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DISSEMINATED FUNGAL INFECTION CAUSING ENDOPHTHALMITIS: A CASE STUDY.

General Medicine

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ABSTRACT

A 39- year- old female, Diabetic, was found to have persistent anemia and thrombocytopenia, and was thus referred to our hospital for further management.Patient was worked up outside and was diagnosed as a case of urosepsis with obstructive uropathy B/L pyelonephritis with uncontrolled DM with anemia secondary to blood loss and thrombocytopenia. Patient was further investigated and found to be a case of disseminated candidiasis secondary to long standing vulvovaginitis missed on multiple PV examinations as patient was having PV and PR bleed at the time of examination with chronic immunocompromised state. She was managed in our hospital and was discharged on antifungals, antibiotics OHAs and insulin glargine. Two days later the same patient was brought to the ER in a drowsy state with hypoglycemia sugars of 30mg/dl and loss of vision in left eye with reduced eye movements. On further investigation patient was found to have fungal ball in her retina with impending endophthalmitis.

KEYWORDS

INTRODUCTION:

The clinical manifestations of infection with *Candida* species range from local mucous membrane infections to widespread dissemination with multisystem organ failure [1]. Although *Candida* are considered to be part of the normal microbiota in the gastrointestinal and genitourinary tracts of humans, they have the propensity to invade and cause disease when an imbalance is created in the ecologic niche in which these organisms usually exist.The immune response of the host is an important determinant of the type of infection caused by *Candida*.

- The most benign infections are characterized by local overgrowth on mucous membranes (oropharyngeal involvement, vaginitis) as a result of changes in the normal microbiota. More extensive persistent mucous membrane infections occur in individuals with deficiencies in cell-mediated immunity, such as AIDS.
- In the neutropenic host or the severely ill patient in the intensive care unit, widespread visceral dissemination occurs when *Candida* species gain access to the bloodstream.
- Invasive focal infections, such as pyelonephritis, endocarditis, and meningitis, most often occur after hematogenous spread or when anatomic abnormalities or devices (eg, prosthetic heart valves or central nervous system shunts) are present. The different *Candida* species generally are capable of producing all of the clinical syndromes, although infection with *Candida albicans* is the most common. It is important to identify the infecting organism because some species are more resistant to the azole antifungal agents than others.

CASE REPORT:

A 39 year old female, was referred to D.Y. Patil hospital, Nerul, Navi Mumbai, when she was found to have dropping hemoglobin levels, low platelets , and raised total counts on her blood picture, and with a USG abdomen report of B/L Bulky kidneys with pyelonephritis , hepatomegaly, uterine fibroid, cystitis and minimal free fluid in the pouch of duglas.

Patient had chief complaints of :
Recurrent fever with chills since 1 month
Generalized weakness since 1 month
Reduced frequency of micturition since 3 days
Abdominal pain and constipation since 3 days
No urine output since 1 day

On further inquiry,
Pt had history of weight loss of 8 Kgs in 2 months,
Black colored stools,
Known case of type 2 diabetes mellitus since 3 years,

Was started on tablet Metformin 500 BD. She had Stopped the medication on her own within one month of treatment due to the episodes of hypoglycemia.

And had started treatment with some homeopathic medication probably containing steroids. Along with some ayurvedic medication for menorrhagia and constipation.

Obstetric history:
G3P2L2A1
2 children
LMP – 5/6/21
Irregular cycle
Associated with blood clots
intermittent bleeding and spotting episodes +

On general physical examination:
Patient was conscious co-operative and oriented to time place and person, Patient had pallor, Icterus, Petechiae on B/L Lower limbs

Temperature of 101 F, a blood pressure of 120/70 mmhg , regular pulse rate of 84 beats/minute, respiratory rate of 22 breaths/ minute, Inguinal lymphadenopathy was present , skin turgor was normal.

On Per abdomen examination, there was B/L flank fullness, 3 cm hepatomegaly. Other systemic examination was unremarkable.

