Iranian Journal of Clinical Infectious Diseases
2007;2(4):199-202

CASE REPORT

Abdominal and cervical lymphadenopathy and multiple abscesses due to mycobacterium tuberculosis; A case report

Shervin Shokouhi[1], Shahryar Nikpour[2], Morteza Sanei Taheri[3], Seyed Amin Zamiri[3]

[1] *Department of Infectious Disease and Tropical Medicine, Shahid Beheshti Medical University, Tehran, Iran*

[2] *Department of Gastroenterology, Shahid Beheshti Medical University, Tehran, Iran*

[3] *Department of Radiology, Shahid Beheshti Medical University, Tehran, Iran*

ABSTRACT

Background: Mycobacterium tuberculosis is the cause of 43% of the peripheral lymphadenopathy cases in developing countries. However, psoas abscesses are usually secondary to the extension of infection from an adjacent site. In the present study, we describe a case of abdominal and cervical lymphadenopathy and multiple abscesses due to mycobacterium tuberculosis.

Patient: A 55 years old man with abdominal and cervical lymphadenopathy, psoas muscle sheath abscess and a large abscess of abdominal wall was admitted. Vertebral column was intact and the patient didn't have immunodeficiency or history of illicit drug use. Analysis of aspirated pus with PCR for mycobacterium tuberculosis was positive, however, Ziehl-Neelsen and gram staining was negative. Culture of pus was positive for mycobacterium. Treatment was commenced with 4 drug antituberculosis regimen. During the treatment period, paradoxical reaction occurred and prednisolone was administered. Following 9 months of treatment, the abscesses resolved and the patient recovered completely.

Conclusion: our patient presented with a group of uncommon extrapulmonary presentations including GI involvement, paraaortic lymphadenopathy and abscess formation secondary to tuberculosis that was aroused without any immunodeficiency context with hematogenous origin. He responded well to our therapeutic protocol.

Keywords: *Tuberculosis, Lymphadenopathy, Psoas abscess, Extrapulmonary TB.*

(Iranian Journal of Clinical Infectious Diseases 2007;2(4):199-202).

INTRODUCTION

One third of the world population is infected with mycobacterium tuberculosis and this microorganism is the cause of 8 million of new infections and about 2 million deaths annually (1). The prevalence of the extrapulmonary cases of tuberculosis was increasing till 1991 because of the increment in AIDS prevalence (2,3). Lymphadenitis is the most prevalent form of extrapulmonary tuberculosis (4). Mycobacterium tuberculosis is the cause of 43% of the peripheral lymphadenopathy cases in developing countries (5). Lymphadenopathy in sites other than the neck and supraclavicular is suggestive of a more severe infection and often with systemic presentations. Biopsy of these lymph nodes shows granuloma, but the smear or culture are usually negative (6). Up to

Received: 10 April 2007 *Accepted*: 28 July 2007
Reprint or Correspondence: Shervin Shokouhi, MD. Department of Infectious Disease and Tropical Medicine, Shahid Beheshti Medical University, Tehran, Iran.
E-mail: sh_sh50@yahoo.com

one third of patients with TB lymphadenitis has a history of pulmonary involvement (7,8). Other sites of TB lymphadenopathy that has been reported are axillary and inguinal, mesenteric, mediastinal and interamammary chain (9). In most cases, gastrointestinal involvement of tuberculosis is secondary to the ingestion of respiratory secretions but in less than 25% of cases radiological evaluations show pulmonary involvement and the diagnosis is made incidentally by endoscopy or surgery (1). Changes like lymph node enlargement, tenderness, suppuration, sinus formation or newly formed lymphadenopathy could seen in 25-30% of cases during or after the completion of therapy (10). In patients with AIDS and tuberculosis, abdominal lymphadenopathy is common and may be massive (11,12). Psoas abscesses are usually secondary to the extension of infection from an adjacent site and primary psoas abscesses are seldom hematogenous (13). Psoas abscess may be a complication of pyogenic or tuberculos osteomyelitis of the vertebral column. Formerly tuberculosis was the leading cause of psoas abscesses but at present it is mostly because of direct expansion of interaabdominal infections (diverticulitis, appendicitis, Crohn's disease) (14). Gastrointestinal involvement by tuberculosis can be seen in 3 forms: ulcerative, hypertrophic and ulcerohypertrophic. The ulcerohypertrophic type often involves ileocecal area (14).

