Ejaculatory Duct Dysfunction and Chronic Pelvic Pain Syndrome in Men

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Chronic pelvic pain syndrome (CPPS) describes unexplained pelvic pain in men associated with irritative voiding symptoms, post-orgasmic pain, and/or pain located in the groin, genitalia, or perineum in the absence of bacterial infection. Many different etiologies of CPPS have been proposed, including roles for immunologic, neurologic, endocrine, and psychologic factors. This article examines one such factor—ejaculatory duct obstruction (EDO). Because EDO is correctable with relatively minor surgery, it should be considered by any clinician caring for a patient who reports symptoms of CPPS.

Introduction

Chronic pelvic pain syndrome (CPPS) describes unexplained pelvic pain in men that is associated with irritative voiding symptoms, post-orgasmic pain, and/or pain located in the groin, genitalia, or perineum in the absence of bacterial infection. The absence of infection differentiates CPPS from chronic prostatitis (CP)/CPPS, which is not the focus of this article. The term CPPS as used in this article is also distinct from the term "urologic chronic pelvic pain syndromes," which the National Institute of Diabetes and Digestive and Kidney Diseases began using in 2007 to describe pain syndromes associated with the bladder (ie, interstitial cystitis/painful bladder syndrome) and the prostate gland (ie, CP/CPPS).

Many different etiologies and mechanisms of pathogenesis of CPPS have been proposed, including roles for immunologic, neurologic, endocrine, and psychologic factors [1]. It has been suggested that CPPS can be caused by many factors, some of which lie beyond the prostate [2]. This article examines one such factor—ejaculatory duct obstruction (EDO). This disorder is relatively uncommon but it is associated with a relatively high prevalence of post-orgasmic pain. A retrospective review of men with symptomatic EDO found that 33% of men reported pain with orgasm [3]. Because EDO is correctable with relatively minor surgery, it should be considered by any clinician caring for a patient who reports symptoms of CPPS. This article briefly reviews the etiology and treatment of EDO as a potentially important factor in the etiology of CPPS.

Anatomical Considerations

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. The ducts are approximately 1 to 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 [4]. Between the two ejaculatory ducts at the verumontanum is the prostatic utricle, a müllerian duct remnant of endodermal origin. The ejaculatory ducts open in most cases anterolateral to the orifice of the utricle. The utricle does not communicate with any other structures. 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. The existence of a sphincter spermaticus has been described, but its role in the pathophysiology of partial or functional EDO remains poorly understood [5].

Etiologies

EDO can be either congenital or acquired [6]. 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 or otherwise, or infectious or inflammatory. Calculus formation secondary to infection may also cause obstruction. Cyst formation from prior instrumentation or infection may also occur [7]. Many times, patients with EDO have no significant antecedent history.

Signs and Symptoms

Patient complaints associated with EDO 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. A retrospective review of men with symptomatic EDO demonstrated that 100% of men complained of a decreased ejaculate, 93% of nonprojectile ejaculation, and 33% of pain with orgasm [3]. Symptoms are generally less pronounced or absent in patients with partial obstructions; however, partial obstructions can progress to complete obstruction. No one symptom or constellation of symptoms can make a definitive diagnosis of EDO.

Patients with suspected EDO 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. However, these patients can, of course, have more than one disorder at the same time. That is, a patient with EDO might also have a varicocele or a patient with testicular failure might also have EDO. Although a patient might seem to demonstrate signs or symptoms only of EDO, complete evaluation for other concomitant, possibly treatable, disorders is necessary.

Semen analysis findings in men with partial EDO include oligospermia or azoospermia, decreased motility, and decreased ejaculate volume [8]. In some men with only mild partial obstructions, semen analyses can approach normal parameters, although motility may remain low. Decreased ejaculate volume—volumes of less than 1 mL—may suggest EDO, but it is by no means pathognomonic. A retrospective review of men with symptomatic EDO from 1995 to 2001 revealed a mean ejaculate volume of 1.1 mL at the time of presentation [3]. With complete ejaculatory obstruction, seminal fluid should theoretically be fructose negative, but often fructose is present, implying the presence of only partial obstruction. Pryor and Hendry [6] 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 EDO.

