Obstructive Azoospermia

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KEYWORDS

• Obstructive azoospermia • Vasal obstruction • Epididymal obstruction
• Ejaculatory duct obstruction • Microsurgical epididymal sperm aspiration • Vasovasostomy
• Vasoepididymostomy • Transurethral resection of ejaculatory ducts

KEY POINTS

- Obstructive azoospermia accounts for 40% of azoospermia and results from obstruction of the excurrent ducts (due to many causes) at any location between the rete testis and the ejaculatory ducts.
- Physical examination, along with possible use of genetic/laboratory testing, ultrasonography, vasography, and/or testis biopsy as indicated helps to definitively diagnose obstructive azoospermia, determine the location of the obstruction, and select appropriate management.
- Treatment options include microsurgical reconstruction (possible in most cases) or sperm aspiration (ie, microsurgical epididymal sperm aspiration) without reconstruction.
- Surgical reconstruction relies on central microsurgical tenets including mucosa-to-mucosa approximation, cutting back to healthy tissue with good blood supply, and assuring a tension-free anastomosis.

Videos of microsurgical vasovasostomy and microsurgical vasoepididymostomy accompany this article at http://www.urologic.theclinics.com/

BACKGROUND

Azoospermia, the absence of sperm in the ejaculate (confirmed by 2 centrifuged semen specimens), is identified in 15% of infertile men and results from pretesticular, testicular, or posttesticular causes, and may be classified as obstructive azospermia (OA) or nonobstructive azoospermia (NOA).[1] OA, which is caused by excurrent duct obstruction, comprises 40% of all azoospermia cases.[2] Excurrent ductal obstruction, which can occur anywhere along the male reproductive tract (rete testis, efferent ducts, epididymis, vas deferens, and ejaculatory duct) is classically characterized by normal spermatogenesis. In most cases of OA, normal or near-normal sperm production continues in the testis.[3] Microsurgical reconstruction, when possible, is a safe, efficacious treatment in most cases of vasal or epididymal obstruction by vasovasostomy (VV) or vasoepididymostomy (VE), respectively. Occasionally, transurethral resection of the ejaculatory ducts (TURED) is necessary for ejaculatory duct obstruction. In unreconstructable cases, viable sperm can almost always be retrieved for use with in vitro fertilization (IVF)/intracytoplasmic sperm injection (ICSI).

CAUSES

The most frequent causes of vasal obstruction are vasectomy and iatrogenic vasal obstruction, which occurs most often after inguinal hernia repair (either pediatric or adult herniorrhaphy especially when mesh is used).[4] Vasal obstruction may also be caused by previous vasography with improper technique or irritation from contrast

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medium. Epididymal obstruction may occur secondary to existing vasal obstruction (which exerts increased intraluminal pressure leading to microrupture and obstruction of the fragile epididymal tubules). Such epididymal obstruction secondary to previous vasal obstruction may occur in up to 60% of men 15 years after vasectomy.[5] In other cases, epididymal obstruction may occur after trauma. Hydrocelectomy is commonly associated with iatrogenic epididymal injury because the epididymis may be difficult to identify especially in large chronic hydroceles.[6] It is important to recognize that one stitch through an epididymal corpus or cauda tubule can result in complete obstruction of this side. Percutaneous epididymal sperm aspiration (PESA), or less frequently microsurgical epididymal sperm aspiration (MESA) or inadvertently performed epididymal incision or biopsies, may result in iatrogenic obstruction. Congenital abnormalities are a common cause of OA, usually congenital unilateral absence of the vas deferens (CUAVD), congenital bilateral absence of the vas deferens (CBAVD), and, less commonly, partial or complete absence of the epididymis, or idiopathic epididymal obstruction.

In addition to intentional and unintentional surgical obstruction or trauma of the vas or epididymis, genitourinary infection may also cause unilateral or bilateral obstruction. Severe acute or chronic epididymitis, and prostatic or seminal vesicle inflammation can lead to scar formation and eventual obstruction. Idiopathic cases of primary epididymal obstruction are often caused by lower genitourinary tract infection. In addition, Young syndrome, which includes sinobronchial disease and OA caused by thickened secretions and impaired ciliary and sperm tail function, is characterized by the absence of structural abnormalities, normal hormones, and normal testicular biopsy.[7]

In addition to vasal and epididymal obstruction, ejaculatory duct obstruction (EDO) should also be considered in the differential for OA (**Box 1**). EDO, present in less than 5% of azoospermic men, may result from trauma, previous surgery, infection, or congenital prostatic, seminal vesicle, or utricular cyst. In the case of cysts, external pressure from cysts can lead to EDO. EDO is classically defined by low semen volume, low semen pH, absent fructose in semen (basic, fructose-positive fluid is the normal contribution of seminal vesicles), and palpable vas deferens (obviously not found in CBAVD) on physical examination. If EDO is a consideration, improper semen collection (the most common cause of reduced ejaculate volume) and ejaculatory dysfunction (including retrograde ejaculation for which postejaculate urinalysis should be completed) must also be ruled out because the presentation could be similar. In addition, if none of the aforementioned apply, then cystic fibrosis transmembrane conductance regulator (CFTR) mutation may be the cause. Markers of an obstructed system include the absence of marker substances from the epididymis (α-glucosidase), seminal vesicle (fructose), and prostate (zinc) in the semen. However, if obstruction is unilateral or partial, these substances may be present.

Box 1
Causes of obstruction of the genitourinary reproductive system

Epididymis

Infection (acute/chronic epididymitis)

Previous iatrogenic epididymal incision for sperm aspiration/biopsy

Previous scrotal surgery (ie, hydrocelectomy)

Congenital (partial or complete absence of epididymis)

Young syndrome

Vas Deferens

Vasectomy

Iatrogenic: herniorrhaphy or other previous scrotal surgery with accidental vas deferens ligation

CUAVD, CBAVD

Vasotomy/vasography with improper technique

Ejaculatory Duct

Cysts (Mullerian utricular, prostastic, seminal vesicular)

Traumatic

Iatrogenic (postoperative)

Infection (ie, prostatic)

DIAGNOSIS

OA is characterized by normal testicular volume (15–25 mL per testis), normal follicle-stimulating hormone (FSH) level, and azoospermia. History and physical examination are critical to identify predisposing factors for OA. To differentiate OA from nonobstructive azoospermia (NOA), patients should be specifically queried regarding OA and NOA risk factors. OA risk factors include previous vasectomy, other previous surgery (inguinal, scrotal, or genitourinary tract), genitourinary infection, chronic bronchopulmonary infection, or gastrointestinal (GI) abnormality associated with