Case Report

Extensive Primary Male Genital Tuberculosis

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Abstract

Genital tuberculosis (GTB) is uncommon, and the most common genital sites of tuberculous infection are epididymis and prostate; isolated testicular TB is extremely rare, comprising only 3% of GTB. The usual modes of genital involvement include descending infection from the kidneys, intracanalicular or direct extension from neighboring foci in the genital tract, and hematogenous dissemination. Ultrasonography (USG) and USG-guided fine-needle aspiration cytology of testicular swelling may confirm the diagnosis of GTB. Anti-TB chemotherapy is the mainstay of treatment to ensure the complete resolution of the lesion. Infertility in GTB is a result of obstruction at the terminal portion of the ejaculatory duct, resulting in dilatation of the proximal ductal system including the vas deferens preventing seminal vesicle secretions from reaching the ejaculate. Seminal vesicle secretions make up the bulk of the ejaculate, contain fructose, and alkalinize the ejaculate, and with obstruction, patients present with azoospermia or aspermia. Here, we present a rare case of extensive primary GTB in a 36-year-old male.

Keywords: *Assisted reproduction techniques, azoospermia, extrapulmonary, infertility, male genital tuberculosis*

Introduction

An estimated one-third of the world's population is infected with *Mycobacterium tuberculosis*.[1] Genital tuberculosis (GTB) often seen in middle-aged men with renal or pulmonary TB, and it is considered an unusual and severe form of extrapulmonary TB.[2] Clinical findings commonly include dysuria with sterile pyuria or a painless testicular mass. GTB is a result of primary reactivation of latent bacilli either in the epididymis or the prostate or by secondary spread from the already infected urinary organs. The epididymis and prostate are the most common involved organs affected primarily by a hematogenous mode of spread. Extensive destruction and fibrosis are the characteristics of GTB, thus an early diagnosis may prevent infertility and organ loss. Ultrasonography (USG) and USG-guided fine-needle aspiration cytology of testicular swelling may confirm the diagnosis of GTB.[3] Antitubercular chemotherapy is the first line of management for all forms of GTB, and a 6-month course is the standard of care. Here, we present a rare case of extensive primary GTB in a 36-year-old male.

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Case Report

A 36-year-old male presented with complaints of fever and painless swelling of the left scrotum with no superficial ulceration or sinus tracts. On palpation, the left scrotum was found to be nontender, hard, not attached to the overlying skin, and not transilluminable. Ultrasound demonstrated a hypoechoic lesion in the left testis with no vascularity, suggestive of abscess. There was adjacent pyocele with scrotal wall thickening, enlarged hypoechoic epididymis with increased vascularity – suggesting epididymitis, enlarged prostate with multiple hypoechoic lesions – suggesting prostatitis and enlarged hypoechoic left seminal vesicle with increased vascularity – suggesting seminal vesiculitis. A provisional diagnosis of extensive male GTB was made [Figure 1a-d]. Semen analysis demonstrated azoospermia at the time of diagnosis [Table 1]. The patient underwent left orchidectomy, and testicular

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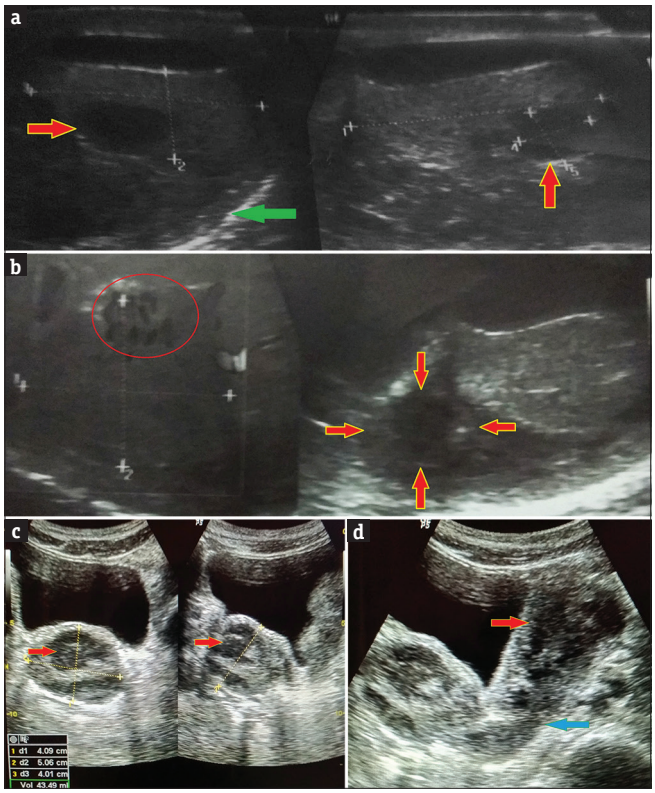

