



## Clinical Case

### Purple Urine Bag Syndrome: a Sometimes Serious Manifestation of Urinary Tract Infection

*Le syndrome du sac urinaire violet: une manifestation parfois grave de l'infection des voies urinaires*

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#### RÉSUMÉ

Purple urine bag syndrome (PUBS) is a sign of urinary infection characterized by a purple staining of the urinary catheter and the urine collector by pigments resulting from the degradation of dietary tryptophan by intestinal and urinary bacteria. It is a benign syndrome but can manifest itself with complications under certain conditions. We report a case of PUBS revealed by a state of septic shock. A patient HM with a history of high blood pressure (hypertension) and stroke with left hemiplegia, bedridden for the last six months was admitted to internal medicine for her bedridden condition, anorexia, and significant weight loss. Twelve days after admission, her clinical condition deteriorated with the onset of septic shock and a change in the urine bag colour to purple. The urinary strip showed a urinary pH at 8.3, leukocyturia (2+) with nitrite (1+) and proteinuria (2+). Urine cytobacteriological examination showed leukocyturia > 10<sup>4</sup>/ml, bacteriuria > 10<sup>5</sup>uF/ml and the presence of K.pneumoniae sensitive to ceftriaxone. Management was based on antibiotic therapy and medical resuscitation. The patient died during resuscitation. Although PUBS is considered benign, the clinician should keep in mind the existence of life-threatening severe forms.

#### ABSTRACT

Le syndrome du sac urinaire violet (PUBS) est un signe d'infection urinaire caractérisé par une coloration violette de la sonde urinaire et du collecteur d'urine par des pigments résultant de la dégradation du tryptophane alimentaire par des bactéries intestinales et urinaires. Il s'agit d'un syndrome bénin mais qui peut se manifester par des complications dans certaines conditions. Nous rapportons un cas de PUBS révélé par un état de choc septique. Une patiente HM ayant des antécédents d'hypertension et d'accident vasculaire cérébral avec hémiparésie gauche, alitée depuis six mois, a été admise en médecine interne pour son état alité, son anorexie et sa perte de poids importante. Douze jours après son admission, son état clinique s'est détérioré avec l'apparition d'un choc septique et un changement de couleur de la poche d'urine qui est passée au violet. La bandelette urinaire montrait un pH urinaire de 8,3, une leucocyturie (2+) avec nitrite (1+) et une protéinurie (2+). L'examen cytobactériologique des urines a montré une leucocyturie > 10<sup>4</sup>/ml, une bactériurie > 10<sup>5</sup>uF/ml et la présence de K.pneumoniae sensible à la ceftriaxone. La prise en charge a été basée sur l'antibiothérapie et la réanimation médicale. Le patient est décédé pendant la réanimation. Bien que le PUBS soit considéré comme bénin, le clinicien doit garder à l'esprit l'existence de formes graves potentiellement mortelles.

#### INTRODUCTION

Purple urine bag syndrome (PUBS) is a rare condition described for the first time in 1978 by Barlow and Dickson [1-2]. It is a sign of urinary infection and manifests as a purple colouring of the urinary catheter and the urine collector. This coloration which characterizes it results from the interaction between constituents of the urinary catheter and the urine collector (polyvinyl chloride) and metabolites from the degradation of food tryptophan by intestinal and urinary bacteria. Often described as benign, PUBS can however lead to potentially life-threatening complications [3-4]. We report a case of PUBS complicated by a state of septic shock.

#### OBSERVATION

Mrs. HM, 66 years old, gravida 6, para 6, with a history of high blood pressure (hypertension) and a stroke with left hemiplegia 6 months previously leading to bed confinement. Mrs. HM was admitted to internal medicine for her bedridden condition, anorexia, and significant weight loss. The physical examination noted a conscious, cachectic patient with a BMI of 9 kg/m<sup>2</sup> and left pyramidal syndrome following her stroke. Twelve days after admission, the patient's condition worsened with the onset of fever of 39 ° C, a respiratory rate of 40 cycles/min, a heart rate of 130 beats/min, blood pressure of 80/50mm Hg and a Glasgow of 8. A change in the colour of the urine collector (placed at admission) was noted, becoming purple while the urine was a normal dark yellow in colour.



**Figure 1:** purple urine bag with urine of normal colour

Biological examination showed an inflammatory syndrome with predominantly neutrophilic hyperleucocytosis at  $12000/\text{mm}^3$ , hypochromic microcytic anaemia at  $9.3\text{g/dl}$ , a C reactive protein (CRP) at  $12\text{mg/l}$ . The human immunodeficiency virus (HIV) serology and the thick blood smear were negative. Blood pressure was  $9.6\text{ mmol/l}$ , serum creatinine was at  $77\text{umol/l}$ , blood glucose at  $5.92\text{ mmol/l}$  and electrolytes were normal. The urine test strip showed a urinary pH of 8.3, leukocyturia (2+) with nitrite (1+) and proteinuria (2+). Urine cytobacteriological examination (ECBU) showed leucocyturia  $> 104/\text{ml}$ , bacteriuria  $> 10^5\text{uFC/ml}$  and the presence of *K. pneumoniae* sensitive to ceftriaxone.

Therapeutic probabilistic antibiotherapy (ceftriaxone-gentamycin) and resuscitation (crystalloid fluid administration, oxygen therapy ....) were instituted. The patient died during the resuscitation.

