AUCTORES
Globalize your Research

Journal of Endocrinology and Disorders

Anthony Kodzo-Grey Venyo *

Open Access

Research Article

Candida Prostatitis: A Review and Update

Anthony Kodzo-Grey Venyo *

North Manchester General Hospital, Department of Urology, Delaunays Road, Manchester, M8 5RB. United Kingdom.

***Corresponding Author:** Anthony Kodzo-Grey Venyo, North Manchester General Hospital, Department of Urology, Delaunays Road, Manchester, M8 5RB. United Kingdom.

Received Date: 27 May 2023 | **Accepted Date:** 12 June 2023 | **Published Date:** 19 June 2023

Citation: Anthony Kodzo-Grey Venyo, (2023), Candida Prostatitis: A Review and Update, *J. Endocrinology and Disorders*. 7(3):

DOI: 10.31579/2640-1045/137

Abstract

Candidiasis is a terminology that is used for a fungal infection that is caused by a yeast which is a fungus that called candida species. Some species of candida can cause infection in human beings and the commonest candida species that can infect human beings is candida Albicans. Candida normally lives upon the skin as well as inside the human body, such as the mouth, the throat, the gut, and the vagina, without causing problems. Candida can cause infections if it grows out of control or if it enters deep into the body. For example, it could cause infections in the bloodstream or internal organs like the kidney, the heart, or the brain. Candida infection can be localized to one body organ or it could be a disseminated candida infection affecting various organs. Candida infection does tend to afflict immune-supressed individuals more often in comparison with immune competent individuals. Candida prostatitis an uncommon condition which clinicians should have a high index of suspicion for because it does manifest with non-specific symptoms that tend to be associated with more common conditions of the prostate and the urinary tract. Candida infection of the prostate gland may be an isolated de novo infection or it may be associated disseminated candida infection, or it may also be associated with prostate cancer or various immune-suppression conditions. Candida infection of the prostate gland could manifest as an acute infection/inflammation of the prostate gland or a chronic inflammation of the prostate gland or it could manifest as prostatic abscess. Some of the potential manifestations of candida prostatitis and or prostatic abscess include: (a) incidental finding upon biopsy of the prostate gland or following trans-urethral resection of prostate gland or prostatectomy. (b) patient may manifest with lower urinary tract symptoms, (c) a patient may manifest with urinary retention that may be acute or chronic, (d) a patient may manifest with raised levels of serum prostate specific antigen (PSA), € at times when digital rectal examination is undertaken on a patient who has Candida prostatitis, the prostate gland may feel benign and in the case of a candida prostatic abscess rectal examination may demonstrate bogginess in the area of the prostate with soft fluctuant feeling. (f) Eosinophil count in some cases of Candida prostatitis would tend to be normal but in some cases of candida infection of the prostate gland, there could be Eosinophilia but this would not be diagnostic of Candida infection, (g) a history of having had coital contact with an individual who has been treated for candida infection or a history of past treatment of the individual should alert all clinicians to exclude the possibility of candida prostatitis. (h) on rare occasions urine culture or culture of expressed prostatic secretions would yield a growth of Candida. Diagnosis of Candida infection or abscess of the prostate gland may be confirmed by positive culture of Candida in prostate biopsy specimen or resected or excised prostate specimen. Treatment of Candida Prostatitis / prostatic abscess does tend to entail: (a) Treatment with appropriate antifungal medicament, plus (b) Complete radiology image-guided aspiration / drainage of any abscess seen plus / minus or endoscopic deroofing trans-urethral resection of the prostate to ensure the abscess drains out completely. Because recurrence of Candida prostatitis or prostatic abscess or Candida infection elsewhere can occur, it is important for patients to have regular follow-up assessments to ensure recurrence disease does not develop and if it develops, it is diagnosed quickly in order to initiate prompt treatment. It is also important to assess all coital contacts of the patient to ascertain if they have Candida infection to enable prompt treatment of their infection. If an individual who has candida prostatitis or prostatic abscess is also found to have contemporaneous adenocarcinoma of the prostate, the carcinoma of the prostate gland should be treated appropriately based upon the Gleason Grade and the Stage of the carcinoma, the performance status and age of the individual patient based upon the national and international guidelines pertaining to prostate cancer as a separate multi-disciplinary team discussion of the patient management.

Key words: candida prostatitis; candidal prostatitis; candida prostatic abscess; candidal prostatic abscess; lower urinary tract symptoms; serum prostate specific antigen; inflammation of prostate; biopsy of prostate

Introduction

Prostatitis is a common clinical entity that tends to be seen by many General Practitioners and Urologists. Prostatitis is said to be an umbrella terminology which is utilized for various medical clinical conditions which incorporate bacterial as well as non-bacterial origin illness within the pelvis region [1]. It has been pointed out that in contrast to the plain meaning of the word prostatitis which does appear to mean inflammation of the prostate gland, the diagnosis or the commonly used terminology of prostatitis may not always include evidence of inflammation within the prostate gland [1]. Prostatitis may be classified at times into acute prostatitis, chronic prostatitis, asymptomatic inflammatory prostatitis and chronic pelvic pain syndrome [1]. others may classify prostatitis into various forms including, acute prostatitis, acute on chronic prostatitis, chronic prostatitis, bacterial prostatitis and non-bacterial prostatitis. acute prostatitis, acute on chronic or chronic prostatitis. Some types of prostatitis may be associated with acute prostatic abscesses or chronic prostate abscesses. Prostatitis may manifest with lower urinary tract symptoms (LUTS) which tends to be attributable to acute and chronic bacterial infections (NIH Category I/II) or as asymptomatic inflammatory prostatitis (NIH Category IV). [2] Patients who have chronic prostatitis/chronic pelvic pain syndrome, (CP/CPPS, NIH Category III) may manifest with a wide range of symptoms resulting from varied aetiology; nevertheless, prostatitis is on rare occasions caused by fungal infections [2].

It has been iterated that within the United States of America (USA), prostatitis is diagnosed in 8% of all male Urologist visits and 1% of all primary care physician visits for male genitourinary symptoms [3].

With regard to classification of prostatitis, it has been iterated that the terminology prostatitis does refer to inflammation of the tissue of the prostate gland [2]. It has been pointed out that prostatitis may occur as an appropriate physiological response to an infection, or it may occur in the absence of infection [3].

It has been pointed out that inn 1999, the National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK) had devised a new classification system as illustrated in table 1. [4,5].

Table 1:

Category 1: Acute bacterial prostatitis, the old name was acute bacterial prostatitis and the new name acute bacterial prostatitis, this is associated with pain, this is associated with presence of bacteria, this is associated with white blood cells; this is associated with bacterial infection of the prostate gland, which requires medical treatment, this is associated with bacteria.

Category 2: Chronic bacterial prostatitis, the old name was chronic bacterial prostatitis, this may or may not be associated with pain, this is associated with white blood cells, this is a relatively rare condition that usually presents as intermittent urinary infections.

Category IIIa: Inflammatory Chronic Pelvic Pain syndrome. [Inflammatory CPPS], the old name was non-bacterial prostatitis, this is associated with pain, no bacteria are found in the prostate, white blood cells are found in the prostate, Category IIIa and III b account for 90% to 95% of prostatitis diagnoses, Category IIIa and Category III b were formerly known as chronic nonbacterial prostatitis.

Category IIIb: Non-inflammatory Chronic Pelvic Pain Syndrome. [Non-Inflammatory CPPS], old name proctodynia, this is associated with pain, no bacteria are found in the prostate, no white blood cells are found in the prostate, Category IIIa and III b account for 90% to 95% of prostatitis diagnoses, Category IIIa and Category III b were formerly known as chronic nonbacterial prostatitis [5].

Category IV: Asymptomatic inflammatory prostatitis, there was no name associated with this category, this is not associated with any pain, no bacteria are found in the prostate, white blood cells are found in the prostate gland, no history of genitourinary pain complaints are made but leucocytosis is noted, usually during evaluation of other conditions. Between 6% and 19% of the men have pus cells within their semen, but no symptoms are manifested [6-8].

It has been documented that in 1968, Meares and Stamey had determined a classification technique based upon the culturing of bacteria. [2-9] This classification is no longer used.

It has been pointed out that the conditions in prostatitis are distinguished by the different manifestation of pain, white blood cells (WBCs) within the urine, duration of symptoms and bacteria cultured from the urine. To help express prostatic secretions that may contain WBCs and bacteria, prostate massage is sometimes used [10].

Occasional case reports had been published on prostatitis which has been caused by Candida. In view of the fact that prostatitis that caused by Candida is not common, it would be envisaged that many clinicians globally may not have encountered or treated a case of Candida prostatitis before and they may not be familiar with the manifesting features, diagnosis and management of candidiasis of the prostate gland before. The ensuing article on Candida prostatitis is divided into three parts: (A) Overview of Prostatitis in general, (B) Overview of Candida, and (C) Miscellaneous Narrations and Discussions from Some Case Reports, Case Series, and Studies Related to Candidiasis and Candida Abscess of the Prostate Gland.

Methods

Internet data search bases were searched including: Google; Google Scholar; Yahoo and PUBMED. The search words that were used included: Candida Prostatitis; Candidal Prostatitis; Candida prostatic abscess: Candidal Prostatic Abscess. Seventy-seven (77) references were identified which were used to write the article which has been divided mainly

into three (3) parts including: (A) Overview of Prostatitis in general, (B) Overview of Candida, and (C) Miscellaneous Narrations and Discussions from Some Case Reports, Case Series, and Studies Related to Candidiasis and Candida Abscess of the Prostate Gland.

Results

[A] Definition / general statements

The ensuing general statements and definitions related to prostatitis had been made [11].

- Prostatitis is a clinical terminology only, so it has been advised that prostatitis should not be diagnosed on pathology reports [11].
- It has been advised that anatomic pathologists should simply diagnose chronic, acute or granulomatous inflammation of the prostate gland [11].
- It has been advised that the diagnosis of prostatitis should be based upon symptoms of pelvic pain and sexual dysfunction, with or without bacteria, based upon quantitative bacterial cultures and microscopic examination of fractionated urine specimens (first 10 mL of urine is urethral, midstream urine is from urinary bladder) and expressed prostatic secretions

Essential features

- It has been documented that with regard to the essential features of prostatitis, either the finding upon pathology examination of specimens of the prostate gland demonstrating lymphocytes, neutrophils, eosinophils or histiocytes should be evident.
- Diagnosis of prostatitis becomes evident or made when the prostate infections are caused mainly by gram negative rods [11].

Terminology

- It has been stated that a terminology that tends to be used for prostatitis is inflammatory disease of the prostate gland [11].

Epidemiology

Some of the salient relevant points related to the epidemiology of the prostate gland which had been made include [11].

- It has been pointed out that chronic prostatitis / chronic pelvic pain syndrome (CPPS), which is also known as NIH category III prostatitis, abacterial prostatitis or prostatodynia, is the most common urological diagnosis in men who are older than 50 years [11].
- It has been pointed out that current evidence had indicated that an inverse relationship has been documented between volume of atrophy with or without inflammatory cells and cancer [12].
- It has been iterated that clinically diagnosed prostatitis might increase prostate cancer risk and this may be race dependent; whereas histological prostatic inflammation can increase serum PSA and produce a false positive result which does decrease the likelihood of cancer detection [13,14]

Sites

- It has been pointed out through a documentation that prostatitis can affect central and peripheral zones of the prostate gland [15].

Pathophysiology

The pathophysiology of prostatitis has been summated as follows [11].

- It has been stated that histological prostatitis is used or diagnosed, when the inflammation features are found present along with hyperplasia (BPH), when it correlates with greater lower urinary tract symptoms, and prostate gland volume and risk of acute urinary retention; and this suggests that prostatitis plays a role in the progression of BPH [16].
- It has been iterated that reflux of infected urinary contents into prostatic ducts does play a part in the pathophysiology of prostatitis and this is a postulated cause documented by some authors to explain the way prostatitis develops in some cases [17].
- Some of the documented risk factors for the development of prostatitis include: indwelling catheter, underlying voiding dysfunction, poorly controlled diabetes, end stage renal disease (ESRD), cirrhosis, immunosuppression [18].

Aetiology

The aetiology of prostatitis has been summed-up as follows [11].

