Antiepileptic therapy was administered during the hospitalization. MSCT of the thorax showed a conglomerate of enlarged lymph nodes in the right mediastinum. There was also an enlarged lymph node in the right supraclavicular fossa that was taken for surgical biopsy and the histologic analysis was performed. The Quantiferon test was positive and the analysis of the lymph nodes showed caseous necrosis as well as Langhans giant cells and the diagnosis of tuberculosis was confirmed. Antituberculotic treatment was started and the patient was referred to a specialized hospital for further evaluation and treatment.

Conclusion: The involvement of the central nervous system is rarely seen as the first manifestation of tuberculosis. However, most seizures caused by it will resolve after a successful treatment of the underlying CNS tuberculoma.

Keywords: Tuberculosis, Central Nervous System, Tuberculoma, Seizures

Sažetak:

Epileptički napad kao prva manifestacija tuberkuloze

Uvod: Tuberkuloza središnjeg živčanog sustava po život je opasno stanje koje se obično manifestira epileptičkim napadima. Najčešće se viđa u nisko i srednje razvijenim zemljama gdje je tuberkuloza endemska.

Prikaz slučaja: 23-godišnji muškarac iz Nepala primljen je u hitnu službu nakon generaliziranog toničko-kloničkog napada u trajanju od nekoliko minuta. Prethodno je bio zdrav, navodi se manji gubitka tjelesne težine i mučnine nekoliko tjedana prije ovog događaja. MSCT mozga pokazao je višestruke prstenaste lezije s postkontrastnom imbibicijom te s okolnim edemom. Učinjena je magnetska rezonancija za daljnju karakterizaciju lezija. Diferencijalno dijagnoza uključivala je neoplastični proces, metastaze i različite zarazne bolesti. Tijekom hospitalizacije započeta je antiepileptička terapija. MSCT toraksa pokazao je konglomerat povećanih limfnih čvorova u desnom medijastinumu. Također je bio povećan limfni čvor u desnoj supraklavikularnoj jami te je učinjena biopsija i patohistološka

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analiza istog. Quantiferonski test je bio pozitivan, a analiza limfnih čvorova pokazala je kazeoznu nekrozu kao i Langhansove gigantske stanice te je potvrđena dijagnoza tuberkuloze. Započeto je s antituberkuloznim liječenjem i bolesnik je upućena u specijaliziranu bolnicu na daljnju procjenu i liječenje. **Zaključak:** Zahvaćenost središnjeg živčanog sustava rijetko se vidi kao prva manifestacija tuberkuloze. Međutim,cnakon uspješnog liječenja tuberkuloma CNS-a postiže se adekvatna kontrola epileptičkih napada.

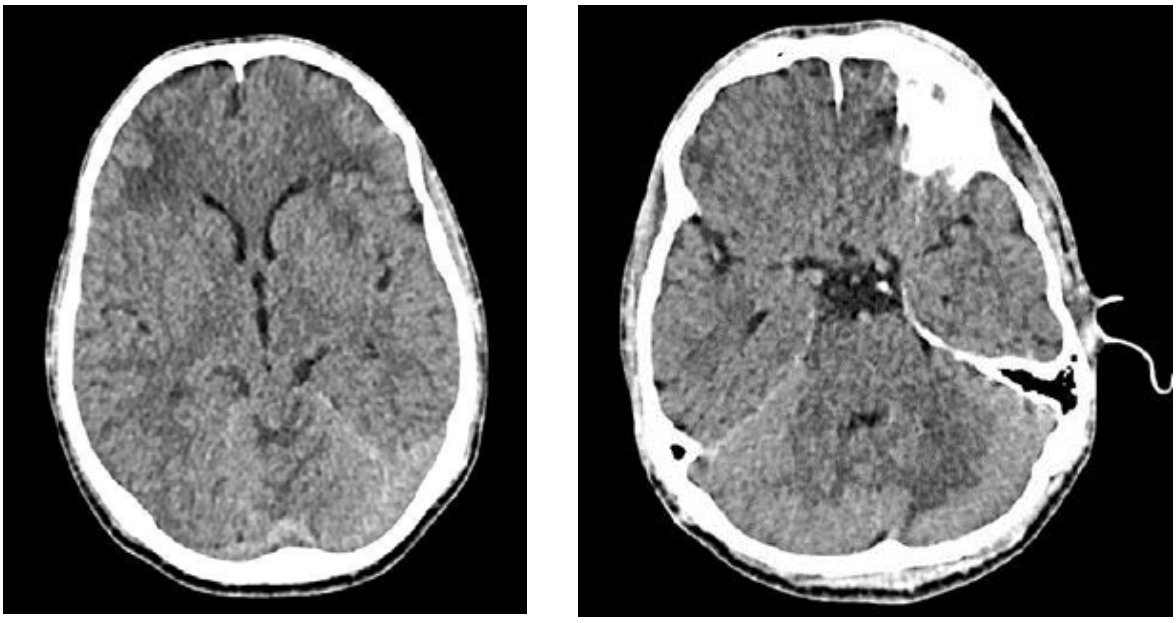
Ključne riječi: tuberkuloza, središnji živčani sustav, tuberkulom, epileptički napad

