MULTIDRUG-RESISTANT TUBERCULOUS ILIOPSOAS ABSCESS

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Psoas abscess is an important disease with subtle and often nonspecific presentation that frequently provides a diagnostic challenge. Of late, it is only rarely caused by *Mycobacterium tuberculosis* (MTB). In a recent retrospective review of extrapulmonary tuberculosis among 196 patients with human immunodeficiency virus (HIV) infection and 153 HIV-negative patients, only one with disseminated infection had psoas abscess at postmortem.[1] However, the recent resurgence of tuberculosis may result in changes in the etiologic trends of psoas abscess. This increase in the incidence of MTB has been reported worldwide, however, there has also been concurrent increase in the prevalence of MTB resistant to first-line antituberculosis agents, especially among patients with HIV infections.[2-5] Multidrug-resistant MTB (MDR-MTB) have been defined as strains resistant to isoniazid and rifampicin, with or without resistance to other drugs.[2]

MDR-MTB producing extrapulmonary manifestations have been well documented. In the above review of patients with extrapulmonary tuberculosis spanning 1983 to 1989, Shafer et al. reported 28 out of 349 patients (8%) to have MDR-MTB, however, none of the patients had MDR-TB psoas abscess.[1] In this communication, we report a case of tuberculous psoas abscess caused by multidrug-resistant *Mycobacterium tuberculosis* in an immunocompetent host. To our knowledge, this is the first case of MDR-TB-causing psoas abscess to be reported in the literature.

Case Report

A 42-year-old female patient was admitted to the hospital complaining of right-sided hip pain and limping of two months' duration. She had been diagnosed seven months earlier at another hospital with tuberculous lymphadenitis based on lymph node biopsy, and was put on antituberculous drugs (not known to the patient), which she took for only three months. There was no history of fever, night sweat or weight loss. She denied any history of cough, shortness of breath, hemoptysis or history of contact with a tuberculous patient or relative. There were no other medical illnesses.

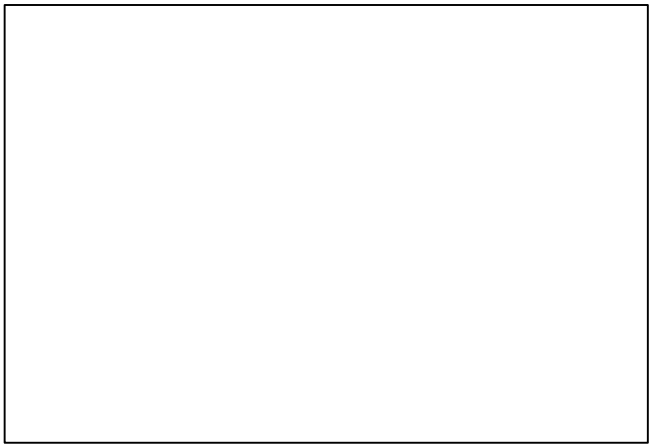

FIGURE 1. Computerized tomography of the pelvis illustrating the fluid collection in relation to the right iliacus and iliopsoas muscles.

Physical examination revealed normal vital signs. The patient looked ill and underweight, but there was no lymph node enlargement or clubbing. Chest and cardiovascular examination was unremarkable. Abdominal examination revealed soft, non-tender abdomen and no organomegaly. There was an 8x4 cm soft, non-tender swelling at the right inguinal region with normal overlying skin.

Laboratory investigations revealed normal full blood count, liver enzymes, electrolytes, urea and creatinine. ESR was 90 and HIV serology was negative. X-rays of the chest, pelvis, and spine were normal. Ultrasound of the right inguinal area showed large fluid collection within the right iliacus and iliopsoas muscles, extending inferiorly into the right hip, consistent with iliopsoas abscess (Figure 1). This abscess was aspirated under ultrasound guidance. Ziehl-Neelsen stain of the aspirate was positive for acid-fast bacilli. The aspirate was inoculated on Lowenstein-Jensen (LJ) slopes and MB/BacT liquid culture bottles for culture of mycobacteria (Organon Teknika, Durham, NC, U.S.A.). Slope cultures were examined weekly for eight weeks, while the liquid cultures were monitored continuously in

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the MB/BacT 2400 cabinet (Organon Teknika) for eight weeks. *Mycobacterium tuberculosis* in the specimens were identified by ligase chain reaction (LCR) mycobacterium assay for mycobacterium DNA (Abbott LCX Probe system, Abbott Diagnostics, Chicago, Ill., U.S.A.).[6] Sensitivity test of the isolate was carried out by Bioscientia Laboratory, Ingelheim, Germany. The patient was started on isonazid, rifampicin, pyrazinamide, and ethambutol. She started to improve clinically and to walk after a few days without support. Pyrazinamide and ethambutol were stopped after two months of initiating therapy.

One month later, the patient started to deteriorate clinically to the degree that she became confined to a wheelchair. She had right-sided hip pain and inability to walk, low-grade fever, poor appetite, and started to lose weight. Repeated ultrasound showed reaccumulation of fluid in the iliac and iliopsoas muscles. CT scan of the pelvis revealed fluid collection in relation to the right iliac muscle, extending down into the pelvic cavity and reaching the symphysis pubis. Evidence of effusion in the right hip and some destruction of the cortex of the head of the femur were also noted with loss of joint space suggesting septic arthritis. ESR was still elevated at 68 mm/hour. The patient was readmitted and, at this stage, the result of the culture of the aspirate was back, revealing *Mycobacterium tuberculosis* resistant to streptomycin, isoniazid, rifampicin and rifabutin, but sensitive to ethambutol, pyrazinamide, prothionamide, capreomycin and amikacin. She was started on pyrazinamide, ethambutol, amikacin, and ciprofloxacin (the latter two medications are the only second-line antituberculous medications available locally). Isoniazid and rifampicin were discontinued. The abscess was then drained under CT guidance and 150 mL of purulent fluid was obtained. A drain was kept in place for seven days.

