Genital and Infectious Emergencies: Prostatitis, Urethritis, and Epididymo-orchitis

Hunter Wessells

Introduction

The management of acute genitourinary tract infections continues to evolve as a result of advances in diagnostic testing, emerging antibiotic resistance, and the absence of new antimicrobial agents. Whereas in the past the ability to recognize and treat lower urinary tract and genital infections required diagnostic acumen based on physical findings and microscopic evaluation of urethral swab specimens, sophisticated molecular and radiologic tests allow rapid identification and treatment of acute prostatitis, urethritis, and epididymo-orchitis. These entities usually involve common pathogenic organisms and high-risk patient populations, and many are sexually transmitted diseases (STD). However, each disease entity has a distinct pathology necessitating individual evaluation and management. This chapter limits its scope to acute bacterial infections of the prostate, urethra, and male reproductive organs.

Acute Bacterial Prostatitis

Definition and presentation. The National Institute of Diabetes and Digestive and Kidney Diseases classification system for prostatitis

H. Wessells, MD, FACS (🖂)

syndromes designates acute bacterial prostatitis (acute infection of the prostate) as Category I [1]. The remaining chronic bacterial, chronic nonbacterial, and asymptomatic prostatitis are classified as Category II, III, and IV, respectively. Acute prostatitis is rare and accounts for less than 5 % of all prostatitis syndromes [2].

Acute prostatitis is characterized by rapid onset of fever, chills, low back, perineal/rectal pain, urinary frequency, urgency, nocturia, hesitancy, or sensation of incomplete bladder emptying [3]. Generalized symptoms, such as malaise, arthralgia, and myalgia, may also accompany urologic symptoms. Past medical history may be significant for a prior history of urinary tract infection, indwelling or intermittent urethral catheterization, urethral instrumentation, recent prostate needle biopsy, diabetes, chronic renal insufficiency, and other immunocompromised states.

Physical examination. Patients typically have elevation in temperature and appear clinically ill. The abdominal examination may reveal bladder distension and the genitalia may show an associated urethritis or epididymitis. An exquisitely tender swollen prostate gland is pathognomonic. The consistency of the gland has been described as boggy, irregular, partially or totally firm, or warm to the touch. Aggressive rectal examination or massage is not recommended due to the risk of severe pain and/or bacteremia [4].

Laboratory. Urinalysis is almost invariably abnormal with acute prostatitis. Bacteriuria, pyuria, and

Department of Urology, University of Washington School of Medicine and Harborview Medical Center, 1959 NE Pacific Street, Seattle, WA 98195, USA e-mail: wessells@u.washington.edu

hematuria are common. The midstream urine should be cultured and antibiotic sensitivity should be tested. Other laboratory studies may be indicated including complete blood count with differential, blood cultures (if the patient is febrile), and chemistry studies. *In men with suspected STDrelated prostatitis, urine should also be sent for additional nucleic acid amplification testing to identify chlamydial and gonococcal organisms.*

Microbiology. Microbial pathogens associated with acute prostatitis and prostatic abscess include *E. coli*, other *Enterobacteriaceae*, *Staphylococcus*, *Enterococcus*, *Neisseria gonor-rhoea*, and *Chlamydia trachomatis* [3].

Treatment. Acute bacterial prostatitis (NIH Category I) should be treated promptly with oral fluoroquinolones or sulfonamides. For severe symptoms and signs, including fever and chills, leukocytosis and hemodynamic alterations, patients should be hospitalized and treated with parenteral antibiotics. Recommended doses of fluoroquinolones are ciprofloxacin 500 mg BID or levofloxacin 500-750 mg QD. Alternatively, acute prostatitis can be treated with oral trimethoprimsulfamethoxazole (160 mg TMP and 800 mg SMX). With associated urosepsis, intravenous antibiotics are indicated (ampicillin 2 g intravenously every 6 h and gentamicin 5 mg/kg every 24 h) [2]. From the results of susceptibility testing from urine culture, appropriate changes in antimicrobial therapy should be made. Incomplete patient response may indicate the need to alter antibiotic therapy or investigate the presence of prostatic abscess. Oral therapy should be continued for a total of at least 4 weeks after the diagnosis of acute bacterial prostatitis to prevent the development of chronic bacterial prostatitis [5]. Additional general supportive measures include hydration, analgesics, stool softeners, antipyretics, and bed rest [5].

