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Ejaculatory Duct Dysfunction and Lower Urinary Tract Symptoms: Chronic Prostatitis

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Abstract Chronic pelvic pain syndrome (CPPS) is the most prevalent form of prostatitis. It is characterized by pelvic pain, voiding symptoms, and ejaculatory symptoms in the absence of bacterial infection. This can be a difficult condition to treat. Many etiologies for CPPS have been proposed including immunologic, neurologic, endocrine, and psychological factors. This article examines a potentially correctable condition that may lead to CPPS, ejaculatory duct obstruction (EDO). EDO is easily correctable with minor surgery. In patients with symptoms of CPPS with associated ejaculatory pain, the diagnosis of EDO should strongly be considered.

Keywords Ejaculatory duct obstruction · Prostatitis · Chronic pelvic pain syndrome · Post-orgasmic pain

Introduction

Chronic lower urinary tract symptoms and pelvic pain can significantly impact the quality of life of patients affected with these conditions. Patient's symptomatic complaints in current classification schemes have brought about the diagnoses of chronic pelvic pain syndrome (CPPS) in men and painful bladder syndrome/interstitial cystitis (PBS/IC) mostly in women. Current knowledge of the etiology and treatment of these disorders is very limited. One potentially treatable cause of this in men is ejaculatory duct obstruction (EDO).

The diagnosis of CPPS as defined by the National Institutes of Health (NIH) is chronic nonbacterial prostatitis (type III) with pelvic pain, voiding symptoms, and ejaculatory symptoms with no identifiable uropathogen or infectious cause [1]. This is a broad definition that includes a wide range of patients and symptoms. This condition is further subdivided as inflammatory and noninflammatory subtypes. This entity is distinct from the National Institute of Diabetes and Digestive and Kidney Diseases definition of PBS/IC.

There have been many different etiologies proposed for CPPS including immunologic, neurologic, endocrine, psychologic, and combinations of these [2]. In this article we discuss a potentially curable cause of prostatitis due to EDO, which we have termed chronic obstructive prostatitis (COP). The diagnosis of EDO is relatively rare with current investigation focusing on patients with complaints related to infertility. The incidence of complete bilateral EDO is less than 1% in infertile men [3]. The incidence of incomplete EDO is unknown as definitions vary among authors. Documented symptomatic complaints of patients treated for EDO include pelvic pain, pain with ejaculation, and hematospermia [4]. The incidence of pain with ejaculation has varied widely among reports and patient populations. In patients with chronic prostatitis, 58% have pain with ejaculation [5]. With similar symptoms seen in patients diagnosed with CPPS, we believe COP is an etiology of pain in a subset of patients with this diagnosis.

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Anatomy

The ejaculatory ducts, seminal vesicle, and vas deferens develop from the Wolffian duct system during embryologic development. The ejaculatory duct begins distal to the confluence of the seminal vesicle and the ampulla of the vas deferens. It courses at an oblique angle from proximal to distal and lateral to medial traversing the prostate and exiting lateral and distal to the verumontanum into the urethra [6]. The ejaculatory ducts are between 1 to 2 cm in length. Between the ostia of the ejaculatory ducts at the verumontanum is the prostatic utricle; a remnant of the Müllerian duct. The utricle does not communicate with any structure. The ejaculatory ducts are lined by cuboidal to pseudostratified epithelium. The proximal and distal portions resemble the epithelial lining of the seminal vesicles [7]. The ejaculatory ducts are surrounded by lamellar tissue and an attenuated continuation of longitudinal muscle fibers from the seminal vesicles [8]. The existence of a distal sphincter, so-called sphincter spermaticus, has been described but its function has remained debatable.

Etiology of EDO

There are congenital and acquired causes of EDO [9]. Congenital abnormalities include atresia and stenosis as well as utricular, Müllerian, and Wolffian duct cyst. Acquired causes include iatrogenic or other trauma, infection, or inflammatory processes. We recently reported the presence of proximal ejaculatory duct in 4% of transrectal ultrasound (TRUS)-guided prostate biopsy specimens [7]. Obstruction from seminal vesicle calculi has also been described [10]. Many patients have no significant history at presentation.

Signs and Symptoms of COP

EDO can present with a number of different symptoms including infertility, decreased force of ejaculate, pain associated with ejaculation, decreased ejaculate volume, hematospermia, and perineal or testicular pain. Other symptoms can include low back pain, urinary obstruction, and dysuria. Some patients will be asymptomatic. In a retrospective review of symptomatic men with EDO, 100% had decreased force of ejaculate, 93% had nonprojectile ejaculation, and 33% had pain at orgasm [4]. The symptoms of partial obstruction are similar to those of complete EDO but are generally less pronounced. Of concern though is the possible progression of a partial to complete obstruction. Although the history may heighten suspicion, the diagnosis of EDO cannot be made with history alone.