- **Abacterial prostatitis:** It has been iterated that abacterial prostatitis has been referred to or defined as a heterogeneous condition with many possible causes; nevertheless, aberrant cytokine function seems to be a final common pathway [19].
    - It has been pointed out that a newly recognized cause of abacterial prostatitis is IgG4 related prostatitis, which presents with obstruction and resolves with steroids and rituximab [20].
- **Bacterial prostatitis**: It has been documented that Escherichia coli does account for > 70% of cases of bacterial prostatitis, and Klebsiella, Pseudomonas, Proteus, Enterobacter, Enterococcus species, Staphylococcus aureus are some of the other common causes of bacterial prostatitis and Staphylococcus aureus can also cause prostatic abscess as one of the causes of prostatic abscesses.

It is worth noting that other causes of acute and chronic prostatic abscess exist apart from the aforementioned bacterial organisms)

Clinical features [11]

- Systemic:
    - Headache, fever, chills and general malaise, low back pain
- Prostate (local) [21].
    - Severely tender prostate with areas of fluctuation on digital rectal examination
    - Perineal pain
    - Dysuria, urinary urgency or frequency, haematuria, purulent urethral discharge
    - Obstructive urinary symptoms

Diagnosis [11]

- Overall, history and physical findings, complete blood count with differential, urine analysis, blood culture, urine culture and even PCR (detection of sexually transmitted organisms) may be used
- For specific types:
    - Chronic abacterial prostatitis / chronic pelvic pain syndrome (CPPS):
        - Includes prostatodynia, category III or abacterial prostatitis
        - Defined by the International Prostatitis Collaborative Network under the National Institute of Health, and diagnosis follows clinical, microbiological and laboratory criteria [22,23]
        - Histopathologic diagnosis is less crucial or may not be required for diagnosis
        - Clinically similar to bacterial prostatitis, with persistent pain, especially after ejaculation; but no bacteria are cultured from expressed prostatic secretions (EPS)
        - Excessive white blood cells (WBC) may be present or absent
        - WBC number fluctuates within the same patient and does not correlate with symptom severity
        - Essentially incurable
    - Acute bacterial prostatitis:
        - Same bacteria types as urinary tract infections (*E. coli*, gram negative rods, enterococci, staphylococci), usually due to reflux, also following surgical manipulation or sexually transmitted disease
        - Usually localized, may cause obstruction, retention, abscess
    - Chronic bacterial prostatitis:
        - Symptoms of low back pain, dysuria, perineal and suprapubic discomfort
        - Often have history of urinary tract infection by same organism
        - May have no symptoms
    - Granulomatous prostatitis:
        - Necrotizing or non-necrotizing granulomas may be seen in men who have undergone bacillus Calmette-Guérin (BCG) treatment for bladder cancer
        - Also occurs post-transurethral resection
        - Otherwise, most cases are idiopathic and do not require acid fast stain
    - Eosinophilic prostatitis:

        - According to case reports, this is capable of increasing serum PSA levels [24].
    - IgG4 related prostatitis:
        - IgG4 related disease is a rare but under recognized type of chronic abacterial prostatitis that can cause urinary obstruction in young men [25].
        - Elevated serum IgG4 levels are diagnostic; serum PSA should be normal
        - There may be chronic sclerosing sialadenitis
        - Biopsy shows prominent plasma cells; if the symptoms resolve following treatment with steroids, there is no need for prostate biopsy

Laboratory tests [11]

- Bacterial: prostatic secretion cultures should have bacterial counts 10x higher than urethral / bladder cultures
- Nonbacterial: > 10 WBC/HPF in prostatic secretions without pyuria
- Acute and chronic bacterial prostatitis can raise the serum PSA above normal [26].

Radiology description [11]

- Computed tomography (CT scan) of the abdomen and pelvis [27].
    - Can better delineate the spread of infection to adjacent organs
- Magnetic resonance imaging (MRI) [28].
    - Hypointense signal on T1 and hyperintense on T2 image

In addition to the above. It is worth pointing out that ultrasound scan can be useful with regard to the assessment of cases of prostatitis and prostate abscess. **T**he value of computed tomography (CT) scan and sonography in the diagnosis and follow-up of abscesses of the prostate was studied in six patients with this disease. Five men had undergone CT scan alone, one had CT scan and ultrasound scan, and one had sonography only. The CT scan findings included an enlarged gland with non-enhancing fluid-density collections which sometimes were multiseptated or had enhancing rims. The sonographic findings were similar, showing a hypoechoic mass with thick walls. Follow-up examinations after antibiotic therapy (one CT, one sonogram) showed improvement or resolution. In the patients who had been studied, CT and sonography were useful methods to detect and follow the course of prostatic abscess. [27].

Prognostic factors [11]

- Depends on the timely diagnosis and treatment and type of prostatitis

Treatment [11]

- Difficult because antibiotics penetrate poorly into prostate

Microscopic (histologic) description [11]

- WBCs in biopsied prostatic tissue do not correlate with the degree of pain in chronic prostatitis / CPPS
- Density of lymphocytes in the prostate is remarkably constant across age groups and races [29].
- Lymphoid aggregates are not specific for prostatitis but may be part of hyperplasia
- Neutrophils tend to be in lumen spaces and macrophages in stroma; however, this is not specific for prostatitis

Positive stains [11]

- For bacterial type, bacteria may be visible upon Gram staining

Differential diagnoses

Some of the documented differential diagnose include the ensuing: [11]

- **Acute bacterial prostatitis**:
    - Clinically and histologically is similar (neutrophils are mostly in lumens and epithelium)
    - No fluctuant mass during a rectal examination
    - No hypoechoic areas on ultrasound
- **Chronic bacterial prostatitis**:
    - Similar clinical symptoms but lower intensity
    - Predominant lymphocytic infiltration (lymphocytes are mostly in the stroma)
- Granulomatous prostatitis
    - Fever and chills are common
    - Irritative voiding symptoms of urgency, frequency
    - May have history of bladder cancer BCG treatment
    - Necrotizing or non-necrotizing granulomas (cohesive clusters of histiocytes, usually with admixed lymphocytes, eosinophils, neutrophils)
- Carcinoma of prostate gland
    - May mimic benign acini with reactive inflammatory atypia
    - Typically lack background inflammation
    - Small glands, sometimes medium to large glands, papillary or cribriform glands or solid growth or single cells
    - Nucleomegaly, prominent and multiple nucleoli, lack basal cell layer

[B] Overview of Candida infections pertaining to the skin

Definition / general

The ensuing statements had been made [30].

- Candida albicans is a part of the normal human skin flora
- Cutaneous candidiasis of candidiasis of the skin is a superficial infection of skin and mucous membranes and the most common Candidal infection

Terminology

Some other terminologies that tend to be used by various people and groups for cutaneous candidiasis include the following [30].

- Oral candidasis
- Candidal intertrigo (affects body folds): acute (wet and red), subacute (red +/- maceration), or chronic (red and dry)
- Candidal diaper dermatitis
- Candidal vulvovaginitis
- Candidal balanitis
- Candidal nail infection: chronic paronychia, onycholysis

Epidemiology

Some of the general summating statements that had been made pertaining to cutaneous candidiasis include the following [30].

- Within the United States of America, (USA) Candida species are a common cause of intertrigo in both elderly and diabetic patients
- Candida species tends to colonize the oropharynx in 30% to 55% of healthy young adults, and tends to be commonly found in normal faecal flora

- Three (3) out of four (4) women would have at least one Candidal vulvoganitis during their lifetime
- With regard to patients who have systemic infections, Candida species, recently, has been the 4th commonest pathogen from blood cultures
- Higher than 90% of HIV population who are not on highly active antiretroviral therapy will develop oropharyngeal candidiasis and 10% develop oesophageal candidiasis
- Internationally, Candida species had replaced Cryptococcus species as the commonest fungal pathogens which affects immunocompromised hosts

Clinical features [30]

- Some relevant summating iterations made in relation to the predisposing factors for opportunistic infection with Candida. *albicans* include:[30]
    - Infancy or elderly
    - Warm climate
    - Occlusive clothing, poor personal hygiene, dental plates
    - Immune deficiencies (low levels of immunoglobulins, HIV, cancer)
    - Broad spectrum antibiotic treatment
    - High dose oestrogen contraceptive pills or pregnancy
    - Chemotherapy or immunosuppressive medications such as systemic steroids
    - Locally applied topical steroids
    - Diabetes mellitus, obesity, Cushing syndrome and other endocrine conditions
    - Iron deficiency
    - Malnutrition
    - Underlying dermatological disease like psoriasis, lichen planus, irritant contact dermatitis
    - Mortality is relatively low for cutaneous Candidal infection in healthy patients; however, the mortality rate is up to 30% to 40% in disseminated / systemic candidasis in immunosuppressed patients

- The characteristic skin manifesting features of Candida skin infections include: red and white patches upon mucosal surfaces that are referred to as leucoplakia. [30]
- In skin folds, candida infection results in moist fissuring with a superficial erythema patch with satellite papulopustules [30]

Diagnosis

Some relevant iterations that had been made regarding the diagnosis of candida infection included the following: [30]

- Potassium hydroxide (KOH) preparation and skin scraping is the easiest and most cost-effective method for diagnosing cutaneous candidiasis

- Culture from intact pustule or skin biopsy tissue is able to support the diagnosis

With regard to candida of the prostate pathology examination of specimens of the prostate gland of biopsy specimen would demonstrate features of candida and inflammation within the prostate as well as culture from aspirates or biopsy of the prostate would grow candida

Treatment

- With regard to the treatment of candida infection, it has been advise**d** that clinicians should confirm the accuracy of medications below before they are used. [30]

- **Oral candidiasis: [30]**
    - Nystatin oral suspension x 10 - 14 days or until 48 - 72 hours after resolution of symptoms
    - The dosage for preterm infants is 0.5 mL (50,000 U) to each side of mouth 4 times / day; for infants is 1 mL (100,000 U) to each side of the mouth 4 times/d; for adults 4 - 6 mL (100,000 U) PO swish and swallow four times per day (QID)

- **Candidal intertrigo:**
    - Clinicians had been advised to keep the skin dry, with the addition of topical nystatin powder, clotrimazole, or miconazole twice daily, often in conjunction with a mid-potency corticosteroid [30].
    - It had also been advised that an extensive infection may require the addition of fluconazole (100 mg orally qd for 1 week to 2 weeks) or itraconazole (100 mg PO qd for 1 week to 2 weeks) [30].

- **Acute intertrigo:**
    - With regard to acute intertrigo, it has been advised that clinicians could use Domeboro solution, Castellani paint or vinegar/water (1 table spoon of vinegar per quart room-temperature water) to apply twice per day for 5 minutes to 10 minutes for 3 days to 5 days as needed [30].
    - Clinicians had been advised to dry the area with a hair dryer (low heat) [30].
    - Clinicians had also been advised that they could also apply triamcinolone-nystatin cream twice daily [30].

- **Subacute intertrigo:**
    - With regard to sub-acute intertrigo, it has been recommended that clinicians can use benzoyl peroxide wash to cleanse the area instead of application of vinegar or Castellani paint [30].
    - It has furthermore been advised that a topical anticandidal cream of choice should be applied twice per day, with or without a mild hydrocortisone cream [30].

- **Chronic intertrigo:**
    - With regard to chronic intertrigo, it has been advised that clinicians could utilize zinc-talc shake lotion once or twice daily, and the hydrocortisone cream / antifungal mixture could be applied at night [30].
    - It has also been stated that Local hyperhidrosis could be treated with antiperspirants (for example, and Extra Dry

Unscented, Dry Idea) on a long-term basis [30].