Related urologic problems. Acute urinary retention due to acute prostatitis (or prostatic abscess) traditionally has been managed with suprapubic cystostomy tube placement. Although anecdotal complications related to urethral catheterization have been reported [6], no prospective studies or large case series have shown a clear relationship between transurethral catheterization and worse outcomes of acute bacterial prostatitis. Thus catheter drainage per urethra has gained acceptance.

Prostatic Abscess

Presentation. Prostatic abscess may be evident at the time of presentation of a patient with acute prostatitis. It may also develop after a course of oral antimicrobial therapy and have a more indolent presentation. Clinical signs of prostatic abscess are variable and include urinary retention, fever, dysuria, frequency, and perineal pain. Presentations can be similar to those of acute prostatitis. Tenderness and fluctuance are unreliable indicators [7]. Patients with diabetes mellitus and acute prostatitis signs/symptoms are predisposed to prostatic abscess formation [8].

Radiologic evaluation. Axial imaging with computed tomography is important to the prompt and accurate diagnosis of prostatic abscess (Fig. 9.1). Transrectal ultrasonography also may be useful for abscess fluid aspiration (perineally or transrectally) for diagnostic and/or therapeutic purposes.

Treatment. Urgent urologic consultation is imperative to ensure prompt drainage of the abscess. In combination with appropriate antimicrobial therapy, incision and drainage will lead to the resolution of most prostatic abscesses. Transurethral, perineal, and transrectal drainage of prostatic abscesses have been described [9–12]. Ultrasound guided transurethral incision or resection (see Fig. 9.2) is now most the common treatment strategy for prostatic abscess [10, 13].

Urethritis

Presentation. Acute urethritis in the male is commonly due to STD (gonococcal and/or chlamydial) and rarely due to *Mycoplasma genitalium*, *Ureaplasma ureolyticum*, or *Trichomonas vaginalis* [14, 15]. Classic gonococcal urethritis produces a profuse, purulent urethral discharge with dysuria. Although gonococcal urethritis can present with scant or absent discharge, this scenario is



Fig. 9.1 CT scan of prostatic abscess demonstrating large low density collection within right side of the prostate (A) distinct from the low density of the urinary bladder (B)

more likely to occur with nongonoccal urethritis (NGU). In all cases, laboratory diagnosis is essential for accurate diagnosis.

Microbiology. Neisseria gonorrhoea (most common cause of urethritis) has an incubation period of 1–9 days. *Chlamydia trachomatis* is responsible for most cases of NGU in the male and has an incubation period of 7–21 days. Coinfection with gonococcal organisms is found in up to 40 % of patients with *C. trachomatis* [16].

Diagnostic testing. Clinic-based diagnostic tools such as Gram-stain microscopy and first void urine microscopy can help establish evidence of urethral inflammation. A urethral specimen collected with a calcium alginate urethro-genital swab is preferred for inoculation of culture medium and gram staining. The gram stain remains highly sensitive and specific for N. gonorrhoeae [17] (gram-negative intracellular diplococci) but not for C. trachomatis. Urethral inflammation, as determined by a gram stain from a urethral swab specimen, in the absence of N. gonorrhoeae, is suggestive of NGU [18]. Standard cell culture techniques remain sensitive and specific for gonococcal and chlamydial urethritis [17, 19]. Nucleic acid amplification testing (NAAT) of both N. gonorrhoeae and C. trachoma*tis* may identify additional infections, either through urethral or urine specimens. These assays enhance the specificity and sensitivity of testing for *C. trachomatis*, especially when the high coinfection rate with *N. gonorrhoeae* is considered [20].

Treatment. Recommended treatments of gonococcal and NGU in men are listed in Tables 9.1 and 9.2 based on data from the Centers for Disease Control and Prevention. Single dose regimens improve compliance, especially if dispensed on site; the first dose can be directly observed.

For *N. gonorrhoeae*, resistance to antimicrobial therapy complicates treatment. Thus, fluoroquinolones are no longer recommended in the US for the treatment of gonorrhea. Furthermore, the frequent coinfection of *N. gonorrhoeae* and *C. trachomatis* now indicate the routine cotreatment with both a cephalosporin *and* azithromycin or doxycycline. For definitive chlamydial infection without gonococcal coinfection, treatment consists of azithromycin or doxycycline. Persistent urethritis after doxycycline treatment may be the result of *Mycoplasma genitalium*, *Ureaplasma ureolyticum*, or *Trichomonas vaginalis*.