PATIENT

A 55 years old man presented with a chief complaint of abdominal mass. He had anorexia since 3 months ago. Since 2 months ago he noticed the right lower quadrant budging that increased in size gradually. He didn't have any complaint of sweating, shaking chills, cough, sputum, diarrhea and abdominal pain. He lost about 10 kg of weight during 2 months. He hadn't family history of tuberculosis. A mass with a size about 8×10cm was palpable in right lower quadrant. Consistency of the mass was pasty in palpation without fluctuation. Other physical examinations were within normal range.

On admission the patient was anemic (Hb=9), PPD>20 mm, and the HIV-ELISA test was negative. In sonography, liver and spleen were reported to be normal. In right lower quadrant a mass sized 4×7×8cm with heterogenic echo was detected in abdominal wall muscle or beneath it without peristalsis.

The abdominopelvic CT scan with IV and oral contrast was performed and showed a hypodense lesion with liquid density and narrow rim enhancement and distinct borders without septation in abdominal wall muscles in hypoderm. Its dimension was 70×120×120mm without any connection with the intraabdominal space, which was suggestive of abdominal wall abscess.

Two adjacent collections were also detected in psoas muscle with rim enhancement from the level of S_1 to the border of acetabulum without extension to the upper or lower parts. Intervertebral discs of L_4-L_5 and L_5-S_1 were normal. Destructive lesions were not detected in the vertebral column. A concentric thickness and irregularity of mucosal foils of terminal ileum and cecum and hepatic flexure of colon was noted. An oval hypodense focus with rim enhancement at the lateral side of the hepatic flexure, with irregularity of colonic wall and fat infiltration was also detected. There was a hypodense lymph node (abscess) in aorto-caval area at the infra renal level. During colonoscopy an area of erythema and edema measured 1×1cm near hepatic flexure was observed. In proximal part of ascending colon, near cecum, a 1×2cm area of edema, vegetation and loss of vascularity was detected. Biopsy was taken from the area and the pathologic evaluation showed severe infiltration of acute and chronic inflammatory cells in mucosa and submucosa with granulomas without central necrosis, scattered in mucosa. The Ziehl-Neelsen staining showed a few acid fast bacilli in

granulomas. Aspiration of abdominal wall abscess was achieved. Analysis of aspirated pus with PCR for mycobacterium tuberculosis was positive, however, Ziehl-Neelsen and gram staining was negative. Culture of pus was positive for mycobacterium. The candida and tetanus skin tests were achieved to evaluate cellular immunity in vivo, however, all were normal. With diagnosis of extrapulmonary tuberculosis, 4 drug anti-tuberculosis regimen was commenced. Two weeks later abdominopelvic CT scan performed and the following changes were observed in comparison with the primary CT scan: the aortocaval lymph node (abscess) was completely subsided, the lesion of the lateral wall of hepatic flexure was improved, thickness and irregularity of the terminal ileum wall decreased, the irregularity and narrowing of the lumen of cecum decreased apparently, and the size of psoas abscess was diminished.

A subcutaneous abscess measured 10×50×120cm with inflammatory reaction, secondary to needle insertion was also noted. Patient discharged with 4 drug regimen. Two months later he presented with pruritus and erythema and enlargement of cervical lymph node. With the diagnosis of paradoxical reaction, he was administered 25mg of oral prednisolone daily. Cervical lymph node became fistulated with little secretions. Prednisolone tapered during one week and then discontinued. Patient had 6kg weight gain during this period of time.