- **Candidal diaper dermatitis:**
    - It has been stated that the goal of treating Candida diaper dermatitis is to minimize the time the diaper area is exposed to hot and humid conditions; air drying, frequent diaper changes and generous use of baby powders and zinc oxide paste are adequate preventive measures [30].
    - It has also been advised that in cases of Candida diaper dermatitis, clinicians should apply topical nystatin, amphotericin B, miconazole or clotrimazole to the affected areas twice daily for 7 days [30].
- **Candidal vulvovaginitis:**
    - It has been stated that with regard to Candida vulvovaginitis, clinicians should recommend the use of topical antifungal agents (Micatin, Monistat-Derm), or clotrimazole (Lotrimin, Mycelex) creams twice daily for7 days or intravaginal appliator QHS x 7 days are curative [30].
    - It had also been iterated that one-time oral treatment with fluconazole (150 mg) or itraconazole (600 mg) is effective and this may be a more attractive alternative treatment to some patients, but it is more costly [30].
- **Candidal balanitis:**
    - It had been stated that with regard to Candida balanitis, topical treatment is effective in majority of patients [30].
    - It has been advised that in cases of Candida balanitis, clinicians should also evaluate asymptomatic sexual partners and treat them if they are infected to prevent recurrence [30].
    - It has furthermore been recommended that with regard to persistent lesions beyond the genitalia, clinicians should consider the possibility of underlying diabetes mellitus or other diseases [30].
- **Candidal paronychia:**
    - With regard to the treatment of candida paronychia it has been stated that topical therapy had usually not ben effective but it should be tried for chronic candida paronychia [30].
    - It had also been documented that drying solutions or antifungal solutions are used for the treatment of Candida paronychia [30].
    - It has furthermore been iterated that oral treatment with either itraconazole (pulse dosing with 200 mg bid for 1 week of each of 3 consecutive months) or terbinafine (250 mg qd for 3 months) is recommended for the treatment of Candida paronychia [30].

Cytology description

- It has been pointed out that with regard to the cytology examination features of Candida, Periodic Acid-Schiff (PAS) staining of the specimen does demonstrate non-septated hyphae, which distinguishes Candida species from tinea [30].

Positive stains

With regard to the staining features of Candida, it has been iterated that Candida specimens do stain for the following: [30].

- Gomori Methenamine-Silver Nitrate (GMS) stain, and for
- Periodic Acid Schiff (PAS) stain.

Differential diagnoses

Some of the differential diagnoses of Candida infections of various parts of the body had been iterated to include the following: [30].

- Bacterial vaginitis.
- Contact dermatitis with or without colonization.
- Intertrigo
- Inverse psoriasis
- Onychomycosis
- Pseudomonas nail bed infection.
- Radiation-induced dermatitis.
- Seborrheic dermatitis.
- Trichomonas infection.

[C] Miscellaneous Narrations and Discussions related to Some Case Reports, Case Series and Studies related to Candida Prostatitis

Septimus et al [31]. stated the following:

- Prostatitis is a common complication of prostate biopsies, which is a procedure that involves the needle aspiration of prostate tissue via a transrectal approach and exposing the prostate to faecal material.
- These infections are most commonly caused by typical faecal organisms, such as Escherichia coli.
- They had reported on the documentation of fungal prostatitis due to Candida albicans.

Septimus et al. [31] reported a 57-year-old gentleman who had a history of hypertension who was in his usual state of health until January of 2004. At that time, he had undergone an ultrasound-guided transrectal biopsy of his prostate gland in view of his elevated serum prostate specific antigen (PSA). He was placed on fluoroquinolone prophylaxis for the procedure. His biopsy was negative for malignant cells. One week later, he had experienced urinary retention and was catheterized with 400 mL of residual urine. He was then placed on tamsulosin and had done well. In April of 2004, while on a business trip, the patient had started to experience dysuria and had commenced taking levofloxacin. He continued to have symptoms and had reported back to his urologist. He had urinalysis while on antibiotics that showed 20 to 30 white cells and 20 to 30 red blood cells per high power field, but the urine cultures were negative. He was asked to continue taking his antibiotics. One week later, he again had urinary retention and underwent urethral catheterization with 450 mL residual urine. His tamsulosin was increased and levofloxacin was continued. He had a second urine culture, without urinalysis, 3 days later, which was again sterile. His symptoms never completely resolved. At no time did the patient experience systemic symptoms, such as fevers or chills. After 1 month of taking antibiotics, his dysuria had progressed to perineal discomfort and burning after micturition. At that time, he was seen once again by a urologist. He had urinalysis again which showed 20 to 30 white blood cells and 20 to 30 red blood cells per high power field, and rectal examination which revealed a slightly boggy prostate with mild tenderness. His urine culture, prostate secretion cultures, and ejaculate cultures all grew Candida albicans at that time. He had ultrasound scan of his prostate which revealed no abscess. The patient was diagnosed at that time as having Candida prostatitis. He was commenced on fluconazole 400 mg daily for 6 weeks, with total resolution of his symptoms

after the first week of treatment. He was at the time of the report of his case symptom-free over a year later, and his follow-up urinalysis after treatment returned to normal.

Septimus et al [31]. made the following follow-up discussion iterations:

- It had been pointed out that fungal urinary tract infections associated with either indwelling catheters or immunosuppression are increasingly recognized in an era of utilization of broad-spectrum antibiotics; nevertheless, prostate infections due to fungal organisms were still relatively uncommon, and Candida prostatitis is rarer still. [32]. with only scattered reports in the literature, most of which report prostatic abscesses due to this organism. [33-35].
- Given his first set of cultures, which were reported as negative upon his presentation with symptoms, they suspected their patient to have developed Candida prostatitis as a secondary complication, likely due to exposure to levofloxacin.
- It has been stated that with utilization of antibiotic prophylaxis, which is now the standard of care, infectious complications from ultrasound-guided transrectal prostate biopsies are infrequent but well documented [36,37].
- With the increased utilization of antibiotics such as fluoroquinolones in these situations as well as widespread use of other broad-spectrum antimicrobials, fungal infections in general had become more prevalent [38].
- Their case adds to this growing body of literature, indicating that clinicians need to consider fungal infections in patients who fail standard therapy for prostatitis, either as a primary causative organism or as secondary agents induced by broad-spectrum antibiotic use.

Golz et al [32]. reported the third case of a culturally and histologically proven candidosis of the prostate gland in the world literature available to them. They reported that autopsy of a 59-year-old man who had metastasizing bronchial carcinoma as predisposing primary disease had revealed a local candidosis of the prostate gland in the left lobe of the prostate, without evidence of a Candida sepsis.

Bartkowski and Lanesky [39]. stated the following:

- Emphysematous cystitis is typified by gas collection within the urinary bladder wall and lumen.
- Often it is the result of aerobic urinary tract infections but it might be caused by gastrointestinal fistulas or iatrogenic surgical and diagnostic instrumentation.
- They had reported a case of emphysematous cystitis owing to Candida albicans with the incidental finding of emphysematous changes within the prostate gland.

Mahlknecht et al [40]. reported a case of an asymptomatic prostatitis due to Candida Albicans that caused a sepsis. They stated that up to June 2005, in literature only 3 cases of Candida infections of the prostate gland without general illness had been described and that in their case the transurethral electro-resection of prostate was the adequate treatment.

Kurnatowska et al [41]. reported three cases of prostatitis caused by the invasion of Trichomonas. vaginalis and Candida. albicans which had been found in different biological materials. After per rectum examination perineum biopsy of prostate gland had been undertaken in all patients; within histopathological preparations of the biopsy specimens there were features found that pointed at the inflammation reaction of that gland within which the fungi were detected. Also, the same microorganisms were proved in sexual partners of those patients but multifocal invasion of Candida albicans, including genital and urinary organs, mouth and alimentary tract, also in members of their family were documented.

Li et al. [42]. stated the following:

- Emphysematous prostatic abscess (EPA) is an uncommon disease, which is characterized by localized collection of gas and purulent exudates within the prostate gland.
- The first case was reported in 1983[1] and only a few cases had been reported since then.
- The pathogens causing EPS included various bacterial and fungal organisms, and the most commonly reported microorganism causing EPA was noted to be Klebsiella pneumoniae [43].
- The management of emphysematous prostatic abscess is not standardized due to the limited number of cases reported.
- They were reporting a rare case of emphysematous prostatic abscess due to Candida tropicalis in a patient with poorly controlled diabetes mellitus and a review of the literature.

Li et al. [42]. reported a 72-year-old man who was admitted to their department because of dysuria for a period of 5 months, and acute urine retention for 6 days preceding his admission. He had ultrasound scan of his prostate gland which showed an enlarged prostate gland that measured 5.71 cm × 5.52 cm × 5.38 cm, without sign of an abscess, and trans-urethral Foley catheter was inserted and kept in place in the emergence department. The patient had a history of type 2 diabetes mellitus for over 10 years. He underwent digital rectal examination which revealed a mild, enlarged prostate gland, with no local tenderness. On the day he was admitted, his body temperature was 37.8°C. His laboratory tests results revealed a white blood cell count of 8.6×10^9/L with 73.1% neutrophils, hemoglobin 144 g/L, alanine aminotransferase (ALT) 11 IU/L, aspartate aminotransferase (AST) 12 IU/L, blood urea nitrogen (BUN) 4.59 mmol/L, fasting glucose 15.09 mmol/L, prostate specific antigen (PSA) 16.023 ng/ml. Urinalysis showed white blood cells 31/μL, red blood cells 449/μL, presence of glucose (4+). They adjusted the oral hypoglycemic agents (OHA) and monitored his blood glucose. On day 3, the patient developed chills and his body temperature was 39°C. His laboratory test results showed a white blood cell count of 14.1×10^9/L with 82.7% neutrophils, C-reactive protein (CRP) 190.2 mg/L. His blood sample was taken for culture immediately. Empiric antimicrobial treatment with intravenous cefoperazone/sulbactam (1:1) 2.0 g was given every 8 hours. On day 4, *Candida tropicalis* was isolated from the culture of his catheter specimen of urine. Fluconazole injection 200 mg every 12 hours was added to his treatment. But the state of his high fever seemed not to have any improvement. On day 8, he had computed tomography (CT) scan of his pelvis, and this revealed swelling of his prostate gland with air and fluid accumulation that measured 4.5 cm × 3.5 cm, which was suggestive of EPA (Fig. (Fig.1).1). On day 9, he underwent trans-rectal ultrasound guided prostate abscess aspiration. Only 5 ml reddish purulent fluid was extracted, saline solution wash did not help to extract more purulent fluid. His blood culture taken before and pus culture both were negative. His body temperature seemed to be improving following the aspiration, but he still got low-grade fever. On day 14, CT scan of his pelvis was undertaken, and gas formation was even bigger which had measured 75mm × 59 mm in the prostate gland (Fig. (Fig.2).2). His laboratory blood tests showed a white blood cell count of 25.9×10^9/L with 88.3% neutrophils, CRP > 270 mg/L. So transurethral unroofing of the prostatic abscess was undertaken immediately. A supra-pubic cystostomy was undertaken during the surgery for urinary diversion. There was not so much purulent fluid within the cavity of the abscess, but lots of necrotic tissue around the abscess cavity. Cefoperazone/sulbactam and fluconazole were administered continuously following the surgery. On day 20, he did not have any fever, and pelvic CT scan was rechecked which had shown a great improvement in the size of the abscess cavity within the prostate gland (Fig. (Fig.3).3). He was discharged

home on day 22. Parenteral antibiotics of fluconazole were kept for 14 days after discharge. He had Pelvic CT scan 1 month after his discharge which showed complete resolution of the EPA (Fig. (Fig.4).4). The cystostomy tube was removed 4 weeks subsequently.

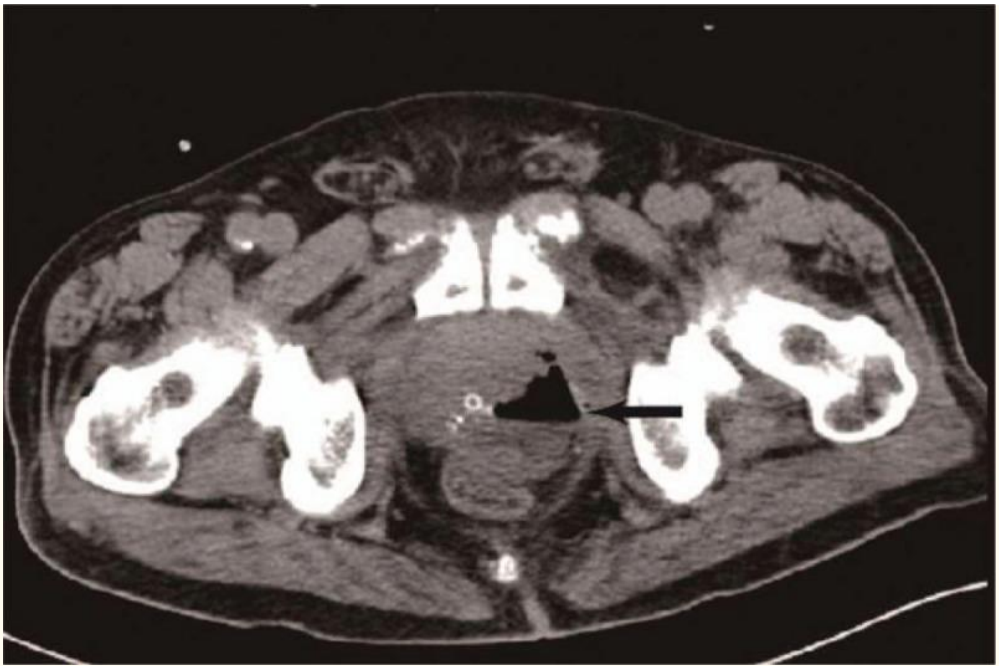

Figure 1: Reproduced from [42] Under Creative Commons Agreement License

Pelvic CT revealed a collection of gas and purulent exudates in the prostate gland (arrow) on day 8 of admission (before aspiration).