Patients suspected of having EDO commonly have no significant physical examination findings. Most patients have normal testicles, vas deferens, rectal examination, and secondary sexual characteristics. Rarely, a mass may be palpated on digital rectal examination representing a swollen seminal vesicle. In the evaluation of CPPS, the use of the four-glass Meares-Stamey localization technique is advocated by most authors. In addition to history, the use of standardized questionnaires such as the NIH chronic prostatitis index and International Prostate Symptom Score help to better define symptoms and gauge effectiveness of treatment. Positive responses to questions regarding pain with climax/ejaculation should raise one's clinical suspicion of possible COP/EDO.

Semen analysis of patients with complete EDO should have low volume, be acidic, and be fructose negative. Pryor and Hendry [11] have stated this description is pathognomonic for EDO in patients with palpable vasa. Semen analysis of patients with partial obstruction includes oligospermia or azoospermia, decreased motility, and decreased volume [12]. In some cases of partial obstruction, semen parameters can approach normal values with decreased motility being the only abnormal finding. Low ejaculate volume (<1 mL) is suggestive of EDO. A retrospective review revealed a mean ejaculate volume of 1.1 mL in patients with symptomatic EDO [4].

TRUS has become the preferred method of diagnosing EDO, replacing the historically gold standard vasography. TRUS has several advantages over vasography including being less invasive, not requiring a general anesthetic, and greater detail of prostate, seminal vesicles, and ejaculatory duct relationships. Most authors agree that seminal vesicles larger than 15 mm are abnormal and may suggest EDO [12, 13]. However, findings on TRUS have been shown to correlate poorly with true obstruction [9]. Adjunctive tests include TRUS-guided injection of a dye material (chromotubation) or x-ray contrast (vesiculography) into the seminal vesicles with concomitant urethroscopy or fluoroscopy to evaluate for outflow obstruction.

In addition to TRUS, Jarrow [14] has described seminal vesicle aspiration as an adjunct to diagnose partial EDO. Seminal vesicle aspiration has also been used to collect sperm for assisted reproductive techniques [15]. A recent study compared chromotubation, vesiculography, and seminal vesicle aspiration with TRUS findings. Twenty-five patients had evidence of obstruction on TRUS. Chromotubation, seminal vesicle aspiration, and vesiculography demonstrated obstruction in 36%, 48%, and 52% of these patients, respectively [16]. This illustrates the difficulty in diagnosing a dynamic process with static imaging.

Efforts have been made to use dynamic tests to assess for EDO as well. Ohran et al. [17] injected Technetium-99m sulphur colloid into the seminal vesicle and then performed

scintography before and after ejaculation. With this technique they found a statistically significant difference in patients with and without evidence of obstruction on TRUS [17]. Limitations of this study are its small sample size (20 patients) and lack of established normal values. Eisenberg et al. [18•] also evaluated the dynamics EDO. They performed seminal vesicle/ejaculatory duct manometry in patients with evidence of EDO and a control arm. In this study they found a statistically significant difference in opening pressures between patients with intraoperative evidence of EDO and the control group [18•]. Although these studies have started to quantify the pathologic dynamics of EDO, they remain investigational at this time. No conclusive test for partial EDO has been defined.

TRUS is very good at detecting anatomic abnormalities such as utricular, Müllerian, or Wolffian duct cyst [13]. Utricular cysts are generally midline near the verumontanum, whereas Müllerian cysts are nearer the base of the prostate. They have different embryologic origins; utricular cysts are of endodermal origin and Müllerian cysts are of mesodermal origin [19]. These cause obstruction by compression of the ejaculatory duct. Wolffian cysts contain sperm and have been called ejaculatory duct cyst or diverticula. These are much less common than utricular or Müllerian cysts [20]. The presence of a midline cyst does not confirm the diagnosis of obstruction but should be considered in the overall clinical picture.

Calcifications can be seen along the ejaculatory duct and may cause obstruction. Calcifications within the substance of the prostate can be associated with prior inflammation. How inflammation can lead to EDO has not been fully characterized; proposed mechanisms include inflammation involving the ducts, changes in compliance, or adjacent tissue impinging upon on the duct [11, 21]. Calcifications have also been described at the junction of the urethra and ejaculatory duct in normal individuals [13]. The presence of calcifications is another indicator of possible pathology leading to EDO.

Treatment

The standard treatment of EDO is transurethral resection of the ejaculatory ducts (TURED); this was originally described by Farley and Barnes in 1973 [22]. Multiple authors have shown this to relieve EDO [3, 4, 11, 12].

