

Lactobacillus Species as an Unexpected Uropathogen in the Third Trimester of Pregnancy leading to premature labour: A Case Report and Clinico- Microbiological Appraisal

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Case Report

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Abstract

Background

Urinary tract infection (UTI) is one of the most frequent bacterial infections during pregnancy and is related with adverse maternal and newborn outcomes, including preterm labour. Although *Lactobacillus* species compose the typical vaginal microbiota and are widely regarded as non-pathogenic commensals, emerging data suggests that they can occasionally behave as opportunistic pathogens under specific clinical situations. We describe a rare instance of a late-pregnancy symptomatic UTI brought on by *Lactobacillus* species, underscoring the difficulties in diagnosing and treating this uncommon uropathogen.

Case Presentation:

A 29-year-old gravida 3, para 1, abortion 1 woman at 37 weeks and 6 days of gestation presented with burning micturition, increased urine frequency, and lower abdomen pain. Pyuria was detected by urine microscopy (8–10 pus cells/high-power field). Significant pure development of *Lactobacillus* species was seen in midstream urine culture; this was verified by repeat culture, ruling out contamination. Conventional microbiological techniques were used to identify the isolate, which showed resistance to ampicillin, cefixime, and cefotaxime but susceptibility to amoxicillin–clavulanate, erythromycin, clindamycin, and vancomycin. After receiving targeted treatment with intravenous amoxicillin–clavulanate, the patient had total clinical recovery despite failing empirical cefixime medication. The microbiological cure was confirmed by a sterile follow-up urine culture. The patient went into labour while in the hospital and gave birth to a healthy baby.

Conclusion

This case shows that when *Lactobacillus* species are regularly recovered in pure culture from symptomatic pregnant individuals with pyuria and chronic urine symptoms, they should not be routinely written off as contaminants. Accurate diagnosis and suitable treatment depend on the correlation of microbiological results with clinical characteristics, repeat urine culture, and culture-guided antibiotic medication. A greater understanding of *Lactobacillus* as a possible opportunistic uropathogen could enhance maternal outcomes and promote antibiotic stewardship during pregnancy.

Introduction

Urinary tract infection (UTI) complicates approximately 8% of pregnancies, spanning a clinical spectrum from asymptomatic bacteriuria (ASB) to symptomatic cystitis and pyelonephritis.^{1,2} Pregnancy-specific anatomical and hormonal changes — progesterone-induced ureteric dilatation, vesicoureteral reflux, reduced bladder capacity from mechanical compression by the gravid uterus, and physiological

hydronephrosis affecting up to 90% of pregnant individuals – collectively create a large reservoir of static urine that facilitates ascending bacterial colonisation.^{1,2} If untreated, bacteriuria progresses to pyelonephritis in up to 30% of cases, and is associated with preterm labour, low birth weight, maternal anaemia, sepsis, disseminated intravascular coagulation, and acute respiratory distress syndrome.^{2,3} Clinically, acute cystitis in pregnancy manifests as dysuria, urinary frequency, urgency, and suprapubic discomfort – symptoms that partially overlap with normal third-trimester physiology, demanding that urinalysis and culture be obtained before initiating therapy.^{1,2} The ACOG 2023 Clinical Consensus defines clinically significant bacteriuria as $\geq 10^5$ CFU/mL of a single uropathogenic organism; pyuria carries up to 97% sensitivity for UTI but lower specificity in pregnancy because vulvovaginal discharge may contaminate voided specimens.^{2,3} Crucially, symptomatic patients with persistent lower urinary tract symptoms despite appropriate empirical therapy warrant microbiological re-evaluation, even when the isolated organism lies outside conventional uropathogen classifications.