Figure 1: (a) Gray scale transverse and longitudinal sonograms demonstrating a well-defined intratesticular hypoechoic lesion with no internal vascularity (red arrows). Note the thickened scrotal wall (green arrow). (b) Gray scale and color sonograms of the scrotum demonstrating an enlarged hypoechoic epididymis (arrow) with markedly increased vascular flow (circle), consistent with epididymitis. (c) Gray scale transverse and longitudinal sonograms demonstrating an enlarged prostate with multiple hypoechoic lesions (arrows), consistent with granulomatous prostatitis in a proven case of genital tuberculosis from the orchidectomy specimen. (d) Gray scale longitudinal sonogram demonstrating an enlarged and hypoechoic left seminal vesicle (red arrow), consistent with seminal vesiculitis. Note the associated enlargement of the spermatic cord (blue arrow)

tissue showed stratified squamous within the stroma, several granulomas, and extensive caseous necrosis. The histological diagnosis was confirmed as TB [Figure 2]. The patient had no other site of TB, and findings of the chest computed tomography and renal ultrasound were normal. Postoperatively, the patient took anti-TB drugs (streptomycin, isoniazid, rifampin, and pyrazinamide). Urinalysis and semen analysis were normal, and scrotal USG showed a normal right epididymis and testis at 6- and 12-month follow-up [Table 2].

Discussion

In adults, TB accounts as the greatest infectious killers worldwide.[4] Eighty percent of male patients with TB are young men, in whom reproductive function is vital.[5] Nowadays, lethal character to TB is due to the emergence of multidrug-resistant and extremely drug-resistant strains.

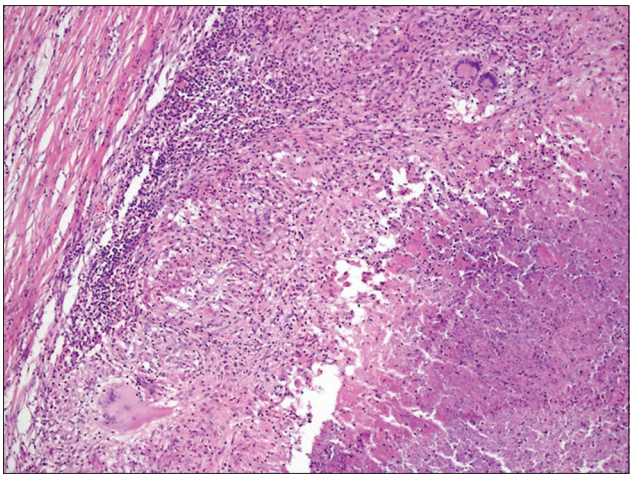

Figure 2: Histopathology image demonstrating testicular tissue with stratified squamous within the stroma, several granulomas, and extensive caseous necrosis (H and E, ×40)

Table 1: Semen analysis report at the time of diagnosis

Parameter	Patient value	Reference range
Count	0 Million/ml	Normal: 60-150 million/ml
Motility	15 Actively motile (%) 30 Sluggishly motile (%) 55 Nonmotile (%)	-
Abnormal forms	28 Percentage	Normal: Up to 20%
Spermatocytes	0-1/HPF	-
Pus cells	3-4/HPF	
RBCs	0-1/HPF	
Parasites	-	

RBCs=Red blood cells, HPF=High power field

Table 2: Semen analysis report at 6-month follow-up postantituberculosis treatment

