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Case Report

Septic Shock with Chlamydia Pneumoniae Secondary to Prostatic Abscess: A Rare Case Report

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Abstract: Prostatic abscesses are a rare clinical entity in current practice due to the widespread use of antibiotics. Management usually imposes a challenge to urologists that is due to the difficult diagnosis, as it may mimic other diseases of the lower urinary tract and the lack of guidelines for treatment. Prostate abscess (PA) usually develops in immunocompromised patients, including diabetic and HIV patients, as a consequence of acute bacterial prostatitis. The reason for the lack of guidelines as regards PA is that most of the published data in the literature are case reports due to the declining incidence of the disease today. We presented a male patient who was not foreknown with pathological or personal antecedents or a promiscuous lifestyle. He was hospitalized in the urology section with subfebrility and lumbar pain. His general condition changed rapidly within the span of a few hours, the patient entering septic shock without an etiology or a determined infection. After careful serial investigations, hemocultures (atypical germs) for IgM antibodies *Chlamydia Pneumoniae* were found in the serological complement fixation (cf) test. The patient responded well to empirically initiated antibiotic treatment upon admission to Intensive Care. Due to a favorable evolution, the patient was declared clinically healthy upon discharge. This was a rare case in medical literature of septic shock of initially unspecified etiology but which, upon thorough investigations and urological reevaluation, revealed a prostatic abscess with Chlamydia Pneumoniae, exteriorized through the urethra and highlighted through positive hemocultures only.

Keywords: *Chlamydia pneumoniae*; healthy; male; prostatic abscesses; septic shock; urethra

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Introduction

Prostatic abscesses are a rare clinical entity in current practice due to the widespread use of antibiotics. Management usually imposes a challenge to urologists that is due to the difficult

diagnosis, as it may mimic other diseases of the lower urinary tract and the lack of guidelines for treatment [1]. Prostate abscess (PA) usually develops in immunocompromised patients, including diabetic and HIV patients, as a consequence of acute bacterial prostatitis [2–4]. The reason for the lack of guidelines as regards PA is that most of the published data in the literature are case reports due to the declining incidence of the disease today [5,6].

Some authors suggest that PA is mostly a complication of bacterial prostatitis, whether acute or chronic, most commonly seen in men in their fifth or sixth decade, but which can occur at any age [7]. Before the advent of modern antibiotic therapy, 75% of prostatic abscesses were attributable to *Neisseria gonorrhea*, and the mortality rate was between 6% and 30% [8]. Currently, Enterobacteriaceae, especially *Escherichia coli*, are the predominant pathogens in acute bacterial prostatitis. Less commonly found organisms are *Klebsiella* sp., *Proteus mirabilis*, *Enterococcus faecalis* and *Pseudomonas aeruginosa*, and *Chlamydia trachomatis* [9]. More recently, there has been a rise in the reported cases of methicillin-resistant *Staphylococcus aureus* (MRSA) as the causative agent of PA in literature. Risk factors for MRSA infection include urinary catheter use, health care exposure, history of genitourinary surgery, presence of comorbidities, and increasing age [10].

As with most urinary tract infections, prostatic abscess tends to develop from urinary reflux from the urethra toward the prostatic acini, favored by the different phases of ejaculation and micturition [11]. This means that prostatic abscesses are made up of small micro abscesses that coalesce in order to form larger ones which, eventually, on their natural course, could complicate spontaneous drainage through the urethra [8]. Hematogenous dissemination has also been described from a septic focus from respiratory, digestive, urinary tracts or of soft tissue. In these cases, the most frequent micro-organisms are *Staphylococcus aureus*, *M. tuberculosis*, *Escherichia coli*, and *Candida* sp. [12].

Predisposing factors for PA include an indwelling catheter, instrumentation of the lower urinary tract, bladder outlet obstruction, acute and chronic bacterial prostatitis, chronic renal failure, hemodialysis, biopsy of the prostate, diabetes mellitus, cirrhosis, and, more recently, acquired immunodeficiency syndrome [6]. Urologists should have a high index of suspicion of PA in those groups of patients which are high-risk.