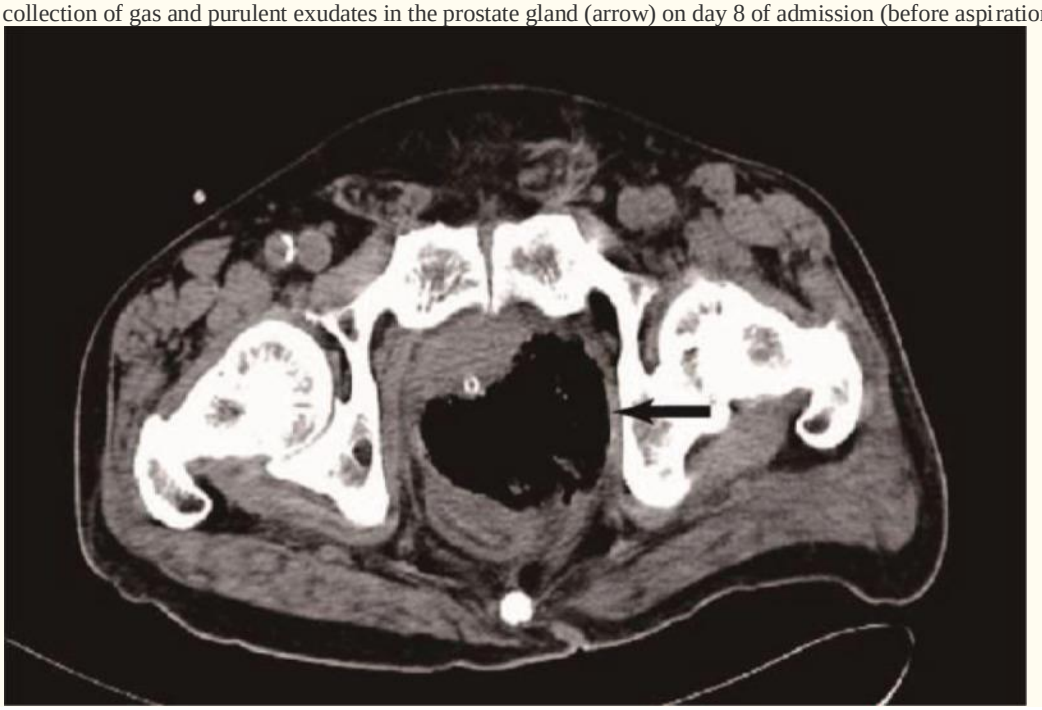

Figure 2: Reproduced from [42] Under Creative Commons Agreement License

Pelvic CT revealed a progress of gas and purulent exudates collection in the prostate gland (arrow) on day 14 of admission (after aspiration).

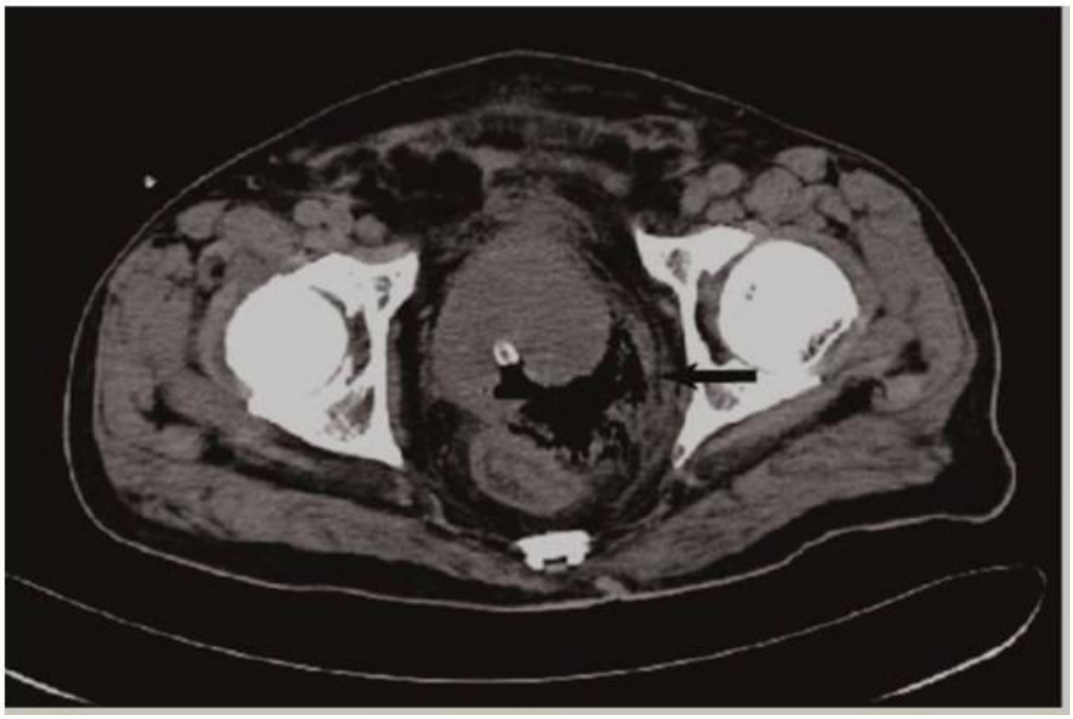

Figure 3: Reproduced from [42] Under Creative Commons Agreement License

Pelvic CT revealed an improvement of EPA in the prostate gland (arrow) on day 20 of admission (after TUR).

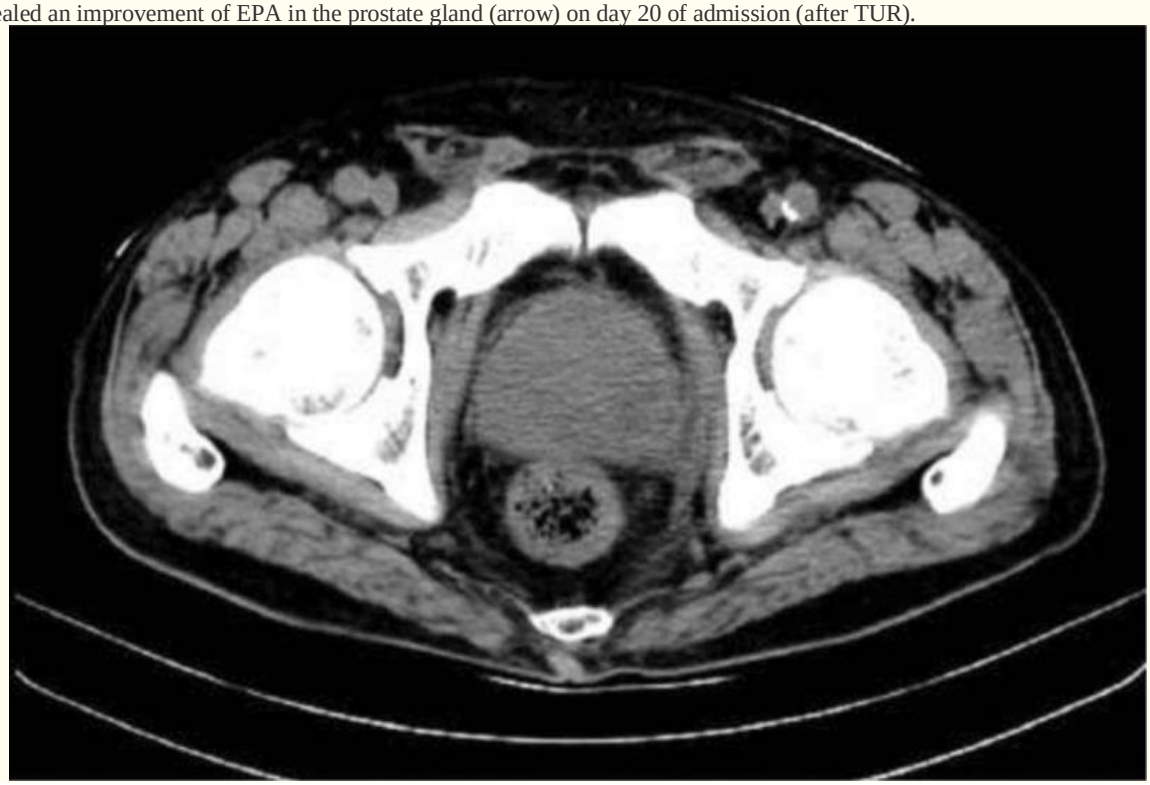

Figure 4: Reproduced from [42] Under Creative Commons Agreement License

Pelvic CT showed almost complete resolution of the EPA 1 month after discharge.

Li et al. [42]. made the ensuing summating discussions:

- Prostatic abscess is a rare complication of acute bacterial prostatitis, which has been reported in 0.5% to 2.5% of patients manifesting with inflammatory prostatitis [44].

- Emphysematous prostatic abscess is a particularly uncommon form of prostatic abscess, which is typified by localized collection of gas and purulent exudates within the prostate gland.
- The classical symptoms and signs of emphysematous prostatic abscess include dysuria, fever, odynuria, increased urinary frequency and urgency to urinate, urinary retention, perineal pain, and fluctuance on digital rectal examination.
- Early diagnosis of prostatic abscess is difficult due to its associated nonspecific symptoms.
- Patients are often initially treated for prostatitis, with a median delay in a correct diagnosis of 8 days [43].
- Radiology imaging techniques such as pelvic computed tomography (CT) and transrectal ultrasonography (TRUS) are most valuable for detecting EPA.
- They had searched PubMed for English language publications utilizing the keywords "emphysematous prostatitis" or "emphysematous prostatic abscess" for the period 1983 to 2019.
- Sixteen cases with EPA were found which had been included in 14 reports.
- They had reviewed the demographic characteristics, age, underlying disease, pathogen, management, and outcomes of the 16 patients with EPA [(see Table 2) table 2]
- Majority of the patients were moted to be from Asia (87.5%, 14/16).
- The median age of patients was 62 years old (range, 45–81 years). 14 (87.5%) had diabetes mellitus.
- Various bacterial and fungal organisms were isolated in urinary and/or pus cultures, with K pneumoniae being the most prevalent pathogen (50%, 8/16).
- Other organisms which had been reported included Escherichia coli (25%, 4/16), Candida species (18.75%, 3/16), Pseudomonas aeruginosa, Bacteroides fragilis, *and* Citrobacter species. Two cases (12.5%) had mixed infections.
- Fifteen (93.8%) underwent drainage of various ways. Except for one (with DM, dementia, advanced gastric cancer) had 8-week antimicrobial therapy, and he was transferred to another hospital for rehabilitation when condition improved, with no further information. Seven of 16 patients (43.8%) underwent a suprapubic cystostomy. The mortality rate was 18.75% (3/16).

