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Secondary Infertility Associated with Intra-testicular Schistosomiasis in an Endemic Area: A Case Report

Mohammed Abdulgabbar Noman [1]*, **Ebraheem Ali Ahmed Al-Nawd** [2] **and Faisal Ahmed** [3]

[1]**Department of Dermatology and Venereology, Faculty of Medicine and Health Sciences, Sana'a University, Sana'a, Yemen,**
[2]**Department of Biochemistry, Faculty of Laboratory Medicine, Jiblah University for Medical and Health Sciences, Ibb, Yemen,**
[3]**Department of Urology, Faculty of Medicine, Ibb University, Ibb, Yemen.**

*Corresponding author: E-mail: noaman670@gmail.com

Abstract

Background: Schistosomiasis remains a significant public health problem in low- and middle-income countries, including Yemen. Although the disease primarily affects the urinary and intestinal systems, involvement of the male genital tract—particularly the testes—is rare and often underrecognized. Testicular schistosomiasis can cause chronic inflammation and granulomatous damage that may lead to male infertility, including secondary infertility.

Case presentation: We report a case of a 30-year-old man with a ten-year history of secondary infertility and progressive deterioration of sperm parameters, culminating in azoospermia. Testicular histopathological examination revealed focal granulomatous inflammation with schistosome ova. Notably, clinical exam, imaging, and repeated stool, urine, and semen analyses showed no bilharzial ova

Conclusions: This case highlights the importance of considering testicular schistosomiasis in the differential diagnosis of male infertility in endemic areas, even when clinical and laboratory findings are unremarkable. Early recognition and treatment may prevent irreversible testicular damage and preserve fertility.

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INTRODUCTION

Schistosomiasis, commonly known as bilharziasis, is a waterborne parasitic disease affecting over 240 million individuals worldwide, with significant prevalence in regions such as Africa and the Middle East, including Yemen [1, 2]. The disease is caused by several species of the genus *Schistosoma*, primarily *S. mansoni*, *S. haematobium*, *S. japonicum*, *S. intercalatum*, and *S. mekongi*. These species can involve various organ systems, leading to a wide range of clinical manifestations [3].

While urogenital schistosomiasis is well documented, involvement of the male reproductive tract, particularly the testes, is rare and often underrecognized [4]. In endemic areas, this condition may be misdiagnosed due to clinical features that resemble sexually transmitted infections [3].

Reported symptoms include hematospermia, dysuria, painful ejaculation, erectile dysfunction, epididymitis, prostatitis, scrotal swelling, and infertility [3, 5–7]. Testicular schistosomiasis is infrequently described in the literature, largely due to its low incidence in this organ [4]. When present, it can clinically mimic a neoplastic lesion, often manifesting as a painless, small, solid testicular nodule.

Additionally, it has been linked to erectile dysfunction and impaired fertility in endemic populations. Severe cases may lead to granulomatous epididymitis and suppression of spermatogenesis, resulting in male infertility [3–5].

Schistosomiasis can impair fertility through multiple mechanisms, including direct gonadotoxic effects of granulomatous inflammation, disruption of the hypothalamic-pituitary-gonadal (HPG) axis, impairment of ejaculation or erectile function, and reduction in libido [8–10]. Diagnosis is challenging owing to nonspecific clinical and radiological findings; histopathological examination remains the definitive method for diagnosis in testicular involvement [2, 4].

In this report, we present the case of a 30-year-old Yemeni male with secondary infertility and progressively worsening sperm parameters, who was incidentally diagnosed with testicular schistosomiasis on histopathological examination despite unremarkable laboratory and ultrasonographic findings.

CASE PRESENTATION

A 30-year-old Yemeni farmer, who attended the Modern Reproduction and IVF Center in Sana'a, Yemen, presented with a 10-year history of secondary infertility following the birth of his daughter. Serial semen analyses showed a progressive decline from oligozoospermia to persistent azoospermia, confirmed over the preceding two years. The patient denied symptoms commonly associated with schistosomiasis, such as hematuria, hematospermia, or rectal bleeding. He reported no history of erectile dysfunction or significant animal exposure but resided in a schistosomiasis-endemic village in Yemen. He also denied systemic symptoms, including night fever, cough, headache, asthenia, rash, gastrointestinal complaints, or genitourinary symptoms such as dysuria or dyspareunia.

