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Clinical and radiological presentation of multiple intracranial tuberculomas mimicking brain metastases: a case report

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ABSTRACT

Cases of brain tuberculoma frequently misdiagnosed as primary or metastatic brain tumors, causes to delay in diagnosis. The delay in diagnosis is even common in patients without a history of pulmonary tuberculosis. In the present case, the patient's history of testicular cancer one year prior, the absence of recent clinical findings suggestive of an infectious process, lack of cough, and the absence of radiological findings indicative of tuberculosis on thoracic CT imaging led clinicians away from considering central nervous system (CNS) tuberculosis in the differential diagnosis. This case is particularly significant in demonstrating that, especially in patients with a known history of malignancy and no evidence of pulmonary tuberculosis, the diagnosis of CNS tuberculosis may be delayed and easily confused with intracranial metastases.

Keywords: Tuberculosis, tuberculoma, intracranial tumor

INTRODUCTION

Tuberculosis, although a preventable and treatable infectious disease, affects more than 10 million individuals worldwide each year and causes over 1 million deaths annually. This makes tuberculosis one of the leading causes of death attributable to a single infectious agent and places it among the top ten causes of mortality globally.[1,2] Approximately 90% of individuals diagnosed with tuberculosis are adults, and the disease is more prevalent in men than in women. Tuberculosis most commonly affects the lungs (pulmonary tuberculosis); however, it may also involve many extrapulmonary organs.[3]

Brain tuberculoma is one of the most severe forms of extrapulmonary tuberculosis and predominantly affects individuals under the age of 40. Nonspecific clinical symptoms and non-characteristic radiological features frequently lead to misdiagnosis.[4] Central nervous system (CNS) involvement represents one of the most serious manifestations of extrapulmonary tuberculosis. While its prevalence in the general population ranges from 2% to 15%, it is observed more frequently in patients with AIDS.[5] CNS tuberculosis accounts for approximately 1% of all tuberculosis cases and is associated with significant morbidity and mortality.[6] Brain parenchymal involvement occurs in 50% of disseminated tuberculosis cases and in approximately 1 out of every 300 untreated pulmonary tuberculosis cases.[7] Studies have shown that more than 75% of patients diagnosed with CNS tuberculosis had a history of pulmonary tuberculosis 6 to 12 months prior to diagnosis. However, pulmonary tuberculosis is absent in 25% to 30% of patients with brain tuberculosis.[8]

Delayed diagnosis and initiation of treatment may be attributed to the absence of a prior tuberculosis history in more than half of patients and the presence of an ambiguous clinical presentation.[9] Tuberculomas can mimic conditions such as glioblastoma, brain metastases, intracranial hemorrhage, and abscesses. The most common radiological appearance of a tuberculoma is a ring-enhancing lesion resulting from the absence of blood flow in the central area of caseous necrosis. However, similar imaging findings may also be observed in other conditions, including thrombosis, toxoplasmosis, bacterial abscesses, cryptococcosis, syphilis, sarcoidosis, inflammatory or vascular abnormalities, and various intracranial tumors.[7]

When treatment is initiated early, more than 85% of tuberculoma cases can be successfully treated. First-line antituberculous therapy consists of isoniazid, rifampicin, ethambutol, and pyrazinamide administered for two months, followed by dual therapy with isoniazid and rifampicin.[10] Corticosteroid therapy is recommended in cases of significant cerebral edema or concomitant meningeal involvement.[10]

Therefore, cases of brain tuberculoma frequently misdiagnosed as primary or metastatic brain tumors, causes to delay in diagnosis. The delay in diagnosis is even common in patients without a history of pulmonary tuberculosis. In this case report, it is aimed to discuss a patient diagnosed with intracranial tuberculoma mimicking multiple cranial metastases, by focusing on diagnostic challenges and differential diagnosis.

