

Purple Urine Bag Syndrome as a Clinical Indicator of Bacteraemic Urosepsis Caused by Extensively Drug-resistant *Escherichia coli*: A Case Report

MITTAL R SINDHAV¹, RONAK SHAH², PARTH SHAH³

ABSTRACT

Purple Urine Bag Syndrome (PUBS) is an uncommon but clinically informative finding in catheterised patients, arising from bacterial enzymatic conversion of urinary indoxyl sulphate to indigo and indirubin within alkaline drainage tubing. Although usually benign in isolation, PUBS can signal severe underlying infection, constipation, alkaline urinary pH, and renal impairment. We report a 77-year-old man with diabetes mellitus and Chronic Kidney Disease (CKD stage 4) who presented with fever, dysuria, and altered sensorium. Following urinary catheterisation, purple-violet discolouration of the drainage tubing developed within six to eight hours, confirming PUBS on bedside assessment; the finding prompted urgent urine microscopy. Urine and blood cultures grew Extensively Drug-Resistant (XDR) *Escherichia coli* with an identical antibiogram, resistant to carbapenems, colistin, and the empirically administered antibiotic, with susceptibility retained only to fosfomycin and nitrofurantoin. He was treated with oral fosfomycin trometamol under nephrology guidance and discharged on day five with clinical resolution and improved renal function. Paired photographs documented the discolouration and its complete resolution within 24 hours. To our knowledge, this is the first photodocumented report of PUBS associated with bacteraemic XDR *E. coli* with concurrent carbapenem and colistin resistance from an emergency department in a low- and middle-income country setting. The case illustrates the value of PUBS as a bedside alarm sign that can accelerate recognition of severe multidrug-resistant sepsis, and underscores the importance of pre-antibiotic culture collection and prompt antimicrobial stewardship in catheterised elderly patients with systemic illness.

Keywords: Anti-bacterial agents, Drug resistance, Multiple, Bacterial, Sepsis, Urinary catheterisation, Urinary tract infections

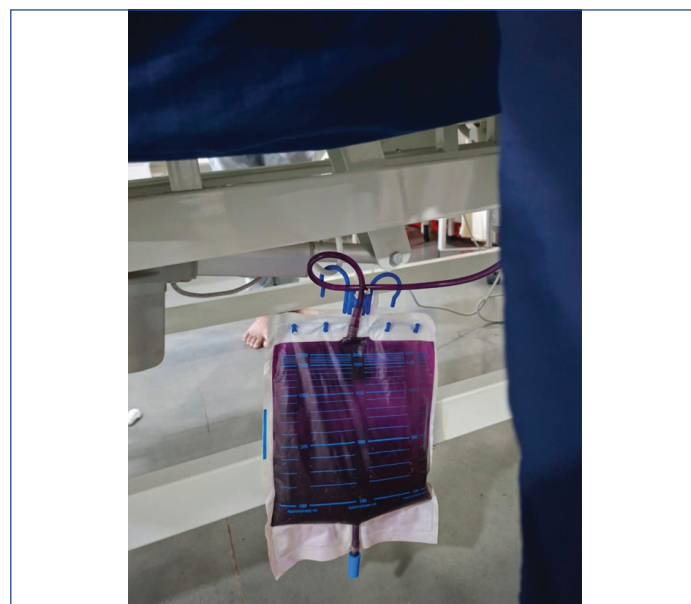
CASE REPORT

A 77-year-old man presented to the emergency department with a three- to four-day history of high-grade fever, dysuria, and progressive alteration in sensorium, brought by family members in a state of reduced responsiveness. He had type 2 diabetes mellitus with suboptimal glycaemic control (HbA1c 8.1%), hypertension, dyslipidaemia, and CKD stage 4, with no history of tobacco or alcohol use, prior urological instrumentation, malignancy, immunosuppression, or antibiotic exposure in the preceding three months. Family history was non-contributory.

On examination, he was acutely unwell, with a pulse rate of 86 beats/min, blood pressure of 108/60 mmHg, respiratory rate of 26 breaths/min, and an estimated Glasgow Coma Scale (GCS) score of 12 (E3V4M5). His quick Sequential Organ Failure Assessment (qSOFA) score was 2, based on altered mental status (GCS <15) and a respiratory rate ≥ 22 breaths/min, with no points assigned for systolic blood pressure ≤ 100 mmHg. Systemic examination revealed mild suprapubic tenderness without peritonism; cardiovascular and respiratory examination were unremarkable. Although he was afebrile at initial nursing contact, this did not exclude sepsis, since the febrile response is recognised to be blunted in elderly patients [1].

