Evaluation and treatment of ejaculatory duct obstruction in infertile men

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Objectives: *The purpose of this review is to survey the growing literature on ejaculatory duct obstruction and to provide a comprehensive overview of the current trends in the diagnosis and management of patients with this disorder.*
Materials and methods: *Review of current literature on ejaculatory duct obstruction.*
Results: *Ejaculatory duct obstruction is a surgically treatable cause of male infertility. It is usually suspected in an infertile male who has low ejaculate semen volume but normal testicular size and normal secondary male sexual characteristics.*
Conclusion: *Transrectal ultrasonography is the initial investigation method used to visualize and locate the presence of a cyst or calcifications that may contribute to the obstruction. Transurethral resection of the ejaculatory ducts represents the best treatment modality, resulting in marked improvement in the semen parameters and pregnancy rate in well-selected cases.*

Key Words: male infertility, ejaculatory duct obstruction, low volume azoospermia, TURED

Introduction

Male factor infertility has been implicated in approximately 50% of all cases of infertility, which affects 15% of the population.[1] Ejaculatory duct obstruction (EDO) is found to be the cause in 1 to 5 % of infertile men.[2] Men with bilateral complete EDO will typically present with low semen volume, and fructose-negative, acidic semen. However, patients with partial EDO may have a more variable presentation of semen parameters.[3] Together, partial and complete EDO represent treatable abnormalities that affect a measurable proportion of patients with male factor infertility.

In the following review, we discuss the anatomy of the ejaculatory duct system with correlation to clinical presentation, semen analysis, and radiological and pathologic findings in patients with EDO. We also present recent trends in its diagnosis and management, to provide a better understanding of EDO and its impact on male infertility.

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Embryology and anatomy

As the male fetus develops, the Mullerian ducts normally disappear from above downwards under the influence of Mullerian inhibitory factor produced by Sertoli cells in the primitive testis. This process is usually completed by the end of the 7th week of gestation. Failure of complete absorption may leave a small Mullerian duct remnant at the lower end, which manifests as Mullerian duct cyst in the adult male. This process is followed by the transformation of the Wolffian duct into the male reproductive tract. The upper segment becomes the epididymis; the middle portion, the vas deferens; and the terminal area, the ejaculatory ducts and seminal vesicles, by day 84 of gestation.[4]

The ejaculatory ducts represent a direct continuation of the seminal vesicles that begins after the ampulla of the vas deferens joins the seminal vesicle duct on its medial aspect.[5] The ducts are approximately 1-2 cm long and enter the prostate

obliquely and posterior at the base. They then course medially and anteriorly through the prostatic glandular tissue, between the middle and the lateral lobes, ending in the prostatic urethra at the verumontanum by slit-like orifices. The ducts diminish in size and also converge towards their termination.

Etiologies of obstruction

The etiology of EDO can be classified into two major categories: congenital and acquired. Congenital causes include congenital atresia or stenosis of the ejaculatory ducts, midline cysts, and Wolffian duct anomalies. Midline cysts are classified into two general categories: those that contain sperm and those that do not contain it.[6] The cysts that do not contain sperm are the Mullerian duct cysts and the utricular cysts. The difference between them is in the embryological origin: the utricular cysts are of endodermal origin and the Mullerian duct cysts are of mesodermal origin. Both contribute to EDO by compressing the orifices of the ejaculatory ducts.[7]

Cysts that contain sperm - Wolffian duct cysts or diverticulae - are less common than the Mullerian duct cysts.[8] Wolffian duct anomalies may be either sporadic, with a localized defect in the proximal part of the vas deferens, or a more generalized maldevelopment due to a systemic genetic abnormality. The latter is usually bilateral and is often associated with carrying the cystic fibrosis gene mutation.[9] Local Wolffian duct abnormality involves loss of a variable amount of the vas deferens, seminal vesicle, and ejaculatory duct, and, sometimes, also parts of the ipsilateral urinary system.

Acquired causes of EDO usually involve cicatrization and obstruction. These include previous surgical trauma such as from bladder-neck incision or transurethral resection of the prostate (TURP);[2] or previous genital infection such as gonorrhea, non-specific urethritis, schistosomiasis in endemic areas, and genitourinary tuberculosis.[10-12] Ejaculatory-duct stones are usually associated with infection but rarely require intervention. Finally, diseases of the prostate such as chronic prostatitis and carcinoma of the prostate may also lead to EDO.

