Bilateral Psoas Abscess: Case Report and Review of Literature

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SUMMARY

Background: Psoas abscess is rare, the presentation confounding and varied thus making the diagnosis and definitive management delayed with resultant severe morbidity and high mortality. The paucity of literature on this disease would appear to account for the low index of suspicion among primary care physicians who often have first contact with these patients.

Methods: We present a case of bilateral psoas abscess in an adult male who presented with acute abdomen and the diagnosis of psoas abscess was made at laparotomy.

Conclusion: We highlight the salient features of the disease, which may assist practitioners in reaching early diagnosis and improve outcome of the disease.

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***Keywords*: Psoas abscess, Confounding diagnosis, Laparotomy**

INTRODUCTION

Psoas abscess is very rare. It is classified as primary or secondary depending on the underlying cause.[1,2] Its clinical features are often varied and the diagnosis is easily missed by the primary care physician.[1] The consequent delay in the diagnosis and definitive treatment often heightens the associated morbidity.[3]

The paucity in medical literature of this disease, coupled with its protean presentations, may partly account for the low index of suspicion among primary physicians.[1] The classical clinical features of fever, loin and hip pain are present in only 30% of cases. The psoas muscle is closely related to the kidneys, ureters, caecum, appendix, sigmoid colon, pancreas, lumbar lymph nodes and nerves of the posterior abdominal wall. Infection of any of these organs may extend to the psoas muscle causing an abscess.[4]

Different microbes are implicated in the aetiology of psoas abscess including *staphylococcus aereus(80%), serratia and haemopilus*.[2] In tropical Africa, *mycobacterium tuberculosis* remains of clinical relevance as a cause of spinal tuberculosis (Tb) with implication for progression to psoas abscess.[5]

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A history of pulmonary tuberculosis may not be obtained and investigations for tuberculosis may be negative. The increasing incidence of immune suppression, in particular by the acquired immune deficiency syndrome (AIDS) may lead to an upsurge in the incidence of this otherwise rare disease.[6]

Increased availability and utilization of modern imaging techniques, in particular ultrasound (US), computerized tomography (CT), and magnetic resonance imaging (MRI) have lead to an increased worldwide diagnosis and reporting of this disease.[7] Treatment may be medical, surgical or a combination of both modalities.

CASE PRESENTATION

A 27-year old male presented at the emergency room with a one week history of severe, constant, non-radiating abdominal pain and distension. The abdomen was grossly distended with oedematous wall and markedly tender. The pulse was 98 per minute, temperature39^{o}C and respiratory rate 22 per minute. He had bilateral pitting poedal oedema. Packed cell volume was 20%. The rectum was empty and markedly tender. Abdominal paracentesis yielded free flowing pus and peritonitis was diagnosed.

Exploratory laparotomy revealed bilateral retroperitoneal abscess cavities in the lumbar region, which gave away the diagnosis. Large tube drains were placed extraperitoneally through bilateral groin incisions and yielded 5.5L of pus. The abdominal incision was closed. Recovery was uneventful. He complained of productive cough on 8th day but a chest radiogram ordered was normal.

Further assessment revealed a gibbus at L_2/L_3, and a thoraco-lumbar x-ray showed wedge compression of the anterior segments of L3 and L4 with no neurological deficits. Mantoux was 12mm. (Acid Fast bacicli on three occasions) and retroviral serology were negative. ESR was 80mm/1st hour and White Blood Cell Count 11X109/L with predominant neutrophils. Culture of the pus yielded growth of *E. coli* but no AFB. An abdominal ultrasound done at one week showed 60ml of residual pus collection. He failed to respond to a combination of ampiclox and metronidazole but improved dramatically of on combination anti- tuberculosis drugs; he stopped coughing, temperature dropped to 37^{o}C in the second week and he gained weight of 1.5 kg by fourth week. Tubes stopped draining after four weeks and were removed. He was discharged after six weeks.

