

# Management of ejaculatory duct obstruction: etiology, diagnosis, and treatment

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**Abstract** Abnormalities of the distal ejaculatory ducts related to infertility have been well-documented. Although there are no specific findings associated with ejaculatory duct obstruction, several clinical findings are highly suggestive. A diagnosis of ejaculatory duct obstruction is suggested 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. Other causes of infertility may be concomitantly present and need to be evaluated and treated. Trans urethral resection of ejaculatory ducts (TURED) has resulted in marked improvement in semen parameters, and pregnancies have been achieved. Proper patient selection and surgical experience are necessary to obtain optimal results. In case of testicular dysfunction, chances of success are minimal. Extended follow-up periods are needed after TURED to examine the long-term effects of this procedure. Better understanding of the anatomy and pathology of the ejaculatory ducts will continue to refine diagnostic and therapeutic procedures for this disorder.

**Keywords** Ejaculatory duct obstruction · Infertility · Transurethral resection of ejaculatory ducts

## Introduction

Although obstructions of the epididymis and proximal vas deferens have become well-recognized and readily treated causes of male infertility [1], more distal obstructions have more recently been recognized and treated [2–9]. Ejaculatory duct obstruction, although rare, is a surgically correctable cause of male infertility [2, 5–15]. Use of high-resolution transrectal ultrasound (TRUS) has resulted in an increased incidence of diagnosis of this disorder [3, 14, 16, 17]. Treatment of ejaculatory duct obstruction by transurethral resection of the ejaculatory ducts (TURED) has also become more common. In addition, there have been several reports of pregnancies following relief of ejaculatory duct obstruction using this technique [2, 4, 5, 13, 11, 15, 18–20]. Although various symptoms, signs, TRUS, radiographic, and cystoscopic findings have been associated with ejaculatory duct obstruction, none is pathognomonic for this disorder [2]. Moreover, the pathogenesis of ejaculatory duct obstruction in association with these findings, and how this obstruction impacts on male fertility, is not well understood. By examining the anatomy of the ejaculatory ductal system, and correlating it with symptomatology, semen analyses, TRUS, and pathologic findings in patients with a presumptive diagnosis of ejaculatory duct obstruction, a better understanding of ejaculatory duct obstruction and its impact on male infertility can be gained.

## Anatomy

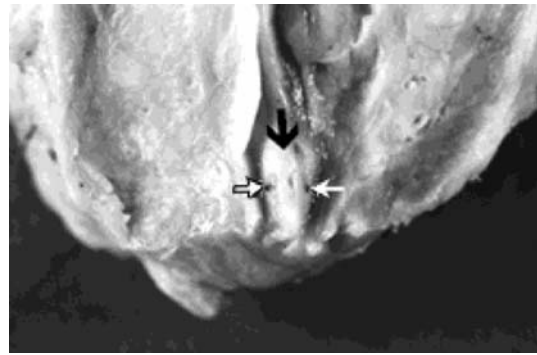
The ejaculatory ducts are derived from the wolffian duct system. The seminal vesicles develop as a blind