Blood Investigations:

Variable	On admission	On discharge
Hb (g/dl)	6.6	10.2
TLC (1000/Ul)	17,000	7300
Plt (10000/ Ul)	20,000	312000
S.creat (mg/dl)	5.93	1.3
FBS (mg/dl)	355	115
PLBS (mg/dl)	406	137
Hba1C (%)	14.5%	

Peripheral smear- large platelets seen, microcytic hypochromic, occ. Basophilic stippling, neutrophilic leukocytosis with 6% band forms. HHH - non-reactive.

Urine routine : Hazy ,pale yellow,
Ph- 6.5, Blood +++
Microscopy: 4-5 rbc/ hpf, plenty of leukocytes (pus cells) / hpf

buddying yeast cells with pseudohyphae.

Urine culture s/o candida.

Two consecutive Blood cultures suggestive of candida.
Gram staining-positive buddying yeast like cells with pseudohyphae.

Radiological investigations:
USG Abdomen: Liver- 16.5 cm,Spleen- 10 cm
Prominent splenic vein, Right kidney -15.7 x 7.3 cm, left kidney- 14.6 x 2 cm
Urinary Bladder – few moving echos and depended debries.
Uterine fibroid- 1.4 x 1.3 cm in posterior uterine wall.

CT KUB plain:
Right kidney – 14.5 x 7.5 cm, left kidney- 14.9 x 6.8 cm
Liver- 20.4 cm, Spleen 13.1 cm
Both kidneys are enlarged in size with perinephric fat stranding.
Mild right hydronephrosis and upper hydroureter.
B/L pyelonephritis, B/L minimal pleural effusion and mild pericardial effusion.

Xray chest- B/L pleural effusion
Bone marrow biopsy- Normal

Patient was diagnosed as : Systemic candidiasis secondary to immunocompromised state with urosepsis, obstructive uropathy, acute kidney injury with uncontrolled sugars with anemia, thrombocytopenia.

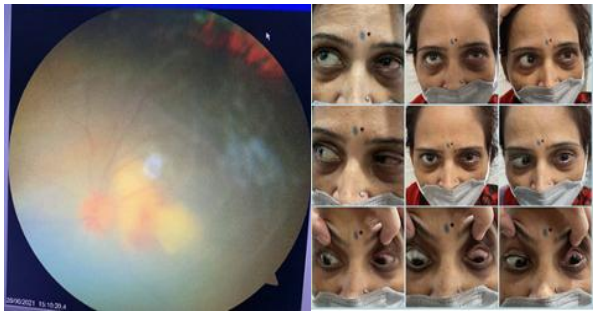
Patient was started on
IV. Fluconazole 400mg BD
IV. Meropenem 500mg TDS
IV Teicoplanin 400mg BD
For 10 days,
Patient was discharged on
Oral fluconazole 200 BD for 10 days
and tab levofloxacin 250 BD for 7 days.
Tab glimiperide + metformin 1/500 BD
Tab teneligliptin 20 OD
Injection insulin glargine (lantus) 10U HS
And clindamycin (100mg) + cotrimazole(100mg) + tinidazole (100mg) vaginal pessary PV for 7 days.

Patient presented in ER 2 days post discharge with drowsiness, unresponsiveness secondary to hypoglycemia and was re-admitted, Hypoglycemia was corrected and ophthalmology consultation was ordered.

On examination patient had reduced left eye movement, redness and progressive blurring of vision. On ophthalmological examination of fundus,

A whitish yellow patch next to the optic disc is seen that is a fungal ball, (seen as a flat patch since this is a 2D image.)

And the yellowish patch between optic disc and fungal ball is retinitis, with vitreitis progressing to endophthalmitis.



On 9 gaze examination it was also seen that there is restriction of moment in Levo-version (looking towards left side) suggestive of restricted abduction due to left lateral rectus involvement/ inflammation.

A differential diagnosis of mucormycosis was considered for the patient given the time when she was admitted , but since mucor is an exogenously acquired infection and candida/ aspergillosis are endogenously acquired infections a diagnosis of candida retinitis was made.