CASE

A 60 year old male patient recording of heart failure (ejection fraction: 25%) and adrenal insufficiency had undergone surgical treatment for testicular cancer one year prior. He went to external emergency department with complaints of intermittent syncopal episodes, somnolence, weakness in the extremities, urinary incontinence, and loss of appetite. Upon reviewing the patient's medical history, it was noted that they had no complaints of cough, phlegm, shortness of breath or haemoptysis. During the initial evaluation, cranial computed tomography (CT) and diffusion magnetic resonance imaging (MRI) were performed. The reports of cranial CT revealed hypodense areas consistent with ischemic changes, and the repot of diffusion MRI demonstrated multiple lesions on T2-weighted sequences involving both the parietal and frontal lobes as well as both cerebellar hemispheres (**Figure 1**). Due to the widespread and multifocal distribution of the lesions (hypointense nodular lesions measuring 16×12 mm in the right parietal lobe, 10×9 mm in the left frontal lobe, 6×5 mm in the left cerebellum, and 10×9 mm in the posterior right cerebellum), the findings were interpreted as being consistent with metastatic brain involvement. On chest CT scans, emphysematous changes are present in both lungs, with millimetre-sized nodular infiltrative opacities noted in the upper and middle zones

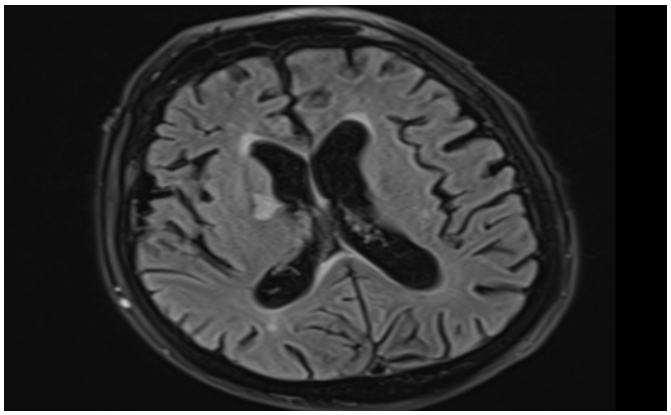

Figure 1. Cranial magnetic resonance imaging (MRI)
MRI: Magnetic resonance imaging

Based on these findings, consultations were requested from the neurology, neurosurgery, and departments as a result, dexamethasone treatment was recommended to reduce intracranial oedema, and no surgical intervention was planned for the patient and the patient was admitted to the medical oncology service. So far, surgical intervention was not a planned option. While during clinical monitoring in the medical oncology unit, positron emission tomography-computed tomography (PET-CT) was performed, which revealed pathologically increased metabolic activity in multiple paraaortic and iliac lymph nodes in the abdomen and pelvis (SUV: max 14.19) (**Figure 2**). Numerous millimetre-sized nodular areas of increased density and infiltration were observed in the upper and middle zones of both lungs, with low metabolic activity (SUV max: 2.90).

A paraaortic lymph node biopsy was performed as a result of the findings. Due to the patient's acute deterioration in consciousness and the presence of strong clinical and radiological findings suggestive of metastases of unknown

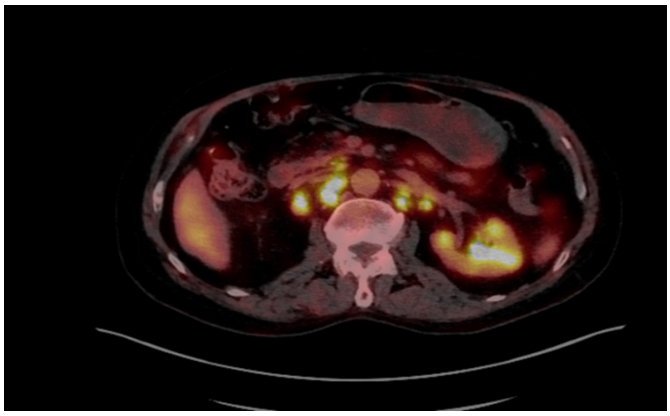

Figure 2. Positron emission tomography-computed tomography (PET-CT)

primary origin, radiotherapy was initiated without awaiting the pathology results, and a total of eight sessions were administered.