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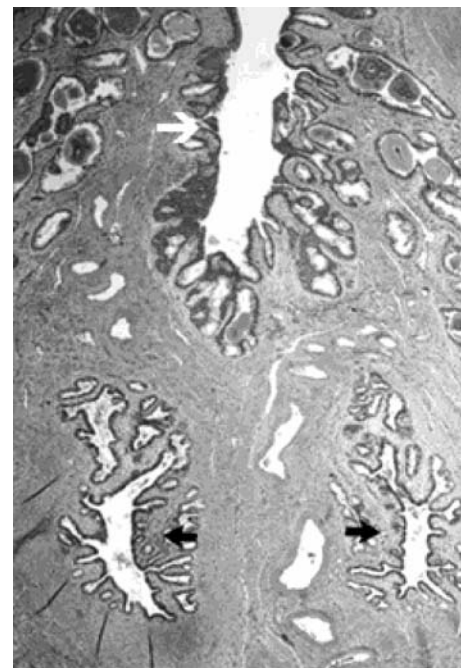
diverticulum at the most terminal end of the vas [21]. The ejaculatory ducts are a direct continuation of the seminal vesicles and, anatomically, begin after the ampulla of the vas deferens joins the seminal vesicle duct on its medial aspect at an acute angle [10, 22, 23]. The ducts are approximately 1–2 cm long and enter the prostate obliquely and posteriorly at the base, course medially and anteriorly through the prostatic glandular tissue, to enter the prostatic urethra at the verumontanum [10, 13, 22, 23]. Between the two ejaculatory ducts at the verumontanum is the prostatic utricle, a müllerian duct remnant of endodermal origin [23]. The ejaculatory ducts open in the majority of cases anterolateral to the orifice of the utricle [23]. In most men, the utricle is less than 6 mm in size but, in up to 10% of men, can exceed 10 mm [24]. The utricle does not communicate with any other structures [10, 22, 23, 25]. Injection of methyl methacrylate into the vas deferens of intact autopsy prostate/seminal vesicles/vasa specimens reveals the ejaculatory ducts exiting close to one another at the verumontanum, with a small utricle lying between them. No methyl methacrylate can be seen exiting the utricle [26] (Fig. 1). In sagittal sections, the ejaculatory duct forms an almost straight course from the prostatic base to the verumontanum. The close relationship of the ejaculatory ducts to the utricle can be seen in transverse section at the verumontanum of a radical retropubic prostatectomy specimen [26] (Fig. 2). The anatomic structures of the ejaculatory ductal system and their relationships can also be demonstrated using rectal coil magnetic resonance imaging (MRI) [5, 27]. In sagittal image, the relationships between the bladder, bladder neck, seminal vesicles, prostate, and ejaculatory ducts are easily demonstrated. Also note that the distal ejaculatory ducts are distal and inferior to the bladder neck. Each duct is surrounded by circular lamellar tissue and, in turn, both ducts are surrounded by a communal muscular envelope [22, 28]. The existence of a sphincter spermaticus has been described, but its role in the pathophysiology of partial or functional ejaculatory duct obstruction remains poorly understood [4, 11]. The ejaculatory ducts are lined by cuboidal to pseudostratified columnar epithelium [22, 28].

### Etiologies of obstruction

Ejaculatory duct obstruction can be either congenital or acquired [13, 15]. Congenital causes include congenital atresia or stenosis of the ejaculatory ducts and utricular, müllerian, and wolffian duct cysts. Acquired causes may be secondary to trauma, either iatrogenic



**Fig. 1** Coronal section of the prostate from an autopsy specimen after injection of methyl methacrylate into the vasa deferentia. Notice methyl methacrylate exiting from distal ejaculatory ducts (white arrows) which sit lateral to the midline utricle (black arrow)



**Fig. 2** Transverse section through the verumontanum from a radical retropubic prostatectomy specimen demonstrating the close relationship of the ejaculatory ducts (black arrows) to each other and to the utricle (white arrow)

or otherwise, or infectious or inflammatory etiologies [13, 15]. Calculus formation secondary to infection may also cause obstruction [4]. Cyst formation from prior instrumentation or infection may also occur [25]. Many times, patients with ejaculatory duct obstruction have no significant antecedent history [10]. Several authors have found that patients with congenital or noninfectious causes of ejaculatory duct obstruction do better after treatment than those with infectious causes [8, 9, 13, 15, 20]. Other authors, however, have not been able to reproduce this finding [10, 29, 30]. In addition, one study demonstrated that patients with partial ejaculatory

duct obstructions have better improvements in their semen parameters than those with complete obstruction [20].

