Purple Urine Bag Syndrome in a Nigerian with Gastrointestinal Bleeding: Case Report

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ABSTRACT

Purple urine bag syndrome (PUBS) is an unusual presentation of urinary tract infection (UTI). It has scarcely been reported from Africa or in patients with gastrointestinal bleeding (GIB). We hereby report a case of PUBS in a Nigerian with GIB and a short duration of urethral catheterization. Furthermore, this report provides pictorial evidence which support the current knowledge on the pathogenesis of PUBS and describes differences between the colors of urine sediments obtained directly from the urethral catheter and that obtained from the urine bag.

Keywords: *Purple urine bag syndrome, Africa, urine sediment, gastrointestinal bleeding, PUBS*

INTRODUCTION

Purple urine bag syndrome is a rare, alarming presentation of UTI which manifests as a sudden purple discoloration of the urine bag. Tryptophan in the colon is broken down to indole which is metabolized by the liver to indole sulfate. When indole sulfate is filtered into the urine, indole sulfatase positive organisms convert it into two pigments – indigo and indirubin- which combine to yield a striking purple color on the urine bag.

CASE REPORT

A 71-year-old female diabetic/hypertensive on twice-weekly maintenance hemodialysis due to diabetic nephropathy, presented with progressively worsening hematochezia of five months. She was treated for lower GIB secondary to diverticulosis. On account of this, she was transfused six units of blood and catheterized to monitor urine output.

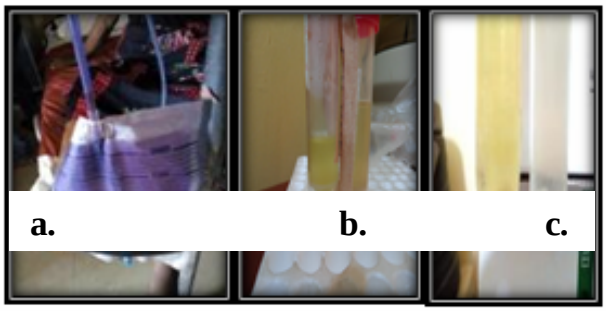

Figure 1a: Purple bag and tubing; Figure 1b: Urine samples from the urine bag and urethral catheter; Figure 1c: Both samples after centrifugation for 5 minutes. The sample on the right is from the urine bag

Two weeks later, while still on admission, she complained of vomiting and generalized body weakness. Her urine bag and tubing were noted to have developed a purple coloration (**Figure 1a**). She had no urinary tract symptoms. Urine samples obtained from the urine bag and from the urethral catheter proximal to the urine bag had normal coloration and were not purple (**Figure 1b**). Her vital

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signs were: T = 36°C, PR = 92b/m, Bp = 110/70mmHg, RR = 22c/m. Serum renal biochemistry revealed Na - 138mmol/l, K – 3.7mmol/L, Cl – 100mmol/L, HCO3 – 28mmol/L, Urea – 10.3mmol/L, Cr - 287µmol/l. Her fasting blood sugar was 122mg/dl and dipstick urinalysis of both samples were similar (**Table 1**).

Table 1: Dipstick urinalysis results of the patient

Urinalysis	WBC	Nitrites	Protein	pH	Blood
	++	Negative	+	8	Trace

After centrifuging the urine samples for five minutes at 2000 rpm, a striking difference was observed. The sample from the urethral catheter had a milky-yellow precipitate whereas the precipitate seen in the sample obtained the urine bag had a periphery that was white in color and a bluish center (**figure 1c**).

Both samples had similar findings on urine sediment microscopy:-numerous leucocytes and coliforms, calcium carbonate and oxalate crystals; though there were much more bacteria and crystals in the sample obtained from the urine bag. The urethral catheter was removed and the urine samples were cultured. Both samples grew *Escherichia coli* sensitive to Ceftazidime, Cefotaxime, Ampicillin, and Gentamycin but resistant to Ceftriaxone and Ofloxacin. She was placed on iv Ceftazidime with subsequent resolution of vomiting and fatigue.