## DISCUSSION

Purple urine bag syndrome (PUBS) is a rare condition described for the first time in 1978 by Barlow and Dickson [1-2]. PUBS is considered a rare phenomenon but current studies even if of low power report a prevalence around 10% in patients with long-term bladder catheterization [3,5-6]. Like our study, most of the authors have described case reports of PUBS as a condition occurring preferentially in women [5,7-14]. Mumoli N et al reported in their hospital study that 73% were women [15]. According to Yu-Jang Su et al. 60% of PUBS cases occur in women [4]. This female predominance is explained by the proximity of the urethra to the anus and by a shorter urethra in women favouring contamination of the urinary tract by digestive germs [2,5]. However, some authors have described PUBS in men and even in children [1,16-21]. As in our study PUBS occurs mostly in the elderly [2, 8-14, 16-17, 19-22]. In the work of Mumoli et al [15], all patients are over 65 years of age with an average age of 78. Yu-Jang Su et al reports [4] in their study an average age of 75.5 years. The more frequent occurrence of PUBS in the elderly may be explained by the fact that during this period of life one is more likely to experience loss of physical and/or cognitive autonomy due to multiple associated conditions such as stroke, dementia. The loss of physical and intellectual autonomy promotes intestinal

and urinary inertia as well as poor urogenital hygiene causing urinary tract infections and PUBS. If Barlow and Dickson were the first to describe PUBS, the merit of ethiopathogenic understanding rests with Dealler et al [23]. According to the latter, the purple colouration observed is due to the presence of two pigments: indigo and indirubine originating from the metabolism of tryptophan. In effect, the colonic bacterial flora metabolizes dietary tryptophan into indole which is then absorbed and conjugated in the liver to eliminated indoxyl sulfate in the urine. At this level the indoxyl-sulfate, in the presence of a bacterial enzyme, indoxyl-sulfatase/phosphatases, is hydrolysed to indoxyl. In a basic medium, the latter is oxidized indigo (a blue pigment) and indirubin (a red pigment). These two pigments mix and interact with the polyvinyl chloride, a constituent of the urinary catheter and the urine collector, to produce a striking purple colour. The bacteria responsible for PUBS possess two types of enzymes necessary for its occurrence: indoxyl-sulfatase/phosphatases and a urease capable of alkalizing the urine. As such, several bacteria have been identified: gram-negative bacilli including *Providencia stuartii*, *Klebsiella pneumoniae*, *Enterobacter agglomerans*, *Pseudomonas aeruginosa*, *Proteus mirabilis*, *E. coli*, *Citrobacter* species, *Enterococcus* species, *Morganella morganii*, and Methicillin-resistant *Staphylococcus aureus*, Group B streptococci [3,24-25]. In our study the isolated germ is *Klebsiella pneumoniae*. According to some studies it is not the presence of these germs that is determinant in the occurrence of this syndrome but rather their concentration (proportional to the intensity of the colouring) in the urine [24-25]. The necessity of an alkaline urinary pH for the transformation of indoxyl is not essential for some authors as some studies have found a basic pH in both the PUBS cases and the controls and PUBS has also been described in an acid medium [6, 25]. In our observation the urinary pH is alkaline. Several risk factors for PUBS are reported in the literature including female sex, dementia, a chronic bedridden state, constipation, alkaline urine, long-term bladder catheterization, high urinary bacterial load, renal failure, the use of a plastic urinary catheter and pouch made of polyvinyl chloride (PVC). The reasons behind the identification of these elements as risk factors are identified. The female sex is more exposed to PUBS because the urethra is shorter and closer to the anus than in men. Dementia is highly related to the recurrence of PUBS because due to the lack of mental autonomy the patients no longer manage to self-insure good urogenital hygiene. Constipation promotes intestinal hypomotility resulting in bacterial overgrowth and increased tryptophan metabolism. Alkaline urine facilitates the precipitation of pigments in the synthetic materials of the catheter and the urine bag. During chronic kidney disease there is an alteration of the clearance of indoxyl sulfate. Long-term bladder catheterization increases the risk of urinary tract infections and therefore of PUBS. Chronic bedridden states with multiple comorbidities require more frequent long-term catheterization. The bacterial

burden during a urinary infection increases the availability of bacterial sulfatases and phosphatases that convert indoxyl sulfate to indigo and indirubin [2, 6,24]. Our patient presents 6 PUBS risk factors, namely female sex, bedridden condition, prolonged bladder catheterization, use of a plastic urine tube and catheter made of polyvinyl chloride, alkaline urine, and a high urinary bacterial load. Most of the work on PUBS considers it a benign condition [1-2,7-9, 11-12, 14, 16,18]. However, some recent studies report cases of severe PUBS with shock or gangrene [4-6, 10,26]. In our case PUBS was complicated by septic shock. According to Yu-Jang Su et al [4] mortality related to PUBS is not negligible and is around 7%. These authors have identified several risk factors for mortality including uraemia, shock, diabetes, leucocytosis and the female sex. Our patient with 4 mortality risk factors died in a state of septic shock. Close monitoring is thus required in patients presenting one of the mortality risks factors. The management of PUBS is based on replacing the urinary catheter, treatment of symptomatic underlying urinary tract infections, control of constipation and good urogenital hygiene. Complicated forms such as ours with shock, gangrene, and immunodepression require more aggressive treatment with parenteral antibiotic therapy and management of these associated complications. Prevention relies on regular replacement and care of bladder catheters [3, 6, 9-10, 12, 24].

## CONCLUSION

Although purple urine bag syndrome is generally considered benign, severe forms with urosepsis, gangrene, and even death is more and more described. Aggressive treatment and close supervision must be applied to any PUBS case with severity risk factors.

## Conflicts of interest

The authors do not declare any conflicts of interest.

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