Diagnosis

Historically, vasography was the gold standard for diagnosis of proximal and distal EDO. However, its invasive nature, with risks of iatrogenic stricture and vasal occlusion, and relative risks of general anesthesia and radiation exposure, have made transrectal ultrasound (TRUS) a more attractive diagnostic technique. TRUS is much less invasive and can demonstrate the anatomic relationships of the prostate, seminal vesicles, and ejaculatory ducts with exquisite detail. Katz et al. [9] 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. This technique eliminates the risks associated with vasography while preserving excellent radiographic visualization of the ejaculatory ducts. Jarow [10] has also shown that TRUS-guided seminal vesicle aspiration was useful in the diagnosis of partial EDO when motile sperm are found in the aspirate.

TRUS findings in suspected EDO include midline cysts, dilated seminal vesicles or ejaculatory ducts, and hyperechoic regions suggestive of calcifications. Although seminal vesicle dilation has been frequently associated with EDO, it is not always present; conversely, fertile men can, at times, have dilated seminal vesicles [11]. Jarow [12] 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 EDO. Seminal vesicles larger than 15 mm in transverse diameter are abnormal and suggest EDO.

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. How prostate inflammation leads to EDO 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]. Prostate or ejaculatory duct calcifications are associated with EDO but have also been described in normal individuals on TRUS, and although suggestive, are not a reliable indicator of obstruction. Jarow [12] found that hyperechoic lesions on TRUS were present in a similar proportion of fertile and infertile men.

Treatment of EDO

In patients with suspected EDO, the standard procedure has become transurethral resection of the ejaculatory ducts (TURED). Originally described by Farley and Barnes [14] in 1973, many reports have now documented its efficacy. TURED requires a setup similar to that of transurethral resection of the prostate. 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. Once this is done, the resectoscope is inserted.

The proximal verumontanum, which may be enlarged, is resected in the midline. TURED is performed using pure cutting current without coagulation. Commonly, one or two chips are resected, removing the proximal verumontanum only. Although, historically, lateral knife incisions were made, resection lateral to the verumontanum is not necessary because the ejaculatory ducts are midline structures in this region.

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 fluid is expressed, another small bite can be taken from the verumontanum, and seminal vesicle pressure applied again. In my 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 to 48 hours may be necessary [15••].

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. Contamination of the ejaculate with urine and seminal vesicle reflux of urine has also been reported, although the clinical significance of this has not been elucidated [16]. Secondary epididymal obstruction can occur after long-term EDO, necessitating scrotal exploration and vasoepididymostomy for patients who fail to improve after TURED and in whom this is suspected.

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

Results Relevant to EDO

A retrospective review of 15 patients treated with EDO demonstrated that men with symptomatic EDO 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 demonstrated a projectile ejaculate. The resolution of hematospermia and painful ejaculation was also noted. Two of the four men who complained of pain with ejaculation reported resolution of the pain. No men reported an exacerbation of their symptoms after TURED. The mean ejaculate volume increased to 2.3 mL and the total motile sperm count to 38.1 million per ejaculate. There were no complications associated with the procedure.

Netto et al. [17] showed that the etiology of the EDO was a significant predictor of success after TURED. In patients with a congenital cause of the obstruction, success rates were excellent, with 100% improvement in semen parameters (motility, volume), 83% improvement in sperm count, and 66% pregnancy rate. In patients with an acquired cause of the obstruction, only 37.5% had improved semen parameters and the pregnancy rate was 12.5%. Furthermore, although 33% of each group had complications, those in the congenital group were more minor.

Aside from TURED, Colpi et al. [18] 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.

Conclusions

Post-orgasmic pain is one component of the larger phenomenon of CPPS. The incidence of post-orgasmic pain is 1% to 9.7% in the general population and as high as 58% in selected populations, such as men with prostatitis [19]. This variable incidence suggests that this component of CPPS should be high on a physician's differential when considering a case of CPPS. Because EDO is a primary cause of post-orgasmic pain, this also suggests that physicians be alert to this possible etiology of a patient's complaints.