Introduction

Central nervous system (CNS) tuberculosis is a rare but extremely dangerous condition that is most frequently seen in low-to-middle income countries where tuberculosis is endemic. It carries high morbidity and mortality among all forms of tuberculosis. Among patients with tuberculosis, approximately 1 to 5 percent are complicated by CNS tuberculosis that may take three clinic-pathological forms: a diffuse form of tuberculous meningitis, a focal form of tuberculoma and spinal arachnoiditis (1). Tuberculoma have been reported to occur in 15% of all CNS tuberculosis cases (2). Patients with CNS tuberculoma can present with a new-onset focal or bilateral tonic-clonic seizures, evidence of systematic tuberculosis or some focal neurological deficit (1). Seizures may occur in 25% of patients with CNS tuberculoma (2). Since clinical, laboratory and radiology manifestation of CNS tuberculosis are nonspecific, its recognition is a challenge. However, an early and accurate diagnosis of CNS tuberculosis is crucial for survival. Treatment efficiency depends upon how early it is administered. Most seizures will resolve after successful treatment of the underlying CNS tuberculoma. Most patients with CNS tuberculoma can be managed nonoperatively, surgical excision is indicated when medical therapy fails.

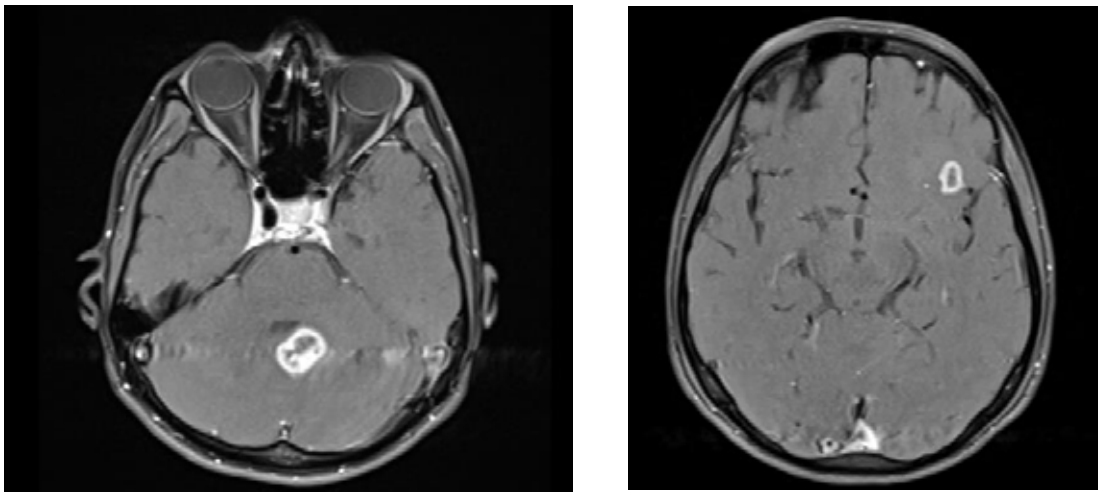
Case report

A young male, from Nepal, presented to our emergency department after loss of consciousness. According to the eyewitnesses, they observed tonic-clonic seizure of unknown onset. He temporarily lost bladder control and bit his tongue. Post-ictal confusion was noticed as well. He was previously healthy, except for the minor weight loss and nausea a few weeks prior to this event. Furthermore, neurological examination showed no neurological deficit. Routine blood tests were done and showed mild hypochromic microcytic anemia. A chest X-ray showed no abnormalities.

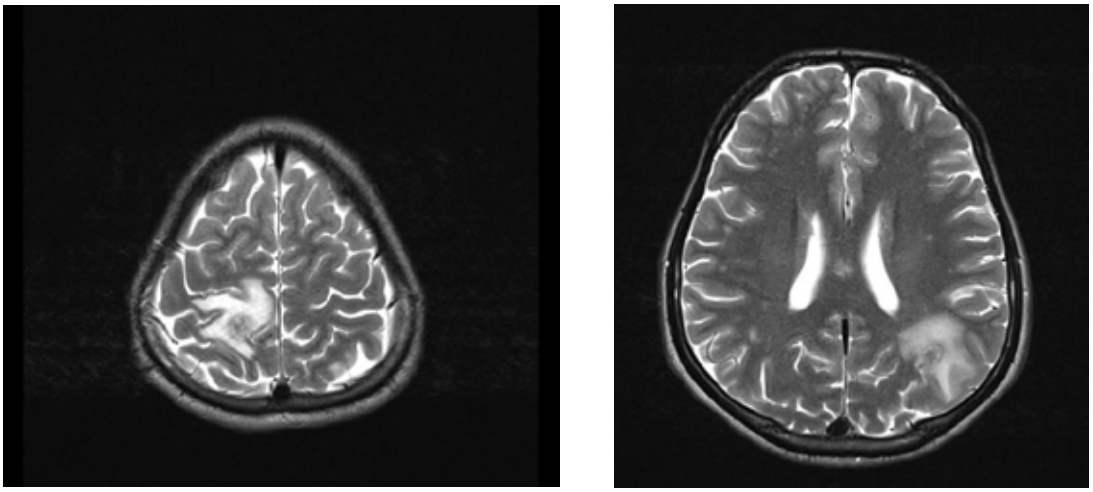


A noncontrast CT scan of the brain showed multiple supra- and infratentorial ring-enhancing lesions with perilesional edema.