## Symptoms

Patient complaints associated with ejaculatory duct obstruction can be quite variable but include infertility, decreased force of ejaculate, pain on or after ejaculation, decreased ejaculate volume, hematospermia, perineal or testicular pain, history of prostatitis or epididymitis, low back pain, urinary obstruction, dysuria, or no symptoms [2, 5, 9, 11, 13, 29]. A retrospective review of men with symptomatic ejaculatory duct obstruction demonstrated that 100% of men complained of a decreased ejaculate, 93% of non-projectile ejaculation, and 33% of pain with orgasm [31]. Symptoms are generally less pronounced or absent in patients with partial obstructions; however, partial obstructions can progress to complete obstruction [4–6]. No one symptom or constellation of symptoms can make a definitive diagnosis of ejaculatory duct obstruction.

## Signs

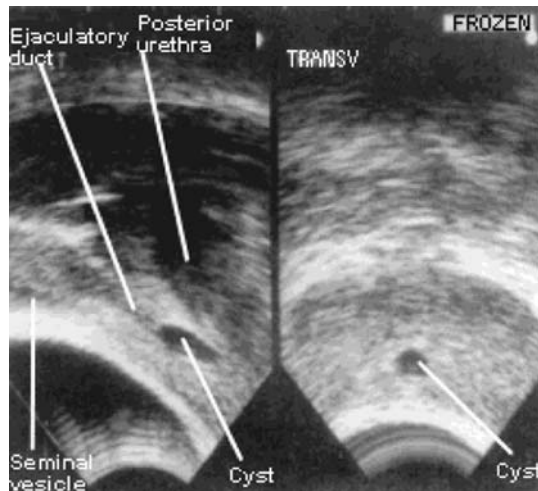
Patients with suspected ejaculatory duct obstruction classically have normal physical examinations, including normal testes, absence of varicoceles, palpable vasa, normal rectal examinations, normal secondary sexual characteristics, and normal hormonal profiles. Occasionally, there will be a palpable seminal vesicle or mass on rectal examination, or prostatic or epididymal tenderness [2, 5, 10, 13, 29]. However, these patients can, of course, have more than one disorder at the same time. That is, a patient with ejaculatory duct obstruction might also have a varicocele or a patient with testicular failure might also have ejaculatory duct obstruction [6]. Although a patient might seem to demonstrate findings only of ejaculatory duct obstruction, complete evaluation for other concomitant, possibly treatable, disorders is necessary.

Semen analysis findings in men with partial ejaculatory duct obstruction include oligospermia or azoospermia, decreased motility, and decreased ejaculate volume [2, 6, 10]. In some men with only mild partial obstructions, semen analyses can approach normal parameters, although motility may remain low [4, 6, 29]. Decreased ejaculate volume, that is, volumes of less than 1 cc (normal 1.5–5 cc), may be suggestive of ejaculatory duct obstruction, but it is by no means

pathognomonic [2, 4, 5, 10, 16]. A retrospective review of men with symptomatic ejaculatory duct obstruction from 1995 to 2001 revealed a mean ejaculate volume of 1.1 cc at the time of presentation [31]. With complete ejaculatory obstruction, seminal fluid should theoretically be fructose negative, but often fructose is present, implying the presence of only partial obstruction [5, 6]. Pryor and Hendry [13] have stated that the finding of a small volume of acid semen, which does not contain fructose, in a patient with palpable vasa, is pathognomonic for ejaculatory duct obstruction.

