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Medicine

PURPLE URINE BAG SYNDROME: A RARE CASE REPORT

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ABSTRACT Purple urine bag syndrome (PUBS) is a rare medical entity, which is observed predominantly in chronically catheterized, constipated patients and is associated with bacterial urinary tract infections that produce sulphatase or phosphatase. We are reporting a case report of this unusual phenomenon in a 45 year male patient which resolved completely with antibiotics.

KEYWORDS : Purple urine bag syndrome, urinary tract infection, chronic urinary catheterisation, indigo, indirubin.

Introduction

Purple urine bag syndrome (PUBS) is an unusual entity, where purple discoloration of urine occurs in people with long term urinary catheters & coexisting urinary tract infection (UTI). It was first described in 1978 by Barlow & Dickson[1]. This phenomenon usually occurs over a two year period in about 8% of patients with indwelling urinary catheters[2]. It is believed to be related to the metabolism of tryptophan[3]. Bacteria in the urine of these patients produce enzyme indoxyl phosphatase or sulfatase. This converts indoxyl sulfate in urine into red & blue colored compounds - indirubin (red) & indigo (blue). The most commonly implicated organisms are gram negative bacteria i.e. Providencia stuartii, Providencia rettgeri, Klebsiella pneumoniae, Proteus mirabilis, Escherichia coli, Morganella morganii, Enterobacter & Pseudomonas aeruginosa[4]. Concerning PUBS, we here present a clinical case.

Case report:

A 45 year old male patient, without any previous comorbidity got an accidental burn injury 26 years back (in 1992) at lower abdomen, inguinal region & bilateral thighs. He was catheterized in view of difficulty in passing urine because of pain. After 2 weeks, catheter was pulled out by a quack without decompression, following which patient developed hematuria and loss of bladder control. Since then, he developed recurrent fever and pain abdomen. Patient had history of urinary catheterisation multiple times. In 2017, he approached PGIMS Rohtak for his above complaints with catheter in situ. On general physical examination, patient was normotensive and had pallor. Systemic examination was within normal limits. Routine blood investigations revealed normocytic- normochromic anemia (Hb- 8.9g/dl) with normal total leucocyte count. Blood urea & serum creatinine were raised (Blood urea- 149 mg/dl; s. creatinine- 6.1mg/dl). S.calcium, phosphorus, sodium and potassium were within normal limits. Blood gas analysis showed metabolic acidosis with pH -7.31, $HCO3^-$ 10.4 meq/l. Urine routine examination revealed albuminuria 2+ & microscopy showed 8- 10 pus cells and crystals of tripple phosphate. Urine was alkaline in pH (pH- 7.6). Urine culture had growth of Acinetobacter & Citrobacter. USG kidneys showed right kidney 9.6×3.4 cm with a calculus of size 7mm in middle calyx with grade III hydronephrosis (HDN). Left kidney measured 9.1×4.1cm with grade II HDN. NCCT KUB was s/o pyonephrosis. In view of above findings, b/l percutaneous nephrostomy (PCN) was done and culture sensitivity specific antibiotics were started. The final diagnosis was kept as obstructive uropathy with urinary incontinence with pyonephrosis with b/l PCN with foley's catheter in situ. After 3 months, patient presented to emergency with c/o breathlessness & purple discoloration of his urobag (Fig 2 & 3). Examination revealed b/l fine crepitations at lung bases s/o pulmonary edema and investigations showed anemia, uremia and metabolic acidosis (Hb- 7.8g/dl, B.urea- 203mg/dl, s.creatinine- 7.8mg/dl; pH- 7.1, $HCO3^-$- 7.6 meq/l). Urine examination showed full of pus cells. Inj. furosemide, Inj. ceftriaxone & Inj. sodium bicarbonate were started. Patient was started on hemodialysis. On urine culture, growth of Pseudomonas aeruginosa sensitive to cefipime, amikacin, imipenem, piperacillin-tazobactam was discovered. Inj. piperacillin-tazobactam in renal modified dose was added to the ongoing treatment along with maintenance hemodialysis. Metabolic acidosis, uremia, pulmonary edema & general wellbeing of the patient improved over next couple of days with disappearance of purple discoloration of urine after 1 wk. At present, patient is doing well on hematinics, tab sodium bicarbonate with maintainance hemodialysis twice weekly.

Discussion :

PUBS is usually more common in female nursing home residents. Other risk factors include:

1) Alkaline urine[5] (predisposes to growth of contributory microbes); but very uncommonly it is still possible for this to occur in acidic urine.
2) Constipation (prolongs tryptophan transit time in intestine that increases indoxyl sulfate levels in urine).
3) Polyvinyl chloride catheter use[6].
4) Dehydrated state or Hypovolemia .

PUBS can be associated with intestinal intussusception[7].

Proposed mechanism of purple urine is shown in Fig.1

Tryptophan ➡ Bacteria in GIT
(In Diet)
⬇
INDOLE
⬇
(Absorbed by Intestine)
⬇
Liver
⬇
Indole converted to INDOXYL SULFATE
⬇
Excreted in Urine
⬇
Bacteria colonising urinary catheters produce Indoxyl sulfatase/ phosphatase
⬇
In presence of alkaline urine[8]
⬇
Indoxyl sulfate oxidation
↙ ↘
Indirubin (Red) & Indigo[9] (Blue)
⬇
PURPLE URINE

Fig 1: Formation of Indirubin & Indigo from Tryptophan.

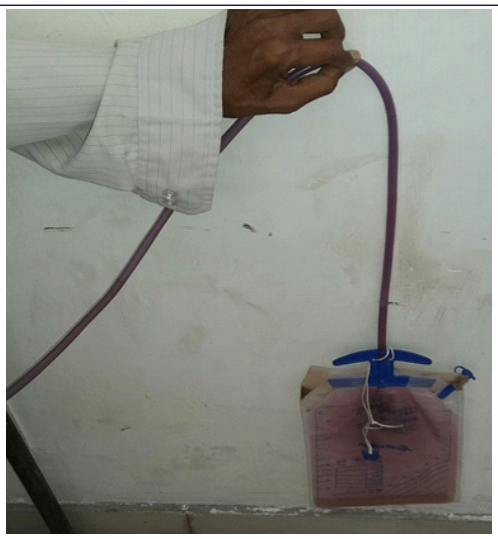

Fig.2 : Purple discoloration of urine in plastic urinary catheter and urobag.

Conclusion

Patients with PUBS usually don't complain of any symptoms. Though purple discoloration of urine may look alarming initially, it is usually considered a benign condition. Although, in the settings of recurrent or chronic UTI, it may be associated with some drug resistant bacteria [10]. PUBS resolves shortly after sensitivity specific antibiotic treatment.

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