LETTER TO THE EDITOR

A nonobstructive azoospermic patient with Trichomonas vaginalis infection in testes

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Dear Editor,

Trichomonas vaginalis (T. vaginalis), a flagellated protozoan parasite emerged as one of the most common nonviral sexually transmitted infections worldwide, often inhabits the vagina, urethra, prostate, and epididymis.1 It has been estimated that there are more than 170 million new cases of T. vaginalis infections per year worldwide. However, current knowledge of T. vaginalis and trichomoniasis is based mainly on studies in female vaginal infections. The prevalence of trichomoniasis in males is far less well characterized than that in females, probably because the infection seems to be asymptomatic in most men and can be resolved after treatment with one dose of metronidazole.1-4

Among men, trichomoniasis has been considered as a cause of nongonococcal urethritis (NGU) and as involvement in the impairment of male fertility.1,3 T. vaginalis is found more often in infertile men than that in fertile individuals and its presence in semen results in significant decreased sperm parameter values, such as motility, normal morphology and viability.4 In vitro studies have also shown that T. vaginalis and its secretory products reduce sperm motility and fertilizing capacity.4,5 Although T. vaginalis has been identified in urethral discharge, urine, semen, and prostatic fluid, its infection may occur in other areas of the urogenital system. In rare cases reported, T. vaginalis infects the epididymis and prostate gland and occasionally, the testis.6,7

Herein, we report a novel case of nonobstructive azoospermia (NOA) with T. vaginalis infection in the testis. A 32-year-old male patient (1.76 m height and 90 kg weight), married for 10 years, presented with the complaint of infertility. General physical examination was normal (1.76 m height and 90 kg weight), anemia, and hypertension (BMI = 29.05). Genetic analysis showed normal 46, XY karyotype and no microdeletion of Yq azoospermia factor gene. No evidence of gross structural pathology was identified according to the formal urological evaluation and the diagnosis of NOA was given. At the same time, vaginal secretions from his wife were tested to be positive for trichomonas.

Therefore, we attempted retrieval of the patient’s spermatozoa via surgical biopsy of the testes, which would be cryopreserved for further in vitro fertilization (IVF) treatment with his wife’s oocytes. Wet preparations from fresh testicular biopsies from four locations in his right testis were examined for sperm under phase contrast microscopy and the pathological examination was performed at the same time. Sections of the testicular biopsies showed that very few germ cells, appearing to be spermatogonia and spermatocytes, were scattered in the seminiferous tubules, while spermatids were rarely detected (Figure 1a). The pathological diagnosis suggested a severe disruption of spermatogenesis. Meanwhile, wet preparations of testicular biopsies failed to demonstrate any sperm cells. However, some flagellated motile protozoa among numerous testicular and red blood cells were observed in one of the wet preparations (Figure 1b and Supplementary Information). In an attempt to identify the protozoa-like structures, Wright-Giemsa staining was made on the same day. On the basis of the morphological features of the cells, namely, an amoeboïd shape, the presence of one elliptically shaped nucleus and poorly defined cytoplasm (Figure 1c), a provisional identification of T. vaginalis parasites was made.

Furthermore, laboratory PCR analysis was notable for the presence of the parasite. Briefly, genomic DNA was prepared from 1.5 ml of semen or 5 ml of urine using DNA extract kit (Omega Bio-Tek, Norcross, GA, USA) according to the manufacturer’s instructions. PCR was performed to check the expression of Tvk and BTUB, according to the procedure described previously.8-10

Table 1: Hormonal profile of the patient

| Hormonal profile                  | Value | Normal range |
|----------------------------------|-------|--------------|
| Follicle-stimulating hormone (mIU ml−1) | 12.2  | 1.5–12.5     |
| Luteinizing hormone (mIU ml−1)     | 5.5   | 1.7–8.6      |
| Prolactin (ng ml−1)               | 4.9   | 4.8–23.3     |
| Estrogen 2 (pg ml−1)              | 53.7  | 7.6–42.6     |
| Testosterone (ng ml−1)            | 1.8   | 2.8–8.0      |

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The results of PCR showed approximately 261 bp fragments representing Tkv 3/7, which has previously been shown to be the most sensitive conventional PCR test for T. vaginalis, in the DNA extracts of both the semen and the urine. Likewise, results also showed an approximately 112 bp fragment representing BTUB 9/2, although a bit weak, in the DNA extract of the semen (Figure 1d). Therefore, both semen and urine from the patient were considered positive for T. vaginalis, and the NOA symptom may be caused by T. vaginalis-induced orchitis.

To our knowledge, this clinical case represents the first report of NOA related to T. vaginalis infection at the level of the testis. Combined with the existing reports, it illustrates that spermaticogenesis failure resulting from T. vaginalis infection in testis may be accompanied by low serum testosterone and atrophic testes, which may reflect the cytotoxicity of T. vaginalis in damaging germ cells and Leydig cells. Therefore, T. vaginalis infection in testis, although occasional, can seriously injure the niche essential for spermatogenesis. Meanwhile, male trichomoniasis is almost asymptomatic and few cases are diagnosed and treated. Hence, the infection persists, and males with male trichomoniasis may develop male infertility with serious harm to the reproductive system. Above all, it is important to improve the microenvironment of the urogenital tract in defending against pathogens during the therapy for trichomoniasis.

**AUTHOR CONTRIBUTIONS**

CX and ZL designed the experiment. YHG and YL performed the experimental work and participated in the pathological work. PL, ZJZ, and YH provided assistance in sample collection and treatment. GHF and YJX participated in the pathological work. All authors read and approved the final manuscript.

**COMPETING INTERESTS**

All authors declared no competing interests.

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Supplementary information is linked to the online version of the paper on the Asian Journal of Andrology website.

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