CASE REPORT

Microsurgical epididymal sperm aspiration with motile trophozoite cells but no spermatozoa

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This paper reports on a patient in whom the clinical diagnosis of obstructive azoosperma was made according to clinical observations, i.e. azoospermia, normal andrological examination, normal follicle stimulating hormone and a misleading histopathological report of a testicular biopsy. Microsurgical vasoepididymostomy failed to restore fertility, and as a last resort, microsurgical sperm aspiration was performed. Although flagellated cells were observed in the epididymal aspiration, no spermatozoa were observed and wet preparation of multiple testicular biopsies failed to demonstrate any spermatozoont. This patient was diagnosed to have a non-obstructive azoosperma, resulting from maturation arrest associated with trichomonas infection at the level of the epididymis.

Key words: azoospermia/epididymitis/infection/male infertility/trichomonas

Introduction

With the introduction of intracytoplasmic sperm injection (ICSI) (Palermo et al., 1992), virtually any form of obstructive azoospermia can be treated either by microsurgery (Silber, 1989) or by ICSI using epididymal or testicular spermatozoa (Schoysman et al., 1993; Tournaye et al., 1994). Furthermore, in about half of the patients showing azoospermia because of deficient spermatogenesis, testicular spermatozoa may also be recovered for ICSI. The differentiation between obstructive and non-obstructive azoospermia can be made correctly only on the basis of a histopathological report. Indeed, neither follicle stimulating hormone (FSH) nor testicular volume can accurately predict normal spermatogenesis (Tournaye et al., 1995). The present paper reports on a patient with a clinical diagnosis of obstructive azoospermia but for whom finally ICSI could not be performed.

Case report

A 34 year old patient presented with azoospermia. He and his spouse had been trying to have children for the previous 8 years. His spouse had an uneventful history and a normal fertility work-up including gynaecological examination, pelvic ultrasonography and endocrine profile. The patient had neither erectile nor ejaculatory problems. His general physical examination was normal. The andrological examination showed a normal penis, testes with a volume of 20 ml, a normal epididymis and a palpable vas deferens at both sides. He showed neither a clinical nor a subclinical varicocele. His semen sample had a volume of 2 ml and a pH of 7.7. Fructose was normal (2766 g per ml). There was a total absence of any spermatozoa, even after centrifugation. His endocrine profile showed a serum FSH value of 9.4 IU/l (normal range 1.5–12 IU/l) and a luteinizing hormone (LH) value of 8.1 IU/l (normal values 0.6–13.5 IU/l). His serum testosterone was 6.3 µg/l (normal range 2.7–10.0 µg/l). His peripheral blood karyotype was 46, XY. In another university-based fertility centre a testicular biopsy had been performed under general anaesthesia which showed normal spermatogenesis. With a final diagnosis of obstructive azoospermia the patient was scheduled for microsurgery 5 months later. A peroperatively performed vasography showed patency up to the level of the seminal vesicles, but a stop at the level of the distal end of the epididymis. Epididymal exploration was performed on one side only (right side); however, no spermatozoa were observed. Nevertheless, it was decided to perform a unilateral microsurgical anastomosis between the ductus deferens and a rete-testis tubulus. The intervention was uneventful. After this microsurgical intervention azoospermia persisted. The couple eventually decided to undergo treatment with insemination with donor sperm. After seven insemination cycles, they decided to abandon this treatment in favour of assisted reproductive techniques (ART) using the husband’s spermatozoa. They were referred to our centre for intracytoplasmic sperm injection (ICSI) with either epididymal or testicular spermatozoa for ICSI. The differentiation between obstructive and non-obstructive azoospermia can be made correctly only on the basis of a histopathological report. Indeed, neither follicle stimulating hormone (FSH) nor testicular volume can accurately predict normal spermatogenesis (Tournaye et al., 1995). The present paper reports on a patient with a clinical diagnosis of obstructive azoospermia but for whom finally ICSI could not be performed.