Clinical Presentation

Prostatic abscess can cause a diagnostic dilemma because, in the early stages, prostatic abscess shares signs and symptoms of other diseases of the lower urinary tract. Symptoms and clinical findings of prostatic abscess are extremely variable. Initially, the disease manifests as dysuria, urgency, and frequency in 96% of the cases, fever in 30% to 72%, perineal pain in 20%, and urinary retention in 1/3 of patients [6,8].

Prostatic abscess should be suspected in high-risk group patients presenting with fever and persistent lower urinary tract symptoms who do not respond to antibiotics. A prostatic abscess may progress to spontaneous fistulation into the urinary bladder, prostatic urethra, rectum or perineum. In some cases, it can lead to severe sepsis and death [13]. One of the theories proposed for the development of sepsis in PA is Panton–Valentine leucocidin (PVL), which is a toxin produced by *Staphylococcus aureus* that leads to persistence of infection and aids in the spread of infection [14].

Diagnosis

The most typical sign of prostatic abscess is a severely tender prostate with areas of fluctuation on digital rectal examination, although those findings range between 16% and 88%. Other focal symptoms include perineal pain, obstructive urinary symptoms, and/or

acute urinary retention [13]. Systemic signs could be fever, leukocytosis, and leukocyturia as well [15].

Transrectal Ultrasound (TRUS)

The diagnostic method of choice, which also serves as a treatment and follow-up tool for patients with prostatic abscess, is transrectal ultrasonography of the prostate. The most common finding is the presence of one or more hypoechoic areas, which contain thick pus primarily in the transition zone and in the central zone of the prostate and which are permeated by hyperechogenic areas and distortion of the anatomy of the gland. Transrectal sonography usually underestimates the real periprostatic extension of the abscess [8].

Tomography (CT)

The role of CT examinations is highlighted in diagnosing PA in cases of extraprostatic collections, as CT can accurately detect the extent of spread of the abscess, particularly to the ischiorectal fossa and perineum [16].

Magnetic Resonance Imaging (MRI)

The use of MRI in PA has not been standardized, and only limited studies are available. The MRI characteristics of an abscess are a hypointense signal on T1 and hyperintense on T2 [17].

Differential Diagnosis [18]:

Diagnosis	Distinguishing Characteristics
Benign prostatic hypertrophy	Obstructive voiding symptoms; enlarged, nontender prostate; negative urine culture
Chronic bacterial prostatitis	Recurring prostatitis symptoms for at least three months; positive urine culture with each episode
Chronic pelvic pain syndrome	Pain attributed to the prostate with no demonstrable evidence of infection
Cystitis	Irritative voiding symptoms; normal prostate examination
Diverticulitis	Left lower-quadrant abdominal pain; acute change in bowel habits; history of diverticulitis; tenderness to palpation localized to the left lower abdominal quadrant
Epididymitis	Irritative voiding symptoms; tenderness to palpation on affected epididymis
Orchitis	Swelling, pain, and/or tenderness to palpation in one or both testicles
Proctitis	Tenesmus; rectal bleeding; feeling of rectal fullness; passage of mucus through the rectum
Prostate cancer	Presence of constitutional symptoms; presence of nodules on prostate examination

Treatment

Initial management entails the use of broad-spectrum parenteral antibiotics. This is usually feasible as a single treatment in cases of monofocal abscess cavity <1 cm in diameter. An abscess that fails to respond quickly to antibiotics with no signs of clinical improvement needs surgical intervention and drainage of the abscess with or without urine diversion [19]. Usually, two weeks are needed before antibiotic treatment is deemed a failure and further surgical intervention would be warranted [20].

Typical antibiotic regimens include ceftriaxone and doxycycline, ciprofloxacin, and piperacillin/tazobactam. The risk of nosocomial bacterial prostatitis can be reduced by using

antibiotics, such as ciprofloxacin, before transrectal prostate biopsy. Several methods have been proposed for surgical drainages, all with reported efficacy and feasibility; these are ultrasound guided drainage, transurethral drainage or open drainage [21–23]. Chlamydiae are obligate intracellular bacteria. They lack several metabolic and biosynthetic pathways and depend on the host cell for intermediates, including ATP. Chlamydiae exist as two stages: (1): infectious particles called elementary bodies and (2): intracytoplasmic, reproductive forms called reticulate bodies. The chlamydiae consist of three species, *C. trachomatis*, *C. psittaci*, and *C. pneumoniae*. The first two contain many serovars based on differences in cell wall and outer membrane proteins. *Chlamydia pneumoniae* contains one serovar-the TWAR organism.

