Purple Urine Bag Syndrome

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Abstract
The purple urine bag syndrome (PUBS) i.e. purple discolouration of urine, is an uncommon and alarming condition in patients with long term urinary catheterisation. Though the condition is benign, it should draw immediate attention to the possibility of an underlying urinary tract infection. The postulated hypothesis for this unusual event is probably a chemical reaction involving tryptophan from food in the gut. We report a case with this unusual and interesting phenomenon in a 65 year old female.

Introduction
PUBS is a rare disorder causing discolouration of urine and urine collection bags due to the presence of indigo and indirubin pigment produced by tryptophan metabolism. This condition is most commonly associated with alkaline urine, female gender, constipation and urinary tract infection. Although this condition is benign, it can be distressing for patients, family members, and health care workers who are unaware of this entity. We present a case with this rare phenomenon.

Case Report
A 65 year old bed ridden female, with a history of type 2 diabetes mellitus and dementia with old fracture of left femur who had an indwelling urinary catheter in place for last 3 months, presented to us in the emergency department with the complaints of nausea, vomiting, decreased oral intake, chronic constipation and purplish discolouration of the urine bag and tubing (Figure 1). She was hemodynamically stable. Physical examination revealed mild palor and features suggestive of left lower limb deep vein thrombosis. Her urine bag was filled with purple coloured urine along with purplish discolourisation of the tubing and the bag. Investigation revealed a haemoglobin of 9.8 gm/dl and total leukocyte count of 8800/mm³. Her blood urea was 68 mg/dl, serum creatinine 1.6 mg/dl, serum sodium 138 mEq/L, potassium 3.07 mEq/L, serum protein 5.7 gm/dl with albumin 1.2 gm/dl. Doppler study of left lower limb was suggestive of deep vein thrombosis (DVT). Urine microscopy revealed 4-5 leucocytes, 3-4 red blood cells and calcium oxalate and phosphate crystals, bacteria 3+ and albumin 2+. Urine culture showed Escherichia coli and she was started on injection Ceftriaxone. Injection low molecular weight heparin was given for DVT. She responded well to treatment with disappearance of purple urine colour.

Discussion
PUBS was first described by Barlow and Dickson in 1978, after purple urine was found in a patient who had prolonged urinary catheterization. Interestingly, even a famous historical figure, England’s King George III (1738-1820) was believed to have been affected by this syndrome or at least a part of it. The prevalence of PUBS ranged from 8.3% to 42.1% in different studies but most of the published data is based on case reports and there is no data regarding prevalence of this rare condition from Indian subcontinent. PUBS has been shown to be associated with female gender, constipation, advanced age, chronic urinary catheterisation, urinary tract infection and use of plastic urinary catheter and bag. Higher bacterial load in urine, in combination with the above factors, facilitates the development of PUBS. The bacteria most commonly associated with PUBS are Escherichia coli, Klebsiella pneumonia, Enterobacter agglomerans, Pseudomonas aeruginosa, Proteus spp., Providencia species, Enterococcus species, Streptococcus spp., Staphylococcus spp., and even MRSA.

The postulated hypothesis regarding aetiologypathogenesis of PUBS is believed to be related to metabolites of dietary tryptophan (Figure 3). The normal flora in the intestine metabolises tryptophan to indole which is absorbed into the portal circulation via the gut wall. Liver conjugates indole into indoxylsulphate which is excreted in urine. Urinary bacteria produce an enzyme indoxylsulphatase, breaking down the indoxylsulphate into indoxyl. Then indoxyl turns into indigo (blue in colour) and indirubin (red in colour). The mixture of these two substances produces purple urine in the urinary catheter and collection bag.

A strong association of PUBS with constipation and intestinal obstruction has also been described. Chronic constipation alters gut motility and prolonged transit time, resulting bacterial overgrowth in the colon. Bacterial flora containing tryptophase converts tryptophan to indole and indole is converted to indigo and indirubin through a chain reaction that gives urine purple colour.

Now, despite the common occurrence of urinary tract infection with all the risk factors of PUBS, it is interesting to note the rarity of this syndrome. There may be few possible explanations: PUBS probably requires the simultaneous presence of multiple risk factors, e.g.
presence of urinary tract infection caused by sulphatase or phosphatase producing bacteria, presence of high tryptophan in diet, being catheterized or constipated. Furthermore, a certain concentration of the pigments may be required for the precipitants to become visible. The presence of alkaline urine and also the type of materials used to manufacture the urinary catheter and bag may be important factors. PUBS is more common following polyvinyl chloride (PVC) plastic catheterisation; PUBS following long term use of silicon based catheter also has been reported but is very rare. Interestingly, PUBS in the presence of acidic urine has also been reported.

Most of the patients with PUBS remain asymptomatic, its clinical course is usually benign and therefore only changing the urinary catheter and urinary bag usually are enough to solve the problem. Aggressive investigations like urine culture or septic work up and treatment with antibiotics are usually not necessary. Antibiotic is only indicated when there is concurrent symptomatic urinary tract infection. For asymptomatic patients, treatment should be aimed at the underlying medical problem rather than purple bag itself and to reduce the likelihood of this problem, it is important that the drainage bags and indwelling catheters may need to be changed on a regular basis.

In conclusion, PUBS is a rare manifestation of urinary tract infection. It often occurs in chronically catheterized and constipated patients who have significant underlying comorbidities. It has a relatively benign course. Considering the known etiologic and pathophysiologic mechanisms of PUBS, it is surprising how rarely this situation is observed and/or reported.

References