Escherichia coli accounts for 60–82.5% of pregnancy-associated UTIs; other established uropathogens include *Klebsiella pneumoniae*, *Proteus mirabilis*, Group B Streptococcus, and *Enterococcus* species.^{1,4} *Lactobacillus* species, by contrast, are the dominant commensals of the healthy vaginal microbiome, protecting the urogenital tract through lactic acid production (maintaining pH 3.8–4.5), hydrogen peroxide, and bacteriocins that suppress uropathogen overgrowth.^{4,5} Both the ACOG and the American Urological Association (AUA) therefore explicitly exclude *Lactobacillus* spp., *Corynebacterium*, and coagulase-negative staphylococci from the diagnostic threshold for clinically significant bacteriuria, classifying them as non-pathogens irrespective of colony count.^{2,4} This protective architecture, however, is not immutable. Predisposing factors – vaginal candidiasis, topical antimicrobials such as clindamycin-clotrimazole preparations, and the relative immunotolerance of pregnancy, which suppresses pro-inflammatory T-lymphocyte signalling to protect the semi-allogeneic foetus – can perturb the vaginal microbiome and attenuate the mucosal immune response within the bladder, creating a permissive window in which a commensal organism of ordinarily low virulence may transition to a clinically relevant uropathogen.^{2,5,9,10}

Lactobacillus species are Gram-positive, non-spore-forming, facultative anaerobic bacilli that constitute the predominant normal vaginal microbiota in healthy women of reproductive age. During pregnancy, hormonal changes, particularly increased estrogen levels, promote glycogen deposition in vaginal epithelial cells, favouring the growth of *Lactobacillus* species.¹¹

A growing body of evidence has systematically challenged the dogma that *Lactobacillus* isolates in urine are invariably contaminants. *Lactobacillus delbrueckii* was identified as the definitive uropathogen in an elderly female patient with chronic pyuria and lower urinary tract symptoms unresponsive to three sequential fluoroquinolone courses – a case that simultaneously highlighted the intrinsic resistance of *Lactobacillus* spp. to quinolones, cephalosporins, and aminoglycosides, which explains why standard empirical regimens so predictably fail.⁶ A subsequent large European case series established the uropathogenic role of *L. delbrueckii* subspecies *allosunkii* in immunocompromised and anatomically

predisposed patients, urging clinical laboratories to reconsider reflexive dismissal of *Lactobacillus* isolates from symptomatic individuals and recommending MALDI-TOF mass spectrometry for reliable species identification.⁷ A neonatal case further demonstrates that *Lactobacillus* uropathogenesis spans the full immunological spectrum, not solely the elderly or immunosenescent.⁸

The clinical significance of *Lactobacillus*-associated UTI in pregnancy is compounded by three converging challenges: first, the diagnostic overlap of cystitis symptoms with normal pregnancy physiology; second, the absence of reliable activity against *Lactobacillus* in the empirical agents most commonly used in pregnancy (cephalosporins, nitrofurantoin); and third, the specific association between third-trimester UTI and elevated risks of preterm birth and neonatal sepsis.^{2,3} When *Lactobacillus* is isolated as the sole organism from a symptomatic pregnant patient with significant pyuria and documented failure of first-line empirical therapy, the possibility of genuine uropathogenesis must be evaluated rather than reflexively dismissed. We report such a case from a NABH-accredited tertiary care hospital in South India and discuss its microbiological, obstetric, and antimicrobial stewardship implications.

Case presentation

Patient Demographics and Presenting Complaints

A 29-year-old female, Gravida 3, Para 1, Living 1, Abortion 1(G3P1L1A1), at 37 weeks 6 days Period of Gestation (POG), was admitted to the Department of Obstetrics and Gynaecology of a NABH-accredited tertiary care teaching hospital in Mysuru, South India, she presented with a principal complaint of burning micturition and increased frequency of urination, along with lower abdominal pain since the day of arrival. She didn't have any bleeding or leaking per vaginum (PV). Throughout the present pregnancy, regular prenatal care was received at the same facility.

Obstetric History

The patient has been married for 7 years. She is G3P1L1A1. Her first pregnancy was a spontaneous conception that resulted in a missed abortion at 6 weeks of gestation, which was managed conservatively as the products of conception were expelled spontaneously without the need for dilatation and curettage. Her second pregnancy was also a spontaneous conception, resulting in a preterm vaginal delivery at 34 weeks of gestation with a male baby weighing 1.5 kg at birth. In view of functional disability noted at birth, the neonate required admission to the Neonatal Intensive Care Unit (NICU) for a period of 3 months. The current pregnancy is her third, conceived spontaneously, during which she presented at 37 weeks and 6 days of gestation with symptoms of urinary tract infection, and subsequently underwent delivery at approximately 8.5 months of gestation, suggesting a possible association between the urinary tract infection and the onset of labour.

