

CASE REPORT

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Purple urine bag syndrome in benign prostatic hyperplasia patient

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Abstract

Background: Purple color of urine is alarming and may indicate a serious underlying pathology. Purple discoloration of the urinary catheter bag is uncommon and is caused by urinary tract infection with certain organisms.

Case presentation: We present a case of this rare phenomenon in a benign prostate hyperplasia patient with indwelling catheter who also has diabetes mellitus and chronic renal disease. We also discussed proposed etiology and pathophysiology of purple urine bag syndrome.

Conclusion: Attempt to make the patient catheter free after stabilizing the patient is also the key point in its management. Although purple urine bag syndrome is not an aggressive pattern, it is an indicator for urosepsis.

Keywords: Purple urine bag syndrome, Benign prostate hyperplasia, Chronic renal disease, Diabetes mellitus

Background

Purple urine bag syndrome (PUBS) is the color of the urine bag rather than discoloration of urine following urinary catheterization for hours and days (Figs. 1 and 2). Risk factors are urinary tract infection due to various pathogens, including *Escherichia coli*, *Klebsiella*, *Morganella*, *Citrobacter*, *Proteus mirabilis*, and *Providencia (rettgeri and stuartii)* [1]. Predisposing factors are chronic constipation, use of laxatives, prolonged urinary catheterization, alkaline urine pH, tryptophan-rich foods, renal failure, and cognitive disorders. Dehydration, hypovolemia, elderliness, and female sex are other risk factors for PUBS. Tryptophan is converted to indigo (blue) or indirubin (red) by sulfatases or phosphatases produced by urinary bacteria. Because these pigments are insoluble, they are attached to a urine bag or catheter, causing purple urine bag syndrome. Mild or asymptomatic infection can also occur with bacteriuria [2] (Tables 1 and 2).

Case presentation

Our patient was a 65-year-aged male patient presented with chronic urinary retention due to BPH. He also had

deranged kidney function, uncontrolled diabetes, hypertension, and chronic constipation, but the patient was ambulatory. USG confirmed 80 g prostate with bilateral hydronephrosis. Initial serum creatinine was 5 mg/dl, serum potassium level was 5.5 mmol/l, electrocardiography findings was normal, serum prostatic specific antigen level was 2.3 ng/dl, blood sugar fasting was 180, and postprandial was 250 mg/dl on oral hypoglycemic. Catheterization was done as there were upper tract changes due to chronic retention. After endocrinology consultation, insulin was started and a cardiologist added two antihypertensive drugs while a nephrologist performed two sittings of hemodialysis. On subsequent visits, his blood sugar was well controlled, and blood pressure and kidney function were stable after 1 month of initial presentation. After 1 month, catheter change was done and the patient is planned for operation and advised to continue tamsulosin and dutasteride combination and laxative till enlistment for admission and operation which usually takes 1 month in our setup.

During his planning for operation, after 2 weeks, the patient presented with discoloration of urobag which was purple in color. Neither fever nor pain was noted in between the period. The patient did not take any other new medication or change his routine diet in the last 2 weeks. Hemoglobin was 9 g/dl, total leucocyte

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count was 8000/mm³ on urine examination, and it was alkaline and culture growth that show *Pseudomonas* and *E. coli*. This was sensitive to levofloxacin. Oral levofloxacin antibiotic was given for 14 days, and catheter change was done.

Subsequently, the patient got well, and after 1 month, the patient got operated for BPH and become catheter free. During 3 months of follow-up, he was doing well.

The patient was male and ambulatory with controlled DM, hypertension, and BPH with chronic retention which makes our case different and rare.

Clinical course

When ingested, tryptophan gets exposed to intestinal flora in patients with altered gut flora and these pigments

Table 1 Risk factors associated with PUBS

Female gender
Dementia (cognitive impairment)
Alkaline urine
Constipation
Chronic catheterization with indwelling catheters
High bacterial loads in the urine
Chronic kidney disease
Diabetes mellitus
Immunocompromised status
Multi organ failure



Table 2 Commonly associated bacteria with purple bag syndrome

Citrobacter species, including <i>C. freundii</i> , <i>C. diversus</i> , and <i>C. koseri</i>
Enterobacter species, including <i>E. aerogenes</i> , <i>E. agglomerans</i> , and <i>E. cloacae</i>
Enterococcus species, including <i>E. avium</i> and <i>E. faecalis</i>
<i>Escherichia coli</i>
<i>Klebsiella pneumonia</i>
<i>Morganella morganii</i>
Proteus species, including <i>P. mirabilis</i> and <i>P. vulgaris</i>
Providencia species, including <i>P. rettgeri</i> and <i>P. stuartii</i>
<i>Pseudomonas aeruginosa</i>
Methicillin-resistant <i>Staphylococcus aureus</i>

evolve. In constipated patients, delayed gut transport allows coliforms to deaminate tryptophan to indole, and after absorption from the gut, it conjugated in the liver to form indoxyl sulfate. Urinary tract infection causes bacteria to produce indoxyl sulfatase, depending on urine pH and oxygen concentration which degrades indoxyl sulfate to indigo (high pH) and/or indirubin (low oxygen). The color of the bag turns to purple, due to the mixture of indigo (blue violet) to indirubin (red violet) that gives the purple color.