Laboratory evaluation at presentation showed elevated inflammatory markers (CRP 119.72 mg/L) and acute-on-chronic renal impairment (creatinine 2.9 mg/dL; eGFR 21.6 mL/min/1.73m²); together with the clinical presentation, these findings supported a provisional diagnosis of urosepsis with altered sensorium. A urinary catheter was inserted for output monitoring; the initially drained urine was turbid amber-yellow. Approximately six to eight hours later, the catheter tubing and collection bag developed deep purple-violet discolouration [Table/Fig-1], while urine sampled directly from the catheter into a specimen container remained yellow-amber, confirming PUBS.

Alkaline urinary pH (7.5-8.0) and a three-day history of constipation supported the diagnosis [Table/Fig-2]. Differential diagnoses of discoloured urine - gross haematuria, rifampicin or methylene blue exposure, beeturia, and porphyria - were excluded on history and on the yellow colour of urine sampled directly from the catheter. Urine and two sets of blood cultures were obtained before antibiotics were given; empirical intravenous cefoperazone-sulbactam and fluid resuscitation were commenced.



[Table/Fig-1]: Photodocumentation of Purple Urine Bag Syndrome (PUBS) in the patient. Deep purple-violet discolouration of urinary catheter drainage tubing and collection bag, observed on Day 1 approximately six to eight hours following catheterisation. The discolouration is uniform throughout the plastic collection system.

Parameter	Admission (17 May)	Day 2 (19 May)	Discharge (22 May)
Haemoglobin (g/dL)	7.1	7.0	9.0*
Total WBC count (/cumm)	10,290	10,530	10,300
Platelets (x10 ⁹ /L)	215	209	236
Creatinine (mg/dL)	2.9	2.9	2.2†
eGFR (mL/min/1.73 m ²)	21.6	21.6	30.1†
Blood urea (mg/dL)	80	75	70
CRP (mg/L)	119.72	149.84	74.66
Serum albumin (g/dL)	2.3	-	-
Urinary pH	7.5-8.0 (alkaline)	-	Normal

[Table/Fig-2]: Serial haematological and biochemical parameters.
*Post-transfusion value on Day 4. †Discharge creatinine/eGFR reflect partial resolution of an acute-on-chronic kidney injury component superimposed on established CKD.

Both urine and blood cultures grew XDR *E. coli* with an identical antibiogram, susceptible only to fosfomycin and nitrofurantoin [Table/Fig-3], establishing a final diagnosis of PUBS secondary to bacteraemic urosepsis. Cefoperazone-sulbactam was discontinued, and, following nephrology review, oral fosfomycin trometamol 3 g every 48 hours was commenced for three doses. The catheter bag was changed, lactulose was administered for constipation, and intravenous hydration was increased; the discolouration resolved completely within 18-22 hours [Table/Fig-4]. The patient received one unit of packed red cells for a haemoglobin of 7.0 g/dL [Table/Fig-2] and was managed in the intensive care unit with nephrology co-management. A structured summary of the clinical timeline, laboratory trend, and antimicrobial susceptibility profile is presented in [Table/Fig-3-5].

Antimicrobial	MIC (µg/mL)	Result	Clinical note
Cefoperazone-sulbactam (empirical)	≥32	R	Empirical agent; discontinued Day 2
Piperacillin-tazobactam	≥64	R	
Ceftriaxone	≥64	R	
Cefepime	≥8	R	
Meropenem	≥4	R	Carbapenem-resistant
Ciprofloxacin	≥4	R	
Colistin	≥4	R	High-level polymyxin resistance
Fosfomycin	≤4	S	Definitive agent - 3 g PO q48h x3
Nitrofurantoin	32	S	Not used (bacteraemia present)

[Table/Fig-3]: Antimicrobial susceptibility profile of the XDR *E. coli* isolate from concurrent urine and blood cultures (CLSI M100, 2024).
R=resistant; S=susceptible. Full 14-agent panel available on request; agents omitted here for length were also resistant except as shown



[Table/Fig-4]: Complete resolution to clear amber-yellow urine on Day 2, following catheter bag change, oral laxative administration, intravenous hydration, and commencement of antimicrobial therapy. Written informed consent for clinical photography was obtained from the patient's family members. All photographs have been cropped to exclude background detail prior to submission.

