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Pediatric Vulvovaginitis Caused by Extended-Spectrum Beta-Lactamase-Producing *Escherichia coli*: A Case Report

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Abstract

Pediatric vulvovaginitis is one of the most common gynecological problems in prepubertal girls and is often attributed to nonspecific or fungal causes. However, recent evidence shows that multidrug-resistant aerobic enteric bacteria are emerging as significant pathogens, complicating diagnosis and limiting treatment options. Extended-spectrum β-lactamase (ESBL)–producing *Escherichia coli* is a particular concern because resistance to commonly prescribed antibiotics restricts effective therapy in children.

We report the case of a 10-month-old female who presented with abdominal distension, fever, and purulent greenish vaginal discharge accompanied by erythema and perineal irritation. Initial topical antifungal therapy proved ineffective. Laboratory investigations revealed leukocytosis and elevated inflammatory markers. Vaginal swab microscopy demonstrated abundant polymorphonuclear leukocytes and Gram-negative bacilli. Aerobic culture yielded lactose-fermenting colonies identified as *E. coli* through biochemical assays, MALDI-TOF mass spectrometry, and 16S rRNA sequencing. Antimicrobial susceptibility testing revealed resistance to third-generation cephalosporins, aminoglycosides, and aztreonam, while maintaining susceptibility to meropenem and ertapenem, confirming ESBL production. The patient responded well to intravenous meropenem combined with oral fluconazole and topical miconazole, showing marked clinical improvement within five days.

This case underscores the susceptibility of prepubertal girls to aerobic vaginitis, the need for accurate microbiological diagnosis, and the therapeutic challenges posed by ESBL-producing organisms. It also highlights broader health implications, emphasizing antimicrobial stewardship, infection prevention, and caregiver education.

Keywords: Pediatric Vulvovaginitis, *Escherichia coli*, ESBL, Antimicrobial Resistance, MALDI-TOF, 16S rRNA Sequencing

Introduction

Pediatric vulvovaginitis is among the most frequent gynecological complaints in prepubertal girls, constituting a significant proportion of pediatric consultations worldwide.[1–3] The condition manifests with vulvar erythema, discharge, irritation, and pruritus, symptoms that often raise concern for both parents and clinicians. The anatomical and physiological characteristics of the prepubertal genital tract[4], including thin and hypoestrogenic vaginal epithelium, proximity to the anus, lack of protective pubic hair, and underdeveloped labia majora, predispose this population to infections.[3,5,6] Behavioral and environmental factors, such as poor perineal hygiene, the use of diapers, and frequent antibiotic exposure, further increase susceptibility to vulvovaginal infections.[5–7]

The etiologies of pediatric vulvovaginitis are multifactorial, involving bacterial, fungal, and parasitic pathogens. Aerobic bacteria, such as *Escherichia coli*, have increasingly been identified as critical causative agents in cases of aerobic vaginitis and vulvovaginitis among children.[3] *E. coli* is a facultative anaerobic Gram-negative bacillus, a common commensal of the gastrointestinal tract, but capable of acting as an opportunistic pathogen.[6,8] Virulence factors, including adhesins, toxins, and biofilm-forming capabilities, enable colonization and infection of the urogenital tract.[9,10] Of clinical concern is the emergence of extended-spectrum beta-lactamase (ESBL)-producing *E. coli*, which confers resistance to penicillins, cephalosporins, and monobactams, leaving limited therapeutic options.[11]

Diagnostic challenges often arise when clinical features overlap with those of other conditions, such as candidiasis, urinary tract infections, or dermatological disorders, leading to misdiagnosis and inappropriate treatment.[12] Such cases can prolong morbidity, promote antimicrobial resistance, and increase the healthcare burden. Therefore, comprehensive microbiological evaluation, including culture, biochemical assays, MALDI-TOF mass spectrometry, and molecular methods such as 16S rRNA sequencing, remains the cornerstone for accurately identifying causative pathogens.[13,14]

In regions with limited resources, such as parts of Southeast Asia, the burden of pediatric vulvovaginitis remains underestimated, and the increasing prevalence of multidrug-resistant organisms heightens clinical concern. Recent studies emphasize the importance of antimicrobial stewardship, infection prevention measures, and clinician awareness in optimizing outcomes.[15] Case reports highlighting resistant pathogens in uncommon pediatric presentations are critical for broadening clinical insights and guiding evidence-based management strategies. The present case describes a 10-month-old female diagnosed with vulvovaginitis caused by ESBL-producing *Escherichia coli*, confirmed through microbiological and molecular diagnostics, thereby underscoring the need for vigilant surveillance and targeted interventions.