DISCUSSION

The primary diagnosis for the patient was an ileocecal mass. To rule out malignancy, endoscopy was performed. Detection of AFB in colon mucosa lesions biopsy confirmed the diagnosis of GI tuberculosis. Visceral tuberculous abscesses include hepatic, pancreatic and splenic abscesses often occur in HIV positive patients (11,12), however, in our case a subcutaneous abscess was found in right lower quadrant and psoas muscle sheath, although the patient was HIV-negative. Subcutaneous abscesses had no association with other sites that could suggest a hematogenous origin. In the reported patient, cervical lymphadenopathy was found at the base of the neck.

Intraabdominal lymph node involvement in tuberculosis is usually in mesenteric area (15). In our case, paraaortic lymphadenopathy was also found. This is an extremely rare presentation for tuberculosis and, to our knowledge, it hasn't been reported yet.

There is a 25-30% probability of lymph node enlargement with sinus formation and becoming supporative during or after the treatment. That is not a sign of treatment failure but an evidence for reaction to residual bacilli antigens that subsides spontaneously, while a short course of corticosteroid accelerate its remission (10). This also happened for our patient. The psoas muscle sheath abscess had no relation with other places and vertebral osteomyelitis was not found, thus, the probable origin of the infection was hematogenous (14). This is also a rare origin for psoas abscess secondary to mycobacterium tuberculosis GI involvement. Ulcerohypertrophic lesions are seen in 30% of all GI TB cases and often involves around ileocecal valve (15).

In conclusion, our patient presented with a group of uncommon extrapulmonary presentations including GI involvement, paraaortic lymphadenopathy and abscess formation secondary to tuberculosis that was aroused without any immunodeficiency context with hemotogenous origin. However, he responded well to our therapeutic protocol.

REFERENCES

1. Dye C, Scheele S, Dolin P, et al. Consensus statement. Global burden of tuberculosis: Estimated incidence, prevalence, and mortality by country. WHO Global Surveillance and Monitoring Project. JAMA 1999;282:677-86.

2. Reider IL, Snider DE, Cauthen GM. Extrapulmonary tuberculosis in the United States. Am Rev Respir Dis 1990;141:347-51.

3. Shafer RW, Kim DS, Weiss LP, et al. Extrapulmonary tuberculosis in patients with human immunodeficiency virus infection. Medicine 1991;70:384-97.

4. Mandell GL, Bennet JE, Dolin R, editors. Principle and practice of infectious disease. 6th edition. Philadelphia: Churchill Livingstone, 2005;p:2881.

5. Summers GD, McNicol MW. Tuberculosis of superficial lymph nodes. Br J Dis Chest 1980;74:369-73.

6. Dandapat MC, Mishra BM, Dash SP, et al. Peripheral lymph node tuberculosis: A review of 80 cases. Br J Surg 1990;77:911-12.

7. McCabe AS. Extrapulmonary tuberculosis revisited: a review of experience at Boston City and other hospitals. Medicine (Baltimore) 1984;63:25.

8. Artenstein AW, Kim JH, Williams WJ, et al. Isolated peripheral tuberculous lymphadenitis in adults: current clinical and diagnostic issues. Clin Infect Dis 1995; 20:876.

9. Yilmaz F, Yagmur Y, Uzunlar AK. Tuberculosis in an intramammary lymph node. Eur J Surg 2000;166:267.

10. Campbell IA. The treatment of superficial tuberculous lymphadenitis.Tubercle 1990;71:1-3.

11. Perich J, Ayuso MC, Vilana R, et al. Disseminated lymphatic tuberculosis in acquired immunodeficiency syndrome: Computed tomography findings. Can Assoc Radiol J 1990;41:353-57.

12. Radin DR. Intraabdominal *Mycobacterium tuberculosis* vs *Mycobacterium avium interacellular* infections in patients with AIDS: Distinction based on CT findings. Am J Roentgenol 1991;156:487-91.

13. Steiner JL, Septimus EJ, Vartian CV. Infection of the psoas muscle secondary to Strcptococcus pneumoniae infection. Clin Infect Dis 1992;15:1047-48.

14. Kyle J. Psoas abscess in Crohn's disease. Gastroenterology 1971;61:149-55.

15. Marshall JB. Tuberculosis of the gastrointestinal tract and peritoneum. Am J Gastroenterol 1993; 88:989.