Historically, vasography was the gold standard for diagnosis of proximal and distal ejaculatory duct obstruction [5, 13, 14, 16]. However, its invasive nature, with risks of iatrogenic stricture and vasa occlusion, and relative risks of general anesthesia and radiation exposure, have made TRUS a more attractive diagnostic technique [2, 9, 14, 16–18, 32]. TRUS is much less invasive and can demonstrate the anatomic relationships of the prostate, seminal vesicles, and ejaculatory ducts with exquisite detail [3, 5, 14, 16, 17, 23, 33, 34]. Katz et al. [35] reported the use of ultrasound-guided transrectal seminal vesiculography under local anesthesia. Under TRUS guidance, a 22 G needle is advanced into the seminal vesicle, and, after its position is confirmed with aspiration, contrast medium is injected. Although not generally accepted as yet, this technique eliminates the risks associated with vasography while preserving excellent radiographic visualization of the ejaculatory ducts. Jarow [36] has also shown that TRUS-guided seminal vesicle aspiration was useful in the diagnosis of partial ejaculatory duct obstruction when motile sperm are found in the aspirate. Orhan et al. [37] describe the use of TRUS-guided seminal vesicle aspiration to both diagnose ejaculatory duct obstruction and to collect sperm for assisted reproduction techniques.

TRUS findings in suspected ejaculatory duct obstruction include midline cysts (Fig. 3), dilated seminal vesicles (Fig. 8a) or ejaculatory ducts, and hyperechoic regions suggestive of calcifications [2–5, 10, 17, 33]. Although seminal vesicle dilation has been frequently associated with ejaculatory duct obstruction, it is not always present; conversely, normal fertile men can, at times, have dilated seminal vesicles [23, 34, 38, 39]. Jarow [16] showed that seminal vesicle width, length, and area did not differ between fertile and infertile men on TRUS; he also stated, however, that cystic dilation of the seminal vesicles in association with abnormally low ejaculate volume is pathognomonic for ejaculatory duct obstruction. Seminal vesicles larger than 15 mm in transverse diameter are abnormal and suggest ejaculatory duct obstruction [1–3, 16].



**Fig. 3** Transrectal ultrasound images transverse on the right and longitudinal on the left, showing a small midline cyst at the distal ejaculatory duct

Midline cysts can be classified into two general categories, those that contain sperm and those that do not [18, 25, 40, 41]. These categories can often be difficult to distinguish [25, 32, 40]. The cysts not containing sperm are generally called utricles or müllerian duct cysts. The difference between utricular and müllerian duct cysts includes embryologic origin, with the utricular cysts being of endodermal and müllerian duct cysts being of mesodermal origin; location, with utricular cysts being midline near the verumontanum and müllerian duct cysts nearer the prostate base; and association of enlarged utricles with intersex disorders [16, 17, 42]. In any case, both types of midline cysts cause ejaculatory duct obstruction by compressing the ducts, and both can be treated by TURED. It is important to note that müllerian duct cysts may be more difficult to resect because of their more posterior location [42]. Cysts that contain sperm have been called wolffian or ejaculatory duct cysts or diverticula and are less common than the müllerian duct cysts [16, 25, 32, 40, 41]. Confusion as to whether a cyst is müllerian or wolffian in origin can be compounded by the fact that secondary epididymal obstruction can occur after long-term ejaculatory duct obstruction, resulting in the possible absence of sperm in a wolffian structure [30, 33]. Midline cysts cause obstruction of the ejaculatory ducts by deviating them laterally or compressing them [10]. Jarow [16] showed, in comparing TRUS findings between fertile and infertile men, that infertile men had a significantly greater incidence of midline müllerian duct cysts than fertile men (11 vs. 0%), but he could not draw any conclusions concerning the functional significance of this finding. As was true for seminal vesicle dilation, the presence of a midline cyst does

not assure the diagnosis of ejaculatory duct obstruction, but certainly suggests obstruction in the correct clinical setting.