In addition, MRI of the brain was preformed. Axial T2- weighted MRI revealed multiple cortico-subcortical isointense lesions supratentorially with the peripheral edema, which show post-contrast ring imbibition with central restriction of diffusion; lesion of the same properties is also seen in the left cereberal hemisphere paramedially and it compresses the 4th ventricle. There are also a few T2/FLAIR- weighted hyperintense lesions supratentorially that are predominantly in the subcortical white matter.



Axial T1-weighted MRI post contrast administration shows nodular lesions with significant ring enhancement after contrast administration.



Axial T2 weighted MRI shows a well circumscribed nodular lesions with a low intensity center on T" and peripheral hyperintensity.

EEG showed focal discharges over right electrodes. Furthermore, lumbar puncture was performed. CSF analysis demonstrated elevated protein concentration and hemato-liquor barrier deficiency. MSCT of thorax, abdomen and pelvis showed a conglomerate of enlarged lymph nodes in the right mediastinum with the central necrosis. Tree-in-bud pattern was described apically bilaterally as well. There was also an enlarged necrotic lymph node in the right supraclavicular fossa that was taken out for surgical biopsy and the histologic analysis was performed. The Quantiferon test was also positive. Surgical biopsy and histologic analysis of the lymph nodes showed caseous necrosis and characteristic Langhans giant cells. Serum and CSF analysis for toxoplasmosis was negative, as well as cysticercosis and serum cryptococcal antigen and the tests for tissue parasites. HIV test, as well as immunologic screening for possible immunodeficiencies were also negative. At the Department of Neurology initial antiedematous therapy was administered, as well as levetiracetam in dose of 500 mg twice a day for seizure prophylaxis. There were no new observed epileptic seizures. Antituberculotic treatment included isoniazide, rifampicin, ethambutol, pyrazinamide for 3 and a half months. Later, patient was referred to a specialized hospital for further evaluation and treatment. In addition, follow-up in the specialized hospital included lab exams (with a special attention to the liver enzymes) and chest X-rays. It is planned to do an additional MRI, EEG and neurological check-up. Treatment includes isoniazid and rifampicin for 3 more months until the next check-up.

Discussion

The involvement of the central nervous system is rarely seen as the first manifestation of tuberculosis. Children younger than 5 years and patients under immunosuppression have higer risk of the CNS involvement of TB. CNS tuberculosis is classified according to anatomical localization (intracranial and spinal). The most common presentation of CNS TB is meningitis (3). Tuberculoma is a rare presentation(<1% of all cases of CNS TB). CNS TB is associated with high mortality because it is hard to diagnose so the start of the treatment is delayed. The most important challenge in managing tuberculoma is its analysis and treatment. Differential diagnosis include intracranial abscess, cerebral metastases, primary lymphoma, meningeoma, glioma and other CNS infections (4). Histologically, tuberculoma consists of a necrotic caseous center surrounded by a capsule containing Langhans giant cells, lymphocytes and fibroblasts (4). Diagnostic procedures include physical examination, neuroimaging techniques (e.g.CT and MRI),extended laboratory blood analysis,CSF analysis and detection of other potentially affected sites (2). Characteristic CSF findings for CNS TB are lymphocytic pleocytosis with an average cell count around 200 cells/µl, moderate to severe proteinorachia and hypoglycorrhachia. It is crucial to perform the smear and the culture of CSF, although it is positive in less than half of patients (4). Treatment regimens include anti-tuberculous therapy and accompanying corticosteroid therapy (5). During the effective anti-tuberculous therapy, worsening of neuroradiological findings can be observed. In one study, more than 64% showed paradoxical deterioration on follow up MRI, but more than half of those patients were asymptomatic. Risk factors related to the development of epilepsy include young age, tuberculoma, cortical involvement, refractory seizures and residual lesions (5). Patients with seizures require antiepileptic drug therapy either as short-course or long- term therapy. Several studies concluded that early usage of antiepileptic drugs reduce risk of developing chronic epilepsy following TBM infection (6). Due to possible adverse drug reactions and potential complications of disease, along with uncertain optimal treatment regimen, it is necessary to monitor all patients during the treatment (5). In the treatment of tuberculosis, isoniazid inhibits the metabolism of carbamazepine, phenytoin and valproic acid, and also can cause toxicity. On the other hand, rifampicin reduces the concentration of carbamazepine, lamotrigine, phenobarbital , phenytoin and valproic acid. (7)

To conclude, CNS tuberculosis is a rare, but potentially fatal disease whose course can be altered by fast recognition and prompt administering of the right therapy.

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