Chlamydiae have a hemagglutinin that may facilitate attachment to cells. The cell-mediated immune response is largely responsible for tissue damage during inflammation, although an endotoxin-like toxin has been described [24,25]. Antibodies develop during infection, but they do not prevent reinfection. The precise role of cell-mediated immunity is not known.

The chlamydiae are a small group of nonmotile coccoid bacteria that are obligate intracellular parasites of eukaryotic cells. Chlamydial cells are unable to carry out energy metabolism and lack many biosynthetic pathways; therefore, they are entirely dependent on the host cell to supply them with ATP and other intermediates. Because of their dependence on host biosynthetic machinery, chlamydiae were originally thought to be viruses; however, they have a cell wall and contain DNA, RNA, and ribosomes and are therefore now classified as bacteria. The group consists of a single genus, *Chlamydia* (order Chlamydiales, class Chlamydiaceae). This genus contains the species *C. trachomatis* and *C. psittaci*, as well as a new organism, the TWAR organism, which has recently been proposed as a third species (*C. pneumoniae*). All three species cause disease in humans. *Chlamydia psittaci* infects a wide variety of birds and a number of mammals, whereas *C. trachomatis* is limited largely to humans. *Chlamydia pneumoniae* (TWAR organism) has been found only in humans [26–31].

Case Report

A 51-year-old previously healthy man with a pathological history of kidney stone removal (URS, ESWL) was brought to the County Emergency Hospital of Oradea, in the urology department with suspicion of pyelonephritis because of a history of several days' progressive right lumbalgia, profuse sweating and shivers. On arrival, the patient was feverish, hypotensive with a blood pressure of 90/70 mmHg (Mean arterial pressure (MAP) 76 mmHg), tachycardic, and oliguric. Glasgow coma score (GCS) was 15, temperature 37.8 °C, respiratory rate 15–20 breaths/min. In 30 min from hospital admission, the blood pressure dropped to 70/50 mmHg (MAP 56 mmHg), lactate level 33 mg/dL, and the patient went into septic shock with unknown etiology and was transferred to the intensive care unit.

According to the latest guidelines from Sepsis Surviving Campaign 2016 (SSC) Hour-1 bundle, the patient was fluid-resuscitated (rapid administration of 30 mL/kg crystalloid), supported with vasopressors (Noradrenaline 0.5 μg/kg/min) to maintain MAP > 65 mmHg, and empiric de-escalation antibiotherapy with broad-spectrum antibiotics (Levofloxacin, Meropenem, Vancomycin, Fluconazole) after obtaining blood cultures. We investigated using CT scans (thoracic, abdominal, pelvic) both native and with contrast, abdominal ultrasound, echocardiography. We collected hemoculture, stool sample, clostridium samples, leptospirosis samples, urethral secretions, peripheral smear, virology, and Venereal Disease Research Laboratory VDRL, a febrile sample for atypical germs and sepsis markers in dynamic (Procalcitonin) and repeated arterial blood samples. The patient had urethral catheterization (Foley catheter), and through prostatic massage resulted a purulent discharge (we collected a specimen and sent it to the laboratory for investigation).

Limited laboratory investigations prior to intensive care unit transfer included: severe pancytopenia: leukocyte count 1.129/10^3/uL, neutrophils 0.880/10^3/uL (77.960%), lymphocytes 0.229/10^3/uL (20.290%), monocytes 0.006/10^3/uL (0.515%), hemoglobin 13.9 g/dL, platelet count 108 $\times$ 10^9/L; creatinine 1.23 mg/dL, T-BIL 1.83 mg/dL, and D-bil 0.81 mg/dL GPT/GOT 247/216 U/L (previously documented to be normal); and international normalized ratio (INR) 1.53. CRP 10.61 mg/dL D-dimers 1292 ng/mL. An arterial blood gas revealed a metabolic acidosis with an anion gap of 8. Elevated lactate 33 mg/dL. Dynamic laboratory analyses are in Table 1.

Table 1. Dynamic laboratory analyses.