Sexual partners (within the preceding 60 days) of patients infected with *N. gonorrhoea* or *C. trachomatis* should be referred for evaluation, testing, and empiric treatment on the



Fig. 9.2 Endoscopic images of transurethral drainage of the prostatic abscess shown in Fig. 9.1. (a) Right lobe of prostate before drainage. (b) Transurethral resection of

basis of contact [21]. Men treated for NGU should abstain from sexual intercourse for 7 days after single dose therapy or until completion of a 7 day regimen. This minimizes transmission or reinfection. Finally, all persons diagnosed with a new STD require testing for syphilis and HIV. portion of right lobe, before unroofing. (c) Purulent drainage into the prostatic fossa

Epididymitis and Orchitis

Etiology. The causes of epididymo-orchitis reflect common causes of genitourinary infection in males based on particular age groups. In children and older men (>35 years), the most common cause of epididymitis is coliform organisms **Table 9.1** Treatment of patients with uncomplicated gonococcal infections of the urethra

Recommended re	egimens
Ceftriaxone	250 mg IM in a single dose
OR, IF NOT AN	OPTION
Cefixime	400 mg orally in a single dose
OR	
Single dose injec	table cephalosporin regimens
PLUS	
Azithromycin	1 g orally in a single dose
OR	
Doxycycline	100 mg orally twice a day for 7 days

Adapted from Centers for Disease Control and Prevention [Sexually Transmitted Diseases Treatment Guidelines, 2010]. MMWR 2010;59(No. RR-5912):[1–116]

 Table 9.2
 Treatment of patients with nongonococcal urethritis

Recommended r	regimens
Azithromycin	1 g orally in a single dose
OR	
Doxycycline	100 mg orally twice a day for 7 days
Alternative regin	nens
Erythromycin	500 mg orally 4 times a day for 7 days
base	
OR	
Erythromycin	800 mg orally 4 times a day for 7 days
ethylsuccinate	
OR	
Levofloxacin	500 mg orally once daily for 7 days
OR	
Ofloxacin	300 mg orally twice a day for 7 days
Adapted from C	enters for Disease Control and Prevention

Adapted from Centers for Disease Control and Prevention [Sexually Transmitted Diseases Treatment Guidelines, 2010]. MMWR 2010;59(No. RR-5912):[1-116]

resulting in bacteriuria. In contrast, the organisms causing urethritis or STD are the common etiologies of epididymitis and orchitis in young adult men (<35 years) [22].

Presentation and differential diagnoses. The clinical syndrome of acute epididymitis (or epididymo-orchitis) results from infection and inflammation of the epididymis and/or testis. It is usually caused by the ascending spread of infection from the urethra or bladder and characterized by progressive increase in pain and swelling of one epididymis and/or testis. It may be associated with fever, lower urinary tract symptoms, and the sensation of a mass in the scrotum [23]. Pertinent information from the history of present illness includes: duration, acuity of onset, location, radiation, associated symptoms, and ameliorating factors. These factors may help to distinguish between epididymo-orchitis and testicular torsion. Epididymo-orchitis usually has a 2-3 day period of progressive increase in scrotal discomfort before severe pain is noted. The pain is localized to the scrotum and does not radiate. Nausea and vomiting are absent. In contrast, testicular torsion has a very acute onset with pain in the testicle possibly radiating into the lower abdominal quadrants. It is usually associated with anorexia, nausea, and/or vomiting.

The past medical history of a patient with epididymo-orchitis may be entirely unremarkable or indicate conditions predisposing to chronic bacteriuria with coliform organisms. These conditions include: congenital urological anomalies (hypospadias, neurogenic bladder, ectopic ureteroceles), practice of anal intercourse, choice of sexual partner (men having sex with men), and acquired obstructive urinary diseases (BPH, prostate cancer, urethral strictures) in older men [24–26].

