Research Article

Role of Trans Rectal Ultrasonography for Evaluation of Male Infertility With Low Semen Volume

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Abstract

Background: Infertility affects an estimated 15% of couples globally, amounting to 48.5 million couples. Males are found to be solely responsible for 20-30% of infertility cases and contribute to 50% of cases overall, the aim is to evaluate the role of trans rectal ultrasonography for evaluation of male infertility with low semen volume. **Methods:** This study was carried out on one hundred and twenty patients attended the outpatient clinic of the diagnostic radiology department at Al-Azhar University Hospital (Assiut) All patients were subjected to the following: Full history taking and thorough clinical examination, Physical examination including genital and digital rectal examination (DRE). Routine laboratory Semen analysis serum follicle stimulating hormone (FSH), testosterone, leutinizing hormone (LH) and prolactin hormone (PRL). Radiological investigations including: Transrectal ultrasound (TRUS). Scrotal ultrasound. **Results:** According to TRUS finding, about 7.5% of patients were normal and 92.5% of patients had abnormal findings. With reference to abnormal TRUS findings, 9.9% of patients had hypoplastic seminal vesicle, 24.3% had dilated seminal vesicle and ejaculatory duct. 5.4% of patients had prostatic midline cyst and the same percentage of patients had prostatic calcification. Varicocele was left sided in 36% of patients and bilateral in 18.9% of patients. **Conclusion**: In transrectal and scrotal ultrasonography are important non invasive diagnostic tools that minimizes the need for more invasive studies in the evaluation of azoospermia or severe oligospermia with low volume ejaculate and can give more information for patients with abnormal testicular biopsy findings.

Keywords: Infertility, azoospermia, ultrasonography, Varicocele

Introduction

Infertility affects an estimated 15% of couples globally, amounting to 48.5 million couples. Males are found to be solely responsible for 20-30% of infertility cases and contribute to 50% of cases overall [(1)]**.** Hypospermia is a condition in which a man has an unusually low ejaculate (or semen) volume, less than 1.5 ml[(2)]. However, Azospermia is defined as the absence of spermatozoa in the semen. (Abdelwahed et al., 2013)[(3)]. Approximately 1% of all men in the general population suffer from azoospermia, and azoospermic men constitute approximately 10 to 15% of all infertile men (Gudeloglu and Parekattil, 2013)[(4)]. The etiologic classification of azoospermia is divided into three primary categories: pretesticular, testicular, and post-testicular. Although the pretesticular and post-testicular causes of azoospermia are generally curable, the testicular causes of azoospermia are generally not (Jungwirth et al., 2012)[(5)].

Methods

This study was carried out on one hundred and twenty patients attended the outpatient clinic of the diagnostic radiology department at Al-Azhar University Hospital (Assiut) between October 2017 and October 2018. All patients were subjected to the following:Full history taking and thorough clinical examination, Physical examination including genital and digital rectal examination (DRE). Routine laboratory Semen analysis (performed by ferti scan CASA pro) 3 analysis separated by one month a part. Hormonal assessment (performed by VIDAS® 30: Healthcare) serum follicle stimulating hormone (FSH), testosterone, leutinizing hormone (LH) and prolactin hormone (PRL). Radiological investigations including: Transrectal ultrasound (TRUS) to assess for obstruction of the ejaculatory duct and the absence or hypoplasia of the seminal vesicles and ejaculatory ducts.

Scrotal ultrasound for assessment of: Testicular volume. Pampiniform plexus of veins. Review any anatomical abnormality as hypoechoic or hyperechoic lesions of the testes.Association of Duplex flow study on testicular veins at rest and during valsalva for diagnosis of varicocele.

Table (2) Demographic characteristics of the studied patients (n=120).

Age (Years):	
(Range) Mean ± SD	(17-63) 30.50 ± 7.8
Residence: n (%)	
Rural	78 (65)
Urban	42 (35)

Discussion

Infertile men with a reduced ejaculate volume have either ejaculatory dysfunction, congenital anomalies of the accessory sex organs or ejaculatory duct obstruction (Worischeck and Parra, 1993).8 The differentiation can be made based on post-ejaculatory urine analysis and transrectal ultrasonography. Previously, vasography had been used as the definitive method of determining ductal patency but the risks of anesthesia, radiation exposure and post-operative vasal obstruction inherent with this procedure cannot be ignored. Transrectal ultrasound is essentially risk-free and inexpensive, and it has proved reliable in the diagnostic scheme of the infertile patient (Yalcin and Yildirim, 2004)9.

Patients with a demonstrable distal ductal abnormality represent only a small fraction (1-2%) of all men who come to medical attention because of infertility. It is crucial, however, to identify those patients with a potentially correctable cause of infertility, to determine appropriate treatment for patients with correctable defects, and to preclude further unnecessary investigation and intervention in patients with clearly non correctable abnormalities, who are best treated with epididymal sperm aspiration and invitro fertilization. In the absence of retrograde ejaculation, neurologic dysfunction, and diabetes mellitus (D.M), patients with azoospermia or severe oligospermia with low ejaculate volumes (<1.5ml) should be examined for congenital defects or obstructive disorders of the distal genital tract. Such abnormalities include absence or hypoplasia of the vas deferens, seminal vesicles and ejaculatory ducts and distal obstruction by fibrosis, calcification, calculi or cysts. Methods for imaging the distal male reproductive tract include vasography, magnetic resonance (MR) imaging and transrectal ultrasound. MR imaging with endorectal coils can clearly demonstrate the distal ductal system and the results are less operator dependent than those of TRUS.