Case	Nation	Age, years	Underlying disease*	Imaging modality†	Pathogen	Drainage‡	Cystostomy	Outcome
Mariani et al, 1983[1]	USA	56	DM	IVP, gallium scan	*aeruginosa, B. fragilis*	TUR	Yes	Survived
Bartkowski and Lanesky, 1988[13]	USA	60	DM	KUB/CT	*Candida*	TUR	Yes	Survived
Lu et al, 1998[14]	China (Taiwan)	45	DM	CT	*K. pneumoniae*	TPNA	No	Died
Lin et al, 2001[15]	China(Taiwan)	55	DM	CT	*K. pneumoniae*	DPID	Yes	Died
Bae et al, 2003[16]	Korea	50	DM	KUB/TRUS/CT	*K. pneumoniae*	TPD	Yes	Survived
Kuo et al, 2007[17]	China(Taiwan)	60	DM,liver cirrhosis	KUB/TRUS/CT	*K. pneumoniae*	TUR	No	Survived
Sampathkumar et al, 2007[18]	India	57	DM, ESRD, with renal transplant	CT	*coli*	TUR	No	Died
Tai, 2007 [19]	China(Taiwan)	60	DM	KUB/TRUS/CT	*K. pneumoniae*	TUR	No	Survived
Thorner et al, 2010 [20]	China(Taiwan)	64	DM, ESRD	CT	*Citrobacter species*	TUR	Yes	Survived
Cheung and Tsang, 2011 [21]	China(Taiwan)	68	DM, stroke	KUB/CT	*E. coli*	TUR	No	Survived
Wen et al, 2012[8]	China(Taiwan)	72	No DM	KUB/TRUS/CT	*E. coli*	CTPD	Yes	Survived
	China(Taiwan)	68	DM, liver cirrhosis	CT	*Candida*	CTPD	No	Survived
	China(Taiwan)	81	No DM	KUB/CT	*Coli, Candida*	CTPD	No	Survived
Hsu et al, 2013[10]	China(Taiwan)	54	DM, liver cirrhosis	KUB/TRUS/CT	*K. pneumoniae*	TPNA and TUR	N/A	Survived
Lee et al, 2014[2]	China(Taiwan)	70	DM	CT	*K. pneumoniae*	CTPD	Yes	Survived
Kiyozumi et al, 2018[6]	Japan	75	DM, dementia, advanced gastric cancer	KUB/CT	*K. pneumoniae*	No drainage	N/A	Survived
Present case	China	72	DM	TRUS/CT	*Candida*	TRNA and TUR	Yes	Survived

* DM = diabetes mellitus, ESRD = end-stage renal disease.
† CT = computed tomography, IVP = intravenous pyelography, KUB = plain film of kidney, ureter and bladder, TRUS = transrectal ultrasound.
‡ CTPD = computed tomography-guided perineal drainage, DPID = direct perineal incision and drainage, TPD, transperineal drainage, TPNA = transperineal needle aspiration, TRNA = transrectal needle aspiration, TUR = transurethral incision or unroofing.

Table 2: Reproduced from [42] Under Creative Commons Agreement License

Cases of emphysematous prostate abscess, including 16 reported patients and the present case.

- EPA had appeared to be geographically more common in Asia, and different from other forms of prostatitis, K pneumoniae rather than E coli appears to be the most common causative pathogen. [45-47].
- Whether some virulent strain of K pneumoniae or host factor had resulted in the phenomenon deserves further investigation [48,49] In their reported case, they had determined EPA was due to C tropicalis infection.
- The diagnosis was confirmed by the undertaking of pelvic CT scan, with about 8 days delay. C species also seemed to be more prevalent in EPA (23.5%, 4/17, with the inclusion of their reported) than in other urinary tract infections. Nevertheless, in view of the small number of cases reported, further study is needed.
- It has been stated that prostatic abscess may develop secondary to reflux of infected urine into the prostate or from hematogenous dissemination [46,50]
- The risk factors for prostatic abscess formation do include urinary bladder outlet obstruction, urethral manipulation and systemic disease such as diabetes mellitus, liver cirrhosis and other immune-compromising conditions. [43,46,49]
- In their reported case, the patient had poorly controlled diabetes, together with urinary tract obstruction and catheterization might be important risk factors which had contributed to prostatic abscess formation.
- The treatment of choice is prompt and thorough abscess drainage with early antibiotic treatment and strict control of blood glucose.
- Abscess drainage may be undertaken by transurethral or trans-perineal way. Open surgery has been less recommended

nowadays. Transurethral incision or unroofing of the prostatic abscess could provide complete drainage but it also increases the risk of sepsis [49,51].

- The surgery should be undertaken in selected patients who are hemodynamically stable and able to tolerate the anaesthesia, and it is best to be quick and effective.
- The trans-perineal route is safer owing to its application under local anaesthesia, but the disadvantage is possible incomplete drainage, abscess recurrence and long-term catheter indwelling needed.
- Because ultrasound waves are reflected by gas, CT-guided trans-perineal abscess drainage for emphysematous prostatic abscess might be more precisely and recommended [45,49].
- For other forms of prostate abscess, based upon outcomes from several large case series studies, TRUS-guided aspiration rather than indwelling drainage has been to be considered the standard treatment before progressing to other therapies. [46,52,53]
- In their reported case, they had reported, TRUS-guided aspiration was obviously not adequate for abscess drainage, and the infection was not controlled until TUR was undertaken.
- They had assumed that the air, thick purulent exudates and necrotic tissue of EPA made it difficult to drain by aspiration and much easier to recur.

Li et al. [42] made the ensuing conclusions:

- Emphysematous prostatic abscess is an uncommon. but highly morbid infectious disease which occurs in immuno-compromised patients, especially in association with diabetes mellitus.
- CT scan or TRUS should be undertaken in the patients with suspected diagnosis.
- Early and appropriate drainage with proper antibiotic therapy is important to achieve a favourable outcome.

Juan et al. stated that prostatic abscess is an uncommon condition and its clinical diagnosis is difficult as well as the classical symptoms and signs of prostatic abscess are variable and nonspecific. Juan et al. [54]. reported a rare case of emphysematous prostatic abscess due to candidiasis in a 68-year-old man and who also had diabetes mellitus and liver cirrhosis. The diagnosis was confirmed by the undertaking of pelvic computed tomography (CT) and it was successfully treated by antibiotics and CT-guided percutaneous abscess drainage. Juan et al. [54]. iterated that their reported case had highlighted the importance of early and accurate diagnosis of emphysematous prostatic abscess followed by appropriate treatment.

Singh et al. [2]. stated the following:

- Prostatitis may manifest with lower urinary tract symptoms (LUTS) which is attributable to acute and chronic bacterial infections (NIH Category I/II) or as asymptomatic inflammatory prostatitis (NIH Category IV).
- Patients who have chronic prostatitis/chronic pelvic pain syndrome, (CP/CPPS, NIH Category III) might manifest with a wide range of symptoms resulting from varied aetiology; nevertheless, seldom caused by fungal infections.
- Occasional case reports had been published on prostatitis due to Candida sp.

Singh et al. [2]. reported a case of an elderly diabetic patient who had undergone per-urethral prostatic resection (TURP) for benign prostatic hyperplasia (BPH) and who returned with complaints of LUTS and perineal discomfort one month later. After repeat surgery, the TURP chips upon histopathology examination showed features of prostate hyperplasia and prostatitis with numerous hyphae and yeast forms of Candida which was admixed with acute and chronic inflammatory exudate. After confirmation by special stains and positive urine culture, a final diagnosis of prostatic candidiasis was made.

Wise and Shteynshlyuger made the ensuing summating iterations [55].

- Epidemiological changes that include immune-compromised patients and drug-resistant fungi had caused an increase in nosocomial infections by Candida albicans and non-albicans Candida species.
- Other fungi, aspergilla and Cryptococcus (environmental contaminants), are opportunistic invaders of the immune-compromised (transplant, HIV) patients.
- The environmental fungi Coccidioides immitis (dry arid areas), Histoplasma capsulatum (Avian-infested areas), and Blastomyces dermatitidis (aquatic areas) could cause infections in immune-competent and immune-deficient patients.
- Each fungus could cause changes in the prostate gland that simulate bacterial infection, benign prostatic hypertrophy, or neoplasm.
- Diagnosis could be established by urine cultures or needle biopsy of the prostate.
- Prostate surgery for carcinoma or benign enlargement of the prostate gland may detect latent fungal infection.
- Different fungal species could have divergent clinical manifestations and require different treatment.
- In some cases, asymptomatic localized fungal prostatitis could be cured by removal of the infected gland.
- Symptomatic and disseminated infection might require prostatectomy and systemic antifungal therapy.

Epstein et al. [56]. stated that fungal prostatitis is exceedingly rare with mostly case reports. Epstein et al. [56]. searched the electronic medical records at three medical centres were for cases of fungal prostatitis due to endemic mycoses and Cryptococcus over the preceding 10 years. Epstein et al. [56]. summarized the results as follows:

- Seven cases were identified from 105 600 prostate biopsies within the Southern California Permanente Medical Group for an incidence of 0.0066%.
- An additional eight cases were identified from two other health care systems.
- With the exclusion of four patients without available clinical data, 11 patients were reviewed, majority of them had undergone biopsy due to elevated serum prostate-specific antigen levels.
- Four of the patients were asymptomatic and the remainder had nonspecific signs or symptoms.
- Pathology examination of all of the biopsies had revealed granulomatous inflammation and fungal organisms. Seven patients had coccidioidomycosis, three patients had cryptococcosis which was confirmed in two cases and suspected by organism morphology in the other, and one patient had likely histoplasmosis based upon organism morphology.
- Prolonged antifungal treatment was standard; and the outcomes were favourable.

Epstein et al. [56]. concluded that fungal prostatitis due to endemic mycoses and Cryptococcus is uncommon and associated with favourable outcomes but generally involves prolonged therapy.

Mayayo et al. [57]. made the following summating statements:

- Prostate pathology is a daily occurrence in urological and general medical consultations.
- Besides hyperplasia and neoplastic pathology, other processes, such as infectious ones, have also been documented.
- Their aetiology is diverse and varied. Within the infectious prostatic processes, fungi could also be a specific cause of prostatitis.
- Fungal prostatitis often appears in patients who have impaired immunity and could also be rarely found in healthy patients. It could result from a disseminated infection, but it could also be localized. Fungal prostatitis is a nonspecific and harmless process. Diagnosis is commonly made by fine needle aspiration cytology or by biopsy.
- A number of fungi could be involved in causing prostatitis.
- Even though there are not many reported cases fungal prostatitis, they are becoming more frequent, in particular in patients who have some degree of immunodeficiency or those who dwell within areas where specific fungi are endemic or in visitors of those areas.
- They had presented a comprehensive review of the various forms of fungal prostatitis, and they had described the morphological characteristics of the fungi that are more frequently reported as causes of fungal prostatitis.
- They had also reported their own experience, aiming to alert physicians, urologists and pathologists of these particular infections (you may refer to the original article for more detailed information.

Demirci et al. [58]. stated the following:

- Chronic prostatitis is a disease which adversely affects the quality of life of patients and does not respond adequately to treatments.
- In their reported case, they would present an immunocompetent patient who had chronic prostatitis due to candida infection and who did not benefit from treatments and who was not diagnosed for a long time in the light of the up-to-date literature.

Demirci et al. [58]. reported a 51 years old man who had lower urinary tract symptoms for 20 years. He was admitted to their clinic due to frequent urination, perineal and suprapubic pain, weak urine stream, and white particles in urine. The patient's IPSS was 20; the pain score was 15, his urinary symptom score was 8, his life quality index score was 9 according to the NIH-CPSI. He did not have any history of comorbidity and operation. He had digital rectal examination which revealed features of a benign prostate gland and during the examination pelvic floor spasm was detected. The results of his serum biochemical analysis included: urea 21.4 mg/dl, creatinine 1 mg/dl, and tPSA 1.12 ng/ml. His urine analysis, revealed 7 erythrocytes and 2 leukocytes, and nitrite was negative. Urine cultures taken before and after his prostate massage were sterile. On urinary tract ultrasound, his upper urinary tract and urinary bladder appeared normal, and his prostate volume was 33 cc. In uroflowmetry analysis, his Qmax was 14 ml/s, volume 539 cc, and his average urine flow rate was 8 ml/s. The patient underwent cystoscopy. The anterior urethra was normal, and the prostate was mildly hyperplastic; there was cloudy urine with dense white particles within his urinary bladder; and there was no mass in the bladder. Cytology examination of the urine specimen revealed uniform bladder epithelial cells. Spore-bearing structures on the ground were seen in smear slides. They were considered as Candida glabrata (see figure 5). The patient began treatment with fluconazole 400 mg/day and doxazosin 4 mg/day. At the end of the first month, white particles in urine were found to be significantly decreased, the patient's IPSS score was 12; the pain score was 6, his urinary symptom score was 2 and his life quality index score was 3 according to NIH-CPSI, and the symptoms were regressed in an obvious manner. In his urine analysis, 48 erythrocytes and 9 leukocytes were detected. His urine culture was negative. In uroflowmetry analysis, his Qmax was 16 ml/s, voided volume of urine was 370 cc, and his average urine flow rate was 12 ml/second, and the doxazosin was stopped. At the end of the second month, white particles in his urine had disappeared completely. Fluconazole 400 mg/day was administered for a total of 8 weeks and then was discontinued. The patient was followed-up for 6 months and no recurrence was observed.