When to suspect EDO in a patient?

The presentation of EDO varies according to type and etiology of the obstruction. Common complaints associated with ejaculatory obstruction include male infertility, decreased volume and force of ejaculate, pain during or after ejaculation, hematospermia, perineal, testicular, or lower-back pain.[13,14] Symptoms are generally less pronounced or absent in patients with partial obstruction. Although physical examinations findings are usually normal in patients suspected of having EDO, there may be a palpable seminal vesicle or mass on rectal examination, with or without prostate tenderness.[13,14]

Complete bilateral EDO leads to only prostatic fluid contribution to the ejaculate. The prostatic fluid ejaculate is typically 0.5 ml-1.0 ml with a watery appearance and an acidic pH (6.5). Both fructose and the seminal coagulum are absent in the ejaculate, as they are produced by the seminal vesicles. Therefore, men with palpable vasa deferentia, and low–volume, acidic semen should be evaluated for EDO.

Since the clinical presentation of partial EDO is highly variable, the indications for evaluation are somewhat controversial. Previously published indications include low ejaculate volume (less than 1.5 ml), very poor sperm motility (less than 30%) or oligospermia (less than 20 million sperm/ml) in the presence of normal testicular volume (greater than 18 ml), normal serum testosterone level, and failure to respond to varicocelectomy.[15]

Evaluation

The optimal methods for the evaluation of patients with suspected EDO are poorly defined. Historically, vasography was the gold standard method for the diagnosis of proximal and distal obstruction. However its invasive nature with risks of iatrogenic stricture and anesthesia has made transrectal ultrasound (TRUS) a more attractive diagnostic modality.[16]

High-resolution TRUS is a minimally invasive procedure to visualize the prostate, ejaculatory ducts, vas deferens and seminal vesicles, and to detect the presence and level of EDO. An obstructed ejaculatory duct may have either anechoic or highly echogenic material within it, Figure 1. It has been previously proposed that an ejaculatory duct lumen of greater than 2 mm is pathological; however, some investigators believe the upper limit of normal should be 1.2 mm.[17]

Multiple attempts have been made to characterize the appearance of the seminal vesicles on TRUS in the presence of EDO. Enlargement of the seminal vesicles is considered when the anterior-posterior dimension exceeds 1.5 cm.[18] However, no universal criteria can be used to define obstructed seminal vesicles seen from TRUS imaging alone. Dilatation of the vas deferens, defined as the visualization of an anechoic fluid or hyperechoic material within the lumen, strongly suggests obstruction of the vas

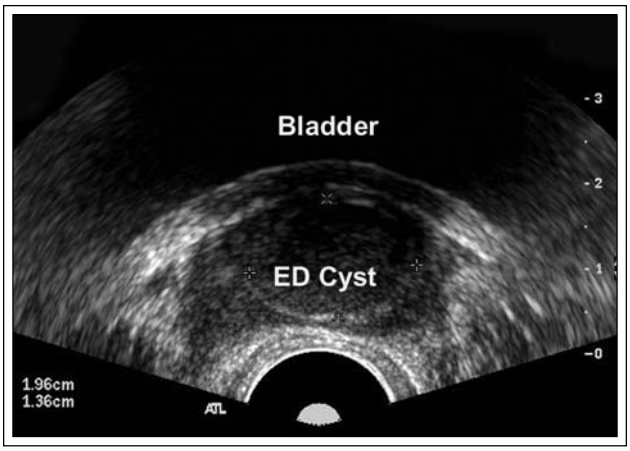

Figure 1. Ejaculatory duct cyst visualized by high resolution transrectal ultrasonography.

deferens, which could be an additional finding in patients with EDO.

Despite the potential risks of vasography, it is still performed mainly due to the low specificity of TRUS. However, there is no consensus among experts regarding its exact indication. Vasography can be performed with an open vasotomy, which is done through a small scrotal incision, or by blind cannulation of the vas deferens. The latter technique is difficult and can result in blockage of the vas deferens due to leakage of the sperm and granuloma formation, perivasal or submucosal injection of dye, and perivasal bleeding.