With the increased use of high-resolution TRUS, abnormalities of the distal ejaculatory ducts related to post-orgasmic pain can be readily assessed. Although there are no pathognomonic findings associated with EDO, 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 EDO is suggested. In selected cases, TURED has resulted in marked improvement in semen parameters and a reduction in post-orgasmic pain. As is the case with all surgical procedures, proper patient selection and surgical experience are necessary to obtain optimal results. Better understanding of the anatomy and pathology of the ejaculatory ducts will continue to refine diagnostic and therapeutic procedures for this disorder, and may illuminate one potential etiological factor in the expression of CPPS.

Disclosure

No potential conflict of interest relevant to this article was reported.

References and Recommended Reading

Papers of particular interest, published recently, have been highlighted as:

- • Of importance
- •• Of major importance

1. Pontari MA, Ruggieri MR: **Mechanisms in prostatitis/chronic pelvic pain syndrome.** *J Urol* 2008, **179(Suppl 5):**S61–S67.
2. Pontari MA: **Etiologic theories of chronic prostatitis/chronic pelvic pain syndrome.** *Curr Urol Rep* 2007, **8:**307–312.
3. Johnson CW, Bingham JB, Goluboff ET, Fisch H: **Transurethral resection of the ejaculatory ducts for treating ejaculatory symptoms.** *BJU Int* 2005, **95:**117–119.
4. McMahon S: **An anatomical study by injection technique of the ejaculatory ducts and their relations.** *J Urol* 1938, **39:**422–443.
5. Hellerstein DK, Meacham RB, Lipshultz LI: **Transrectal ultrasound and partial ejaculatory duct obstruction in male infertility.** *Urology* 1992, **39:**449–452.
6. Pryor JP, Hendry WF: **Ejaculatory duct obstruction in subfertile males: analysis of 87 patients.** *Fertil Steril* 1991, **56:**725–730.
7. Mayersak JS: **Urogenital sinus-ejaculatory duct cyst: a case report with a proposed clinical classification and review of the literature.** *J Urol* 1989, **142:**1330–1332.
8. Meacham RB, Hellerstein DK, Lipshultz LI: **Evaluation and treatment of ejaculatory duct obstruction in the infertile male.** *Fertil Steril* 1993, **59:**393–397.
9. Katz D, Mieza M, Nagler HM: **Ultrasound guided transrectal seminal vesiculography: a new approach to the diagnosis of male reproductive tract abnormalities [abstract 330].** *J Urol* 1994, **151:**310A.
10. Jarow JP: **Seminal vesicle aspiration in the management of patients with ejaculatory duct obstruction.** *J Urol* 1994, **152:**899–901.
11. Wessels EC, Ohori M, Grantmyre JE, et al.: **The prevalence of cystic dilatation of the ejaculatory ducts detected by transrectal ultrasound (TRUS) in a self-referred (screening) group of men [abstract 973].** *J Urol* 1992, **147:**456A.
12. Jarow JP: **Transrectal ultrasonography of infertile men.** *Fertil Steril* 1993, **60:**1035–1039.
13. Worischeck JH, Parra RO: **Transrectal ultrasound in the evaluation of men with low volume azoospermia.** *J Urol* 1993, **149:**1341–1344.
14. Farley S, Barnes R: **Stenosis of ejaculatory ducts treated by endoscopic resection.** *J Urol* 1973, **109:**664–666.
15. •• Fisch H, Lambert SM, Goluboff ET: **Management of ejaculatory duct obstruction: etiology, diagnosis, and treatment.** *World J Urol* 2006, **24:**604–610.

This article provides a comprehensive review of the clinical consequences of EDO.

16. Malkevich DA, Mieza M, Nagler HM: **Patency of ejaculatory ducts after transurethral resection of ejaculatory ducts verified by reflux seen on voiding cystourethrogram [abstract 295].** *J Urol* 1994, **151:**301A.
17. Netto NR, Esteves SC, Neves PA: **Transurethral resection of partially obstructed ejaculatory ducts: seminal parameters and pregnancy outcomes according to the etiology of obstruction.** *J Urol* 1998, **159:**2048–2053.
18. Colpi GM, Negri L, Patrizio P, Pardi G: **Fertility restoration by seminal tract washout in ejaculatory obstruction.** *J Urol* 1995, **153:**1948–1950.
19. Ilie CP, Mischianu DL, Pemberton RJ: **Painful ejaculation.** *BJU Int* 2007, **99:**1335–1339.