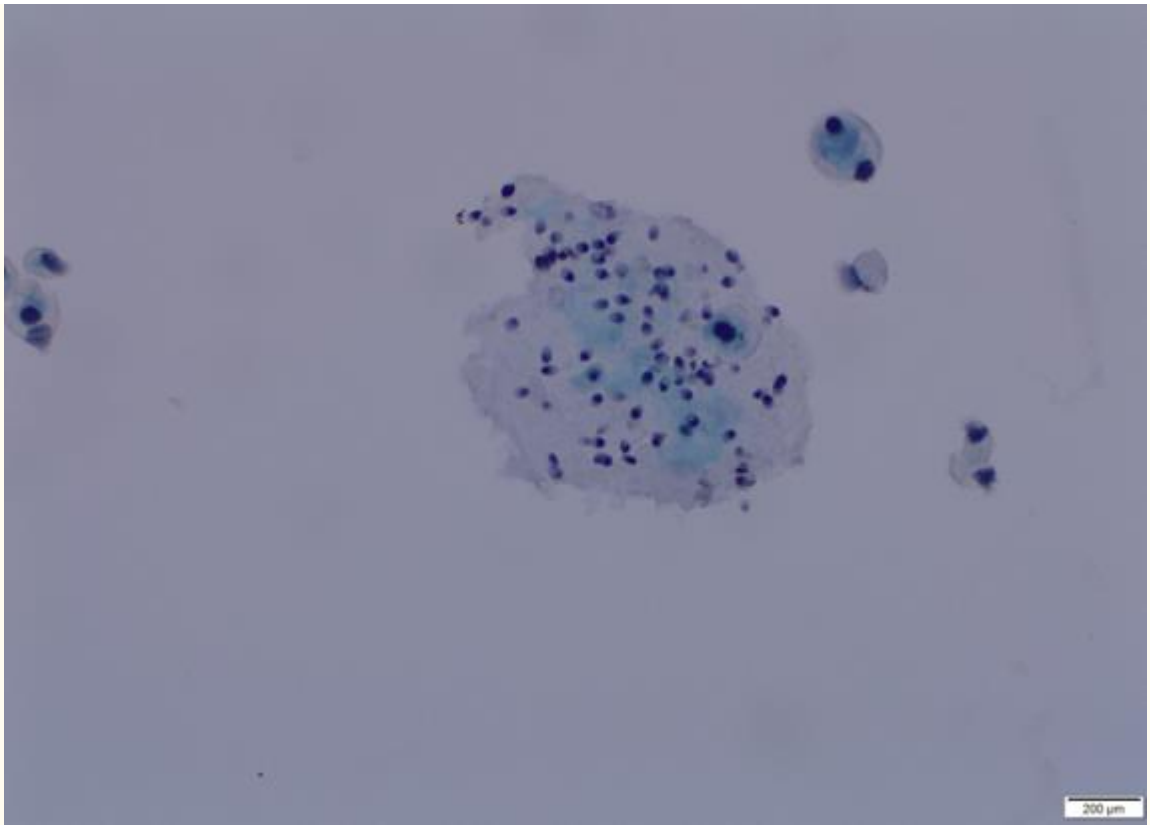

Figure 5: Microscopic findings of urine cytology. Reproduced from: [58] under Creative Commons Attribution License.

Demirci et al. [58]. made the following summating discussions:

- The National Institute of Health had divided prostatitis into four main groups: acute bacterial prostatitis, chronic bacterial prostatitis (CBP), chronic prostatitis/chronic pelvic pain syndrome (CP/CPPS), and asymptomatic inflammatory prostatitis. [5]
- According to this classification, the prevalence of chronic prostatitis ranges between 1.8% and 8.2%. Even though CP is characterized by pelvic pain and urinary symptoms lasting more than three months and presence of infection source.
- They used the Modified Meares-Stamey test (2-glass test) in their patient and they found that the urinary system infection was not bacteria-oriented.
- It has been pointed out that urologists do have difficulties in making the diagnosis and giving medical treatment in this group. [59].
- Their patient had many previous admissions to urology clinics for 20 years due to pelvic pain, lower urinary tract symptoms, and white particles in urine. He was followed and treated with benign prostatic hyperplasia and CBP. During this period, inability to diagnose CP associated with candida infection had indicated that the syndrome should be understood more in urological practice.
- In view of the fact that traditional therapies had failed in clinical practice in patients who have chronic prostatitis, new approaches had gradually increased in recent times.
- It has been pointed out that a 6-point clinical phenotyping classification system [UPOINT] had begun to take its place in urology practice [60].
- They had shaped their approach by paying attention to this.
- They also used methods such as cystoscopy and cytology.
- It has been pointed out that fungal prostatitis is encountered especially in the elderly, hospitalized, catheterized, and immunosuppressed patients [61]
- Over the preceding two decades, the frequency of non-albicans Candida species had increased steadily.
- Even though it is thought that CP which is caused by fungal infection can be encountered in this group, their patient was in the rare group.
- They had detected C. glabrata, which is difficult to grow in urine culture, by lack of microscopic observation of hypha formation and by seeing spore-bearing structures in cytology.
- The number of publications on this subject in the literature is insufficient, and mycotic factors should be considered especially in patients with CP who do not benefit from medical treatment for symptoms of prostatitis.
- Chronic prostatitis is mostly difficult to treat.
- Nickel et al [62]. undertook a study on 100 patients and had reported that only one-third of them had benefited from the treatment [62].
- As the first step in the treatment of chronic prostatitis, even though fluoroquinolone treatment is used for 4–6 weeks, it is influential only in half of the patients whose symptoms start newly. In the second step, anti-inflammatory agents and alpha blockers are used; nevertheless, mostly the patients who have not used alpha blockers before benefit from this treatment.
- In the third step, 5 alpha reductase inhibitors, glycosaminoglycans are used and surgery is suggested for patients who are more resistant [59].
- In some studies, it had been suggested that multimodal treatments should be undertaken.

- Candida glabrata species are systematically treated with amphotericin B or fluconazole when they cause symptomatic urinary tract infection. [61].
- Their patient began treatment with fluconazole 400 mg/day and doxazosin 4 mg/day. At the end of the first month, white particles in urine were significantly decreased, and lower urinary tract symptoms regressed. They had determined that their patient was cured 6 months after the end of treatment.

Demirci et al. made the following conclusions [58].

- Their case had shown that fungal agents should be considered especially in patients who do not respond to medical treatment.
- There are very few publications on this subject in the literature.
- It was their opinion that the fact that the diagnosis of CP caused by C. glabrata which is rarely seen could not be made during years and thus the patient could not receive adequate treatment is valuable in terms of contributing to the literature as a case study.

Mahlknecht et al [40]. reported a case of a case of asymptomatic prostatitis due to Candida Albicans which had caused a sepsis. They stated that up to the time of the report of their case in 2005 June in literature only 3 cases of Candida infections of the prostate gland without general illness had been reported. In their reported case the transurethral electro-resection of prostate was the adequate treatment.

Indudhara et al [35]. stated that fungal prostatitis is an uncommon entity. Indudhara et al [35]. reported a case of isolated candidal prostatitis in an elderly patient who had manifested with acute urinary retention and who was clinically diagnosed as having benign hypertrophy of the prostate. Histology of his resected prostate gland demonstrated invasive prostatic involvement by Candida albicans. There was no evidence of systemic involvement by Candida.

Elert et al [33]. in 2000 reported a case of isolated Candidal prostatitis and the details of the case report can be seen in the original article.

Goltz et al [63]. stated that the third case of a culturally and histologically proven candidosis of the prostate in the world literature available to them had been reported by them. They reported that autopsy of a 59-year-old man with metastasizing bronchial carcinoma as predisposing primary disease had revealed a local candidosis of the prostate in the left lobe of the prostate, without evidence of a Candida sepsis.

Gupta et al. [64]. made the ensuing introductory iterations:

- Surgical or radiological drainage is the treatment of choice for prostatic abscesses, in conjunction with appropriate antibiotics.
- Fungal prostatic abscesses are uncommon, and amphotericin or azoles are the usual agents used for treatment.
- Up to 2008, by the time of the report of their case, echinocandins had not been reported as agents of choice for fungal abscesses because of a concern regarding their large molecular size and high protein binding.
- They were reporting a case of Candida glabrata prostatic abscess which was treated successfully using micafungin. Adequate intra-abscess concentrations of the drug were demonstrated.
- Majority of prostatic abscesses occur in patients with diabetes mellitus, in immunocompromised patients, and in patients who may not have received appropriate treatment for acute prostatitis.
- Foreign bodies and urinary tract obstruction are other predisposing factors.
- It has been pointed out that the infection / prostatic abscess generally does tend to occur by the ascending route and is caused by common uropathogens [65].
- Infection with fungi, mycobacteria, and other granuloma-causing organisms is uncommon. Among the fungi, *Cryptococcal* prostatic abscesses had been reported in HIV-positive patients.
- The prostate gland is known to be a reservoir site for *Cryptococcus* species. Few cases of prostatic abscesses with *Aspergillus*, and with dimorphic fungi such as *Blastomyces* and *Histoplasma*, had been described.
- Among *Candida* spp., 2 cases each of *C. tropicalis* and *C. albicans*, and 1 case of *C. glabrata* had been described.
- The treatment of choice is transurethral resection and/or intravenous amphotericin B. Fluconazole has been used for treatment of some deep-seated abscesses.
- Up to the time of the report of their case, echinocandins had not been reported as agents of choice for prostatic abscesses.
- They were describing a case of prostatic abscess which had been caused by *C. glabrata*, successfully treated with micafungin.

Gupta et al [64]. reported a 73-year-old man who was sent to their hospital from an extended care facility, with lethargy, chills, perineal pain, and tenesmus, which had progressively worsened over 2 days. He had denied having fevers, dysuria, abdominal pain, nausea, vomiting, diarrhoea, cough, weight loss, and night sweats. His medical history included end-stage renal disease requiring peritoneal dialysis, insulin-dependent diabetes mellitus, hypertension, coronary artery disease, hypothyroidism, benign prostatic hypertrophy, and peripheral vascular disease requiring multiple revascularization surgeries. Ten months preceding his admission, he had undergone extensive perineal resection and partial colectomy for Fournier gangrene. For 6 months prior to his admission, the patient had been given systemic antibiotics for recurrent urinary tract infections with Escherichia coli *and* Candida glabrata. Until 2 months before his admission, he had received urinary bladder irrigations with amphotericin B and neomycin/polymyxin B. During his admission, the patient's vital signs were stable, with temperature of 98.6°F, blood pressure of 140/80 mm Hg, pulse rate of 78 beats/min, and blood oxygen saturation of 97% on breathing air. His cardiopulmonary examination was unremarkable, and his abdomen was noted to be soft, non-tender, and distended with peritoneal dialysate. No groin lesions or skin rashes were found during his examination. He had digital rectal examination which revealed an extremely tender enlarged prostate gland. The results of his laboratory tests were notable for leukocytosis (18.6 cells per μL/mm^3 with 89% granulocytes), hypokalaemia (2.9 mEq/L), and hyperglycaemia (269 mg/dL). His serum creatinine level was elevated (5.5 mg/dL) as expected with his renal insufficiency. His serum electrolytes were repleted. His urine cultures grew Escherichia. coli and multidrug-resistant Proteus mirabilis, which were treated with intravenous ertapenem. He had Computed tomographic (CT) scan of abdomen and pelvis with oral and intravenous contrast which had revealed a multi-loculated prostatic collection, that measured 6.0 cm × 4.5 cm in dimensions. Peritoneal dialysate and dialysis catheter were visualized; no other significant abnormalities were demonstrated. Percutaneous radiologically guided catheterization of prostate was undertaken, and purulent fluid was obtained. Cultures of this fluid resulted in abundant growth of Candida. glabrata. Given his previous hospitalizations and nosocomial acquisition of C. glabrata urinary infection in the past, the authors had predicted probable resistance to azole antifungals and, therefore, deferred using these for his treatment. Later, this was confirmed when the isolate had demonstrated minimal inhibitory concentrations (MICs) ≥256 μg/mL for fluconazole and ≥2 μg/mL for voriconazole. A long course of amphotericin B was relatively contraindicated in this patient to preserve his residual renal function and to avoid potential hyperkalaemia [66]. In view of concern of maintaining residual renal function, the authors recommended micafungin in addition to catheter-drainage to treat the prostatic abscess. The C. glabrata isolate was found to be susceptible to echinocandins, with an MIC of 0.06 μg/mL for caspofungin, which is therapeutically equivalent to micafungin [67]. In order to determine tissue penetration of micafungin into the prostate, the authors measured levels of the drug in serum as well as abscess fluid. Micafungin level in serum was 1.28 μg/mL, and in abscess fluid, it was 0.43 μg/mL, well

above the MIC for the isolate. The patient received micafungin 100 mg/d intravenously for 37 days. The patient was determined not to be a candidate for prostatectomy because of his underlying comorbidities. Many attempts at radiologically guided catheter-drainage failed because of multiple loculations in the abscess. Eventually, transurethral unroofing of the abscess was undertaken with decortication of loculations, which had resulted in optimal drainage of abscess fluid via the urethra. Computed tomographic (CT) scan was undertaken 45 days after his admission which had shown complete resolution of the prostatic abscess.