Case Report

A 10-month-old female, born on July 5, 2023, was admitted to Dr. Wahidin Sudirohusodo Hospital on May 18, 2024, with a primary complaint of abdominal distension noted in the last six months before admission. One week before admission, the patient developed intermittent fever responsive to antipyretics, without associated seizures, cough, or dyspnea. Two days before admission, a greenish vaginal discharge was observed, accompanied by pruritus and erythema in

the perineal region. The parents reported that the child frequently scratched the genital area and was irritable, especially during urination and defecation.

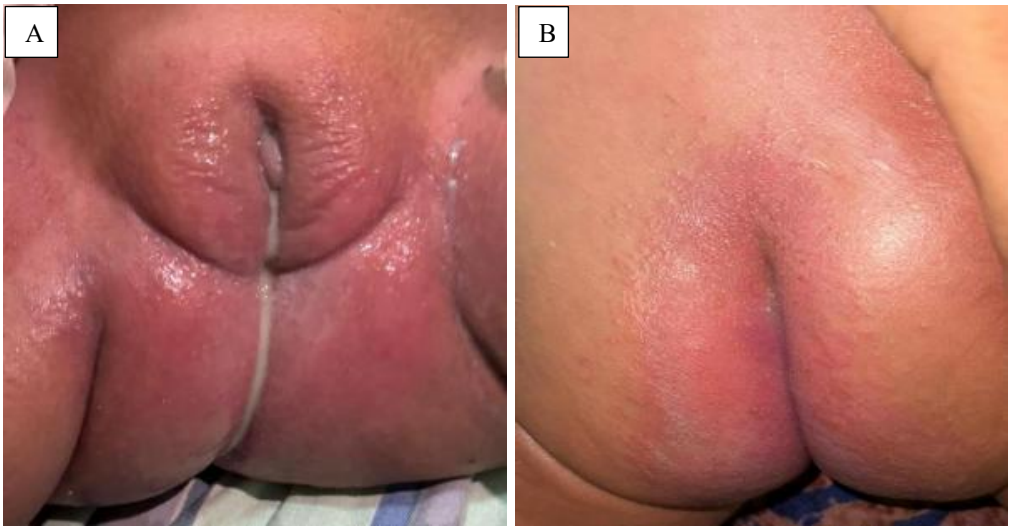


Figure 1. A. Location: Vaginal Region: Erythematous Macules, Greenish-white Discharge, Swollen Labia Majora
B. Location: Gluteal Region: Erythematous Macules, Erythematous Plaques, and Maceration

On May 21, 2024, erythematous lesions extended to the thighs and buttocks. Initial management with topical Myco-Z ointment provided no improvement. The child had a history of hospitalization in April 2024 for pneumonia, an intra-abdominal mass, gastrointestinal bleeding, and iron deficiency anemia, treated with intravenous ampicillin, gentamicin, and dexamethasone. There was no family history of similar complaints or malignancy.

On admission, vital signs included a temperature of 38°C, a pulse of 120 beats per minute, a respiratory rate of 28 breaths per minute, and oxygen saturation of 88% on room air, which improved to 99%. Physical examination revealed a mobile, firm mass in the left lumbar region measuring 5×3×1 cm, with no tenderness or organomegaly. Local examination showed erythematous macules, white-greenish discharge, and swelling of the labia majora, with satellite lesions and maceration noted in the gluteal region.

Laboratory investigations demonstrated leukocytosis (WBC 28,160/µL) and anemia (Hb 8.7–9.9 g/dL). Urinalysis revealed proteinuria (+1), hematuria (+3), and pyuria (+3). Imaging studies included chest radiography showing bronchopneumonia, abdominal plain film suggesting a mesenteric cyst, and an abdominal MSCT scan demonstrating right renal hypoplasia with hypoperfusion, uteromegaly with hydrocolpos, left hydronephrosis grade II, cystitis, and spina bifida. Procalcitonin was elevated (0.16 ng/mL), and CRP was markedly increased (13.9 mg/L).