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zoo. Although his general physical examination and andrological examination were unchanged, his serum FSH level was now 13.3 IU/l. Because of his previous history and work-up, the patient and his spouse were scheduled for ICSI with epididymal or testicular spermatozoa. On the day of ovum retrieval, the husband had a scrotal exploration under general anaesthesia. After microsurgical epididymal sperm aspiration, motile cells were observed on peroperative microscopical examination at ×400. Since the motile cells observed in the epididymal aspirates were not readily recognized as spermatozoa, testicular biopsies were taken for wet preparation. After mincing using two glass slides in order to break the seminiferous tubules, the cell suspension was centrifuged at 500 g for 5 min and the preparation was checked at ×400 (Verheyen et al., 1995). On the contralateral side also, microsurgical epididymal sperm aspiration failed to recover spermatozoa. A total of 12 testicular specimens was taken from both testes, but no spermatozoa were observed in the wet preparations. Specimens were sent for microbiological and histopathological examination. The histopathology of the testicular biopsies showed a maturation arrest at the spermatocyte level on both sides. Only a few seminiferous tubules contained some rare spermatids, but no spermatozoa were observed. The microbiological examination of the epididymal aspirates showed the presence of a trichomonas species. Trophozoites of *Trichomonas vaginalis* were seen by low power direct microscopic examination (magnification ×100) and identified by high power microscopy (magnification ×400). Both the patient and his partner received antibiotics and eventually they decided to continue ART with donor spermatozoa. After the final diagnosis of azoospermia because of maturation arrest, the patient was screened for microdeletions of the Y-chromosome (Reijo et al., 1995) and was found to have no deletion.

**Discussion**

This paper reports on a rather unusual case of azoospermia. Although the diagnosis of obstructive azoospermia was made both from the clinical picture and from preliminary histopathology, this patient turned out to have azoospermia because of primary testicular failure. It is probable that in this case a mistake was made in analysing or reporting the preliminary histopathology. This may explain the failure to observe any spermatozoa during the microsurgical exploration and perhaps even the microsurgical failure. It is unlikely that testicular failure resulted from an infection arising as a result of the preliminary biopsy: the patient did not remember any complications and this biopsy was performed on one side only. This case clearly illustrates the importance of careful assessment of azoospermia before, but also during, any surgery. No causative element was present in this patient’s history to suggest obstruction. At physical examination no epididymal induration or swelling was noted. The key elements for assuming obstruction were the normal FSH, the normal testicular volumes and finally the misleading histopathological report. The failure of microsurgery is not surprising in itself, since only sporadic successes have been reported after vas-to-rete testis re-anastomosis (Silber, 1988). But this case demonstrates that there is an important role for peroperative wet preparation of a testis biopsy whenever spermatozoa are not observed in the epididymal fluid during male microsurgery. Since the final histopathology of the testicular biopsies showed the presence of some rare spermatids, the couple could have been treated by ICSI with spermatids (Fishel et al., 1995). However, at that time we decided not to attempt this approach because the couple had not received appropriate counselling and because the safety of this technique has not been verified (Tournaye, 1996). The role of chronic infection by trichomonas on male infertility is unknown. Sexually transmitted diseases have been shown to affect fertility negatively, possibly by causing obstruction of the secretory ducts or by damaging the seminiferous tubules and eventually leading to complete testicular atrophy. Trichomonas is a sexually transmitted protozoan parasite that inhabits the human genital tract and is considered to be strictly limited to the lower urogenital tract in men (Csonska, 1989). The reported incidence of trichomonas in the human male population is stated to vary between 8.9 and 18% (Hager, 1994). Trichomonas is a motile, ovoid, flagellated parasitic protozoan with a nucleus near the flagellum. The organism has been noted to be responsible for a variety of clinical manifestations including urethritis (Lewis et al., 1981). Very little has been reported about other clinical manifestation in men. Only few studies report on the role of trichomonas in male infertility and the issue is a matter of debate (Moskowitz and Mellinger 1992; Verges 1979). In the present case report, the patient was found to be infected with trichomonas at the level of the epididymis. The characteristic of the semen in terms of viscosity, sperm motility and vitality are reported to be affected in infected samples. Hynie et al. proposed in 1960 that toxic secretions from trichomonas may affect fertility. Tuttle et al. (1977) showed that sperm motility decreased when spermatozoa were exposed *in vitro* to trichomonas. Recently Gopalkrishnan et al. (1990) compared the characteristics of asymptomatic males affected by trichomonas, and showed an increased viscosity, decreased motility and a lower vitality in the infected group. To our knowledge, this is the first report of the presence of trichomonas at the level of the epididymis in an asymptomatic man with non-obstructive azoospermia. It remains unclear whether the infection by trichomonas was causative, associated with another infection of the genital tract causing testicular failure, or was just coincidental. This case also illustrates the importance of careful assessment when MESA is performed. The presence of moving flagella is not always a sign of the presence of sperm cells.

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**References**


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