DISCUSSION

Purple urine bag syndrome is a rare form of UTI whereby the urine confers a purple discoloration to the urinary catheter bag and tubing. . It is an unusual presentation of UTI. It can be a source of great distress to the patient and of significant concern to the managing physician.

The earliest documentation of PUBS was in 1978 by Barlow and Dickson [1]. This condition has been scarcely documented in Sub-Saharan Africa. A careful search of Google Scholar and Medline showed only three articles from Sub-Saharan Africa with none from Nigeria [2,3,4].

PUBS is caused by UTI secondary to indole sulfatase (IS) secreting organisms such as *E. coli, Proteus mirabilis, Klebsiella pneumonia, Providencia spp, Pseudomonas spp, Enterobacter spp, Enterococcus spp, Morganella morgagni, Citrobacter spp* [5]. Worldwide, E.coli is the most common cause of PUBS accounting for 23% of cases [5].

In the colon, tryptophan is metabolized by bacteria into indole. Indole is absorbed into the portal circulation and transported to the liver where it is converted to indole sulfate. Indole sulfate is filtered into the urine where it is catabolized by IS positive bacteria into two products: indigo, which has a blue color and indirubin, which is red in color [6]. Indirubin dissolves in the plastic material of the urine bag while indigo sticks to the plastic surface. The mixture of both colors with the plastic stains the urine bag purple [6]. However, the urine itself does not have the purple coloration. This is exactly as shown in figures 3 and 4; urine samples from the bag and directly from the catheter were amber.

Other conditions identified as common risk factors for PUBS include constipation, enemas, institutionalization, chronic urinary catheterization, alkaline urine, abnormal urinary tracts such as the presence of nephrostomy tubes, or ileal diversion of urine [2,6,7]. Constipation is a significant risk factor for PUBS [7]. This is because the bowel stasis in constipation promotes colonic bacterial overgrowth and also increases the retention time of tryptophan within the large intestine. This enhances the metabolism of tryptophan to indole leading to increased concentrations of indole sulfatase within the portal circulation. Bowel enemas may alter the intestinal flora to favor the dominance of indole sulfatase producing organisms while institutionalization, chronic urinary catheterization, and abnormal urinary tract increase the risk for UTI and consequently PUBS. Dealler et al observed that urease positive organisms tend to produce indigo faster on culture compared to the negative strains; this might account for the strong association of PUBS with alkaline urine [6]. Most cases of PUBS reported in the 20th century were in women but more recent reports published since 2011 suggest that the incidence in both sexes may be equal [5].

Our patient had several risk factors that are in agreement with the majority of the literature on PUBS. She was an elderly woman (71 years) who had alkaline urine. However, it is interesting to note that she was not constipated, contrary to reports in the literature, rather she had lower GIB. This is the first report of PUBS in the setting of GIB. The presence of chronic GIB in the index case may have increased the tryptophan load in the colon and also altered the balance of colonic flora leading to

overgrowth of indole sulfatase secreting organisms and increased indole sulfate synthesis [6].

Another unusual feature of this case is the onset of PUBS following catheterization for just 2 weeks. This may be because the combination of the short urethra in females and her diabetic status put her an increased risk for catheter-associated UTI [8]. Hemodialysis (HD) may have increased her risk for PUBS even though PUBS in HD patients is rare [9]. Uremia encourages the growth of aerobes like E.coli which increases the production of indole [9]. Additionally, indole sulfate is poorly cleared by hemodialysis because it is protein bound [10].

The white sediment in the sample from the urine bag was due to the alkaline pH of the urine which was left to stand in the bag; these conditions favored precipitation of calcium crystals. Whereas the milky color of the sediment from the fresh urine sample reflects its high concentration of leucocytes.

Purple urine bag syndrome generally has a good outcome as seen in our patient where it resolved after the catheter was changed and the appropriate antibiotic was administered [5].

CONCLUSION

We have described a patient with purple urine bag syndrome in an elderly Nigerian woman with chronic GIB and short duration of catheterization. We postulate that chronic GIB led to increased tryptophan load and overgrowth of indole sulfatase secreting organisms within the colon. The urine color changed back to normal following administration of antibiotics.

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