Physical examination. The physical exam should be focused to differentiate epididymo-orchitis from other urologic and nonurologic conditions. Vital signs are usually normal, but if the temperature is elevated in the patient with epididymitis, a severe infection or associated abscess should be suspected. The abdomen is examined to exclude other intra-abdominal processes (renal colic, appendicitis, hernia) causing radiation to the groin. The penis and urethral meatus are inspected for signs of urethritis, and the scrotum is carefully examined with the patient in the supine position. The overlying scrotal skin is inspected for erythema, fixation, or fluctuance indicating abscess. The palpation of scrotal contents is directed first to the contralateral uninvolved side and subsequently to the affected side. The presence of an ipsilateral cremasteric reflex is a useful adjunctive sign suggesting epididymis rather than torsion [27]. Within the epididymis, the head or tail may be enlarged and tender with or without involvement of the testis and/or surrounding structures. The position of the testis may be helpful in differentiating torsion and infection. Epididymo-orchitis is associated with a normal vertical orientation of the testis and relief of pain with scrotal elevation (Prehn's sign). Torsion may be associated with a high horizontal lie of testis. However, this sign is not uniformly present. With epididymitis, the spermatic cord may be tender and swollen possibly extending into the groin. Importantly, the prostate should be examined to exclude concurrent prostatitis.

Diagnostic testing. Urinalysis or urethral smear can usually determine the microbial cause of epididymitis [23]. For suspected cases of STD (especially in men younger than 35 years), gram stain of the urethral smear is advisable along with subsequent NAAT. A midstream urine specimen should be performed in all patients and examined for the presence of gram-negative bacteria. In less obvious cases of testicular pain without clear evidence of abnormalities on physical exam, the presence of blood on urinalysis should raise the possibility of renal colic, masquerading as torsion or epididymitis, with radiation of pain to the scrotum.

Microbiology. In men under 35 years of age, the most common bacteria causing epididymo-orchitis are STD organisms. The percentage of men in this age group with E. coli infection ranges from 0 to 24 %, most commonly due to anal intercourse [25]. In men over the age of 35, the majority of pathogenic bacteria are due to E. coli, although STD organisms may still cause a substantial amount of epididymo-orchitis [22]. Other rare pathogenic bacteria include Haemophilus influenzae [28]. Rare systemic infections causing epididymitis include: tuberculosis, atypical microbacteria, cryptococcus, brucellosis, and schistosomiasis [29–31].

Radiologic evaluation. Color-duplex scrotal ultrasonography can help to visualize the epididymis, testis, and surrounding tissues and distinguish epididymitis from torsion [32]. Sonographic features of acute epididymitis include enlargement of the epididymis and a primarily inhomogeneous echogenic texture (Fig. 9.3). Echogenic areas within the swollen epididymis and a reactive hydrocoele may be present. Usually the visualized testis is normal, although with orchitis the testis may have increased blood flow with color Doppler (Fig. 9.4) or a diffusely abnormal echogenicity with no residual normal tissue. A helpful finding in inflammation of the scrotal contents is thickening of the superficial skin overlying the testis or epididymis, useful in differentiating a very extensive orchitis from neoplasm. Diffuse inflam-mation of the testis usually causes mild to moderate enlargement with preservation of the normal oval shape and smooth contour of the testis. Focal inflammation of the epididymis can also be delineated on ultrasonography. A localized lesion (enlarged and hypoechoic or with mixed echogenicity) may be compared with the normal contralateral epididymitis. Focal orchitis may show localized areas of decreased echogenicity secondary to the close proximity of the inflamed epididymis [33].

Associated testicular infarction. Testicular infarction (due to compromise of testicular blood flow from edema and a compartment-like syndrome) may occur secondary to epididymo-orchitis [34– 36]. The infarction secondary to acute inflammation may be difficult to differentiate from torsion and requires exploration and orchiectomy [37].

Associated abscess. Abscesses of the epididymis, testis, or scrotum related to acute bacterial or mycobacterial infection are easily detected on ultrasonography. Focal hypoechoic or anechoic regions in the epididymis or testis with involvement of the overlying scrotal soft tissue and skin can usually be differentiated from simple reactive hydroceles (Fig. 9.5).

Treatment. Appropriate antimicrobial therapy for acute epididymitis is based on history, physical examination, and findings from urinalysis and/or urethral smear. Therapy should be instituted empirically and follow-up adjustment based on culture and sensitivities are indicated. For mild to moderate epididymo-orchitis due to bacteriuria, a



Fig. 9.3 Ultrasonographic appearance of acute epididymitis. Note that the epididymis (left-sided structure demonstrating increased color flow on Doppler) is equal in size to the adjacent testis (photograph courtesy of T. Dubinsky, M.D.)