Antenatal History

First Trimester (T1)

Conceived spontaneously; Urine Pregnancy Test (UPT) confirmed; dating scan and NT/NB (Nuchal Translucency/Nasal Bone) scan performed and reported normal. Two doses of Inj. Tetanus Toxoid administered one week apart. Folic acid supplements taken regularly. No history of exposure to radiation, teratogens, hyperemesis gravidarum, or fever with rash.

Second Trimester (T2)

Quickening was perceived at 5 months of gestation. The patient took iron and calcium supplements regularly. An early anomaly scan was reported as normal, showing no detectable congenital anomalies. Couple karyotyping revealed normal chromosomal patterns in both partners (46, XX and 46, XY), with no evidence of chromosomal abnormalities. There was no history of elevated blood pressure recordings. An oral glucose tolerance test (OGTT) performed and showed a result of 127 mg/dL, following which non-nutritive therapy (NNT) was advised. She denied any history of bleeding per vaginum or leaking per vaginum.

Third Trimester (T3)

The patient continued to perceive adequate foetal movements throughout the third trimester. She remained compliant with iron and calcium supplementation and attended regular follow-up visits for genetic counselling.

At 37 weeks and 6 days of gestation, she presented with complaints of burning micturition, increased urinary frequency, and pain over the medial aspects of both thighs. In view of her urinary symptoms, a midstream urine sample was sent for culture and sensitivity testing, and empirical oral cefixime 200 mg twice daily was initiated. However, her symptoms persisted despite treatment.

On obstetric examination, the cervix was approximately 2 cm dilated (admitting two fingers), suggestive of the onset of labour. The initial urine culture yielded significant growth of *Lactobacillus species*. To rule out contamination/colonization and confirm the finding, a repeat urine culture was performed, which again demonstrated significant growth of *Lactobacillus species*.

Based on the repeated culture findings and antimicrobial susceptibility results, intravenous amoxicillin-clavulanate 1.2 g stat dose was administered. In the meantime, the patient progressed into labour and subsequently delivered a healthy baby. By this time her urinary symptoms were relieved. Following completion of antibiotic therapy, a repeat urine culture was performed, which showed no bacterial growth, indicating microbiological clearance of the infection. As shown in the Table 1

Table 1
Physical and systemic examinations and radiological and Laboratory findings

Parameter	Observation
Physical examination	
Blood pressure	127/87 mmHg
Pulse rate	77 beats/minute
Febrile status	Afebrile
Systemic examination	
Cardiovascular system	NAD
Respiratory system	NAD
Per-abdomen examination	Uterus involuting well
Laboratory findings	
CBC - COMPLETE BLOOD COUNT	
Hemoglobin (Hb)	11.8 g/dl
Total Leucocyte count (TLC)	8920 cells/cumm
Neutrophils	82.0
Lymphocytes	13.9
Eosinophils	0.4
Monocytes	3.6
Basophils	0.1
PCV/HCT (Packed Cell Volume / Haematocrit)	36.4
RBC Count	4.20
Platelet Count	2.22
MCV (Mean Corpuscular Volume)	86.7
MCH (Mean Corpuscular Haemoglobin)	28.1
MCHC (Mean Corpuscular Haemoglobin Concentration)	32.4
Liver Function Test	
Albumin	3.2
Total Proteins	5.6

Parameter	Observation
Physical examination	
A/G Ratio	1.4
AST (Aspartate Aminotransferase),	17
Bilirubin Direct	0.20
ALT (Alanine Aminotransferase),	9
Alkaline Phosphatase	261
Gamma Gt	5
Bilirubin Total	0.33
AST/ALT Ratio	1.97
Serum Globulin	2.380
Serum Indirect Bilirubin	0.13
Renal Function Test	
Glucose - Random	87
Urea	12
Creatinine	0.46
Uric Acid	5.1
Sodium	135
Potassium	4.4
Chloride	103
APTT (Activated Partial Thromboplastin Time)	
Test	32.2
Control	31.4
Ratio	1.03
PT- Prothrombin Time	
Test	11.6
Control	13.5
INR	0.84
Ratio	0.86

