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Purple Urine Bag Syndrome in 55- Years-Old Sudanese Women with Renal Failure: Case Report

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Abstract **Case Report**

Purple urine discoloration, or Purple Urine Bag Syndrome (PUBS), is a prevalent condition in bed ridden people who used a urinary catheter for a long time. The purple discoloration is alarming to the patients and everyone around them, even though it is usually benign. ***Case Study***: This case study describes a 55-year-old woman as having PUBS. She has had type II diabetes for 20 years, hypertension for 10 years, and an ischemic stroke during the last five months. Her symptoms of renal impairment have required her to be on a catheter for the previous week. She reported to the ER that she had experienced an alteration in the color of her urine along with overwhelming symptoms that had persisted for several days. A new catheter was used. Laxatives and antibiotics were also upgraded, and urine culture samples were taken in. ***Clinical Discussion*:** Although it has also been described in acidic urine, PUBS is typically linked to alkaline urine and in patients who endured catheterization for an extended period, as this patient was. ***Conclusion***: Patients and their family may experience anxiety when dealing with PUBS, an uncommon sign of a urinary tract infection that has an unsettling appearance. Purple discoloration is a relatively benign and asymptomatic condition that serves only as a warning sign of underlying bacteriuria, with little prognostic implications.

Keywords: Purple urine bag syndrome, renal failure, Women.

INTRODUCTION

Purple urine bag syndrome (PUBS) can occur hours or days after catheter insertion, with rates of 9.8%, 8%, 27%, and 42.1% in chronic care units, chronic indwelling catheters, dementia patients, and nursing homes, respectively [1-3].

Purple staining of the urine bag and tube is an unusual clinical manifestation of urinary tract infection. Urine turns purple due to the metabolization of tryptophan by specific bacteria, which results in the production of enzymes and a continuous urinary tract infection. Indigo is blue, whereas indirubin is red. Barlow and Dickson reported it for the first time in 1978 [4].

Alkaline urine, female gender (increased likelihood of UTI due to shorter urethra), constipation (allows more time for bacterial action on tryptophan metabolites), and chronic renal failure (diminished clearance of tryptophan metabolites) are all risk factors for PUBS [5]. The bluish discoloration is thought to be caused by microorganisms breaking down tryptophan metabolites. This is most common in alkaline urine, but PUBS in acidic urine has also been documented [2]. Many bacteria, including E. coli, Proteus mirabilis, Pseudomonas aeruginosa, Klebsiella, Enterococci, and Group B Streptococci, have been implicated in the pathogenesis of PUBS.

The treatment of PUBS is mostly focused on determining the underlying cause, which is usually a bacterial urinary tract infection and constipation. Within one week, symptoms are resolved by replacing the Foley catheter with a fresh bag and treating the infection with antibiotics. Ciprofloxacin (a quinolone) is the most often used antibiotic for PUBS and is deemed adequate empirical therapy [1]. Using non-plastic urine bags is also thought to be a PUBS preventive technique [2]. Many specialists believe that increasing mobility and using safe and sanitary measures during catheterization

will lower the risk of getting purple urine bag syndrome. Other risk factors, such as constipation, can be treated to reduce the likelihood of getting this problem.