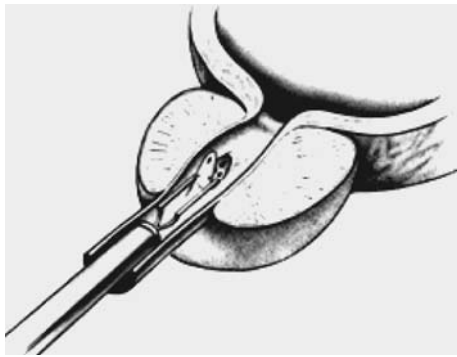
Calcifications along the course of ejaculatory ducts might be directly involved in obstruction, but those in the prostate itself are associated with prior inflammation, although not necessarily with symptomatic prostatitis [3, 4, 10]. How prostate inflammation leads to ejaculatory duct obstruction has not been well characterized. It is theorized that inflammatory involvement of the ducts themselves leading to stenosis or obstruction could cause a mechanical obstruction whereas changes in compliance of the ejaculatory duct walls or of the adjacent prostatic tissue could cause a functional obstruction [13, 15, 17]. Prostate or ejaculatory duct calcifications are associated with ejaculatory duct obstruction but have also been described in normal individuals on TRUS and although suggestive are not a reliable indicator of obstruction [3, 16]. Jarow [16] found that hyperechoic lesions on TRUS were present in a similar proportion of fertile and infertile men.

## Treatment

In patients with suspected ejaculatory duct obstruction, the standard procedure has become TURED [2, 4, 8–10, 17]. Originally described by Farley and Barnes [7] in 1973, several reports have documented its efficacy [2–5, 7, 8, 10–13, 15, 19, 20].

Trans urethral resection of ejaculatory ducts (TURED) requires a setup similar to that of transurethral resection of the prostate [6]. An OÆConnor drape is used. Cystourethroscopy is performed to rule out strictures in the anterior and bulbar urethra, as well as for evaluation of the posterior urethra. Cystoscopic findings include distorted verumontanum anatomy, splaying of the ejaculatory ducts, bulbous or bi-lobed verumontana, midline cysts, and inflammatory calcifications [10]. Once this is done, the resectoscope is inserted. The proximal verumontanum, which may be enlarged, is resected in the midline (Fig. 4). TURED is performed using pure cutting current without coagulation. Commonly, one or two chips are resected, removing the proximal verumontanum only. Although, historically, lateral CollingÆs knife incisions were made, [17] resection lateral to the verumontanum is not necessary because the ejaculatory ducts are midline structures in this region [10].

With the bladder filled with irrigation fluid, palpation of the seminal vesicles is made easier. Mild pressure is exerted on the seminal vesicles, resulting in fluid expressed from the respective ejaculatory ducts. If no



**Fig. 4** Schematic diagram of resection of the proximal verumontanum

fluid is expressed, another small bite can be taken from the verumontanum, and seminal vesicle pressure applied again. In our experience, operative success for TURED is defined as fluid expression from both ejaculatory ducts at the termination of the procedure. If bleeding is encountered, gentle coagulation is recommended, taking care to avoid the ejaculatory ducts. A catheter is inserted into the bladder and is left in place for a few hours in the recovery room. Postoperative urinary retention can occur after catheter removal, particularly in patients with prior voiding dysfunction. In these cases, reinsertion of the catheter for an additional 24–48 h may be necessary [10].

Complications due to TURED are rare if the procedure is done carefully and with expertise. Obviously, if resection is performed too proximally, damage to the bladder neck can result in retrograde ejaculation postoperatively. Resection too distally can cause damage to the external sphincter with subsequent urinary incontinence. Excessive postoperative fibrosis may result in scarring and subsequent azoospermia, implying reocclusion of the ejaculatory ducts. If this occurs, a repeat TURED may be necessary [10]. Contamination of the ejaculate with urine and seminal vesicle reflux of urine have also been reported, [43, 44] although the clinical significance of this has not been elucidated. We reported on a patient with seminal vesicle urinary reflux following TURED, causing significant post-void dribbling [45]. Secondary epididymal obstruction can occur after long-term ejaculatory duct obstruction, necessitating scrotal exploration and vasoepididymostomy for patients who fail to improve after TURED and in whom this is suspected [30, 33].

The patient is asked to refrain from sexual activity for 7–10 days. When sexual activity is resumed, hematospermia may be evident but is self-limited; the patient should be warned of this occurrence and reassured. A semen analysis is obtained 1 month following the resection.