Parameter	Observation
Physical examination	
Blood Group	O Positive
TSH (Thyroid-Stimulating Hormone)	1.590
Anti HCV By Clia (Chemiluminescence Immunome Assay)	Non-reactive
Anti-HIV By Clia (Chemiluminescence Immuno Assay)	Non-reactive
HBsAg By Clia (Chemiluminescence Immuno Assay)	Non-reactive
Urine Routine	
Urine Sugar (Glucose)	Negative
Urine - Albumin (Protein)	Positive 1+ (25 mg/dL) (Day 1)
Urine Microscopy	
RBCs	Nil
Pus Cells	8–10
Epithelial Cells	2–4
Casts	Nil
crystals	Nil

Menstrual History

Menarche at 13 years. Regular cycles of 28–30 days duration, flow 4 days, moderate in amount, no dysmenorrhoea, no clots.

Medical / Surgical History

No history of hypertension, diabetes mellitus, cardiac or renal disorders. No history of major surgeries (asthma, epilepsy noted as past, no active disease). The observations are shown in the Table 1, No history of blood or blood product transfusion. No known drug allergies.

Microbiological evaluation

A midstream clean-catch urine sample was collected under aseptic precautions and processed for culture in the Microbiology department. Urine routine microscopy revealed 8–10 pus cells/high-power field, suggestive of urinary tract inflammation. The specimen was inoculated onto HiCrome UTI agar plate using a calibrated loop technique and incubated aerobically at 37°C for 24–48 hours. Significant

bacteriuria with a colony count of 10^5 CFU/mL was observed, yielding pure growth of tiny purple-colored colonies on the culture plate.

Gram staining demonstrated Gram-positive bacilli arranged singly and in short chains. The organism was non-motile, catalase-negative, oxidase-negative, indole-negative, citrate-negative, urease-negative, and Triple Sugar Iron (TSI) agar showed an acid slant and acid butt (A/A) without hydrogen sulphide production or gas formation, indicating carbohydrate fermentation by the isolate. Based on colony morphology, Gram staining, and conventional biochemical reactions performed manually, the isolate was identified as *Lactobacillus* species.

Table 2
Antimicrobial susceptibility pattern of
Lactobacillus species.

Antimicrobial agent	Interpretation
Ampicillin	Resistance
Erythromycin	Sensitive
Clindamycin	Sensitive
Amoxicillin/clavulanic acid	Sensitive
Vancomycin	Sensitive
Cefixime	Resistance
Cefotaxime	Resistance

As the laboratory did not have access to the VITEK® 2 ANC card for automated identification and antimicrobial susceptibility testing (AST) of *Lactobacillus* species, and broth microdilution testing facilities were unavailable, antimicrobial susceptibility testing was performed manually using the Kirby–Bauer disk diffusion method. The isolate was tested on Mueller–Hinton agar supplemented with 5% sheep blood, and the inoculated plates were incubated under anaerobic conditions at 37°C for 24–48 hours.¹⁴ Due to the limited availability of standardized CLSI clinical breakpoints for *Lactobacillus* species,¹⁴ inhibition zone diameters were recorded and interpreted descriptively rather than categorically. The susceptibility profile was subsequently correlated with published literature and the known intrinsic resistance patterns of *Lactobacillus* species to guide clinical interpretation.¹⁵

As shown in the Table 2

Discussion

Lactobacillus species have been recognised as opportunistic pathogens that can cause invasive infections in humans, despite the fact that they are typically thought of as beneficial commensals of the gastrointestinal and female genital tracts. *Lactobacillus* species were linked to bacteraemia, infective

endocarditis, meningitis, liver abscess, intra-abdominal abscesses, peritonitis, pneumonia, and sepsis in newborn.¹⁶ Rossi et al. found 48 cases of invasive *Lactobacilli* infections in their systematic evaluation of case reports published between 2019 and 2021.¹⁷ The most frequent presentations were bacteraemia and infective endocarditis. Additionally, they reported less common but clinically significant infections, such as meningitis, pneumonia, pyelonephritis, chorioamnionitis, intra-abdominal abscess, liver abscess, and neonatal sepsis, especially in patients with diabetes mellitus, immunosuppression, cancer, or other predisposing conditions.¹⁸ Evidence from the study suggests that *Lactobacillus* isolated from normally sterile clinical specimens should be considered a potential pathogen rather than a contaminant when correlated with compatible clinical findings¹⁸.