During inpatient clinical monitoring after radiotherapy, the patient's fever increased and a marked deterioration in the level of consciousness. As the Glasgow Coma Scale (GCS) score was assessed as 9, admission to the intensive care unit (ICU) was indicated. The patient was subsequently transferred from the external center to our ICU. During ICU hospitalization, the patient was monitored while receiving oxygen support via face mask. Isocoric pupils with bilaterally positive light reflexes were determined during neurological examination and no lateralized motor or sensory deficits were observed in the upper extremities. In addition to absence of neck stiffness, minimal motor responses were elicited in all four extremities with minimal stimulation.

The results of laboratory evaluation revealed a C-reactive protein (CRP) level of 54 mg/L and a procalcitonin level of 0.12 ng/Ml. Moreover, the result of anteroposterior chest radiograph wes normal (**Figure 3**). Based on findings, septic emboli, cerebral vasculitis, and CNS infection were considered in the initial differential diagnosis. Empirical treatment with vancomycin, ceftriaxone, sulbactam, and acyclovir, which had previously been initiated due to suspected CNS infection, was continued following consultation with the infectious diseases department.

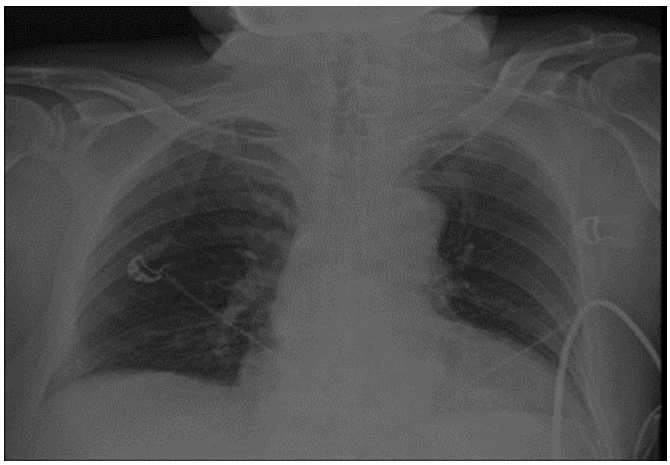

Figure 3. Anteroposterior chest radiograph (PA-AC)

On the second day of antibiotic therapy, histopathological examination of the paraaortic lymph node biopsy revealed granulomatous inflammation composed of extensive

necrosis, small lymphocytes, epithelioid histiocytes, and giant cells. Accordingly, sputum samples were obtained for acid-fast bacilli (AFB) staining, mycobacterial culture, and tuberculosis polymerase chain reaction (PCR) testing. The sputum sample was positive for tuberculosis PCR, and microscopic examination of the sputum AFB smear revealed 1-9 bacilli per 100 fields. Antibiotic susceptibility testing has shown sensitivity to isoniazid, streptomycin and rifampicin.

As a result of reevaluation, the cranial MRI findings metastasis were considered to be consistent with intracranial tuberculoma. However, the cranial MRI findings initially interpreted in favor of metastasis. Therefore, antituberculous therapy consisting of pyrazinamide, rifampicin, isoniazid, and ethambutol hydrochloride was initiated. In addition, treatment with dexamethasone 4 mg four times daily has been initiated for cerebral oedema. The treatment regimen was adjusted following consultation with the Department of Infectious Diseases and Tuberculosis. Given the extensive cranial involvement, the severity of the clinical presentation, and the patient's need for intensive care, an expanded antimicrobial treatment strategy was adopted in addition to the standard antituberculous regimen. In this context, after consultation with the tuberculosis clinic, amikacin, linezolid, and moxifloxacin were added to the treatment regimen to ensure adequate CNS penetration and to cover potential drug resistance.