Day	Clinical events	Key investigations	Management
Day 0 (17 May)	Presentation to ED: fever, dysuria, altered sensorium. BP 108/60 mmHg, qSOFA 2/3. Urinary catheter inserted. Urine and 2 sets blood cultures sent before antibiotics.	Hb 7.1 g/dL. Creatinine 2.9 mg/dL (eGFR 21.6 mL/min/1.73m ²). CRP 119.72 mg/L. Urine: alkaline pH 7.5-8.0, pyuria, haematuria.	i.v. fluid resuscitation. Empirical cefoperazone-sulbactam 3g i.v. stat, then 1.5 g BD. Catheter output monitoring.
Day 1 (18 May)	Purple-violet discolouration of catheter tubing and collection bag observed; PUBS confirmed (urine yellow on direct sampling from the catheter). Constipation confirmed (nil stool x 3 days).	Ongoing pyrexia. Haemodynamic parameters monitored.	Catheter bag changed. Oral lactulose 30 mL given. i.v. hydration increased. Urine sent for urgent microscopy. PUBS documented with photograph.
Day 2 (19 May)	PUBS fully resolved - clear yellow urine. Blood and urine cultures reported: XDR <i>E. coli</i> (>100,000 CFU/mL in urine). Empirical antibiotic discontinued. Transferred to MICU.	CRP 149.84 mg/L (peak). WBC 10,530/cumm. Creatinine 2.9 mg/dL.	Cefoperazone-sulbactam stopped (MIC ≥32, resistant). Oral fosfomycin trometamol 3 g every 48 h commenced (nephrology-guided). Nephrology review obtained.
Day 4 (21 May)	Clinical improvement. Sensorium normalised. Afebrile. Hb 7.0 g/dL - transfusion threshold met.	Post-transfusion Hb 9.0 g/dL. Vitals stable.	1 unit packed red cells transfused (Group A+ve, compatible). Medications dose-adjusted for CKD.
Day 5 (22 May)	Discharged in stable condition. Full clinical resolution. Urine clear throughout preceding 48 hours.	CRP 74.66 mg/L (declining). WBC 10,300/cumm (normal). Creatinine 2.2 mg/dL (eGFR 30.1 mL/min/1.73 m ²).	Discharge on usual medications plus completing fosfomycin course. XDR profile flagged in discharge summary. Nephrology follow-up in 2 weeks.

[Table/Fig-5]: Structured clinical timeline documenting PUBS observation and resolution, microbiological reporting, antibiotic decision-making, and clinical course across the five-day admission. eGFR calculated using the CKD-EPI 2021 race-free formula.

Clinical improvement was progressive, with resolution of fever, normalisation of sensorium, declining inflammatory markers, and improvement in renal function (creatinine 2.9 to 2.2 mg/dL; eGFR 21.6 to 30.1 mL/min/1.73 m²). He was discharged on day five on his usual medications, to complete the fosfomycin course. At the two-week nephrology follow-up, the patient remained clinically stable and was doing well. Renal function was stable, with serum creatinine approximately 0.8 mg/dL. No new renal or clinical concerns were noted at follow-up.

DISCUSSION

PUBS is a visually striking but under-recognised finding in catheterised patients, first described by Barlow GB and Dickson JA in 1978 [2]. It results from bacterial enzymatic cleavage of urinary indoxyl sulphate - a hepatic conjugate of the colonic tryptophan metabolite indole - by sulphatase- or phosphatase-producing organisms such as *Providencia stuartii*, *Klebsiella pneumoniae*, and *Escherichia coli* [3]. In an alkaline environment, the liberated indoxyl oxidises spontaneously within plastic drainage tubing to indigo and indirubin, producing the characteristic purple discolouration. PUBS requires the simultaneous presence of catheterisation, alkaline urine, an enzymatically active organism, constipation, and factors that concentrate urinary indoxyl sulphate, such as renal impairment and hypoalbuminaemia [4-7]. Although often regarded as a benign curiosity, its occurrence in an acutely unwell patient should prompt evaluation for coexisting severe infection.

In the present patient, all recognised risk factors were satisfied except female sex, which predominates in published series largely because of demographic patterns in long-term institutional catheter care rather than a biological predisposition [4,5]. The distinguishing feature of this case is the causative organism: an XDR *E. coli* isolate resistant to carbapenems, ciprofloxacin, third- and fourth-generation cephalosporins, and colistin (Minimum

Inhibitory Concentration [MIC] ≥ 4 $\mu\text{g/mL}$), meeting the Magiorakos AP et al., criteria for XDR [8]. While *E. coli* is an established PUBS-associated organism, a systematic PubMed and MEDLINE search using the terms 'PUBS', 'XDR', 'carbapenem-resistant', 'E. coli', and 'bacteraemic urosepsis' did not identify a prior report of PUBS caused by bacteraemic, carbapenem- and colistin-resistant *E. coli*, particularly from a low- or middle-income country emergency department. This resistance pattern reflects the broader landscape of Enterobacteriaceae resistance reported across Indian and other low- and middle-income country settings, where carbapenem and polymyxin resistance among urosepsis isolates has risen substantially [9-11]. Growth of *E. coli* with an identical antibiogram from concurrent blood and urine cultures confirmed bacteraemia, escalating the case from simple urosepsis to bacteraemic urosepsis, with corresponding implications for antibiotic selection.

