

Candiduria as a Diagnostic Clue for HIV Infection in an Adolescent with Type 1 Diabetes Mellitus: A Case Report

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ABSTRACT

Candiduria is an uncommon urinary tract infection in immunocompetent individuals and is challenging to diagnosis. Urinalysis is the initial tool for detecting Urinary Tract Infections (UTIs), but candida species do not convert nitrates to nitrites, necessitating careful diagnostic practices and confirmation through urine cultures. This report focuses on a 16-year-old female with type 1 diabetes who developed Diabetic Ketoacidosis (DKA) and candiduria. Urinalysis indicated DKA with glycosuria and ketonuria, masking the UTI. While DKA was managed with insulin and fluids, the UTI did not respond to cefuroxime, and urine cultures showed no bacteria. After 48 hours, *Candida albicans* was identified via microscopy, and treatment with oral fluconazole was successful. The candiduria diagnosis also led to positive HIV screening, and the report discusses the challenges of managing her condition, especially after initiating highly active antiretroviral therapy.

Keywords: Candiduria, Diabetes mellitus, Immunosuppression, Retroviral infections

CASE REPORT

A 16-year-old female with a four-year history of Type 1 Diabetes Mellitus presented to the children’s emergency unit with progressive vulvar swelling and generalised weakness. The swelling, initially peanut-sized, had enlarged fivefold over three days. She reported fever and two episodes of non-bilious, non-bloody vomiting, but denied abdominal pain, sexual activity, pregnancy, or drug use. She had been non-compliant with insulin therapy due to financial constraints. On examination, she was severely dehydrated, febrile (37.9°C), tachycardic (120 bpm, weak pulse), and weighed 47 kg. Blood pressure was 101/66 mmHg. Oral examination revealed whitish plaques on the palate and tongue with pharyngeal erythema. Perineal examination showed a discrete, tender, cystic swelling of the left vulva with differential warmth, foul-smelling vaginal discharge, whitish patches, and exfoliated lesions on the left labia majora. Other systemic findings were normal.

Initial investigations revealed hyperglycaemia (random blood sugar 30 mmol/L) and HbA1c of 14.1%. Electrolytes and renal function were within normal limits except for metabolic acidosis [Table/Fig-1]. Urine culture grew *Candida albicans*, while retroviral screening was positive on day 5. Full blood count showed leukocytosis (18,700/mm³) on admission, which normalised by day 5 [Table/Fig-2]. Urinalysis demonstrated ketonuria and glycosuria at presentation, both resolving with treatment.

Electrolytes and urea (Day 1)	
Sodium	134 mmol/L
Potassium	4.1 mmol/L
Chloride	100 mmol/L
Bicarbonate	13 mmol/L
Urea	3 mmol/L
Creatinine	60 µmol/L
Hepatotropic viruses screening results	
HBsAg	Negative
HCV	Negative
Infection panel results	
Blood culture	No growth

Urine microscopy	Epithelial cells - ++ White blood cells - 12-14 High power field Red blood cells - 1 Yeast cells - +++
Urine culture	<i>Candida albicans</i> species
VDRL	Negative
Gene expert	Negative
Retroviral screen (Day 5)	Positive

[Table/Fig-1]: Blood chemistry and microbiology results of the patient.

Parameter	Day 1	Day 3	Day 5
Full blood count			
White blood cell count	18,700/mm ³		5100/mm ³
Platelets	Not done		298000/mm ³
Differential neutrophil	78%		74%
Differential lymphocytes	20%		22%
Differential monocytes	02%		04%
CD4			367 cells/mm ³
Urinalysis			
Leucocytes	Negative	Negative	Negative
Nitrite	Negative	Negative	Negative
Bilirubin	Negative	Negative	Negative
Urobilinogen	Normal	Normal	Normal
Protein	Negative	Negative	Negative
PH	6.0	6.0	7.0
Blood	Negative	Negative	Negative
Specific gravity	1.030	1.025	1.020
Ketones	+++	++	Negative
Glucose	+++	++	+

[Table/Fig-2]: Patient’s serial blood cellular profile and urinalysis result.

A provisional diagnosis of DKA secondary to UTI with an infected Bartholin’s cyst was made. Oral thrush, vaginal candidiasis, and possible genital herpes were considered presumptive diagnoses, supported by clinical findings and resolution after fluconazole therapy.

The DKA was managed with intravenous fluids, insulin infusion (0.1 IU/kg titrated to euglycaemia), and potassium supplementation. Cefuroxime (150 mg/kg/day in two divided doses) was administered for the Bartholin's gland infection. Vaginal candidiasis was initially treated with topical nystatin and Mycoten cream, and later switched to fluconazole following urine culture confirmation of *Candida albicans*. Despite antibiotics, vulvar swelling persisted, requiring incision and drainage by a gynaecologist.