Physical local examination revealed bilateral medium-sized, non-tender testes with normal consistency and intact vasa deferentia. No clinical evidence of varicocele was detected. Abdominal examination showed no hepatosplenomegaly. Hormonal assays demonstrated mildly elevated serum follicle-stimulating hormone (FSH) at 13.1 IU/L (reference range: 1.5–12.4 IU/L) and luteinizing hormone (LH) at 9.8 IU/L (reference range: 1.7–8.6 IU/L), while total testosterone was within normal limits at 14.5 nmol/L (reference range: 8.6–29 nmol/L). Testicular ultrasonography revealed homogeneous medium echotexture testicular parenchyma without focal lesions, microlithiasis, or masses (**Fig.1.A**). Repeated urine and semen microscopic examinations were negative for schistosome ova.

In light of unexplained azoospermia, the patient underwent bilateral testicular sperm extraction (TESE). Few motile spermatozoa were retrieved exclusively from the left testis and cryopreserved to be used later on for Intracytoplasmic sperm injection (ICSI). Histopathological analysis of bilateral testicular biopsies showed disrupted seminiferous tubule architecture with sloughing of germinal epithelium, alongside focal granulomatous inflammation containing schistosome ova (**Fig.1 B–C**). Species identification was not feasible due to the localization of ova within the tissue sections.

Following the histopathological diagnosis, repeat stool, urine, and semen testing remained negative for bilharzial ova. The patient and his wife were counseled about ICSI as a fertility option, and he was prescribed weight-based praziquantel therapy to target schistosomiasis.

Written informed consent was obtained from the patient for publication of the clinical data. No patient identity information has been linked to this case report.

Figure.1 Incidental diagnosis of testicular schistosomiasis in an azoospermic patient.(A) Testicular ultrasonography demonstrating normal testicular echotexture without evidence of hypoechoic lesions or microlithiasis.(B) Testicular histopathology (hematoxylin and eosin stain, original magnification $\times$40) showing disrupted spermatogenesis (asterisk) accompanied by focal granulomatous inflammation (black arrow).(C) Higher magnification view of granulomatous lesion (H&E, $\times$200) revealing a schistosomal egg (black arrow); species identification was not possible due to the absence of visible spine morphology.

DISCUSSION

This case highlights the incidental diagnosis of testicular schistosomiasis in a patient presenting with secondary infertility and progressive deterioration of semen parameters despite normal clinical and ultrasonographic evaluations. The diagnosis was confirmed histopathologically by identification of schistosome ova within granulomatous formations in the testicular tissue. Importantly, routine investigations, including stool, urine, and semen analyses, were repeatedly negative for bilharzial ova, underscoring the diagnostic challenges encountered in such presentations.

Testicular involvement by schistosomiasis is exceptionally rare, with few cases reported worldwide [4, 9]. Its underrecognition stems from often asymptomatic or nonspecific manifestations that mimic other conditions, including testicular neoplasia or sexually transmitted infections [10]. This rarity makes the present case valuable for raising clinical awareness, particularly in endemic regions where schistosomiasis remains prevalent but is not commonly included in the differential diagnosis of male infertility [3, 9].

The pathogenesis involves deposition of schistosome eggs within the testicular tissue, eliciting a chronic granulomatous inflammatory response characterized by fibrosis and disruption of seminiferous tubules, thereby impairing spermatogenesis. This local immune reaction