Patient was advised and posted for intravitreal injections of liposomal amphotericin B, but she did not give consent for the same,

Thus, she was started on tablet Posaconazole and after the creatinine was within normal limits she was treated with injectable amphotericin B. recovery was good.

Patient was discharged on oral Tab. Posaconazole 200 mg BD.

DISCUSSION:
Vulvovaginitis-Vulvovaginal candidiasis is the most common form of mucosal candidiasis. It most often occurs in situations associated with increased oestrogen levels, such as oral contraceptive use and pregnancy. Antibiotics, glucocorticoids, diabetes mellitus, HIV infection, intrauterine devices, and diaphragm use are also risk factors.

The clinical manifestations of *Candida* vulvovaginitis are primarily itching and discharge. Dyspareunia, dysuria, and vaginal irritation also may be present. Physical examination shows vulvar erythema and swelling and vaginal erythema and discharge, which is classically white and curd-like but may be watery. Some patients, primarily those with *Candida glabrata* infection, have little discharge and often only erythema on vaginal examination.

The diagnosis of *Candida* vulvovaginitis is typically made clinically, but confirmation is easily obtained by observing budding yeast, with or without pseudo-hyphae, on a wet mount or KOH preparation of vaginal secretions.

Risk factors for invasive infection:
Candida infections are most often associated with candidemia, which primarily occurs in immunocompromised patients and those requiring intensive care. Immunocompromised patients at special risk for candidemia include:

- Those with hematologic malignancies
- Recipients of solid organ or hematopoietic cell transplants
- Those given chemotherapeutic agents for a variety of different diseases

Neutropenia is common in these settings, and most transplant recipients are also receiving glucocorticoids.
Other risk factors in these patients include chemotherapeutic agents, especially those associated with extensive gastrointestinal mucosal damage, broad-spectrum antibiotics, and central venous catheters.
Patients in intensive care units (ICUs) account for the greatest number of episodes of candidemia at most hospitals.
Surgical units, especially those caring for trauma and burn patients, and neonatal units have the highest rates of *Candida* infections. Other risk factors commonly associated with invasive candidiasis in ICU patients include:

- Central venous catheters
- Total parenteral nutrition
- Broad-spectrum antibiotics
- High APACHE scores
- Acute kidney injury, particularly if requiring hemodialysis
- Prior surgery, particularly abdominal surgery
- Gastrointestinal tract perforations and anastomotic leaks

Candidemia And Invasive Candidiasis:
Candidemia refers to the presence of *Candida* species in the blood.

The clinical manifestations vary from minimal fever to a full-blown sepsis syndrome indistinguishable from severe bacterial infection. Invasive candidiasis occurs when visceral sites are infected as a result of hematogenous spread.

Clinical clues to the occurrence of hematogenous spread of *Candida* include characteristic eye lesions, skin lesions, and, less commonly, muscle abscesses. The skin lesions tend to appear suddenly as clusters of painless pustules on an erythematous base; they can occur on any area of the body. The lesions vary from tiny pustules that can be easily missed to others that are nodular, up to several centimeters in diameter, and may appear necrotic in the center.

In addition to these typical peripheral sites of involvement, signs of multiorgan system failure may be present. Autopsy studies reveal widespread visceral microabscesses that are especially prominent in the kidneys, heart, liver, spleen, lungs, and brain.

INVASIVE FOCAL INFECTIONS:

Urinary tract infection-Funguria is common in hospitalized patients, although it is often difficult to distinguish colonization from infection of the bladder. *Candida* infection of the bladder must be distinguished from infection of the kidney, although the two entities can coexist.

Invasive infection of the kidney is unusual and is more difficult to treat than infection of the bladder.

Renal infection can be secondary to hematogenous seeding and is typically characterized by multiple microabscesses in the setting of disseminated candidiasis. Ascending infection develops more insidiously, is usually unilateral, and can be complicated by development of an obstructing bezoar or fungus ball.

Endophthalmitis-*Candida* endophthalmitis can develop exogenously following trauma or eye surgery or endogenously through hematogenous seeding of the retina and choroid as a complication of candidemia.