Patient	Admission	At 6 Hours	Day 2	Day 3	Day 5 and Discharge
WBC (*10^3/uL)	1.129	4.041	9.178	10.090	7.412
NEU (*10^3/uL)	0.880		8.694	8.890	5.415
LYM (*10^3/uL)	0.229		0.156	0.662	1.320
PLT (*10^3/μL)	108.900	94.280	107.400	84.630	131.000
T-BIL (mg/dL)	1.83	1.39		0.91	0.57
GPT/GOT (U/L)	247/216	160/104		101/43	
CRP (mg/dL)		10.61		20.704	
Procalcitonin (ng/mL)		16.45	21.71	21.64	1.34
ASTRUP (arterial)					
pH	7.421		7.405	7.415	
Lactate (mg/dL)	33		13	8	
Base deficit (mmol/L)	−8.6		−9.1	−5.6	
Hco_3 (mmol/L)	18.3		17.9	20.5	
Anion Gap (mmol/L)		8.8	8.6	6.1	
D-dimers (ng/mL)		1292		886	498
Fibrinogen (mg/dL)		208		480	
PO_2	58.3	78.3	83.4	88	
PCO_2	23.6	28.9	32.2	35.3	
SO_2	91.8	96.5	97	98	

Admission scores: The APACHE II score, calculated within the first 24 h of admission, was 25 points, with a predicted mortality of 60.3%, SOFA score 6 points, and **qSofa 3 points**.

The evolution of the patient under empirical antibiotherapy therapy of de-escalation and volumetric filling is clinically and paraclinical favorable (according to the serial blood analyses in Table 1), and the vasoactive support is progressively reduced, suppressing the second day of therapy. The general condition is improved, the patient being conscious, cooperative, hemodynamically and respiratory stable, afebrile, with present diuresis, mobilized, and fed through the mouth from the second day of admission to intensive care. On day 3, the antibiotic was adjusted according to the antibiogram for *Chlamydia Penumoniae*, the germs being sensitive to Levofloxacin and Meropenem, and the patient was transferred to the Urology department, where the targeted antibiotic therapy was continued. On the fifth day from admission, the patient was discharged clinically healthy. The CT scan showed no pulmonary dysfunction, normal kidney without hydronephrosis (though light perirenal fat infiltration could possibly suggest pyelonephritis), the prostate 44/63 mm without focal lesions, and hepatomegaly (Figures 1–3). The abdominal and pelvic echography showed only hepatomegaly and a large prostate, homogenous. The echocardiography: normal value. TRUST (Transrectal Ultrasound) was not performed on this patient during his Intensive Care Unit stay.

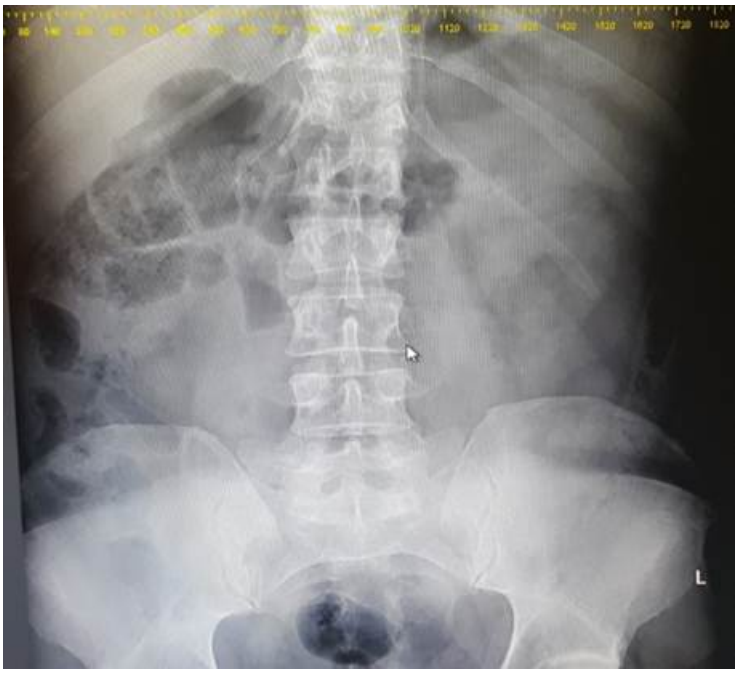

Figure 1. Abdominal scan (hepatomegaly, without hydronephrosis but light perirenal fat infiltration, normal spleen, and normal pancreas).