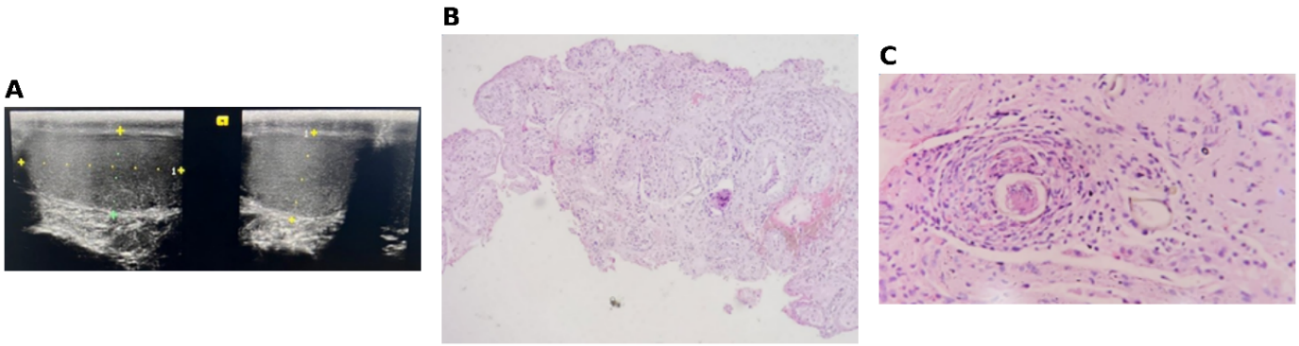

Figure 1

may compromise the blood-testis barrier, promoting immune cell infiltration and tissue damage. Moreover, schistosomal infection can alter the hypothalamic-pituitary-gonadal axis, leading to hormonal imbalances affecting reproductive function. Inflammatory and fibrotic changes may cause obstructive pathology impeding sperm transport and contribute to ejaculatory dysfunction. Systemic and local effects may reduce libido and erectile function. Emerging evidence suggests that schistosome-derived substances, such as estrogen-like metabolites, may exert endocrine-disrupting and genotoxic effects, further impairing fertility [1, 8–10]. Collectively, these multifactorial mechanisms contribute to the complex infertility observed in schistosomiasis.

Unlike most reported cases presenting with palpable masses, testicular pain, or radiological abnormalities, our patient's silent presentation is unusual. Literature often describes testicular nodules or epididymo-orchitis as initial clinical features [7, 9]. The incidental diagnosis here underscores the importance of considering schistosomiasis in the differential diagnosis of unexplained male infertility in endemic areas and supports the use of tissue biopsy when standard evaluations are inconclusive.

Scrotal ultrasonography demonstrated homogeneous testicular parenchyma without focal lesions or masses, consistent with the limited sensitivity of imaging modalities to detect schistosomal granulomas [3, 11]. Negative urine and semen examinations for schistosome ova reflect intermittent and low egg excretion in minimal parasitic burden cases. Definitive diagnosis was achieved by histopathological identification of granulomas containing schistosome ova, which remains the diagnostic gold standard for extraintestinal schistosomiasis [3, 5, 7, 12]. Conventional parasitological methods, such as Kato-Katz stool microscopy and urine filtration, offer high specificity but rely heavily on egg load and timing of sample collection, requiring multiple samplings [3]. Antigen detection assays and molecular diagnostics improve sensitivity in ova-negative cases but are often unavailable in resource-limited settings [6, 8, 9]. This case thus highlights the indispensable role of histopathology in diagnosis when non-invasive tests are negative.

Therapeutic management poses challenges. Praziquantel at 40 mg/kg remains first-line treatment, though evidence for its efficacy in reversing infertility is limited and not supported by randomized trials [3]. Sperm retrieval using TESE or micro-TESE followed by intracytoplasmic sperm injection (ICSI) offers the best reproductive outcomes in azoospermic patients, with live birth rates reported between 25–40% [3, 13]. Chronic granulomatous inflammation may cause irreversible testicular damage, underscoring the importance of early detection and treatment [5, 6, 9]. Multidisciplinary management involving infectious disease specialists and reproductive andrologists is essential in endemic regions. Further research is needed to establish optimal timing for assisted reproductive technologies post-treatment and to clarify praziquantel's effect on spermatogenesis recovery.

CONCLUSION

Schistosomiasis is an underrecognized cause of male infertility in endemic areas. Clinicians should maintain high suspicion for schistosomal involvement in patients with unexplained or progressive semen quality and quantity deterioration, especially when routine investigations are inconclusive. Early diagnosis through histopathological examination and prompt antiparasitic treatment may prevent irreversible testicular damage and improve fertility outcomes.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

Approval for the study protocol and all related procedures was granted by the Institutional Ethics Committee of The Modern Reproduction and IVF Center, Sana'a, Yemen (Reference number: 0141MN-2023, dated 13 May 2023). The study was conducted in accordance with the ethical standards of the institutional research committee and with the 1964 Helsinki Declaration and its later amendments. Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

CONSENT FOR PUBLICATION

Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

AVAILABILITY OF DATA AND MATERIALS

All data generated or analyzed during this study are included in this published article.

COMPETING INTERESTS

The authors declare that they have no competing interests.

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AUTHORS' CONTRIBUTIONS

All authors contributed equally to the conception, design, data acquisition, analysis, and interpretation of the study; participated in drafting and critically revising the manuscript; approved the final version for publication; agreed on the journal for submission; and accept responsibility for all aspects of the work.

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