Weintraub et al. [5] reported on eight patients with ejaculatory duct obstruction diagnosed by TRUS, rectal coil MRI, and vasography. Eighty percent of patients were improved symptomatically after TURED, the majority had improvements in sperm density or volume, or both, and 25% were able to impregnate their wives [5]. Hellerstein et al. [4] reported on 2 patients with infertility, 1 with a large midline cyst and 1 with dilated seminal vesicles, who underwent TURED for presumed ejaculatory duct obstruction; both had significant improvements in semen parameters and both were able to impregnate their wives.

Meacham et al. [2] reported on 24 patients with clinical profiles consistent with ejaculatory duct obstruction, all of whom underwent TURED. Fifty percent had an increase in sperm density or motility and 29% had an increase in ejaculate volume only. Seven of 24 (29%) were able to impregnate their wives [2]. Again, none of these studies report on the long-term effects of this procedure. Turek et al. [46] showed a greater than 50% improvement in semen parameters in 65% of patients after TURED. Twenty percent were able to initiate a pregnancy; there was a 20% overall complication rate with the most common being a watery ejaculate.

In a retrospective review of 15 patients, Johnson et al. [31] demonstrated that men with symptomatic ejaculatory duct obstruction have marked subjective and objective improvements in signs and symptoms post TURED. Ninety-three percent of men reported an improvement in volume of ejaculate and demonstrate a projectile ejaculate. The resolution of hematospermia and painful ejaculation was also noted. In addition, the mean ejaculate volume increased from 1.1 cc preoperatively to 2.3 cc after TURED. Preoperatively the mean total mobile sperm count was 8.1 million per ejaculate that increased substantially to 38.1 million per ejaculate after surgical intervention. Of the six men available for long term follow up, four men had successfully fathered a child without the use of assisted reproduction techniques. There were no reported complications of the procedure.

Netto et al. [47] showed that the etiology of the ejaculatory duct obstruction was a significant predictor of success after TURED. In those patients with a congenital cause to the obstruction, success rates were excellent with 100% improvement in semen parameters (motility, volume), 83% improvement in sperm count, and 66% pregnancy rate. In those patients with an acquired cause to the obstruction, only 37.5% had improved semen parameters and 12.5% pregnancy rate. Furthermore, although 33% of each group had

complications, those in the congenital group were more minor in nature.

This notion of central cystic lesions responding best to TURED was confirmed by Kadioglu et al. [20] and Schroeder-Printzen et al. [8]. In addition, Kadioglu et al. [20] demonstrated that response to TURED depended on the degree of obstruction; improvements in semen parameters were significantly better in patients with partial obstruction (94%) than those with complete obstruction (59%).

Aside from TURED, Colpi et al [48] describe antegrade seminal tract washout to relive ejaculatory obstruction. In one patient, the vasa were exposed scrotally and saline was injected antegrade to the seminal vesicles until the obstruction was relieved. Fertility was restored in this patient.

## Conclusion

With the increased use of high-resolution TRUS, abnormalities of the distal ejaculatory ducts related to infertility have been well-documented [2–5, 10, 16]. Although there are no pathognomonic findings associated with ejaculatory duct obstruction, several clinical findings are highly suggestive. 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, the diagnosis of ejaculatory duct obstruction is suggested [2, 9, 10, 16, 17]. Of course, other causes of infertility may be concomitantly present and need to be searched for and treated as well. In selected cases, TURED has resulted in marked improvement in semen parameters, and pregnancies have been achieved [2–5, 10, 16]. As is the case with all surgical procedures, proper patient selection and surgical experience are necessary to obtain optimal results. In patients with evidence of testicular dysfunction, chances of success are minimal. In addition, extended follow-up periods are needed after TURED to examine the long-term effects of this procedure. Better understanding of the anatomy and pathology of the ejaculatory ducts will continue to refine diagnostic and therapeutic procedures for this disorder.

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