Because of the risk of endophthalmitis from hematogenous seeding, a dilated retinal examination by an ophthalmologist should be performed in all candidemic patients.

The primary presenting symptom is a decrease in visual acuity; pain can sometimes occur. The classic findings of chorioretinal involvement are focal, white, infiltrative, often mound-like lesions on the retina.

When vitreous extension is present, a vitreal haze is present; sometimes fluffy white balls or "snowballs" in the vitreous are noted. The infection can be sight-threatening if it is not treated promptly.

Summary And Recommendations:

- *Candida* in a blood culture should never be viewed as a contaminant and should always prompt a search for the source. For many patients, candidemia is a manifestation of invasive candidiasis, whereas for others it reflects colonization of an indwelling intravenous catheter.
- The clinical manifestations of candidemia vary from minimal fever to a full-blown sepsis syndrome that is indistinguishable from severe bacterial infection. Invasive candidiasis is defined as hematogenous spread to multiple viscera (eg, eye, kidney, heart valves, brain).
- Clinical clues on physical examination to hematogenous spread of *Candida* include characteristic eye lesions (chorioretinitis with or without vitritis), skin lesions, and, much less commonly, muscle abscesses.
- The gold standard for the diagnosis of candidemia is a positive blood culture; blood cultures should be obtained in all patients with suspected candidemia. However, blood cultures can be negative in patients with invasive candidiasis, and the diagnosis in such cases is often based on clinical suspicion.
- In patients with focal findings (eg, skin lesions or parenchymal involvement), biopsy should be performed for staining, culture, and histopathologic evaluation.
- When available, we suggest obtaining a beta-D-glucan assay as an adjunct to blood cultures and biopsy. It appears to be particularly useful in patients with deep-seated candidiasis (eg, intra-abdominal candidiasis), given the low sensitivity of blood cultures in this setting. However, beta-D-glucan is present in the cell wall of many fungi, so therefore is not specific for *Candida* spp.
- The clinical manifestations of infection with *Candida* species range from local mucous membrane infections to widespread dissemination with multisystem organ failure. Although *Candida* species are considered to be part of the normal microbiota in the gastrointestinal and genitourinary tracts of humans, they have the propensity to invade and cause disease when an imbalance is created in the ecologic niche in which these organisms usually exist.
- Oropharyngeal candidiasis, or thrush, is a common local infection seen in infants; older adults who wear dentures; patients treated with antibiotics, chemotherapy, or radiation therapy to the head and neck; and those with cellular immune deficiency states, such as AIDS. Patients with xerostomia and those treated with inhaled glucocorticoids for asthma or rhinitis are also at risk.
- Esophageal candidiasis is most common in HIV-infected patients, in which it is an AIDS-defining illness, and in patients with hematologic malignancies.
- Vulvovaginal candidiasis is the most common form of mucosal candidiasis. It most often occurs in situations associated with increased estrogen levels, such as oral contraceptive use and pregnancy. Antibiotics, glucocorticoids, diabetes mellitus, HIV infection, intrauterine devices, and diaphragm use are also risk factors.
- Chronic mucocutaneous candidiasis is a rare syndrome that usually has its onset in childhood. Some patients have autosomal recessive polyglandular autoimmune syndrome type I, which is also referred to as the autoimmune polyendocrinopathy-candidiasis-ectodermal dystrophy syndrome.
- Candidemia refers to the presence of *Candida* species in the blood. Immunocompromised hosts and patients in intensive care units are at the highest risk of candidemia.
- Hepatosplenic candidiasis (also called chronic disseminated candidiasis) is seen almost entirely in patients with hematologic malignancies who have just recovered from an episode of neutropenia. Some patients have a documented prior episode of candidemia; in others, it is likely that invasion of *Candida*species occurred through the portal vasculature.
- Invasive focal infections include urinary tract infection, endophthalmitis, osteoarticular infections, meningitis, endocarditis, peritonitis and intra-abdominal infections, empyema, mediastinitis, and pericarditis.
- Pneumonia due to *Candida* is extremely rare.

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