INTRODUCTION

Acute prostatitis is a usual clinical entity affecting men and is most commonly caused by members of the Enterobacteriaceae family. Varicella-zoster virus (VZV) is one of eight herpes viruses known to infect humans. The genome is a linear duplex DNA molecule first sequenced in 1986. Primary VZV infection results in varicella. Varicella is associated with complications such as pneumonia, bronchitis, and encephalitis. VZV has the ability to lie latent within a cell in the nervous system. In 10–20% of patients, VZV reactivates later in life, causing herpes zoster. Its presence in the male genital tract is not previously described. We report the case of an immunosuppressed man discovered to have an acute prostatitis caused by VZV. To the best of our knowledge, this is the first report in the literature.

CASE REPORT

A 66-year-old Caucasian male presented himself in the emergency room (ER) with difficulty in passing urine in the last couple of days, lower abdomen pain, fever, and suspected anuria. Anamnestically, he suffered from rapid progradient glomerulonephritis on the ground of a granulomatous polyangiitis and chronic renal failure stadium V. About 5 weeks before his coming to the hospital, he had the last doses of cyclophosphamide (750 mg, in total 9000 mg). At that time, the kidney retention values remained at a high level, and he started a conservation therapy with azathioprine (150 mg/day). In this setting, the patient was immunosuppressed. In the last week and at the appearance of herpes zoster (buttock left, thigh inside left, penis), the doses of azathioprine were reduced (100 mg/day) and a therapy with acyclovir began (800 mg, twice a day).

Due to this background, an internist treated the patient originally in the ER. He placed a transurethral catheter, because of the suspected anuria, in aseptic conditions and without a complication. The catheter drained 1000 ml of urine from the bladder. At this point, a urological evaluation was requested. The clinical examination of the external genitalia revealed the herpes zoster exanthem of the penis. Testicles

ABSTRACT

Acute prostatitis is a common disease affecting men. In general, viral causes are rare. A total of 7 cases are reported in the literature (6 due to cytomegalovirus and 1 due to adenovirus). We present the first case of an acute prostatitis caused by varicella-zoster virus (VZV). All patients, including ours, were immunosuppressed. A 66-year-old Caucasian male presented himself with lower urinary tract symptoms, lower abdomen pain, fever, and suspected anuria. Anamnestically, he suffered from rapid progradient glomerulonephritis on the ground of a granulomatous polyangiitis and in the last week from herpes zoster. The laboratory results verified the diagnosis of an acute prostatitis due to VZV. Taking into account the clinical findings, we should be aware of the possibility that a lower urinary tract infection affecting an immunosuppressed person could be due to a virus and we should proceed to appropriate laboratory tests. The verification of a viral cause demands an adaptation of the implemented therapy.

Key words: Acute prostatitis, immunosuppression, varicella-zoster virus
and epididymides were without pathologic findings. Digital rectal examination of the prostate was omitted, cause a clinical indication of acute prostatitis was established. The ultrasound imaging showed no hydronephrosis bilaterally, an empty bladder, a correctly-lying catheter balloon, and a prostate volume of approximately 27 ml. The laboratory studies were as follows: creatinine = 7.7 mg/dl, urea = 179 mg/dl, creatinine clearance = 6.7 ml/min, C-reactive protein = 8.01 mg/dl, white blood cells = 7000 T/μl, urine analysis: 4–8 leukocytes/ high power field and 9–15 erythrocytes/high power field, urine culture: sterile (urine sample was obtained through the catheter), and blood cultures for aerobic and anaerobic microorganisms: sterile, prostate-specific antigen (PSA) = 8.24 ng/ml (blood sample was obtained before the catheterization).

Considering the clinical symptoms and findings, a urine test looking for VZV was specifically asked. A polymerase chain reaction testing for the glycoprotein-E region (gpE) of the viral genome was positive, providing the evidence of VZV-DNA, which suggests the presence of VZV in the examined urine sample.

The patient was admitted to the department of internal medicine. After 3 days of an oral intake of tamsulosin 0.4 mg/day, an unsuccessful trial without a catheter was performed and a suprapubic catheter was placed until the transurethral resection of the prostate (TURP), which was scheduled after the definitive treatment of the herpes zoster and the glomerulonephritis.

**DISCUSSION**

Prostatitis is a common diagnosis, but <10% of cases have proven bacterial infection.[3] *Escherichia coli* is the predominant pathogen in acute bacterial prostatitis.[3] Viruses represent a rare causative factor and usually affect immunosuppressed people. Cytomegalovirus (CMV) and adenovirus (ADV) are previously reported to be associated with prostatitis.

McKay et al. first reported in 1994 the case of a patient undergoing chemotherapy for multiple myeloma discovered to have a CMV prostatitis. The diagnosis was histologically verified on transrectal specimen biopsies because of an elevated PSA.[5] Yoon et al. reported a series of 4 cases of CMV prostatitis, which were detected on needle biopsy (3/4) and on TURP specimen (1/4). Only two patients reported lower urinary tract symptoms that could lead thinking toward a diagnosis of prostatitis.[6] Adopting the classification suggested by the National Institute of Diabetes, Digestive and Kidney Diseases (NIDDK) of the National Institutes of Health (NIH), 3 of the above cases are Type IV prostatitis (histological variant). The other 2 cases could be regarded as Type I-like prostatitis since there is no bacterial cause. Moreover, Macasaet et al.[7] Benson et al.[8] and Mastroianni et al.[9] reported CMV prostatitis in autopsy tissue.

Dikov et al. reported a case of necrotizing ADV prostatitis in a patient with terminal AIDS and generalized adenoviral infection.[10] Electron microscopy and immunohistochemistry confirmed the presence of ADV.[10] This case is also a Type IV prostatitis according to the NIDDK-NIH classification.

Taking into account the clinical symptoms, signs, and findings, the laboratory results as well as the fact that VZV was the only pathogenic factor isolated in urine, we consider this case as the first reported acute prostatitis caused by VZV. At this point, we would like to make some comments regarding the reliability of the technique used to isolate the VZV. Whole-VZV-infected cell lysates or purified glycoproteins are used as antigens to detect antibodies against VZV.[11] gpE, B, H, and L, which are structural components of the viral envelope, are the major viral antigens of VZV.[12] VZV gpE is the most abundant viral glycoprotein expressed in VZV-infected cells[13] and has been demonstrated to be highly immunogenic.[14] The purified gpE is proved to be a highly sensitive and specific ELISA antigen for the detection of intrathecal antibody production against VZV.[15] Moreover, a recent research showed that the use of immunohistochemistry for viral infections without a high degree of clinical or histologic suspicion is unnecessary in most cases.[16] On this base, we think that our hypothesis is supported by robust evidence.

Furthermore, in this way, it is demonstrated another aspect of the diversity of the organ manifestations seen in a VZV infection. Another important point is that viruses (CMV, ADV, and VZV) appear to cause prostatic inflammation to men suffering from immunodeficiency (either because of a treatment like post-transplantation antirejection treatment or as a consequence of a disease like AIDS). Within this context, we find it orthologic that viral prostatitis could be distinguished as a separate type of prostatitis and that this fact could be incorporated in the NIDDK-NIH classification.

**CONCLUSIONS**

We should suspect viruses as a potential cause of prostatitis in immunosuppressed men. Thus, we should be alert to make the proper diagnosis on time and modify our therapy accordingly, aiming to maximize the clinical benefit for our patients.

**REFERENCES**