Gupta et al. [64] made the ensuing summating discussions:

- Surgical or radiologically guided drainage is the primary component of treatment of prostatic abscess.
- Amphotericin B has remained the gold standard antifungal agent for most fungal abscesses.
- For sensitive Candida species, azole antifungals are a good option.
- Because of their small size molecules, the azoles have the ability to penetrate tissues well and attain high concentrations in deep tissues and abscess cavities. Nevertheless, with the widespread utilization of fluconazole, azole-resistant Candida isolates are now being encountered.
- Recent results from the Global Antifungal Surveillance Study had demonstrated the existence of cross-resistance between fluconazole and voriconazole, with the greatest emphasis on C. glabrata.
- Among 137,487 isolates of Candida *spp*. that had been tested against voriconazole, less than 30% of fluconazole-resistant isolates of C. albicans, C. glabrata, C. tropicalis, and *C.* rugosa had remained susceptible to voriconazole [68].
- Susceptibility of a Candida *spp*. to voriconazole was predicted by utilizing fluconazole MICs ≤ 32 μg/mL to identify voriconazole-susceptible isolates and MICs ≥ 64 μg/mL to identify voriconazole resistance [69].
- Echinocandins had not been reported as therapeutic agents for fungal prostatic abscess. In fact, these newer antifungals had not been proven to eradicate fungi within abscesses in general.
- Echinocandins are large lipoprotein molecules with a relative molecular weight of approximately 1200 and with high protein binding and, therefore, are initially confined to the plasma compartment.
- The volume of distribution subsequently expands slowly to the extravascular space [70].
- Data exist regarding successful use of echinocandins in necrotizing pulmonary aspergillosis and aspergillomas, as well as in some cases of brain abscesses from Aspergillus [71,72].
- Survival is prolonged in such patients, but a moderate number of fungal elements may persist in tissues, partly because echinocandins are only fungistatic for *A*spergillus spp.
- For majority of Candida spp., echinocandins are highly fungicidal. Activity is less against *C.* parapsilosis and C. guilliermondii. The mean trough concentrations of echinocandins can be maintained over the MIC90 for clinically relevant Candida spp. when given in recommended doses. Tissue concentrations of these drugs had not been studied in human subjects with deep-seated fungal infections. Caspofungin tissue distribution has been quantitatively analyzed in rats [73].
- Tissues containing the highest amounts of drug were liver, kidney, lung, and spleen, with drug concentrations more than 4-fold the MIC90s. Therapeutic levels of micafungin have been achieved in lung, liver, spleen, and kidney of rabbits after long-term intravenous administration of 0.5 to 2 mg/kg [74].
- Likewise, multiple doses of 0.1 to 10 mg/kg per day of anidulafungin yielded therapeutic trough concentrations in lungs, liver, spleen, and kidney of rabbits, and a substantial accumulation in brain tissue was achieved at dosages ≥ 0.5 mg/kg per day [75].
- Micafungin efficacy had been found to be equal to that of fluconazole at 10% the dosage for therapy of deep-seated candidiasis in a cyclophosphamide-induced immunosuppressed mouse model, by inducing intraperitoneal abscess by C. albicans [76]. The treatment effect of micafungin was similar to and superior to that of fluconazole at 24 hours and 8 days after the end of therapy, respectively. With regard to invasive candidiasis and candidemia in humans, micafungin (100 mg/d) is as effective as, and causes fewer adverse events than, liposomal amphotericin (3 mg/kg per day) [77]. Efficacy is independent of the Candida spp., primary site of infection, neutropenic status, Acute Physiology and Chronic Health Evaluation II score, and catheter removal.
- With the emergence of azole resistance among Candida spp. and the usual comorbidities of chronically ill patients who are more susceptible to fungal infections, echinocandins are rapidly emerging as antifungal agents of choice, given their efficacy against most Candida spp. and their relative safety compared with amphotericin. The major limiting factor for these drugs is their cost. Empiric use of these agents should be discouraged to avoid selection of fungal isolates resistant to yet another class of antifungal agents. For deep-seated fungal abscesses, the first-line treatment still is surgical drainage, along with an antifungal agent prudently chosen based on the susceptibility of the isolate, geographic and institutional prevalence of azole-resistant fungi, host factors, and cost-effectiveness. Based upon the therapeutic success in our patient, echinocandins may be reasonably considered for deep-seated abscesses with susceptible Candida spp., when indicated.

Conclusions

- Candida prostatitis and Candida prostatic abscess have tended to be reported on rare occasions, and perhaps there has been underreporting of these conditions due to the fact that the conditions manifest with non-specific symptoms that tend to be more commonly encountered with more common conditions including benign prostatic hyperplasia (BPH), bacteria prostatitis, and carcinoma of the prostate gland.
- If an individual has undergone treatment for a provisionally diagnosed bacterial prostatitis and has not been responding to appropriate antibacterial medications, then clinicians should have a high index of suspicion to exclude rare types of prostatitis and prostatic abscess including Candida prostatitis, tuberculous prostatitis, and cryptococcus prostatitis so as to undertake biopsy of the prostate and / or radiology-image-guided aspiration of the abnormal looking area in the prostate for pathology and microbiology examination to establish the correct diagnosis and to provide effective treatment.
- Patients who are treated for Candida prostatitis / prostatic abscess should be followed up regularly over a long period of time in order to establish a quick diagnosis of any subsequent recurrence of candida infection
- Pursuant to the treatment of any individual who has been treated for candida prostatitis/ candida prostatic abscess, there should be contact tracing and assessment of any / all coital contacts of the individual in order to ascertain any candida infection that would need to be treated promptly.

Conflict of interest – None

Acknowledgements

Acknowledgements to:

- Medicine (Baltimore) and Wolters Kluwer for granting permission for reproduction of figures and contents of their journal article to be reproduced under copyright: Copyright © 2020 the Author(s). Published by Wolters Kluwer Health, Inc. This is an open access article distributed under the Creative Commons Attribution License 4.0 (CCBY), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited. http://creativecommons.org/licenses/by/4.0

- Urology Case Reports and Elsevier for granting permission for reproduction of figures and contents of their journal article under copyright: Copyright © 2018 Published by Elsevier Inc. This is an open access article under the CC BY-NC-ND license: (http://creativecommons.org/licenses/by-nc-nd/4.0/).

References

1. WIKIPEDIA the free encyclopedia. Prostatitis.
2. Singh S, Singh M, Bains L, Sagar T. (2023). Candida prostatitis: A rare entity. Tropical Doctor, 53(2):282-284.
3. Collins MM, Stafford RS, O'Leary M P, Barry M J. "How common is prostatitis? A national survey of physician visits J. Urol, **159** (4):1224-1228.
4. Kirby, Roger; Carson, Culley C. (January–February. "Editor's Comment on Diagnosis and treatment of chronic prostatitis/chronic pelvic pain syndrome". Trends in Urology and Men's Health, **6** (1):17.
5. Krieger JN, Nyberg L, Nickel JC (July 1999). "NIH consensus definition and classification of prostatitis". JAMA. **282** (3): 236-237.
6. J. Curtis Nickel. (1999). Textbook of prostatitis. Taylor & Francis, 27.
7. Habermacher G M, Chason J T, Schaeffer A J. (2006). Prostatitis/chronic pelvic pain syndrome". Annu. Rev. Med, **57**:195-206.
8. Korrovits P, Ausmees K, Mändar R, Punab M. (2008). Prevalence of asymptomatic inflammatory (National Institutes of Health Category IV) prostatitis in young men according to semen analysis. Urology. 2008 June; **71** (6): 1010-1015.
9. Meares EM, Stamey T A. (1968). Bacteriologic localization patterns in bacterial prostatitis and urethritis. Invest Urol. 1968 March; **5** (5): 492-518.
10. The Manual Editorial Staff. Quick Facts Prostatitis: Benign Prostate. MSD Manual Consumer Version. Full review / revision. 2023.
11. Iczkowski KA. Prostatitis. PathologyOutlines.com website. https://www.pathologyoutlines.com/topic/prostateprostatitis.html. Accessed May 12th, 2023.
12. Iczkowski KA, Torkko KC, Wilson RS, Lucia MS, Bostwick D G. (2014). Prostatic atrophy: its spatial proximity to carcinoma and intraepithelial neoplasia based on annotation of digital slides. Hum Pathol, 45(1):54-58.
13. Rundle AG, Sadasivan SM, Chitale DA, Gupta NS, Williamson SR, Kryvenko ON, Chen Y, Bobbitt K, Tang D, Rybicki BA. (2021). Racial differences in the systemic inflammatory response to prostate cancer. PLoS One. 9;16(7): 0252951.
14. Kryvenko ON, Jankowski M, Chitale DA, Tang D, Rundle A, Trudeau S, Rybicki BA. (2012). Inflammation and preneoplastic lesions in benign prostate as risk factors for prostate cancer. Mod Pathol. Jul;25(7):1023-1032.
15. Barozzi L, Pavlica P, Menchi I, De Matteis M, Canepari M. (1998). Prostatic abscess: diagnosis and treatment. AJR Am J Roentgenol, 170(3):753-757.
16. Li J, Li Y, Cao D, Huang Y, Peng L, Meng C, Wei Q. (2022). The association between histological prostatitis and benign prostatic hyperplasia: a single-center retrospective study. Aging Male, 25(1):88-93.
17. Tiwari P, Pal DK, Tripathi A, Kumar S, Vijay M, Goel A, Sharma P, Dutta A, Kundu AK. (2011). Prostatic abscess: diagnosis and management in the modern antibiotic era. Saudi J Kidney Dis Transpl, 22(2):298-301.
18. Brede CM, Shoskes DA. (2011). The etiology and management of acute prostatitis. Nat Rev Urol. 2011 Apr;8(4):207-212.
19. Iczkowski KA. Chronic pelvic pain syndrome: a role for aberrant cytokine function. J Urol. (2010).184(4):1253-1254.
20. Jazdarehee A, Ahrari A, Bowie D, Chang SD, Tran H, Jamal S, Chen LYC, Tran KC. (2022). IgG4-related prostatitis manifesting as urinary obstruction in a 28-year-old male. BMC Urol. 11:22(1):35.
21. Jang K, Lee DH, Lee SH, Chung BH. (2012). Treatment of prostatic abscess: case collection and comparison of treatment methods. Korean J Urol, 53(12):860-864.
22. Krieger JN, Nyberg L Jr, Nickel JC. (1999). NIH consensus definition and classification of prostatitis. JAMA, 21:282(3):236-237.
23. Polackwich AS, Shoskes DA. (2016). Chronic prostatitis/chronic pelvic pain syndrome: a review of evaluation and therapy. Prostate Cancer Prostatic Dis. 19(2):132-138.
24. Liu S, Miller PD, Holmes SA, Christmas TJ, Kirby RS. (1992). Eosinophilic prostatitis and prostatic specific antigen. Br J Urol, 69(1):61-63.
25. Bourlon MT, Sánchez-Ávila M, Chablé-Montero F, Arceo-Olaiz R. (2013). IgG4-Related Autoimmune Prostatitis: Is It an Unusual or Underdiagnosed Manifestation of IgG4-Related Disease? Case Rep Urol, 295472.
26. Yamamoto M, Hibi H, Miyake K. (1993). Prostate-specific antigen levels in acute and chronic bacterial prostatitis. Hinyokika Kiyo, 39(5):445-449.
27. Thornhill BA, Morehouse HT, Coleman P, Hoffman-Tretin JC. (1987). Prostatic abscess: CT and sonographic findings. AJR Am J Roentgenol. 1987148(5):899-900.
28. Singh P, Yadav MK, Singh SK, Lal A, Khandelwal N. (2011). Case series: Diffusion weighted MRI appearance in prostatic abscess. Indian J Radiol Imaging. 21(1):46-48.
29. Bostwick DG, de la Roza G, Dundore P, Corica FA, Iczkowski KA. (2003). Intraepithelial and stromal lymphocytes in the normal human prostate. Prostate. 2003 May 15:55(3):187-193.
30. Do HK. (2011). Skin non tumor Infectious disorders Fungi-Candida. PathologyOutlines.com website. Author Update July 01; Last Staff Update 2021 January 18.
31. Septimus, J.D. Septimus, E.J. Prostatitis Due to Candida albicans. Infectious Diseases in Clinical Practice 14(4): 2392006.
32. Golz R, Mendling W. (1991). Candidosis of the prostate: a rare form of endomycosis. Mycoses. 34(9-10):381-384.
33. Elert A, von Knoblock R, Nusser R, Heidenreich A, Hofman R. (2000). Isolated candidal prostatitis. J Urol, 163(1):244.
34. Collado A, Ponce de Leon J, Salinas D, Salvador J, Vicente J. (2001). Prostatic abscess due to Candida with no systemic manifestations. Urol Int, 67(2):186-188.
35. Indudhara R, Singh SK, Vaidyanathan S, Banerjee C K. (1992). Isolated invasive candidal prostatitis. Urol. Int, 48(3):362-364.
36. Sieber PR, Rommel FM, Agusta VE, Breslin J A, Huffnagle H W, Harpster L E. (1997). Antibiotic prophylaxis in ultrasound guided transrectal prostate biopsy. J Urol, 157(6):2199-2200.