On the fifth day of ICU hospitalization, the marked improvement was shown in clinical condition of patient. The patient's level of consciousness fully recovered, and the GCS score was assessed as 15. New developed focal neurological deficits were not observed on neurological examination. The findings of laboratory examination demonstrated a CRP level of 24 mg/L and a procalcitonin level of 0.04 ng/ml. After clinical and laboratory improvement, the patient was transferred to the tuberculosis service.

DISCUSSION

CNS tuberculosis, also known as neurotuberculosis, is a severe form of extrapulmonary tuberculosis that develops as a result of the dissemination of Mycobacterium tuberculosis into the cerebrospinal fluid (CSF) and meninges.[10] The most common clinical presentation of neurotuberculosis is tuberculous meningitis. Other manifestations include brain tuberculoma, encephalopathy, brain abscess, Pott's paraplegia, meningoencephalitis, arteritis, and arachnoiditis.[11] CNS tuberculosis accounts for approximately 1-2% of all tuberculosis cases worldwide and is associated with high rates of morbidity and mortality, with more than 100,000 new cases reported annually.[12,13] Current treatment regimens involve prolonged courses of high-dose oral antituberculous medications, typically lasting six to nine months.[14]

One of the primary challenges in establishing a diagnosis in these cases is the presence of nonspecific and ambiguous clinical manifestations, such as altered consciousness, seizures, headache, and focal neurological deficits. The diagnosis of tuberculoma is often based on neuroimaging modalities, including brain CT and MRI; however, no radiological finding is pathognomonic or confirmatory for tuberculoma. Tuberculomas may occur in any intracranial location and can present as solitary or multiple lesions. While infratentorial involvement is more common in children, adults typically exhibit supratentorial localization. Therapeutic challenges include the management of cerebral edema, hydrocephalus, seizures, psychosis, and vasculitis. Moreover, the recent increase in drug-resistant tuberculosis cases has further complicated the treatment of CNS tuberculosis.[15] Long-term antituberculous therapy remains the cornerstone of treatment and is frequently supplemented with corticosteroids to control inflammation and prevent complications such as hydrocephalus. Treatment duration may range from 9 to 24 months, and when initiated early, the prognosis is generally favorable.[16]

In the present case, the patient's history of testicular cancer one year prior, the absence of recent clinical findings suggestive of an infectious process, lack of cough, and the absence of radiological findings indicative of tuberculosis on thoracic CT imaging led clinicians away from considering CNS tuberculosis in the differential diagnosis. Consequently, the lesions observed on cranial imaging were interpreted as mass lesions rather than intracranial tuberculomas. Additionally, the presence of multiple lesions on cranial MRI, a distribution pattern frequently associated with metastatic brain involvement in the literature, further strengthened the preliminary diagnosis of metastasis. However, following the detection of caseating granulomatous inflammation on histopathological examination and a positive tuberculosis PCR result in the sputum sample, reevaluation revealed that the imaging findings were consistent with intracranial tuberculoma.

CONCLUSION

This case is particularly significant in demonstrating that, especially in patients with a known history of malignancy and no evidence of pulmonary tuberculosis, the diagnosis of CNS tuberculosis may be delayed and easily confused with intracranial metastases. In the differential diagnosis of multiple intracranial lesions, even when clinical and radiological findings strongly suggest metastatic disease, the possibility of intracranial tuberculoma should always be considered in regions where tuberculosis is endemic.

ETHICAL DECLARATIONS

Informed Consent

Written informed consent was obtained from the patient(s) included in this report. Signed consent forms are retained by the authors and are available upon request.

Peer Review Process

This report underwent external peer review.

Conflict of Interest

The authors declare no conflicts of interest.

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Author Contributions

Concept: HÇA, EBÇ; Design: Control: HÇA, EBÇ; Data Collection and/or Processing: HÇA, EBÇ; Analysis and/or Interpretation: HÇA, EBÇ; Literature Review: HÇA, EBÇ; Article Writing: HÇA, EBÇ; Critical Review: HÇA, EBÇ.

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