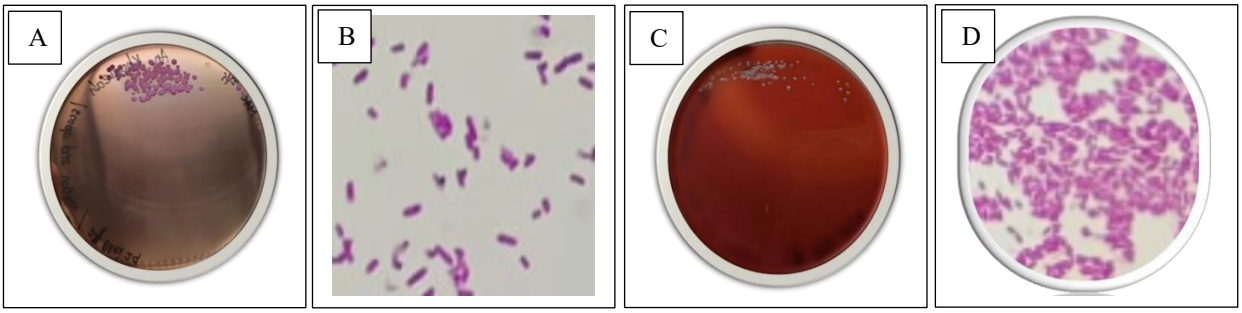
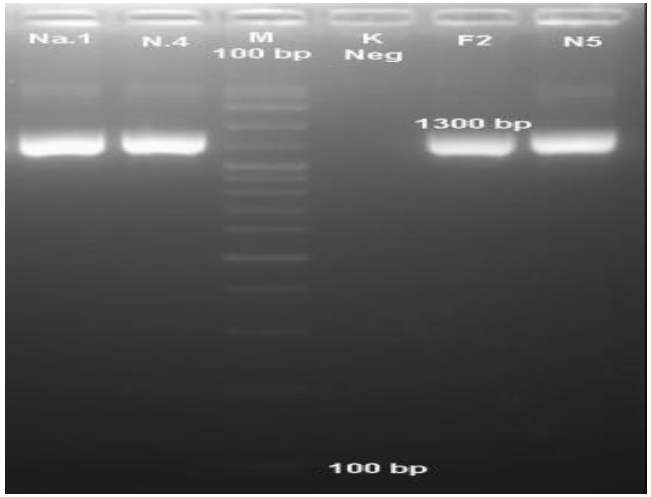
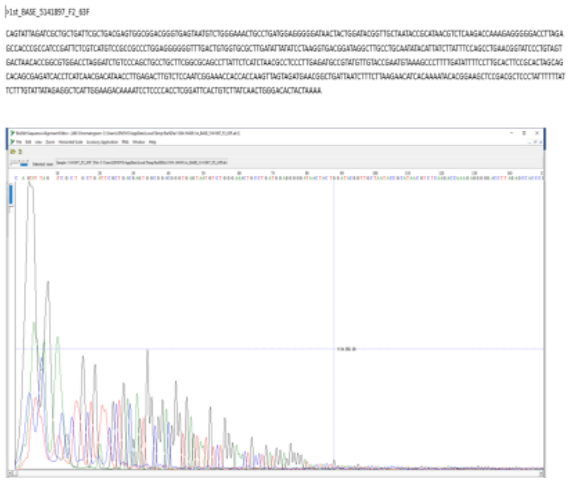


Figure 2. A. Colonies on MacConkey Agar. Colony Characteristics: Round Shape, Flat Edges, Smooth Surface, Convex Elevation, Cloudy Clarity, Light Pink Color, Lactose Fermenter. B and D Indirect Gram from Colony, C. Colonies on Blood Agar: Colony Characteristics: Round Shape, Flat Edges, Smooth Surface, Convex Elevation, Cloudy Clarity, Gray Color, and Gamma Hemolysis.

Assessment**,** taken together, the clinical findings (purulent greenish vaginal discharge, perineal and gluteal erythema with satellite lesions and maceration), laboratory results (leukocytosis, elevated procalcitonin and CRP, and urinalysis showing pyuria and hematuria), microbiological identification (abundant polymorphonuclear leukocytes and Gram-negative bacilli on direct smear, with culture, MALDI-TOF MS, and 16S rRNA sequencing confirming ESBL-producing *Escherichia coli*), and radiological findings (bronchopneumonia, a mesenteric cyst, right renal hypoplasia with hypoperfusion, left grade II hydronephrosis, cystitis, uteromegaly with hydrocolpos, and spina bifida) led the attending pediatrician to establish a working diagnosis of pediatric vulvovaginitis caused by ESBL-producing *Escherichia coli* (aerobic vaginitis) with a regressing superficial candidal dermatitis component, complicated by community-acquired bronchopneumonia, iron-deficiency anemia, an intra-abdominal mass suspected to represent a mesenteric cyst, and underlying congenital urogenital anomalies (right renal hypoplasia, left hydronephrosis grade II, cystitis, and uteromegaly with hydrocolpos) together with spina bifida. The treatment plan that follows was established on the premise of this comprehensive clinical and microbiological evaluation.