Lactobacillus species have many virulence factors, such as surface adhesins and extracellular matrix-binding proteins that interact with host fibronectin, fibrinogen, and collagen to facilitate tissue adhesion, early colonisation, and evasion of host immune responses, are thought to be responsible for their pathogenic potential. Furthermore, the synthesis of enzymes such as aryl amidase proteases and glycosidases facilitate the breakdown of host glycoproteins, which encourages tissue invasion. Certain strains also contain the potential to bind fibrinogen and stimulate human platelet aggregation, a critical mechanism contributing to the development of systemic infections such as infective endocarditis.^{19,21,23}

The current case demonstrates how *Lactobacillus* species are becoming recognised as possible uropathogens during pregnancy. Even though *Lactobacillus* species are usually thought of as normal vaginal commensals and are frequently written off as contaminants when isolated from urine cultures, there is growing evidence that they can occasionally cause urinary tract infections that are clinically significant, especially when predisposing host factors are present¹².

Darbro et al. reported a case of urinary tract infection caused by *Lactobacillus delbrueckii* in which repeated urine cultures consistently yielded the organism in association with pyuria and urinary symptoms. The patient showed clinical features compatible with true urinary tract infection rather than contamination, emphasizing that *Lactobacillus* species, although generally regarded as commensal organisms, can occasionally act as opportunistic uropathogens when isolated repeatedly in an appropriate clinical setting²⁰. Similar to their findings, our patient had persistent urinary symptoms, pyuria, repeated isolation of *Lactobacillus* species from urine cultures, and poor response to empirical cefixime therapy, supporting the diagnosis of a true infection rather than colonization or contamination.

Rossi et al. reviewed published infections caused by *Lactobacillus* species and concluded that, although these bacteria are generally regarded as non-pathogenic commensals, they can cause clinically significant infections, including urinary tract infections, particularly in patients with predisposing conditions such as diabetes mellitus, immunosuppression, urinary tract abnormalities, pregnancy, and previous antimicrobial exposure²¹. Pregnancy was the principal predisposing factor in our patient, supporting the hypothesis that physiological alterations during gestation may facilitate opportunistic infection by *Lactobacillus* species.

Malézieux-Picard et al. reported a series of urinary tract infections caused by *Lactobacillus delbrueckii* subspecies in elderly and immunocompromised patients. The authors highlighted that repeated isolation of *Lactobacillus* species from symptomatic individuals should not be dismissed as contamination but should instead raise suspicion of a true urinary tract infection. Consistent with these findings⁷, our patient demonstrated repeated pure growth of *Lactobacillus* species on urine culture in association with persistent urinary symptoms and pyuria, supporting its etiological role in the infection.

Bhatt et al. reported a neonatal urinary tract infection caused by *Lactobacillus* species and highlighted that these organisms can occasionally behave as opportunistic pathogens outside their normal ecological niche. Their findings support the growing recognition that *Lactobacillus* species are capable of causing clinically relevant infections under specific host conditions²². Pregnancy-associated immunological alterations may have contributed to the pathogenic potential of *Lactobacillus* species in this patient.

Lewis et al. demonstrated that disruption of the normal vaginal microbiota, altered host immunity, and local mucosal factors may facilitate migration of vaginal microorganisms into the urinary tract. Pregnancy is associated with physiological urinary stasis, ureteric dilatation, and altered immune responses, all of which predispose to urinary tract infections⁹. These mechanisms may explain the development of *Lactobacillus*-associated UTI in the present case.