Within 72 hours, ketoacidosis resolved with normalisation of electrolytes, bicarbonate, and urine ketones. Blood glucose stabilised between 4.2 and 7.7 mmol/L. Fever and dehydration resolved within 48 hours of therapy. *Candida* was no longer isolated after fluconazole treatment. The patient was discharged on day 8 in good condition (random blood sugar 6.6 mmol/L).

Retroviral screening confirmed HIV infection. She was referred for endocrinology and paediatric antiretroviral follow-up. Initiation of Dolutegravir-based HAART led to hyperglycaemia, necessitating significant escalation of insulin doses for glycaemic control.

DISCUSSION

Candiduria is frequently encountered, but in immunocompromised patients such as those with HIV infection, it carries a higher risk of morbidity and mortality because it may signal systemic candidiasis. Clear criteria to distinguish colonisation from infection, or upper from lower urinary tract involvement, are lacking. In one case study, the overall prevalence of candiduria among HIV patients was 13.5%, with HAART-naïve individuals showing a significantly higher prevalence compared to those on therapy. Female gender was also identified as a significant risk factor for candiduria [1]. In HIV, the spectrum ranges from asymptomatic colonisation to invasive disease, with immune compromise driving dissemination [2]. In this patient, features suggestive of sexually transmitted infection and invasive *Candida* disease prompted HIV screening despite known diabetes mellitus.

Diabetes mellitus itself is associated with increased candiduria, with reported prevalence of 12.5-14.3% [2]. Severe or recurrent candidiasis should trigger evaluation for immune suppression and diabetes. In this case, candiduria was initially difficult to interpret: *Candida* is not a common UTI pathogen, nitrite was absent (as *Candida* does not reduce nitrates), and the dipstick failed to detect blood or leukocytes despite microscopy findings. Nonetheless, fluconazole therapy led to the resolution of fever and candiduria, supporting pathogenic infection rather than contamination. Prompt antifungal treatment may have prevented dissemination [3].

Management was limited by financial constraints, precluding serial CD4 counts or viral load testing, a common challenge in resource-limited settings. Such assays could have correlated immune status with infection severity. Post-discharge, glycaemic control was complicated by Dolutegravir-based HAART, the national first-line regimen for adolescents in Nigeria [4]. Dolutegravir has been linked to hyperglycaemia through mechanisms such as magnesium chelation and mitochondrial dysfunction, with prior reports confirming this adverse effect. This necessitates careful insulin titration and consideration of more diabetes-friendly HAART guidelines [5,6].

The presence of *Candida* in urine should always prompt evaluation for predisposing factors such as catheterisation, antibiotic use, urinary tract anomalies, or systemic immunosuppression. In this case, candiduria likely contributed to DKA, as UTI is a known precipitant. Failure to treat candiduria risks dissemination and may accelerate HIV progression. Conversely, poor HIV control worsens *Candida* infection, while uncontrolled diabetes promotes both insulin resistance and fungal overgrowth. Thus, HIV, candiduria, and diabetes exert a collateral negative impact if inadequately managed. Optimal outcomes require strict glycaemic control, adherence to HAART, and timely treatment of co-infections [7].

Niu S et al., described a case of a 55-year-old male with poorly controlled diabetes and multiple urinary calculi who repeatedly passed visible urinary fungal masses due to *Candida tropicalis* candiduria, requiring adjunctive caspofungin, bladder irrigation, and intensified glycemic control. In contrast, our patient was a 16-year-old girl with no urinary tract anomalies or calculi, presenting instead with vulvar swelling, oral thrush, and diabetic ketoacidosis [8].

Similarly, Ahmadi F et al., reported a 63-year-old man with uncontrolled diabetes who developed candidal cystitis with intravesical fungal balls mimicking an enterovesical fistula. Management required bladder irrigation with amphotericin B and oral fluconazole. In contrast, our patient's candiduria was accompanied by genital candidiasis and HIV immunosuppression, with resolution achieved through systemic fluconazole and gynecological drainage of an infected Bartholin's cyst [9]. Taken together, these comparisons emphasise that while adult cases often involve urinary obstruction, calculi, or catheterisation, our adolescent case uniquely combines uncontrolled Type 1 diabetes, candiduria, and newly diagnosed HIV without prior instrumentation.

Sustained investment is essential to prevent HIV escalation and mitigate the burden of diabetes in resource-limited settings.

CONCLUSION(S)

This case highlights the complexity of managing candiduria in adolescents with Type 1 Diabetes Mellitus with coexisting immunosuppression from HIV infection. Early recognition of candiduria and prompt adjustment of therapy are crucial for favourable outcomes, particularly in resource-limited settings. The case also underscores the importance of comprehensive patient education, intensive monitoring of blood glucose and viral suppression, adherence to treatment, and multidisciplinary follow-up for chronic conditions like diabetes and HIV.

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