Fig. 9.4 Orchitis. The testis in enlarged and has increased blood flow visible on color Doppler scanning (photograph courtesy of T. Dubinsky, M.D.)



Fig. 9.5 (a, b) Epididymitis with abscess formation. Note reactive hydrocele (dark anechoic fluid) and low density collection within epididymis

10-day course of broad-spectrum fluoroquinolone antibiotic, such as levofloxacin 500 mg orally daily, is recommended. In severe epididymoorchitis associated with systemic illness, a combination of intravenous beta-lactam and aminoglycoside antibiotics is indicated. For epididymo-orchitis due to STD organisms, treatment should include single dose therapy to cover N. gonorrhea and a longer course of therapy for nongonococcal urethritis. Thus the recommended regimen includes ceftriaxone 250 mg IM in a single dose plus doxycycline 100 mg orally twice a day for 10 days. In all cases of acute epididymoorchitis, supportive measures including bed rest, scrotal elevation, nonsteroidal anti-inflammatory drugs, and/or local anesthetic spermatic cord block may be helpful [23].

Indications for scrotal exploration, drainage of abscess, and/or orchiectomy. Antibiotic therapy and supportive measures will allow most cases of epididymo-orchitis to resolve without need for surgical intervention. However, scrotal exploration/drainage or orchiectomy is indicated for abscesses or an infarcted testis secondary to severe infection, respectively. The ultrasonographic presence of fluid adjacent to the testis does not necessitate incision and drainage in all cases. Systemic illness, obvious fluctuance, significant inflammatory changes of the scrotum, spermatic cord, or perineum, or obvious abscess formation with echogenic material indicate the need for scrotal exploration and drainage. A decision regarding orchiectomy can be difficult. However, in many cases, the testis will be extensively involved in inflammatory and infectious processes and requires removal. Abscesses arising solely from an acutely inflamed and infected epididymis may be drained and debrided without the need for orchiectomy. If exploration and drainage is not elected, hospitalization with broad-spectrum intravenous antibiotics, frequent physical examination, and repeat imaging of the scrotum and testicle is advised.

Conclusions

The persistence of unprotected sexual exposures in the United States and other developed and developing countries indicates the continued need to identify and treat prostatitis, urethritis, and epididymitis. Nucleic acid amplification testing and high resolution scrotal ultrasonography are important adjuncts to accurate diagnosis and treatment. For men with prostatitis, the diagnosis rests on accurate history and detailed physical examination. Appropriate use of antimicrobial agents is critical in order to avoid progressively increasing rates of bacterial resistance.

References

- Krieger JN, Nyberg Jr L, Nickel JC. NIH consensus definition and classification of prostatitis. JAMA. 1999;282:236–7.
- Lipsky BA. Prostatitis and urinary tract infection in men: what's new; what's true? Am J Med. 1999; 106:327–34.
- Roberts RO, Lieber MM, Bostwick DG, Jacobsen SJ. A review of clinical and pathological prostatitis syndromes. Urology. 1997;49:809–21.
- Krieger JN. Prostatitis syndromes: pathophysiology, differential diagnosis, and treatment. Sex Transm Dis. 1984;11:100–12.
- Meares Jr EM. Prostatitis syndromes: new perspectives about old woes. J Urol. 1980;123:141–7.
- Pfau A. Prostatitis. A continuing enigma. Urol Clin North Am. 1986;13:695–715.
- Weinberger M, Cytron S, Servadio C, Block C, Rosenfeld JB, Pitlik SD. Prostatic abscess in the antibiotic era. Rev Infect Dis. 1988;10:239–49.
- Dajani AM, O'Flynn JD. Prostatic abscess. A report of 25 cases. Br J Urol. 1968;40:736–9.
- Barozzi L, Pavlica P, Menchi I, De Matteis M, Canepari M. Prostatic abscess: diagnosis and treatment. AJR Am J Roentgenol. 1998;170:753–7.
- Meares Jr EM. Prostatic abscess. J Urol. 1986; 136:1281–2.
- Kadmon D, Ling D, Lee JK. Percutaneous drainage of prostatic abscesses. J Urol. 1986;135:1259–60.
- Cytron S, Weinberger M, Pitlik SD, Servadio C. Value of transrectal ultrasonography for diagnosis and treatment of prostatic abscess. Urology. 1988;32:454–8.
- Trauzzi SJ, Kay CJ, Kaufman DG, Lowe FC. Management of prostatic abscess in patients with human immunodeficiency syndrome. Urology. 1994; 43:629–33.
- Harrison WO. Gonococcal urethritis. Urol Clin North Am. 1984;11:45–53.
- Bowie WR. Nongonococcal urethritis. Urol Clin North Am. 1984;11:55–64.
- Lin JS, Donegan SP, Heeren TC, et al. Transmission of Chlamydia trachomatis and Neisseria gonorrhoeae among men with urethritis and their female sex partners. J Infect Dis. 1998;178:1707–12.
- Manavi K, Young H, Clutterbuck D. Sensitivity of microscopy for the rapid diagnosis of gonorrhoea in men and women and the role of gonorrhoea serovars. Int J STD AIDS. 2003;14:390–4.
- Chernesky MA, Jang D, Lee H, et al. Diagnosis of Chlamydia trachomatis infections in men and women by testing first-void urine by ligase chain reaction. J Clin Microbiol. 1994;32:2682–5.
- 19. Jensen IP, Fogh H, Prag J. Diagnosis of Chlamydia trachomatis infections in a sexually transmitted disease clinic: evaluation of a urine sample tested by enzyme immunoassay and polymerase chain reaction