37. Otrock ZK, Oghlakian GO, Salamoun MM, Haddad M, Bizri A R. (2004). Incidence of urinary tract infection following transrectal ultrasound guided prostate biopsy at a tertiary-care medical center in Lebanon. Infect Control Hosp Epidemiol. 25(10):873-877.
38. Clark TA, Hajjeh RA. (2002). Recent trends in the epidemiology of invasive mycoses. Curr Opin Infect Dis,15:569-574.
39. Bartkowski DP, Lanesky JR. (1988). Emphysematous prostatitis and cystitis secondary to Candida albicans. J Urol, 139(5):1063-1065.
40. Mahlknecht A, Pecorari V, Richter A. (2005). Sepsis due to asymptomatic Candida prostatitis. Arch Ital Urol Androl, 77(3):155-156.
41. Kurnatowska A, Kurnatowski A, Mazurek L, Wedzikowski P. (1990). Rzadkie przypadki zapalenia stercza wywołane inwazja Trichomonas vaginalis z Candida albicans [Rare cases of prostatitis caused by invasion of Trichomonas vaginalis with Candida albicans]. Wiad Parazytol. 36(5-6):229-236.
42. Li Z, Wen J, Zhang N. (2020). Emphysematous prostatic abscess due to candidiasis: A case report and review of the literature. Medicine (Baltimore), 99(9): 19391.
43. Lee CY, Tsai HC, Lee SS, Chen YS. (2014). Concomitant emphysematous prostatic and periurethral abscesses due to Klebsiella pneumoniae: a case report and review of the literature. Southeast Asian J Trop Med Public Health, 45(5):1099-1106.
44. Langer JE, Cornud F. (2006). Inflammatory disorders of the prostate and the distal genital tract. Radiol Clin North Am, 44(5):665-767.
45. Thomas AA, Lane BR, Thomas AZ, et al. (2007). Emphysematous cystitis: a review of 135 cases. *BJU Int*, 100:17-20.
46. Ackerman AL, Parameshwar PS, Anger JT. (2018). Diagnosis and treatment of patients with prostatic abscess in the post-antibiotic era. Int J Urol, 25:103-110.
47. Suzuki K, Yamaguchi T, Yanai M. (2018). Simultaneous occurrence of hypermucoviscous Klebsiella pneumoniae emphysematous prostatic abscess, emphysematous cystitis, and renal abscess. IDCases, 14: 00464.
48. Fung CP, Chang FY, Lee SC, Hu B S, Kuo B I-T, Liu C-Y, Ho M, Siu L K. (2002). A global emerging disease of Klebsiella pneumoniae liver abscess: is serotype K1 an important factor for complicated endophthalmitis, 50:420-424.
49. Wen SC, Juan YS, Wang CJ, Chang K Shih M C, Shen J T, Wu W J, Jang M Y. (2012). Emphysematous prostatic abscess: case series study and review. *Int J Infect Dis*, 16: 344-349.
50. Roberts RO, Lieber MM, Bostwick DG, Jacobsen S J. (1997). A review of clinical and pathological prostatitis syndromes. Urology, 49:809-821.
51. Hsu LN, Chiang PH, Kang CH. (2015). Emphysematous prostatic abscess: rare case and systematic review. J Formos Med Assoc, 114:292-293.
52. Vyas JB, Ganpule SA, Ganpule AP, Sabnis R B, Desai M R. (2013). Transrectal ultrasound-guided aspiration in the management of prostatic abscess: A single-center experience. *Indian* J Radiol Imaging, 23:253-257.
53. Elshal AM, Abdelhalim A, Barakat TS, Shaaban A A, Nabeeh A, Ibrahiem E-H. (2014). Prostatic abscess: objective assessment of the treatment approach in the absence of guidelines. Arab J Urol, 12(4):262-268.
54. Juan YS, Huang CH, Chang K, Wang CJ, Chuang SM, Shen JT, Wu WJ. (2008). Emphysematous prostatic abscess due to candidiasis: a case report. Kaohsiung J Med Sci, 24(2):99-102.
55. Wise GJ, Shteynshlyuger A. (2006). How to diagnose and treat fungal infections in chronic prostatitis. Curr Urol Rep, 7(4):320-328.
56. Epstein DJ, Thompson LDR, Saleem A, Kao CS, Epstein JI. (2020). Fungal prostatitis due to endemic mycoses and Cryptococcus: A multicenter case series. Prostate. 80(12):1006-1011.
57. Mayayo E, Fernández-Silva F. (2014). Fungal prostatitis: an update. Anal Quant Cytopathol Histpathol, 36(3):167-176.
58. Demirci A, Bozlak N, Turkel S. (2018). Chronic prostatitis developing due to candida infection: A case diagnosed 20 years later and review of up-to-date literature. Urol. Case Rep, 18:20:88-89.
59. Murphy A.B.1, Macejko A, Taylor A, Nadler R.B. (2009). Chronic prostatitis: management strategies. *Drugs*, 69(1):71-84.
60. Shoskes D.A.1, Nickel J.C. Classification and treatment of men with chronic prostatitis/chronic pelvic pain syndrome using the UPOINT system. World J Urol. 2013 Aug;31(4):755-760.
61. Fidel P L., Jr., Vazquez J A., Sobel J D. (1999). Candida glabrata: review of epidemiology, pathogenesis, and clinical disease with comparison to C. albicans. Clin Microbiol Rev, 12(1):80-96.
62. Nickel J.C.1, Downey J, Ardern D, Clark J, Nickel K. (2004). Failure of a monotherapy strategy for difficult chronic prostatitis/chronic pelvic pain syndrome. *J Urol*, 172(2):551-554.
63. Golz R, Mendling W. (1991). Candidosis of the prostate: a rare form of endomycosis. Mycoses. 34(9-10):381-384.
64. Gupta, Shaili MD, Liu-Young, Gustine MD, Mahnensmith, Rex MD, Topal, Jeffrey E. MD. (2008). Prostatic Abscess with Candida glabrata Treated with Micafungin. Infectious Diseases in Clinical Practice 16(6): 387-389.
65. Weinberger M, Cytron S, Servadio C, Block C, Rosenfeld J B, Pitlik S D. (1988). Prostatic abscess in the antibiotic er*a*. Rev Infect Dis, 10:239-249.
66. Rocco M, Soucie JM, Pastan S, McClellan W M. (2000). Peritoneal dialysis adequacy and risk of death. Kidney Int, 58:446-457.
67. Pfaller MA, Boyken L, Hollis RJ, Messer S A, Tendolkar S, Diekema D J. (2006). Global surveillance of in vitroactivity of micafungin against Candida*:* a comparison with caspofungin by CLSI-recommended methods. J Clin Microbiol, 44(10):3533-3538.
68. Pfaller MA, Diekema DJ, Gibbs DL, Newell VA, Meis JF, Gould IM, Fu W, Colombo AL, Rodriguez-Noriega E. (1997). Global Antifungal Surveillance Study. Results from the ARTEMIS DISK Global Antifungal Surveillance study, 1997 to 2005: an 8.5-year analysis of susceptibilities of Candida species and other yeast species to fluconazole and voriconazole determined by CLSI standardized disk diffusion testing. J Clin Microbiol, 45(6):1735-1745.
69. Pfaller MA, Messer SA, Boyken L, Rice C, Tendolkar S, Hollis R J, Diekema D J. (2007). Use of fluconazole as a surrogate marker to predict susceptibility and resistance to voriconazole among 13,338 clinical isolates of Candida spp. Tested by clinical and laboratory standards institute-recommended broth microdilution methods. J Clin Microbiol, 45(1):70-75.
70. Wagner C, Graninger W, Presterl E, Joukhadar C. (2006). The echinocandins: comparison of their pharmacokinetics, pharmacodynamics and clinical applications. Pharmacology, 78(4):161-177.
71. Maertens J, Raad I, Petrikkos G,Boogaerts M, Selleslag D, Petersen F B, Sable C A, Kartsonis N A, Ngai A, Taylor A, Patterson T F, Denning D W, Walsh T J. (2004). Efficacy and safety of caspofungin for treatment of invasive aspergillosis in patients who are refractory to or intolerant of conventional antifungal therapy. Clin Infect Dis, 39:1563-1571.

72. Colombo AL, Rosas RC. (2003). Successful treatment of an Aspergillus brain abscess with caspofungin: case report of a diabetic patient intolerant ofamphotericin B. Eur J Clin Microbiol Infect Dis, 22(9):575-576.
73. Stone JA, Xu X, Winchell GA, Deutsch P J, Pearson P G, Migoya E M, Mistry G C, Xi L, Miller A, Sandhu P, Singh R, DeLuna F, Dilzer sc, Lasseter K C. (2004). Disposition of caspofungin: role of distribution in determining pharmacokinetics in plasma. Antimicrob Agents Chemother, 48(3):815-823.
74. Groll AH, Mickiene D, Petraitis V, Ibrahim K H, Piscitelli S C, Bekersky I, Walsh J T. (2001). Compartmental pharmacokinetics and tissue distribution of the antifungal echinocandin lipopeptide micafungin (FK463) in rabbits. Antimicrob Agents Chemother, 45(12):3322-3327.
75. Groll AH, Mickiene D, Petraitiene R, Petraitis V, Lyman C A, Bacher J S, Piscitelli S C, Walsh J. (2001). Pharmacokinetic and pharmacodynamic modeling of anidulafungin (LY303366): reappraisal of its efficacy in neutropenic animal models of opportunistic mycoses using optimal plasma sampling. Antimicrob Agents Chemother, 45:2845-2855.
76. Ninomiya M, Mikamo H, Tanaka K, Watanabe K, Tamaya T. (2005). Efficacy of micafungin against deep-seated candidiasis in cyclophosphamide-induced immunosuppressed mice. J Antimicrob Chemother, 55(4):587-590.
77. Kuse ER, Chetchotisakd P, da Cunha CA, Ruhnke M, Barrios C, Raghunadharao D, Sekhon JS, Freire A, Ramasubramanian V, Demeyer I, Nucci M, Leelarasamee A, Jacobs F, Decruyenaere J, Pittet D, Ullmann AJ, Ostrosky-Zeichner L, Lortholary O, Koblinger S, Diekmann-Berndt H, Cornely OA. (2007). Micafungin Invasive Candidiasis Working Group. Micafungin versus liposomal amphotericin B for candidaemia and invasive candidosis: a phase III randomised double-blind trial. Lancet, 5:369(9572):1519-1527.

DOI:10.31579/2640-1045/137