Empirical cefoperazone-sulbactam, selected per institutional guidelines, was subsequently confirmed resistant (MIC ≥ 32 $\mu\text{g/mL}$), representing documented antibiotic failure in a bacteraemic patient and reinforcing the principle that culture must be obtained before empirical therapy and acted upon within 24-48 hours [12]. Definitive therapy required an agent active against the XDR isolate; fosfomycin was the only oral option available locally [13]. In severe CKD (eGFR 21.6 mL/min/1.73 m²), renal clearance of fosfomycin is reduced, prolonging its elimination half-life and increasing the risk of systemic accumulation with standard dosing intervals; the dosing interval was therefore extended to every 48 hours rather than shortened. Repeat dosing beyond a single 3 g dose was nonetheless still required, because complicated bacteraemic infection needs sustained urinary drug exposure that a single dose does not provide, even allowing for reduced renal clearance. Published data report peak urinary fosfomycin concentrations of approximately 1053-4415 $\mu\text{g/mL}$ after a single 3 g dose, values that comfortably exceed the isolate's MIC of ≤ 4 $\mu\text{g/mL}$ even at the lower end of the range expected with reduced renal function [14]. Nitrofurantoin, although susceptible in vitro, was withheld because of inadequate systemic tissue concentration in bacteraemia and its contraindication below an eGFR of 30 mL/min [15]. Intravenous fosfomycin, pharmacokinetically preferable for bacteraemic infection, was unavailable at this institution, and its absence is acknowledged as a resource limitation of this setting [16].

This case demonstrates that PUBS functioned as a bedside diagnostic prompt that accelerated recognition of bacteraemic XDR infection; without this visible sign, the severity of the presentation might have been appreciated later. Emergency physicians managing catheterised elderly patients should treat new purple discolouration of catheter tubing as an indication for bedside confirmation by sampling urine directly from the catheter into a clear container, urinary pH testing, and urgent culture, together with blood culture collection where systemic illness coexists. Limitations of this report include the absence of molecular characterisation of the resistance mechanisms and the unavailability of intravenous fosfomycin; these gaps reflect constraints common to case reporting from resource-

limited emergency departments and do not diminish the educational value of the photodocumented findings.

CONCLUSION(S)

This case documents, to the best of the authors' knowledge, the first photographically recorded instance of PUBS associated with bacteraemic, carbapenem- and colistin-resistant *E. coli* managed in a tertiary emergency department in India. PUBS served as an early bedside signal of severe infection that prompted urgent evaluation; pre-antibiotic cultures then guided modification of an ineffective empirical antibiotic and initiation of oral fosfomycin under nephrology guidance, contributing to full clinical recovery. Emergency physicians should recognise new catheter-bag discolouration as a clinically actionable sign warranting systematic evaluation, including blood culture collection, in catheterised elderly patients with systemic illness.

Consent for Publication Written informed consent for publication of this case report and the accompanying clinical photographs was obtained from the patient's family members.

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PARTICULARS OF CONTRIBUTORS:

1. Assistant Professor, Department of Emergency Medicine, Smt. B. K. Shah Medical Institute and Research Centre, Sumandeep Vidyapeeth Deemed to be University, Vadodara, Gujarat, India.
2. Associate Professor, Department of Critical Care Medicine, Smt. B. K. Shah Medical Institute and Research Centre, Sumandeep Vidyapeeth Deemed to be University, Vadodara, Gujarat, India.
3. Assistant Professor, Department of Anaesthesiology, Ananya College of Medicine and Research, Vadodara, Gujarat, India.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:

Dr. Mittal R Sindhav,
1388, Akashganga, Behind Akashvani Radio Station, Vadodara-390002,
Gujarat, India.
E-mail: mittalmeghraj7@gmail.com

PLAGIARISM CHECKING METHODS:

- Plagiarism X-checker: Jul 10, 2026
- Manual Googling: Aug 29, 2026
- iThenticate Software: Aug 31, 2026 (2%)

ETYMOLOGY: Author Origin

EMENDATIONS: 6

AUTHOR DECLARATION:

- Financial or Other Competing Interests: None
- Was informed consent obtained from the subjects involved in the study? Yes
- For any images presented appropriate consent has been obtained from the subjects. Yes

Date of Submission: Jun 26, 2026

Date of Peer Review: Aug 01, 2026

Date of Acceptance: Sep 02, 2026

Date of Publishing: Nov 01, 2026