DISCUSSION

The global reporting of psoas abscess has been on the increase with 12 cases per annum in 1991, as against 4 cases

annually in 1985. This is attributed to increased availability and utilization of modern imaging modalities –US, CT and MRI.[7] Under-reporting is a major problem in developing countries probably due to absent diagnostic tools.[1] Reports from Johns Hopkins University Medical School show a predominance of secondary psoas abscess and a close association between primary psoas abscess, intravenous drug abuse and acquired human immune-deficiency syndrome. The average age of patients is 30 to 40 years and male: female is 2:1.[1,2] Conditions associated with secondary psoas abscess are listed in Table 1.

Table 1: Conditions associated with secondary psoas abscess

Gastro-intestinal	Appendicitis, appendiceal tumor, strangulated obturator hernia, diverticulitis, colorectal carcinoma
Musculo-skeletal	Vertebral osteomyelitis, septic arthritis, infectious sacroilitis
Genito-urinary	Urinary tract infection, extra-corporeal shock wave lithotripsy, cancer
Others	Haemo/ peritoneal dialysis, trauma, spinal surgery sepsis, endocarditis

A thorough appraisal of the history and physical signs including a high index of suspicion remarkably improves the diagnostic yield. The classical symptom of fever, loin and hip pain are present in only 30% of patients,[7] most have low back pain, abdominal pain and other non-specific symptoms like nausea, vomiting, and malaise.[3] Patients usually maintain a flexed knee with hip externally rotated; this is the position of greatest comfort. The symptoms of marked abdominal pain and distension as well as the physical findings in our index patient were unusual; they rather simulated peritonitis and few physicians encountered with these varied features will make a diagnosis of psoas abscess firsthand. It is noteworthy that we missed the gibbus which could have suggested spinal tuberculosis and psoas abscess. Laboratory findings in psoas abscess include moderate to severe anaemia, leucocytosis, and a raised ESR. These may not be apparent early in the disease.[1] A plain x-ray film is indispensable in the diagnosis and is recommended as a first line investigation. It reveals bony destruction of the spine (see fig.1), soft tissue gas, mass effects and abnormal psoas margin. However, minimal skeletal lesions will produce negative results.[7]

Ultrasound produces fast and cheap results; it differentiates between solids and fluids, it is less sensitive because it cannot penetrate gas or bone. MRI and CT scans produce better tissue resolution but are employed when plain films are negative or difficult to interpret.[8]

Issues of interest in our index patient are late presentation and a remarkable response to anti- tuberculosis drugs, which prompted the likely aetiology of the abscess. Late presentation is the norm in spinal tuberculosis; the organism may remain dormant in the skeletal system for prolonged periods prior to detection and the history and physical findings may not reliably suggest the diagnosis.[7,8] Spinal tuberculosis is the most common form of extra-pulmonary tuberculosis, consisting 50 to 80% of all cases.[5,9] It affects the anterior segment of two or more vertebrae. Spread is through haematogenous or lymphatic routes.[8] An abscess cavity forms locally and may involve contiguous structures. Collapse of the involved vertebral bodies and loss of disc space eventually leads to kyphosis and scoliosis. The thoracic and lumbar spines are commonly affected.[6,9] Systemic symptoms set in as the disease progresses. Paralysis is a late sign, arising from cord compression.[8]

The positive Mantoux test, raised ESR, failure to respond to earlier introduced antibiotics and the thoracolumbar segment collapse on plain radiogram enhanced the diagnosis. Failure to find *mycobacteria tuberculosis* in sputum and pus is not unusual; bone biopsy is specific. CT is the gold standard in skeletal tuberculosis; it can detect very early changes. Treatment is by draining the abscess, spinal fusion and combination anti-tuberculosis drugs. Drainage may be open or closed; the latter under CT guidance. We did open drainage and the tubes were removed when they ceased to drain. Ultrasound was used to assess any residual abscess collection. Anti-tuberculosis drugs and high calorie/high protein diet was our main stay of treatment.

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