TRUS-guided seminal vesicle aspiration has proved to be useful in the diagnosis of EDO when sperms are found in the aspirate within 24 hours of ejaculation. This technique can also be used to collect sperm for assisted reproductive techniques, and can be combined with the injection of contrast media for vesiculography.[19,20]

Chromotubation of the ejaculatory ducts or TRUS-guided seminal vesicle chromotubation has also been described in the evaluation of patients with EDO. The technique involves the injection of dye such as indigo carmine or methylene blue, then observation for the release dye from the ejaculatory ducts cystoscopically. EDO is suspected when no dye is seen effluxing from the ejaculatory ducts. This procedure may also serve to guide the EDO resection until the expression of dye from the ejaculatory ducts.[21]

Treatment

The standard treatment of EDO involves transurethral resection of the ejaculatory ducts (TURED).[22] Before the TUR of the presumed obstructed ejaculatory duct in an azoospermic man, it is important to document the presence of normal sperm production in the testes. This is can be done via a testicular biopsy and confirmatory wet prep. TURED can be done with concomitant TRUS for the identification of ejaculatory duct cysts, chromotubation, simultaneous vasotomy and vasography, or without any adjunctive guides. Vasotomy/vasography is the most invasive procedure among the options, and the potential risks described previously must be considered and avoided. In addition, the surgeon should have the capacity to perform vasovasostomy or epididymovasostomy in the event that a proximal obstruction is discovered.

The TURED procedure involves the resection of the prostatic side of the verumontanum using only cutting current. If a Mullerian duct cyst is present in the midline, additional resection is performed just proximal to the verumontanum to completely "unroof" the cyst. If not, the resection of the ejaculatory ducts is carried out until a dilated region is seen.[23]

The typical infertile male is young and has a small prostate; therefore, resection is carried out in proximity to the rectum, urethral sphincter, and bladder neck. A simultaneous TRUS guidance during the TURED is ideal to minimize the risk of injury to the above-mentioned regions. If radiological guidance is not available, an O'Connor sheath can be placed in the rectum to digitally assess the proximity of resection to the rectum. If bleeding is encountered during the procedure, haemostasis is achieved with a minimum of cauterization to avoid stricturing the ejaculatory ducts.

Complications of the TURED may be severe and significant; therefore, proper attention to resection of only the ejaculatory ducts must be sought. The most common complications are listed in Table 1.

Balloon dilatation is an alternative minimally-invasive approach that utilizes minimum resection

TABLE 1. **Potential complications from the transurethral resection of ejaculatory duct**

Impairment of semen parameters
Retrograde ejaculation
Urinary reflux into the ejaculatory ducts
Postoperative bleeding
Rectourethral fistula
Incontinence (sphincter injury)
Bladder-neck contracture
Erectile dysfunction

until direct access to the ducts is obtained. The obstructed ejaculatory ducts are then cannulated and dilated to 4 mm using a 2-cm long catheter.[15]

Results of treatment

Overall, about 50% of azoospermic patients who undergo surgical reconstruction will have sperm in the ejaculate post operatively, and half of them will achieve a pregnancy with their partners.[13] Several factors may contribute to the failures: restricturing of the ejaculatory ducts, presence of obstruction in the epididymis or vas deferens, or the poor quality of sperm after prolonged obstruction. Another anatomical factor is that after resection, the prostatic urethra may become widened and fill with urine; hence, with emission, the semen is mixed with the urine, which can adversely affect the semen quality.

Given the potential failure from the treatment, options of cryopreserving retrieved sperm at the time of reconstruction should be discussed with the patient prior to the procedure.

Summary

EDO diagnosis and treatment represents one of the bench-marks of progress in the field of male reproductive medicine. The presence of obstruction can be diagnosed from patient's history, semen analysis, and simple, non-invasive imaging. Careful discussion with patients about the treatment options, risks, and realistic expectations is essential. With the available treatments, infertile couples can achieve spontaneous pregnancy, and in some instances, avoid costly and emotionally draining procedures such as in-vitro fertilization or intracytoplasmic sperm injection. □

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