Enlarge prostate leads to chronic retention that land up into back pressure changes to the kidney causing renal failure, and associated diabetes make kidney more susceptible to uremia and make the bowel movement sluggish. Usually, indoxyl sulfatase is normally excreted in urine, but in uremic patients, its excretion raised markedly due to impaired renal clearance. Due to toxins in uremic patients, there are chances to increase aerobacteria such as *E. coli* and decrease anaerobacter such as bifidobacteria in the intestine. Indole is chiefly produced by the aerobe which is the chief precursor of indoxyl sulfate. Raised serum indoxyl sulfate causes raised urinary indoxyl sulfate. Even after hemodialysis, protein-bound indoxyl sulfate is difficult to wash out. This explains the probable pathophysiology of our case (Additional file 1). The clinical course of PUBS is not aggressive if treated with proper antibiotic in time and if the catheter is changed.

Review of literature

The physical examination of the urine includes an evaluation of color, turbidity, specific gravity and osmolality, and pH. The color identification does not require any especial series of test; it is simply determined by the naked eye in a clear glass test tube. The normal color of urine is pale yellow due to the presence of the pigment urochrome [3]. Mostly, urine color variation is due to its

Table 3 Common causes of abnormal urine color

Color	Cause	
Colorless	Very dilute urine	
	Overhydration	
Cloudy/milky	Phosphaturia	
	Pyuria	
	Chyluria	
Red	Hematuria	
	Hemoglobinuria/myoglobinuria	
	Anthocyanin in beets and blackberries	
	Chronic lead and mercury poisoning	
	Phenolphthalein (in bowel evacuant)	
	Phenothiazines (e.g., Compazine)	
	Rifampin	
	Orange	Dehydration
		Phenazopyridine (Pyridium)
		Sulfasalazine (Azulfidine)
Yellow	Normal	
	Phenacetin	
	Riboflavin	
Green-blue	Biliverdin	
	Indicanuria (tryptophan indole metabolites)	
	Amitriptyline (Elavil)	
	Indigo carmine	
	Methylene blue	
	Phenols (e.g., IV cimetidine [Tagamet], IV promethazine [Phenergan])	
	Resorcinol	
	Triamterene (Dyrenium)	
	Brown	Urobilinogen
		Porphyria
Aloe, fava beans, and rhubarb		
Chloroquine and primaquine		
Furazolidone (Furoxone)		
Metronidazole (Flagyl)		
Nitrofurantoin (Furadantin)		
Brown-black		Alcaptonuria (homogentisic acid)
	Hemorrhage	
	Melanin	
	Tyrosinosis (hydroxyphenylpyruvic acid)	
	Cascara, senna (laxatives)	
	Methocarbamol (Robaxin)	
Methyldopa (Aldomet)		
Sorbitol		

From [4]

Table 4 Associated mechanisms of PUBS

Increased available tryptophan substrate for conversion
Alkaline urine facilitates indoxyl oxidation
Increased time for bacterial deamination in constipation and slow gut motility
Female short urethra prone to UTI
Catheter in situ bacteriuria
In UTI, more bacterial sulfatase/phosphatase availability
In uremic patients, impaired clearance of indoxyl sulfate

concentration. Many foods, medications, metabolic products, and infection may also produce abnormal urine color [4] (Table 3). Change of urine color is often noticed by the patient him/herself.

Historically, in 1812, purple discoloration is noticed by a physician in King George III urine who also has chronic severe constipation [5]. But in literature, purple urine bag syndrome was first reported in 1978 [6].

Dealer et al. study stats the incidence of purple bag syndrome mentioned about 9.8% in institutionalized patients mostly bedridden, cognitively impaired, and constipated females but it very rarely seen in daily practice and very few literature is present [7, 8].

Nakayama and Kanmatsuse found that serum levels of tryptophan and valine significantly fell down in PUBS patients. They also documented that amino acid absorption is altered by disturbances of colonic motility and intestinal overgrowth [9, 10].

Discoloration of urine is alarming and may be an indicator of early sign of serious underlying pathology. However, most of the cases are benign and occur as a result of ingestions of substances such as medications or food dye or post traumatic instrumentations (Table 4) [11].

Some untreated cases of PUBS also reported high morbidity and mortality especially in immunocompromised and multiple organ disease patients [12].

Discussion and conclusion

Purple discoloration of the urinary catheter bag is relatively uncommon. And it is caused by urinary tract infection with certain organisms in patients with certain features. Although PUBS is not an aggressive pattern, it is an indicator for urosepsis. The catheter-associated UTI is most commonly seen in females. So PUBS is more common in female patients [1].

To our knowledge, it is the first case in a male patient with chronic urinary retention for BPH and who has PUBS.

Attempt to make the patient catheter free after stabilizing the patient is also the key point in its management.

Additional file

Additional file1: Metabolic pathway and proposed pathophysiological mechanism (DOC 39 kb)

Abbreviation

BPH: Benign prostate hyperplasia; PUBS: Purple urine bag syndrome; UTI: Urinary tract infection

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Availability of data and materials

The data and materials were all included in the manuscript.

Authors' contributions

AK is responsible for the manuscript. AK and CLB performed the acquisition of the data and interpretation of the data and drafted and revised the manuscript. SD helped to draft the manuscript. BCM and TKM participated in the design and coordination. All authors read and approved the final manuscript.

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Written informed consent was obtained from the patient. For this type of case report, formal consent from a local ethics committee is not required.

Consent for publication

Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

Competing interests

The authors declare that they have no competing interests.

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