TURED requires set up of standard equipment for endoscopic resection of prostatic tissue. To aid in palpation and expression of seminal vesicles an O'Connor drape is used for this procedure. Urethroscopy is performed to rule out stricture disease or other unrecognized pathology. The verumontanum should be inspected carefully for the presence any distortion, midline cyst, splaying of ejaculatory ducts, or inflammatory calcifications. Once a thorough examination is completed the resectoscope is inserted. The proximal verumontanum is resected in the midline. The resection is carried out using a pure cutting current. Coagulation should be used only to control bleeding, and then only sparingly away from the orifice of the ejaculatory duct. Commonly only one or two chips are resected removing the proximal verumontanum. Lateral incisions have been described but are unnecessary because the ejaculatory ducts are midline structures at the verumontanum [23].

The seminal vesicles are more easily palpated with the bladder full of irrigation. With the index finger in the rectum gentle pressure is applied to the seminal vesicle with expression of fluid from the respective ejaculatory duct. If no fluid is expressed additional resection can be performed. In our series intraoperative success was defined as expression of fluid from both ejaculatory ducts. A catheter is routinely left for 24 h. Postoperative urinary retention is possible, with those at highest risk being patients with preoperative voiding dysfunction [23].

Complications after TURED are rare if performed carefully. If resection is carried out too proximally bladder neck integrity may be compromised and result in retrograde ejaculation. Resection too distally risks damage to the external sphincter and urinary incontinence. Postoperative fibrosis may develop and necessitate repeat TURED. Urinary reflux into the seminal vesicles has been described as well as contamination of the ejaculate with urine [24, 25]. The clinical significance of these findings has not been elucidated. In our experience, seminal vesicle reflux resulted in post void dribbling after TURED [26]. Epididymitis is another well-documented complication of this procedure [16, 18•].

Patients with long-term EDO may also develop secondary epididymal obstruction. This may require subsequent vasoepididymostomy if an infertile patient's semen parameters fail to improve after TURED and this is suspected [27].

Patients are asked to abstain from sexual activity for 7 to 10 days. When sexual activity is resumed, a self-limiting hematospermia may be observed. Patients should be informed of this and reassured. A semen analysis is obtained 1 month after resection.

In a case report antegrade seminal tract washout has been described as an alternative to TURED. The vasa are exposed scrotally and flushed with normal saline until the obstruction has been relieved [28].

Results

In a retrospective series 14 out of 15 patients reported subjective improvement in ejaculation with increases in volume and projectile ejaculate. Mean volume and number of sperm increased from 1.1 to 2.3 mL and 8.1 to 38.1

million, respectively [4]. Two of four patients with painful ejaculation had resolution after TURED. In the series by Purohit et al. [16], 7 of 8 patients improved after TURED. Success was defined as 50% increase in motile sperm. Two patients complaining of pain had resolution postoperatively.

Netto et al. [29] demonstrated that the cause of obstruction was a significant predictor of successful outcome. In patients with congenital causes of obstruction, 100% saw increased motility and volume, and 83% had improved sperm count with a pregnancy rate of 66%. In patients with acquired obstruction, only 37.5% had improved semen parameters with a pregnancy rate of 12.5%.

Conclusions

COP is a small component of the larger CPPS. With large variations in the incidence of symptoms suggestive of EDO/COP, and rates as high as 58% in select populations, urologists should be attuned to these symptoms in patients with CPPS. EDO can also cause ejaculatory pain in patients without CPPS and should prompt investigation when this is encountered clinically.

With the increasing resolution of TRUS the ejaculatory ducts and seminal vesicles can be more easily assessed as a cause of ejaculatory pain. Currently, EDO cannot be diagnosed with TRUS alone; however, in the setting of an infertile patient with oligospermia or azoospermia, low semen volume, normal secondary sexual characteristics, and normal hormone profile dilated seminal vesicles and calcifications along the ejaculatory duct are highly suggestive. The ideal diagnostic study has yet to be defined.

TURED has been shown to be an effective therapy in properly selected patients with marked improvements in semen parameters and resolution of post-orgasmic pain. Optimal results require good surgical technique and proper patient selection. Better understanding of the mechanisms of EDO and refinement of diagnosis will help us to better identify patients with this disorder and hopefully treat those patients with COP leading to CPPS.