CASE PRESENTATION

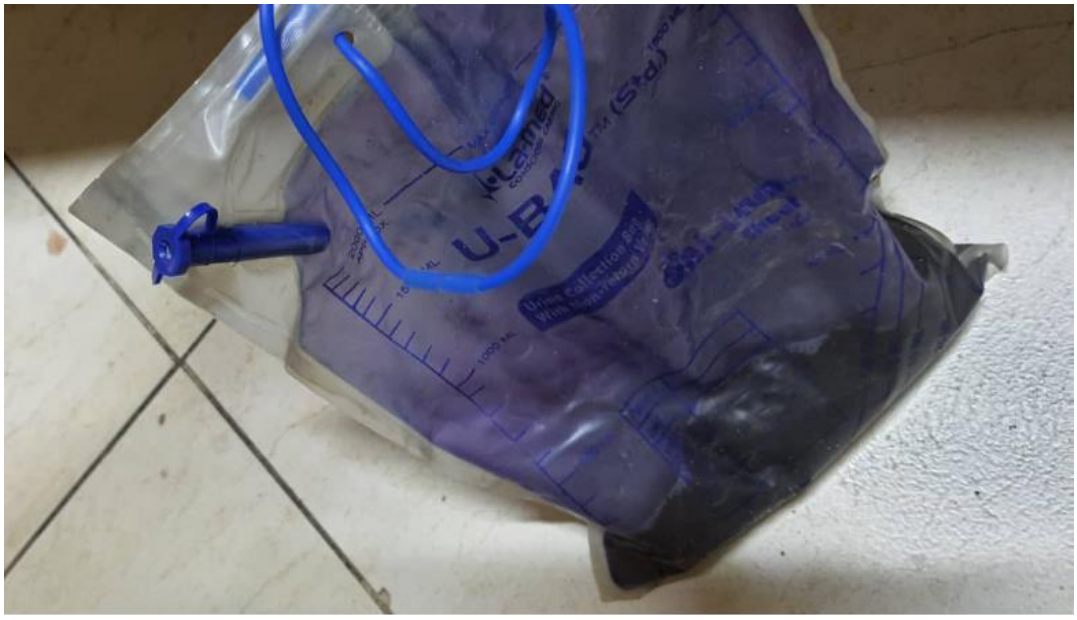
A 55-year-old female known to be diabetic for 20 years on Glimepiride 4 mg and hypertensive for 10 years on amlodipine 5 mg. She has a past medical history of an ischemic stroke five months ago, after which she regained full function and put on aspirin and clopidogrel. She presented to the ER with nausea, severe vomiting more than three times per day, and itching. Apart from that, the other systemic review was clear. The patient was vitally stable, with a BP of 150/90 mmHg and urine output of 6 ml/kg/hr. The Physical examination was unremarkable, apart from scratch marks all over the body. General investigations were abnormal, including a low HB of 7.7, and all indices (MCV, MCH, and MCHC) were low. Additionally, an iron study was done and showed iron 23, ferritin 282, TIBC 162, and an iron saturation of 14.4%. ABG was done and suggested acidosis; her RFT were a little bit deranged, as creatinine 2, urea 50, Na 141, K 3.7, and HBA1c 6.4%. Ultrasound revealed small shrinkage kidneys and uremic gastropathy, which was suggestive of end-stage renal disease.

Therefore, the hemodialysis decision was considered along with fluid intake based on the severity of the patient's symptoms and the continuous rise in renal function test markers.

Regarding the treatment plan, the patient was catheterized and put on a renal chart. Moreover, loop diuretics were added in addition to ceftriaxone, metronidazole, pantoprazole, and cautious fluid intake. Some symptoms, like vomiting and itching, were stopped subsequently. Even so, on day three, new complaints like repeated episodes of hypoglycemia, headaches, and constipation emerged, which were treated accordingly with D50%, paracetamol tabs, and laxatives.

On Day 4, patient level of consciousness deteriorated as GCS declined to 13/15 along with episodes of retrograde amnesia. On physical examination, she was pale and tachypneic. Chest auscultation showed bibasal crepitation in addition to lower limb edema and purpura over the upper limbs and chest. New investigations were ordered and showed the following: urea 89, creatinine 5.7, K 3.0, Na 132, S. calcium 9.5, S.mg 2.9, HB 7.7, WBC 10, and PLT 292. Accordingly, a blood transfusion was started, and received two units in addition to continuing the same plan.

On day five, the patient had a change in urine color in the urine bag, which turned purple. She started to gain consciousness gradually, and her urine output became 0.5mg/kg/hr. Subsequently, on days 6 and 7, two hemodialysis sessions were done, and the catheter was changed again. As well as that, urine culture samples were taken, and laxatives and antibiotics were upgraded.



Following, patient condition improved gradually, constipation was relieved, and urine color returned to normal. It should be noted that she was discharged in good condition and put on multivitamin and calcium supplements in addition to restricted diabetes and hypertensive medications. She was also referred to the nephrology department to schedule future regular hemodialysis sessions and regular follow-ups.

DISCUSSION

Purple Urine Bag Syndrome (PUBS), initially documented in 1978 (5), (However, it had an effect on King George III in 1812 Despite its rarity, it has been observed in institutionalized patients on long-term urinary catheters to reach up to 27%. However, only five cases were discovered during a seven-year study that ended in 2020.Only one of them was male, while the other four were female). It is an infrequent manifestation associated with urinary tract infections. While it is considered a rare phenomenon, its prevalence has been noted to reach up to 9.8% among individuals residing in institutionalized settings who use long-term indwelling urinary catheters [1].