However, MR imaging rarely provides information that cannot be obtained by means of a thorough TRUS evaluation, and its use is limited by cost and availability (Kuligowska and Fenlon, 1998).10 The finding of dilated seminal vesicles with or without dilatation of the ejaculatory ducts established the diagnosis of ejaculatory duct obstruction that was found in 14 patients in this study and seminal vesicle hypoplasia, aplasia or atrophy in 6 patients. Our results agree with the results of Yalcin and Yildirim, 2004.9 They studied 50 patients and found ejaculatory duct obstruction in 14 patients, seminal vesicles dilatation in 12 patients and seminal vesicle hypoplasia, aplasia or atrophy in 6 patients and these findings were bilateral. Also in our study there was high prevalence of primary infertility versus secondary infertility.

Also Worischeck and Parra, 1993)8 studied 25 infertile male patients using TRUS and found ejaculatory duct obstruction in 5 cases, seminal vesicle dilatation in 3 and seminal vesicle aplasia in 2 and these results are comparable with our results.Carter and Coworkers (1989)11 documented the presence of isolated seminal vesicle anomalies in 30 of 85 infertile men with

low volume azoospermia or severe oligospermia. TRUS identified 12 patients with bilaterally absent and 18 with unilaterally absent seminal vesicles. Twenty-five patients had atrophic seminal vesicles, in six was a bilateral condition. Of 40 ductal systems explored and proved to have absent or atretic vasa, TRUS defined 21 as having absent seminal vesicles and the remainder to be atrophic (8), thin (4), dilated (4), and normal (3). In our study the cause of ejaculatory duct obstruction was mullerian duct cyst in 2 cases and calcification in 2 cases, the remaining 10 cases shows the appearance of distal ductal stenosis. Worischeck and Parra (1993)[8] reported the cause of ejaculatory duct obstruction was ductal calculi in 1 case, mullerian duct cyst in 1 case and distal ductal stenosis in 3 cases.

However, Yalcin and Yildirim (2004)[9] reported that the exact cause of ejaculatory duct obstruction was ductal calculi in 3 patients, ductal cyst in 4 patients and distal ductal stenosis in 7 cases, that may be due to inflammation by venereal diseases or genitourinary tuberculosis. To confirm the diagnosis of ejaculatory duct obstruction a less invasive alternative, seminal vesicle aspiration is used and can be performed under TRUS guidance. The finding of numerous sperms in the seminal vesicles confirms the presence of ejaculatory duct obstruction in the azoospermic patient and rules out concomitant ipsilateral epididmal obstruction. In addition methylene blue can be injected into the seminal vesicles after aspiration to assist in identifying the level of obstruction and the adequacy of the resection during surgical therapy.

The distally obstructed ejaculatory duct appears clearly on sagittal and transverse sonogram images as a hypoechoic tubular structure (Yalcin and Yildirim, 2004). Yalcin and Yildirim (2004), demonstrated that atrophic seminal vesicles and aplasia or hypoplasia were found in six patients. Because of the 80% incidence of ipsilateral renal agenesis reported to be presented in this group of patients (Yalcin and Yildirim, 2004), a concomitant renal sonogram was performed to rule out such an anomaly. No additional pathology was seen. Our results comparable with Yalcin and Yildirim, 2004 as seminal vesicles hypoplasia aplasia or atrophy were found in 6 patients (9.4%). Also a concomitant renal sonogram was performed and there was one case of renal agenesis and in the other 5 cases, no additional renal pathology was seen. In these patients surgical reconstruction was unlikely to be successful and after documentation with TRUS, epididymal aspiration and ICSI were offered to the patients. Ejaculatory duct obstruction is a rare but surgically correctable cause of male infertility.

Although there are no pathognomonic findings associated with ejaculatory duct obstruction, the diagnosis should be suspected in an infertile male with oligospermia or azoospermia with low ejaculate volume, normal secondary sex characteristics, testes, and hormonal profile, and dilated seminal vesicles, midline cyst, or calcifications on TRUS (Fish et al., 2002). Zakarisson et al., (2000) reported that the mean width of both Rt and Lt normal seminal vesicles was 10.6 $\pm$ 2.9mm. Our study showed that the mean width of normal Rt and Lt seminal vesicles were 8.5$\pm$2.4 mm and 7.6$\pm$2.7mm respectively. Dimensions of the mean width of the dilated Rt and Lt seminal vesicles were 25$\pm$4.8mm and 20.3 $\pm$ 3.2mm respectively also dimensions of the mean width of the hypoplastic Rt and Lt seminal vesicles were 2.7 $\pm$ 0.6 mm and 2.7 $\pm$ 0.9mm respectively. These findings are unique to our study as no other study in the literature up to our knowledge has measured the mean size of the hypoplastic and dilated seminal vesicles. Pierik et al., (1999) reported that the prevalence of scrotal abnormalities in infertile patients detected by scrotal colour Doppler ultrasonography in 38% of the infertile men. Examination of the scrotal contents with color Doppler ultrasonography was primarily performed for detection of varicocele.

Varicocele was the most frequent finding and was noted in 33% of the infertile patients. These varicocele were generally on the Lt side and bilateral in 11% of these case. Our results are comparable with the results of Pierik et al., 1999 as varicocele was the most common prevalent lesion. Varicocele prevalence was 50% and it was prevalent on the Lt side and bilateral in 12%.

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