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References

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

- Of importance

1. Kreiger JN, Nyberg L Jr, Nickel JC: NIH consensus definition and classification of prostatitis. JAMA 1999, 282:236–237.
2. Pontari MA: Etiologic theories of chronic prostatitis/chronic pelvic pain syndrome. Curr Urol Rep 2007, 8:307–312.
3. Jarow JP, Espeland MA, Lipshultz LI: Evaluation of the azoospermic patient. J Urol 1989, 142:62–65.
4. Johnson CW, Bingham JB, Goluboff ET, Fisch H: Transurethral resection of the ejaculatory ducts for treating ejaculatory symptoms. BJU Int 2005, 95:117–119.
5. Litwin MS, McNaughton-Collins M, Fowler FJ Jr, et al.: The National Institutes of Health chronic prostatitis symptom index: development and validation of a new outcome measure. Chronic Prostatitis Collaborative Research Network. J Urol 1999, 162:369–375.
6. McMahon JF, Ritter S, Klemperer P: Anatomical and histological study of the verumontanum with especial reference to the ejaculatory ducts. J Urol 1924, 17:1–16.
7. McIntyre M: The incidence of proximal ejaculatory duct in transrectal ultrasound guided biopsy specimens. Presented at the South Eastern Section American Urological Association annual meeting. Miami, FL; March 11–14, 2010.
8. Nguyen HT, Etzell J, Turek PF: Normal human ejaculatory duct anatomy: a study of cadaveric and surgical specimens. J Urol 1996, 155:1639–1642.
9. Hellerstein DK, Meacham RB, Lipshultz LI: Transrectal ultrasound and partial ejaculatory duct obstruction in male infertility. Urology 1992, 39:449–452.
10. Philip J, Manikandan R, Lamb GH, Desmond AD: Ejaculatory-duct calculus causing secondary obstruction and infertility. Fertil Steril 2007, 88:706.e9–706.e11.
11. Pryor JP, Hendry WF: Ejaculatory duct obstruction in subfertile males: analysis of 87 patients. Fertil Steril 1991, 56:725–730.
12. Meacham RB, Hellerstein DK, Lipshultz LI: Evaluation and treatment of ejaculatory duct obstruction in the infertile male. Fertil Steril 1993, 59:393–397.
13. Jarow JP: Transrectal ultrasonography of infertile men. Fertil Steril 1993, 60:1035–1039.
14. Jarow JP: Seminal vesicle aspiration in the management of patients with ejaculatory duct obstruction. J Urol 1994, 152:899–901.
15. Orhan I, Onur R, Cayan S, et al.: Seminal vesicle sperm aspiration in the diagnosis of ejaculatory duct obstruction. BJU Int 1999, 84:1050–1053.
16. Purohit RS, Wu DS, Shinohara K, Turek PJ: A prospective comparison of 3 diagnostic methods to evaluate ejaculatory duct obstruction. J Urol 2004, 171:232–235.
17. Orhan I, Duskal I, Onur R, et al.: Technetium Tc 99m sulphur colloid seminal vesicle scintigraphy: a novel approach for the diagnosis of the ejaculatory duct obstruction. Urology 2008, 71:672–676.
18. • Eisenberg ML, Walsh TJ, Garcia MM, et al.: Ejaculatory duct manometry in normal men and in patients with ejaculatory duct obstruction. J Urol 2008, 180:255–260. *This article begins to explore the dynamics of EDO.*
19. Van Poppel H, Vereecken R, De Geeter P, Verduyn H: Hemospermia owing to utricular cyst: embryological summary and surgical review. J Urol 1983, 129:608–609.
20. Kirkali Z, Yigitbasi O, Diren B, et al.: Cysts of the prostate, seminal vesicles and diverticulum of the ejaculatory ducts. Eur Urol 1991, 20:77–80.
21. Worischeck JH, Parra RO: Transrectal ultrasound in the evaluation of men with low volume azoospermia. J Urol 1993, 149:1341–1344.
22. Farley S, Barnes R: Stenosis of ejaculatory ducts treated by endoscopic resection. J Urol 1973, 109:664–666.
23. Fisch H: Transurethral resection of the ejaculatory ducts. Curr Surg Tech Urol 1992, 5:2–7.

24. Malkevich D, 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.
25. Vazquez-Levin MH, Dressler KP, Nagler HM: Urine contamination of seminal fluid after transurethral resection of ejaculatory ducts. J Urol 1994, 152:2049–2052.
26. Goluboff ET, Kaplan SA, Fisch H: Seminal vesicle urinary reflux as a complication of transurethral resection of ejaculatory ducts. J Urol 1995, 153:1234–1235.
27. Fisch H, Kang YM, Johnson CW, Goluboff ET: Ejaculatory duct obstruction. Curr Opin Urol 2002, 12:509–515.
28. Colpi GM, Negri L, Patrizio P, Pardi G: Fertility restoration by seminal tract washout in ejaculatory duct obstruction. J Urol 1995, 153:1948–1950.
29. Netto NR Jr, 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.