The etiology of PUBS begins with the digestive and absorptive processes of tryptophan in the gastrointestinal tract. Tryptophan undergoes bacterial metabolism in the intestines, leading to the formation of indole. Subsequently, hepatic enzymes facilitate the conversion of indole to its conjugate, indoxyl sulfate, which is then excreted into the urine by the kidneys. Within the urinary system, phosphatases and sulfatases produced by gram-negative bacteria play a role in metabolizing indoxyl sulfate into indoxyl. Further oxidation processes may result in the transformation of indoxyl into indigo and indirubin. In patients with urinary catheters, the mixture of blue indigo deposited on the surface of the urine bag and red indirubin which is mainly mixed with urine gives us the purple discoloration of the urine. According to a case-control study, individuals with Purple Urine Bag Syndrome (PUBS) exhibited significantly elevated bacterial counts in their urine, ranging from 1 to 2 logs higher compared to those without the syndrome. This indicates that a heightened bacterial load in the urine plays a crucial role in the development of PUBS [6, 7]. Other important risk factors for the development of PUBS are hospitalization and urinary catheterization, constipation and the female gender. Additionally, other factors that play a role are the pigments concentration, the presence of alkaline urine, and the urine bag manufacturing materials [7]. However, other studies did report PUBS in acidic urine [8].

PUBS is frequently linked with bacterial species such as Providencia spp., E. coli, Proteus spp., Pseudomonas spp., Klebsiella pneumoniae, Morganella spp., and Enterococcus spp. Occasionally, less common associations involve Citrobacter spp., Staphylococcus spp., Streptococcus spp., and MRSA [9]. As in our case, the patient was a female, she had the risk for PUBS as she was hospitalized with indwelling catheter, she started having constipation on day three of hospitalization, two days before urine discoloration along with (her UTI and alkaline urine).

In a limited cohort study involving Taiwanese patients, chronic kidney disease (CKD) emerged as a notable risk factor for Purple Urine Bag Syndrome (PUBS) [6]. Elevated levels of indoxyl sulfate in the serum and urine were prominently observed in individuals with CKD or undergoing dialysis, reflecting compromised renal clearance [9]. Within the scope of the current investigation, 18.8% of PUBS cases had a documented history of uremia. As our patient had a history of CKD, this study supports the addition of CKD to the other risk factors that our patient had. Moreover, Previous research has also highlighted that comorbid conditions such as diabetes mellitus, dementia, and iron deficiency anemia independently contribute to the risk of asymptomatic Bacteriuria (ABU) and UTI [9, 10] like our case.

The comparison of our case to a similar case by Pillai *et al*., [11], reveals similarities in the complexity of

medical histories, including type 2 diabetes with renal complications, and hypertension. Both cases have the risk factors of constipation and urinary catheterization prior to the development of PUBS and they both involve complications requiring hemodialysis in addition to UTI and antibiotic use, emphasizing the need for careful monitoring and adjustment of treatment plans. Furthermore, almost all patients who developed PUBS had a chronic indwelling catheterization due to significant disabilities [12] unlike our patient who was catheterized for only 5 days before the change of her urine color to purple.

Although the PUBS is benign, the concomitant UTI may have unforeseen consequences in a vulnerable patient. If the recurrence is frequent, sanitation measures such as replacing the catheter and bag with the same or a different material may be sufficient in some situations, but oral antibiotics may be required to treat UTI. Intravenous antibiotics may be reserved for those with repeated infections or who are immunocompromised. In addition, regular hydration and treatment of other risk factors such as constipation and chronic renal failure are critical. Urine acidity can also be useful when combined with vitamin C supplementation. It is also critical to reassure the patient and their relatives.1However, because there are no treatment guidelines, whether antibiotics are used to treat it is up to the practitioners. Although in this case, no additional antibiotics were given, catheter care was outstanding, and constipation was managed (Before prognosis).

Despite being relatively benign and easily treatable, the sudden discoloration of urine and urine bags in Purple Urine Bag Syndrome (PUBS) can cause distress among family, friends, and healthcare workers who may be unfamiliar with this phenomenon. It often leads to undue alarm due to the unexpected nature of the discoloration. It is crucial for physicians to be cognizant that PUBS serves as a signal for underlying recurrent urinary tract infections (UTIs), often stemming from inadequate care of urinary catheters and poor sanitation practices. While the syndrome itself is manageable, if left untreated, it can potentially contribute to significant morbidity and mortality. Therefore, raising awareness about PUBS and its association with recurring UTIs is essential for ensuring timely and appropriate medical intervention [5, 1, 13].

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