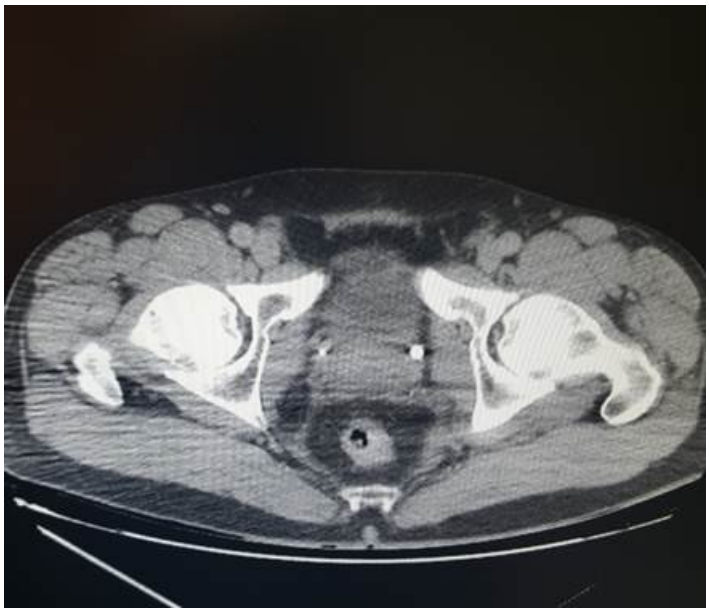

Figure 2. Pelvis scan (prostate 44/63 mm without focal lesions, no lymphadenopathy, no intraperitoneal fluid).

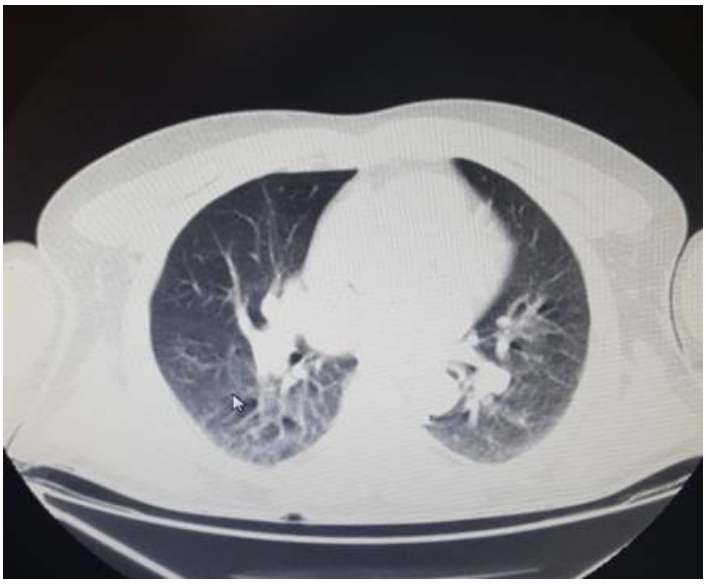

Figure 3. Thorax scan (normal).

Conclusions

We presented a male patient who was not foreknown with pathological or personal antecedents or a promiscuous lifestyle. He was hospitalized in the Oradea County Hospital, in the urology department with subfebrility and lumbar pain. His general condition changed rapidly within the span of a few hours, the patient entering septic shock without an etiology or a determined infection. After careful serial investigations, blood samples (atypical germs) for IgM antibodies Chlamydia Pneumoniae in the titer of 1:80 were found positive in the serological complement fixation (cf) test. The patient responded well to the antibiotic treatment initiated empirically upon admission to the Intensive Care Unit, the germs being sensitive to some antibiotics in the scheme. Due to a favorable evolution, the patient was declared clinically healthy upon discharge, having recovered 100% only 5 days after his hospital admission.

Particularities

This was a rare case in medical literature of septic shock of initially unspecified etiology but which, upon thorough investigations and urological reevaluation, revealed a prostatic abscess with Chlamydia Pneumoniae, exteriorized through the urethra and highlighted through positive blood samples only.

Conflicts of Interest: The authors declare no conflict of interest.

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