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Case Report

A case of psoas abscess, discitis and meningitis: A tuberculous triology

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Abstract

Psoas abscess is an unusual manifestation of extra-pulmonary tuberculosis. A combination of psoas abscess, discitis and meningitis is a rare triology in medical science, with a paucity of differentials. This case report describes a 33-year-old-lady with a pre-vertebral abscess and meningitis caused by psoas abscess, who was diagnosed to be suffering from extra-pulmonary tuberculosis. The discussion highlights the multiple aetiologies of psoas abscess and the importance of prompt diagnosis and management to reduce morbidity and mortality. There is an emphasis to identify meningitis in a case of psoas abscess to prevent life-threatening complications.

Keywords: Psoas, Abscess, Discitis, Meningitis, Tuberculosis.

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1. Introduction

Psoas abscess is an uncommon form of extra-pulmonary tuberculosis with indistiinct clinical presentation. The classical triad of 'fever, limp, and back pain' is observed in <30% of patients.[1] This entity was first described as psoitis by Mynter in 1881. However, psoas abscess along with discitis and meningitis is rarely encountered in clinical practice. In this case report, we discuss a 33-year-old female with fever and backache who was found to have a pre-vertebral abscess and meningitis due to tuberculosis.

2. Case Description

A 33-year-old female presented with high-grade fever with chills and rigor for 17 days, dull-aching pain in left lower back radiating to left lateral thigh. She further developed weakness of left lower limb for 15 days and weakness of right lower limb 4 days back.

On physical examination, she had normal higher mental functions, cranial nerves including fundus. Motor examination showed normal finding in upper limbs, while lower limb showed hypotonia, grade 4 power in right lower limb, grade 3 power in left lower limb, absent knee and ankle reflexes bilaterally and flexor plantar response bilaterally, suggestive of lower motor weakness. There was dermatomal involvement of L1-L5 segments in the left side of all the modalities of sensation. Kernig's sign was present and neck rigidity was present. Straight leg raising test was positive at 45 degree on left side.

Initial laboratorical work-up showed neutrophilic leukocytosis and prerenal azotemia. A lumbar puncture was performed which showed 340 cells/cumm (10% neutrophil, 90% lymphocytes), protein 59.3mg/dL, sugar 55.6 mg/dL (corresponding blood sugar-101.5mg/dL), India ink staining was negative and cryptococcal antigen test was negative. Cerebrospinal fluid (CSF) virology was negative for Cytomegalovirus, Ebsstein Barr virus, Herpes Simplex Virus. CSF CBNAAT was negative. Her HIV status was negative. Contrast computed tomography (CT) imaging of abdomen and pelvis showed left psoas abscess of low density (30mL) collection with patchy enhancing wall in computed tomography. (**Figure 1**) It was associated with discitis of L4/L5 intervertebral disc in T2-weighted non-contrast magnetic resonance imaging of spine. (**Figure 2**). Contrast-imaging of brain and chest imaging was normal.

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Patient was started on empirical broad spectrum antibiotics (injection ceftriaxone 2g intravenous twice daily, vancomycin 1 gram intravenous twice daily infusion) initially for 14 days, standard anti-tuberculous therapy (oral therapy of isoniazid 300mg, rifampicin 450mg, pyrazinamide 1250 mg, ethambutol 800 mg, pyridoxine 20 mg, dexamethasone 8mg thrice daily) in view of clinical presentation of psoas abscess, spinal arachnoiditis (part of tuberculous meningitis), with discitis; and a CSF picture of lymphocytic-predominant, with hypoglycorrhachia along with sterile culture favoring TBM. CT-guided aspiration (20mL) was done and cultures were sent which were sterile, microscopy was negative. However, the patient symptomatically improved during hospital stay and was discharged (on anti-tubercular therapy and steroids) with advice to follow-up regularly.

At 3-months of follow-up, patient showed clinical improvement and was able to do activities of daily living independently without any discomfort. She gained her normal limb power and she had no sensory complaints as well.

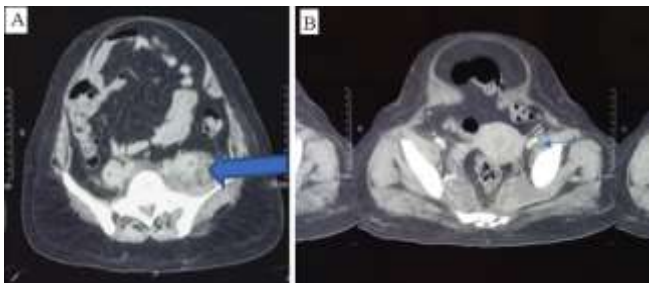

Figure 1: A: Non-contrast computed tomography (CT) of abdomen showing hypodense collection in psoas muscle in left side (blue solid arrow); **B:** Contrast CT of abdomen showing patchy enhancement with central hypodense area in left psoas muscle (blue arrow).