On May 27, 2024, a vaginal swab specimen was collected under aseptic conditions using Amies transport medium. Microscopic examination revealed polymorphonuclear leukocytes (3+/LPK) and Gram-negative bacilli. Aerobic culture demonstrated lactose-fermenting colonies on MacConkey agar. Identification using VITEK® and MALDI-TOF MS confirmed *Escherichia coli* with high probability (95–99.9%). Molecular confirmation using 16S rRNA PCR and sequencing further validated *E. coli* identity. Antimicrobial susceptibility testing indicated resistance to ceftriaxone, cefotaxime, ceftazidime, cefepime, aztreonam, and gentamicin, while susceptibility to meropenem and ertapenem was preserved. Extended-spectrum beta-lactamase (ESBL) production was confirmed.





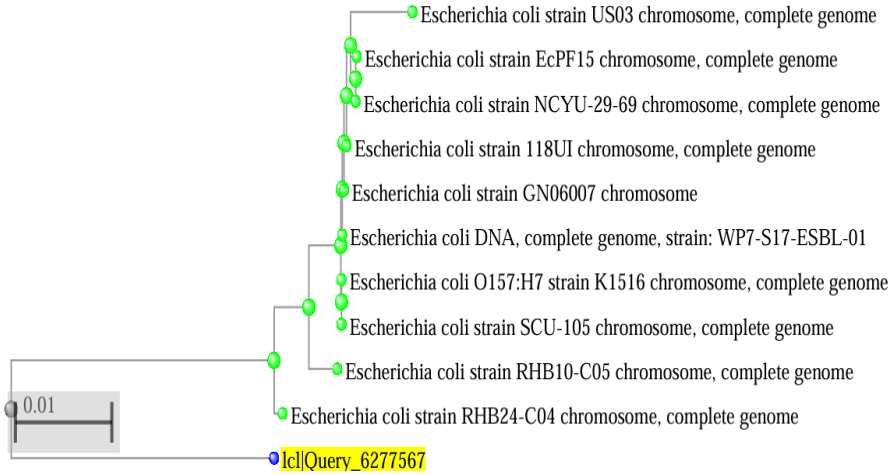


Figure 3. A. PCR Results of 16s RNA of *E. coli* Bacteria, B and C Sequencing results of Lactose Fermented Colonies

The patient received ceftriaxone intravenously (18–24 May 2024), followed by meropenem intravenously (25–29 May 2024). Concurrently, fluconazole (42 mg/day orally) and topical miconazole were administered. Which had been empirically initiated prior to the microbiological results, was maintained due to the clinically suggestive nature of the perineal and gluteal lesions (satellite papules and maceration superimposed on the erythematous plaques) suggesting a potential candidal component. Clinical improvement was noted by May 29, 2024, with a reduction in discharge, resolution of erythema, and improved patient comfort. The patient was discharged with oral fluconazole and topical miconazole, with follow-up cultures showing no fungal growth.

This case illustrates pediatric vulvovaginitis caused by ESBL-producing *Escherichia coli*, with diagnostic confirmation through microbiological culture, biochemical testing, MALDI-TOF MS, and molecular sequencing, highlighting the complexity of management in resistant pediatric infections.

Discussion

The present case underscores the intersection of host, behavioral, and iatrogenic factors that predispose prepubertal children to inflammatory vulvovaginitis driven by aerobic enteric organisms. Hypoestrogenic vaginal epithelium, the short anogenital distance, the absence of protective pubic hair, frequent diaper use, maceration, and alkaline shifts in the local milieu collectively weaken colonization resistance and facilitate the translocation of fecal flora to the vulva and distal vagina.[3,5,6] In this biological context, the recovery of an ESBL-producing *Escherichia coli* from a carefully collected vaginal swab, accompanied by purulent greenish discharge and abundant polymorphonuclear leukocytes, is pathognomonic of an inflammatory aerobic process rather than mere colonization and is concordant with contemporary descriptions of aerobic vaginitis in which Lactobacillus dominance is replaced by aerobic commensals/pathogens with robust neutrophilic inflammation.[7,10,16]

The layered laboratory approach strengthens diagnostic validity in this child. The specimen was obtained after saline cleansing and transported in Amies medium. The Gram stain revealed a heavy presence of PMNs and Gram-negative bacilli. Culture yielded lactose-fermenting colonies. Species-level identification was concordant across biochemical profiling, MALDI-TOF MS, and 16S rRNA sequencing. The clinical trajectory improved while receiving a carbapenem active in vitro. These steps align with evidence that rapid organism identification using MALDI-TOF can expedite antimicrobial optimization and reduce inappropriate exposure in pediatric infections, particularly when coupled with stewardship review.[13,14] Importantly, Over-attribution to Candida in diapered infants is common; in this case, prior empiric antifungal use without compatible

culture findings, along with a neutrophilic smear, favors a bacterial primary process with a possible superficial candidal dermatitis component that had begun to regress.[15]

