Table 1. Summary of data from cases of aeromonas arthritis.

Patient no.	Reference	Age (y)/ sex	Predisposing factor(s)	Extraarticular infection	Blood culture	Portal of entry	Involved joint	Synovial fluid WBC count in mm^3 (% neutrophils)	Organism
1	[4]	45/M	Alcoholic liver disease	Peritonitis	+	HS	Bilateral glenohumeral	259,000 (NA) 85,000 (NA)	*A. hydrophila*
2	[5]	15/M	None	None	−	Penetrating wound	Knee	NA	*A. hydrophila*
3	[6]	65/F	Acute myeloblastic leukemia	None	+	HS	Left knee	9,800 (45)	*A. hydrophila*
4	[6]	36/M	Chronic myelogenous leukemia	None	+	HS	Right knee	90,000 (97)	*A. hydrophila*
5	[7]	16/M	Acute myelogenous leukemia	Cellulitis	+	HS	Second right metacarpal phalangeal	NA	*A. hydrophila*
6		31/M	Cirrhosis, OLT	None	−	HS	Left knee	104,000 (89)	*A. veronii* (*sobria*)

NOTE. HS = hematogenous spread; NA = data not available; OLT = orthotopic liver transplantation; + = positive; − = negative.

Intestinal permeability is also elevated in cirrhotic patients and has contributed to bacterial infections [10]. *A. veronii* biotype *sobria* differs from the other pathogenic *Aeromonas* species in that it remains susceptible to first- and second-generation cephalosporins [5]. Co-trimoxazole and ciprofloxacin are good choices for oral therapy [2]. Septic arthritis due to *Aeromonas* species is rare. This infection usually occurs in immunocompromised patients.

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Acute Prostatitis with Prostatic Abscess Caused by Group B *Streptococcus*

Group B *Streptococcus* (GBS), or *Streptococcus agalactiae,* causes puerperal sepsis and neonatal infections [1]. The occurrence of invasive infections due to GBS in nonpregnant adults is now well recognized [2]. Numerous cases of upper and lower urinary-tract infections in nonpregnant adults have been reported, but we have found only two cases in the literature (MEDLINE search) of probable prostatitis due to GBS [3] and none of prostatic abscess. We describe a case of prostatic abscess due to GBS.

A 45-year-old male with diabetes mellitus type II controlled by diet was evaluated for complaints of dysuria and perineal discomfort. He started receiving therapy with ofloxacin as an outpatient. Two days later he presented to the emergency room of University Hospital of Geneva (Geneva) for evaluation of suprapubic pain. An urethral bladder catheter was inserted because of acute urinary retention, and a urine culture yielded pure GBS, $>10^5$ cfu/mL. This result was neglected for unknown reasons. Four days later, the patient complained of progressive, unbearable perineal pain as well as fever and chills. He was febrile (temperature, 39°C). The prostate was soft and extremely tender on digital examination. Ultrasonography did not reveal signs of abscess. The urethral catheter was replaced by a suprapubic catheter. Two pairs of blood cultures (BACTEC, Becton Dickinson Europe, Meylan, France)

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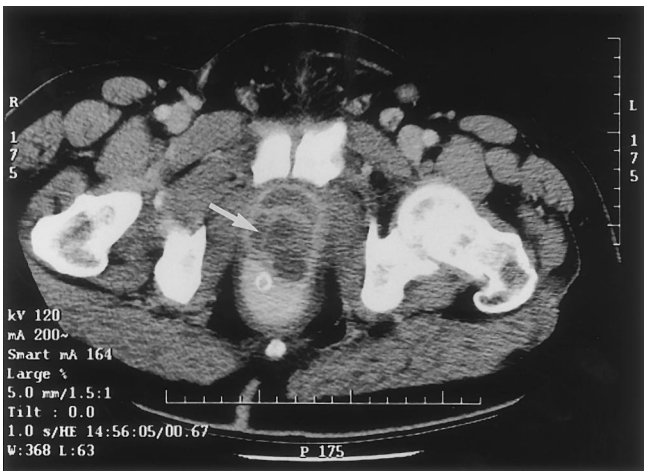

Figure 1. CT scan of a patient with acute prostatitis due to group B *Streptococcus* demonstrates prostatic abscess (*arrow*).

were obtained. All blood cultures yielded GBS, and treatment with penicillin G iv, 3 million units q4h, was started.

Two days later the fever recurred, and the patient complained of new localized perineal pain. On physical examination a tender induration was found in the inguinocrural area behind the spermatic cord. A CT scan showed a significant prostatic abscess (figure 1). Surgical drainage was performed through an inguinocrural approach. Cultures of evacuated purulent material yielded GBS in pure culture. The patient completed a 15-day course of iv penicillin G and was discharged to receive trimethoprim-sulfamethoxazole (TMP-SMZ) double-strength b.i.d. for 1 month.

In a population-based study of nonpregnant adults, Farley et al. [2] found 140 cases of invasive GBS infections, representing an annual incidence of 4.4 per 100,000. Significant risk factors included diabetes (31%), neurological disease (30%), renal failure (18%), and other conditions. Only 1% of cases had no underlying diseases. Although patients with urinary-tract infections were included only if they had concurrent bacteremia, urosepsis occurred in 14%.

In a prospective study of nonpregnant adults, GBS accounted for 2% of urinary-tract infections [4], and 95% of the patients had an underlying condition: intrinsic urinary-tract abnormalities or stones (60%), chronic renal failure (27%), or diabetes (22%). No underlying urinary-tract abnormality was evident on examination of our patient.

Penicillin G is the antibiotic of choice for treatment of infections due to GBS, given that GBS are uniformly susceptible in vitro. High doses (10–12 million units q.d.) are recommended because MICs for GBS are higher than those for group A strains [1]. Quinolones have only moderate in vitro activity against GBS [1, 5].

Our patient developed a prostatic abscess. Diabetes mellitus, insertion of an urethral catheter, and inappropriate initial antibiotic treatment may have all contributed to the occurrence of this rare complication [6, 7]. Various drainage procedures have been described [6, 7]. Although transurethral interventions are preferred, due to the extent of the abscess in our patient, perineal drainage by means of incision was undertaken.

In conclusion, prostatitis, prostatic abscess, or infection due to an unusual pathogen like GBS should be a consideration for male patients with urinary-tract infections that do not respond to standard antimicrobial treatment.

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Marburg and Ebola Hemorrhagic Fevers: Does the Primary Course of Infection Depend on the Accessibility of Organ-Specific Macrophages?

Viral hemorrhagic fevers (VHFs) are prime examples of emerging/reemerging infectious diseases that have increased in frequency worldwide in the past. Of the human VHFs, Marburg and Ebola hemorrhagic fevers are characterized by extreme, severe courses and high case-fatality rates. After the onset of nonspecific symptoms (e.g., fever, headache, and asthenia), patients infected with filoviruses (Marburg and Ebola viruses) display generalized fluid distribution problems, hypotension, coagulation disorders, and hemorrhages, finally resulting in fulminant shock and death [1, 2]. These symptoms are comparable to those of the cytokine-induced systemic inflammatory response syndrome that is a surplus reaction of the host triggered by pathogens or their products [3]. Since filoviruses do not produce substances comparable to the endotoxins or exotoxins of bacteria, the pathophysiology of these devastating infections remains unknown. Because filoviruses are classified as

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