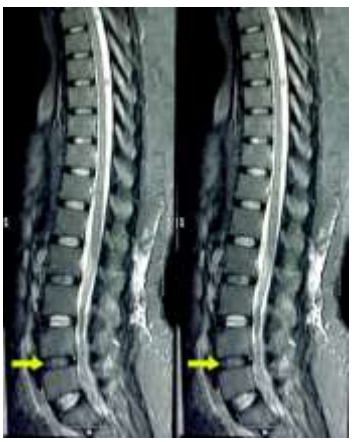

Figure 2: Non-contrast-T2 weighted magnetic resonance imaging of spine showing discitis of L4-L5 level (yellow arrow).

3. Discussion

Psoas abscess (PA) is an important infectious disease which can be caused multiple pathogenic organisms.[2] It is further classified into primary and secondary on the basis of an identifiable underlying disease.[3] Primary occurs in the background of diabetes mellitus, intravenous drug abuse, chronic alcoholics, Acquired immunodeficiency syndrome, chronic kidney disease, malignancy, chemotherapy/ radiotherapy-induced immunosuppression or malnutrition. In, addition, other important risk factors are young age (<20 years), male gender (3:1), and poor socio-economic status. Secondary PA is caused by microbial agents of mixed enteric flora like Escherechia coli and bacteroides. Pott's disease (mycobacterium tuberculosis infection of the spine) is the most common cause of secondary PA in under-developed and developing nations. While the route of causation in Primary PA is haematogenous or lymphatic spread from a distant site, secondary PA occurs by direct spread of a proximately located infectious/inflammatory process into the ilio-psoas muscle.

The psoas muscle which is located in between the transversalis and the psoas fascia, originates from the Thoracic 12 to Lumbar 5 vertebrae. Due to its location, there is a possibility of spread from diaphragm, pancreas, intestine, kidneys and ureters, the spinal column, hips, along with the mesenteric, celiac and lumbar lymph nodal system.

Four main mechanisms of abscess formation have been mentioned previously.[4]

1. Direct spread from a neighboring site (e.g. in Crohn's disease, osteomyelitis, diverticulitis)
2. Hematogenous spread.
3. Purulent lymphadenitis
4. Traumatic hematoma with further infection.

Psoas abscess associated with meningitis is rarely reported in literature. In a case reported by M Horiya et al it, discitis was observed in a 62-year-old female patient suffering from psoas abscess along with bacterial meningitis (Staphylococcus aureus), wherein diabetes mellitus was the prominent risk factor.[5] However no such risk factor was identified in our patient.

Contrast-enhanced computed tomography is considered gold standard for diagnosis of psoas abscess.[6] The finding of edge reinforcement with partly irregular contouring (rim enhancement) and hypo-dense centers (central low density areas) is characteristic of tuberculosis, however it is non-specific.[7]

Empirical antibiotic therapy for psoas abscess includes beta-lactams with beta-lactamase inhibitor, metronidazole, third-generation cephalosporin and MRSA coverage. Anti-tuberculous therapy with corticosteroid is to be initiated on clinical suspicion. Surgical or percutaneous drainage (PCD)

of the abscess can definitely reduce morbidity and mortality.[8] PCD has become the treatment of choice in primary PA due to its non-invasive nature and better results. Surgical drainage is however indicated in case of failure of PCD, or any contraindication to PCD procedure or the presence of any abdominal pathology requiring intervention. Surgical drainage has shown better outcome in secondary PA due to the presence of definite concurrent intra-abdominal pathology. The fatality rates for primary and secondary PA are approximately 2.4% and 18.9% respectively.[3] Death is usually due to inadequate or delayed treatment, and it can be 100% fatal if untreated in immunosuppressed state because of florid sepsis. In tuberculosis, psoas abscess can be a prominent finding in Pott's spine which seldom requires drainage in addition to anti-tubercular treatment.[9] It can be an accompaniment of tuberculous meningioencephalitis, as in our case.[10] Other complications like hydrocephalus, vascultic infarcts, paradoxical reaction in the form of arachnoiditis or tuberculomas can also occur in tuberculous meningitis which needs prompt treatment to prevent morbidity and mortality.[11] Furthermore, outcome also depends upon factors like immune-compromised status of the patient, co-morbidities, bacterial load and resistance, socio-economic status and treatment continuity.[12]

4. Conclusion

Meningitis in presence of psoas abscess is an important although uncommon coexistence. It is important that this condition is recognised early and treated promptly. The identification of meningitis and early treatment has equal importance to prevent morbidity and mortality.

5. Source of Funding

None.

6. Conflict of Interest

None.

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