The coexisting radiologic impressions of cystitis, left hydronephrosis, and uteromegaly with hydrocolpos widen the pathophysiological lens. Hydrometrocolpos/hydrocolpos in infants can impair the clearance of secretions, favor stasis, and secondarily alter the microbial ecosystem, creating a permissive niche for ascending infection or persistent vaginal inflammation.[7,12,17] While causality cannot be assigned from a single observation, the temporal clustering of urinary and genital findings, together with the organism recovered, supports a contiguous pathway hypothesis. At a minimum, longitudinal follow-up is warranted to ensure the resolution of hydrocolpos and to exclude persistent outflow obstruction or urogenital anomalies that might predispose to recurrence.

Therapeutically, the ESBL phenotype explains the non-utility of third-generation cephalosporins.[18,19] Contemporary guidance recommends a carbapenem as first-line therapy for serious ESBL-Enterobacterales infections when oral options are limited, with de-escalation once susceptibilities and clinical stability permit.[8–10] In localized pediatric vulvovaginitis without systemic features, targeted narrower agents would be preferable from a stewardship perspective; however, child-appropriate oral agents with reliable activity against ESBL-*E. coli* remain few, and this patient presented with overlapping signals (fever history, elevated inflammatory markers, radiologic cystitis) that justified short parenteral therapy. Stewardship-aligned actions include daily reassessment for the shortest effective duration, discontinuation of unnecessary antifungals, and consideration of non-antibiotic adjuncts (such as barrier repair, meticulous hygiene, and proper diapering practices) to reduce recurrence.

From a public health vantage point, the recovery of ESBL-*E. coli* in a 10-month-old aligns with surveillance signals indicating that third-generation cephalosporin resistance in *E. coli* remains high globally and regionally, with implications for empirical pediatric care.[19,20] Inpatient settings should implement contact precautions proportionate to risk and reinforce hand hygiene to prevent nosocomial transmission. At discharge, caregiver education emphasizing front-to-back wiping, frequent diaper changes, gentle cleansing (avoiding irritant wipes), and avoidance of unnecessary antibiotics is a low-cost strategy likely to reduce recurrence. Finally, future research should characterize virulence factors and resistomes in pediatric genital *E. coli* isolates and prospectively evaluate short, targeted regimens—including potential non-antibiotic topical options—in the context of aerobic vaginitis.

Conclusion

This case demonstrates the clinical relevance of extended-spectrum β-lactamase (ESBL)–producing *Escherichia coli* as a causative pathogen in pediatric vulvovaginitis. The infant's presentation, marked by purulent vaginal discharge, perineal erythema, and local irritation, illustrates the susceptibility of prepubertal girls to aerobic enteric infections due to anatomical, physiological, and behavioral predispositions. Rigorous diagnostic confirmation, utilizing Gram stain, culture, biochemical testing, MALDI-TOF mass spectrometry, and 16S rRNA sequencing, substantiated the role of *E. coli,* thereby minimizing the risk of misclassification as candidiasis or nonspecific dermatitis. The presence of ESBL resistance limited standard empirical options and necessitated the use of carbapenem therapy, which resulted in rapid clinical improvement.

This report reinforces three critical messages for clinical practice. First, pediatric vulvovaginitis should not be reflexively attributed to fungal infection, particularly in infants with prior antibiotic exposure and inflammatory discharge. Second, microbiological precision, achieved through a layered diagnostic strategy, is essential for directing effective therapy and preventing inappropriate antimicrobial use. Third, the identification of ESBL-producing *E. coli* in a young child highlights the growing challenge of multidrug-resistant organisms in both community and hospital settings, underscoring the need for antimicrobial stewardship, meticulous infection-prevention measures, and caregiver education to reduce recurrence and transmission.

In summary, pediatric vulvovaginitis caused by multidrug-resistant enteric bacteria requires a high index of suspicion, robust microbiological confirmation, and individualized treatment guided by susceptibility data. By reporting this case, we aim to raise awareness of ESBL-producing pathogens in pediatric gynecologic infections and to emphasize the imperative of integrating microbiological evidence into clinical decision-making to optimize outcomes.

Conflict of Interest

The authors declare that there is no conflict of interest regarding the publication of this case report. The research, writing, and submission of this manuscript were conducted independently and without any financial or personal relationships that could inappropriately influence or bias the content of the article.

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