Parameter	Patient value	Reference range
Count	66 Million/ml	Normal: 60-150 million/ml
Motility	60 Actively motile (%) 05 Sluggishly motile (%) 35 Nonmotile (%)	-
Abnormal forms	16 Percentage	Normal: Up to 20%
Spermatocytes	2-3/HPF	-
Pus cells	0-2/HPF	
RBC's	0-1/HPF	
Parasites	-	

RBC's=Red blood cells, HPF=High power field

In our case, seminal analysis demonstrated azoospermia at the time of diagnosis of male GTB with sonological findings of testicular abscess, epididymitis, prostatitis, and seminal vesiculitis. Subsequent to orchidectomy and anti-TB drug treatment for 6 months, follow-up seminal analysis report demonstrated improvement with a sperm count of 66 million/ml.

Genital TB is the third most frequent extrapulmonary tuberculous infection.[6] GTB in males may lead to infertility, thus reducing the quality of life. High index of suspicion for the in-time diagnosis of GTB is necessary to prevent genital complications. Isolated involvements of genital organs in TB were reported in 5%–30% of the cases.[7] GTB can involve any part of the reproductive tract, including the testis, epididymis, vas deferens, seminal vesicles, prostate, and the ejaculatory ducts. GTB is characterized by extensive destruction, fibrosis, and scarring of prostate and ejaculatory ducts, resulting in low seminal volume. Ejaculatory duct obstruction due to inflammation is a common cause of male infertility in 22%–50% of men.[8]

Hypoechoic enlargement of the epididymis, concomitant hypoechoic lesions in the testis with associated sinus tract, and extratesticular calcifications on USG are typical for tuberculous epididymitis and orchoepididymitis. However, testis is a rare site for tuberculous involvement due to the presence of a blood–testis barrier that may impede seeding of testicular parenchyma.[9] Testicular involvement usually occurs contiguous to the epididymal involvement. Tuberculous infection of the seminal vesicles or the prostate may be diffuse and result in aspermia without a demonstrable obstruction of the ejaculatory ducts. On sonography, proximal ductal system will not be dilated.

Infertility is an uncommon manifestation of genitourinary tract TB. Distortion of the normal anatomy by fibrosis and anatomic obstruction by granulomas is the most common cause of infertility. The diagnosis is usually based on a suggestive history along with evidence of granulomatous infection on a tissue sample. Fraietta *et al*.[10] described a case of aspermia as a complication of TB of seminal vesicles and Tzvetkov and Tzvetkova[11] have analyzed history cases of 69 male genital TB patients with infertility. TB of the genital tract results in calcification of the seminal vesicles, prostate, and the vas deferens.[12] Fibrotic and atrophic seminal vesicles with ejaculatory duct obstruction have been considered a diagnostic feature for TB. TB may even involve penile shaft and the glans penis and can result in ulcers, bulbous enlargement, and severe disfigurement.[13] The deformity precludes normal sexual intercourse and may result in subsequent infertility. Urethral strictures and urethrocutaneous fistulae may also prevent deposition of the semen during intercourse, leading to infertility.

Most other patients may be candidates for *in vitro* fertilization and have a prognosis similar to that in men with other causes of obstructive azoospermia.[14] Management of tuberculous infertility may be either medical or surgical. Medical management aims to treat infection with antitubercular drugs and may also restore fertility in patients who were diagnosed early and have not developed bulky granulomas resulting in obstruction. However, in majority of the cases, the presentation is late, and antitubercular treatment does not improve the fertility status. Infertility is the result of anatomic obstruction, and surgical management depends on the site of obstruction and feasibility of reconstruction and is helpful only in cases with discrete ejaculatory duct obstruction.

Conclusion

This case report emphasizes the meticulous analysis of imaging findings to ensure early diagnosis and complications of GTB to prevent infertility in a case of extensive male TB. GTB is a rare cause of male infertility, and its diagnosis is important not only for managing infertility but also for preventing systemic ramifications of the disease. Most cases of infertility complicated by GTB are not amenable to surgical correction and will require assisted reproduction techniques which provide the results comparable to those in men without this disease.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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