Three months after starting the second-line anti-tuberculous agents, the patient improved clinically, gained weight (2 kg), and started to walk with crutches. Her ESR decreased to 28 mm/hour. Amikacin was stopped after completion of four months of therapy. Repeated CT scan of pelvis revealed no more fluid collection, however, the right femoral head showed ill-defined border with widening of joint space and erosions on the acetabular roof. A period of 18 months of therapy was completed. At the end of the treatment course, the patient gained 13 kg and remained asymptomatic, apart from mild residual limping.

Discussion

Psoas abscess is often a diagnostic challenge due to its nonspecific symptomatology and subtle physical findings. Plain radiograph may be suggestive, but ultrasonography and CT scan are the keys to definitive diagnosis. When considering the etiology of psoas abscess, *Mycobacterium tuberculosis* should always be considered, as well as other pyogenic organisms, such as *Staphylococcus aureus*, *Escherichia coli* and other Enterobacteriaceae. Ziehl-Neelsen staining of any aspirated material should be carried out together with culture on Lowenstein-Jensen's media. In addition to Gram's stain, blood cultures and routine culture of the material, PPD test and HIV serology should also be performed.

TABLE 1. *Prevalence of MDR tuberculosis in previous studies in Saudi Arabia.*

	No. of patients	% of all resistant	% of INH resistant	% of RIF resistant	% of MDR
Schiott et al.[16]	108	43.7	40.8	20.4	19.4
Al-Orainy et al.[17]	432	21.3	19.4	9.7	8.8
Jarallah et al.[18]	678	22.6	6.5	15	3.8
Ellis et al.[19]	289	8.7	7.2	3.1	2.8
Kinsara et al.[20]	78	11.6	10.2	5.1	5.1

*Modified from Wali et al.[21]

The source of psoas abscess is usually a distant site, with presumed hematogenous spread at the time of primary tuberculous infection, which remains quiet until reactivation. In some cases, infection may be due to direct spread from a ruptured viscus or skeletal tuberculosis. In our reported case, since there was no evidence of either of these, and since chest radiograph was normal and sputa for acid-fast bacilli was negative, we can only presume that hematogenous spread occurred at the time of initial infection.

The management of tuberculous psoas abscess is similar to that of non-tuberculous lesions, comprising a combination of antituberculous chemotherapy and operative drainage, which has recently been replaced by percutaneous drainage (PCD) under ultrasound or CT guidance.[7-12] CT is more sensitive than ultrasound in showing the exact localization and the extent of the abscess.[13] When performed by experienced personnel, PCD has a low risk and is a fairly easy procedure for the management of psoas abscess, especially in countries where tuberculosis is widespread.[11] Reports regarding PCD in treating tuberculous psoas abscess are scanty.[11,12] Pombo et al. reported six patients with TB psoas abscess who were treated successfully by CT-guided PCD and chemotherapy despite extensive bony and soft tissue disease.[12] There was only one relapse on follow-up, which was probably related to poor adherence to anti-TB chemotherapy. Dinc et al. treated 10 tuberculous and five pyogenic psoas abscesses, four of which were multiloculated, by PCD and chemotherapy.[11] None required surgery and relapse was reported in only one patient.

Our patient had the additional challenging problem of multidrug-resistant TB, which is reported to be a devastating illness, even with good compliance to therapy. It has been suggested that a tuberculosis retreatment regimen should always include at least four, but possibly as many as six or seven drugs, since the four-drug regimen has been reported to be inadequate in some instances.[3] The number of drugs used will vary according to the extent of

the disease, potency of the available agents and the in vitro susceptibility results.[2,3] Regimens of multiple drugs which are generally poorly tolerated and more toxic than traditional regimens must be administered for 18 to 36 months.[2,3,14] Some reports suggest that even the best available treatment is often unsuccessful, with only about a 65% success rate.[15]

The prevalence of MDR tuberculosis in Saudi Arabia is largely unknown, except for a few studies confined to the large centers,[16-20] however, it has been reported to vary from 2.8% to 19.4% (Table 1).[21]

Drug-resistant MTB may arise from pitfalls in the management of tuberculous infections. These include monotherapy, poor patient compliance, and failure to supervise treatment.[22] Other risk factors are the history of previous antituberculous therapy, history of contact with a known tuberculous case, history of hospitalization, institutionalization or incarceration at a facility with a known MDR-TB outbreak, and recent immigration from Asia, Africa, and Central and South America to developed countries.[3] There are some additional risk factors which may contribute to the development of MDR-TB in Saudi Arabia. These include availability of antituberculous drugs without prescription, the use of some antituberculous drugs for the treatment of conditions such as brucellosis and leprosy, and the presence of large numbers of expatriate workers in the Kingdom from countries where TB is highly endemic.[19,23] In our patient, the initial poor adherence to antituberculous chemotherapy was the major risk factor for the development of secondary resistance to first-line anti-tuberculous drugs.

This reported case established the development of psoas abscess by MDR-TB in an immunocompetent patient, which is still rare. With the increasing prevalence of MDR-TB worldwide, more cases are to be expected. Proper diagnosis and treatment requires facilities for culture and susceptibility testing of *M. tuberculosis*.

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