in comparison with a cervical and/or a urethral swab tested by culture and polymerase chain reaction. Clin Microbiol Infect. 2003;9:194–201.

- Taylor-Robinson D. Tests for infection with Chlamydia trachomatis. Int J STD AIDS. 1996;7:19–25.
- Orr DP, Johnston K, Brizendine E, Katz B, Fortenberry JD. Subsequent sexually transmitted infection in urban adolescents and young adults. Arch Pediatr Adolesc Med. 2001;155:947–53.
- Berger RE, Alexander ER, Harnisch JP, et al. Etiology, manifestations and therapy of acute epididymitis: prospective study of 50 cases. J Urol. 1979;121:750–4.
- Luzzi GA, O'Brien TS. Acute epididymitis. BJU Int. 2001;87:747–55.
- 24. Mittemeyer BT, Lennox KW, Borski AA. Epididymitis: a review of 610 cases. J Urol. 1966;95:390–2.
- Berger RE, Kessler D, Holmes KK. Etiology and manifestations of epididymitis in young men: correlations with sexual orientation. J Infect Dis. 1987; 155:1341–3.
- Hoppner W, Strohmeyer T, Hartmann M, Lopez-Gamarra D, Dreikorn K. Surgical treatment of acute epididymitis and its underlying diseases. Eur Urol. 1992;22:218–21.
- Rabinowitz R. The importance of the cremasteric reflex in acute scrotal swelling in children. J Urol. 1984;132:89–90.
- Thomas D, Simpson K, Ostojic H, Kaul A. Bacteremic epididymo-orchitis due to Hemophilus influenzae type B. J Urol. 1981;126:832–3.
- Skoutelis A, Marangos M, Petsas T, Chionis I, Barbalias G, Bassaris H. Serious complications of tuberculous epididymitis. Infection. 2000;28:193–5.
- Mitchell CJ, Huins TJ. Letter: acute brucellosis presenting as epididymo-orchitis. Br Med J. 1974; 2:557–8.
- Kazzaz BA, Salmo NA. Epididymitis due to Schistosoma haematobium infection. Trop Geogr Med. 1974;26:333–6.
- Hendrikx AJ, Dang CL, Vroegindeweij D, Korte JH. B-mode and colour-flow duplex ultrasonography: a useful adjunct in diagnosing scrotal diseases? Br J Urol. 1997;79:58–65.
- Herbener TE. Ultrasound in the assessment of the acute scrotum. J Clin Ultrasound. 1996;24:405–21.
- Sue SR, Pelucio M, Gibbs M. Testicular infarction in a patient with epididymitis. Acad Emerg Med. 1998;5:1128–30.
- Desai KM, Gingell JC, Haworth JM. Fate of the testis following epididymitis: a clinical and ultrasound study. J R Soc Med. 1986;79:515–9.
- Kirk D, Gingell JC, Feneley RC. Infarction of the testis: a complication of epididymitis. Br J Urol. 1982; 54:311–2.
- Vordermark JS, Favila MQ. Testicular necrosis: a preventable complication of epididymitis. J Urol. 1982; 128:1322–4.