Lactobacillus species exhibit intrinsic resistance to several antimicrobial agents commonly used for empirical treatment of urinary tract infections, including fluoroquinolones, aminoglycosides, metronidazole, and many cephalosporins⁶. Consequently, infections caused by these organisms may fail to respond to conventional empirical therapy directed against common uropathogens. In contrast, ampicillin and amoxicillin–clavulanate have demonstrated reliable *in vitro* activity against many *Lactobacillus* species isolated from symptomatic urinary tract infections, providing biological plausibility for the favourable clinical response observed following targeted therapy⁷. In the present case, the patient failed to respond to empirical cefixime therapy, which may be attributed to the intrinsic resistance of the infecting *Lactobacillus* species to cephalosporins. Following administration of amoxicillin–clavulanate, the patient's urinary symptoms resolved completely, and follow-up urine culture demonstrated microbiological clearance, further supporting the pathogenic role of the isolate and the importance of culture-guided antimicrobial therapy.

Regarding antimicrobial susceptibility, Darbro et al. demonstrated that successful management of *Lactobacillus* urinary tract infection depends on antimicrobial susceptibility testing because empirical therapy may be ineffective. In their case, the patient experienced persistent urinary symptoms despite three courses of ciprofloxacin, and susceptibility testing revealed high-level resistance to ciprofloxacin while showing susceptibility to ampicillin, erythromycin, vancomycin, and other agents. Clinical resolution occurred only after administration of clarithromycin, emphasizing the importance of culture-guided therapy²⁰. Similarly, our isolate exhibited resistance to cefixime, cefotaxime, and ampicillin but remained susceptible to amoxicillin-clavulanate, erythromycin, clindamycin, and vancomycin. The

patient's symptoms resolved following treatment with amoxicillin-clavulanate, further supporting the pathogenic role of the isolate and highlighting the importance of individualized antimicrobial selection based on susceptibility testing rather than empirical therapy alone

Kieffer et al. reported that urinary tract infections during pregnancy are associated with adverse obstetric outcomes, including preterm labour and preterm delivery. Infection-induced inflammatory responses may stimulate cervical ripening and uterine contractions³. In the present case, the patient presented with symptomatic UTI at 37 weeks and 6 days of gestation and subsequently progressed into labour, suggesting a possible association between infection and labour onset.

The American College of Obstetricians and Gynaecologists (ACOG) recommends that persistent urinary symptoms despite empirical therapy should prompt repeat urine culture and reassessment of the causative organism². Our case reinforces this recommendation, as repeated urine cultures were crucial in establishing *Lactobacillus* species as the causative pathogen and guiding appropriate antimicrobial therapy.

Overall, the present case contributes to the growing but still limited evidence supporting *Lactobacillus* species as a true uropathogen. The repeated isolation of the organism in pure culture, in association with significant pyuria, persistent urinary symptoms, failure of empirical therapy, and complete clinical and microbiological response to targeted antimicrobial treatment, strongly supports its etiological role in this infection. Clinicians and microbiologists should therefore exercise caution before dismissing *Lactobacillus* isolates as commensals or contaminants, particularly when they are repeatedly isolated from symptomatic patients with supportive clinical and laboratory findings. Early recognition of such uncommon pathogens may improve patient management and promote appropriate antimicrobial stewardship.

Conclusion

This report emphasizes the importance of correlating microbiological findings with the clinical presentation and avoiding the routine dismissal of *Lactobacillus* isolates in symptomatic pregnant patients. Repeat urine culture and appropriate antimicrobial susceptibility testing are essential when empirical treatment fails, as they facilitate accurate diagnosis and guide effective therapy. Increased awareness of uncommon urinary pathogens among clinicians and microbiologists may improve maternal outcomes, optimize antimicrobial stewardship, and contribute to the growing evidence that *Lactobacillus* species should be considered a potential cause of urinary tract infection in selected clinical settings.

Declarations

Ethical Statement: In compiling our case report exclusively from secondary data sources, including case summaries, case reports, and case sheets, we have maintained ethical integrity by prioritising patient

confidentiality and privacy rights. As such, ethical committee clearance is not applicable, but written informed consent was obtained from the patients for the publication of this case report.

Clinical trial: Not applicable

Conflict of Interests: The authors declare no conflict of interest.

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Author Contribution:

Study conception and design, Manuscript rewriting, editing: MNS

Manuscript writing, rewriting, editing, methodology and data curation: SSSC, GKM.YDM.

Analysis, Manuscript rewriting, editing: SRS, YDM.VSGS, CSE.

Manuscript rewriting, editing, and critical review: NSM and RSG ,DR.BSS

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