Purple Urine Bag Síndrome: Pathophysiology Involved

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ABSTRACT

Purple urine bag syndrome is a rare but dangerous entity that indicates an important colonization of the urinary tract by multiple multiresistant bacteria in most cases. The low incidence means that this entity is not only underdiagnosed but also treated erroneously. The culture, as well as a general urine test, are key to its correct diagnosis and treatment.

INTRODUCTION

The purple urine bag syndrome is a rare pathology, related to important bacterial resistance, as well as the presence of multiple risk factors and with little clinical follow-up by the doctors who let it pass, which makes this syndrome a important phenomenon to study.1

Purple urine bag syndrome (PUBS) is a rare condition seen primarily in elderly patients with long-term urinary catheters, almost always due to benign prostatic hyperplasia for urinary drainage, and is characterized by a purple color intensity of urine only when the collection system is made of plastic.1,2

The risk factors associated with this entity are: female sex, advanced age, diet rich in tryptophan, constipation, debilitating chronic diseases and alkaline urinary pH.1,3,4

The most common cause of elevated urinary pH is urinary tract infection by phosphatase- and sulfatase-producing pathogens, this is mainly caused by their metabolism. The most frequently isolated bacteria are, in order of frequency, E. coli, Proteus mirabilis, Klebsiella pneumoniae, Enterococcus spp, among others, two

The pathophysiology of PUBS involves the metabolism of tryptophan, whose 2 final metabolites are responsible for giving the characteristic purple color: indirubin and indigo2,3.

In the small intestine, tryptophan is converted to indole by the intestinal microbiota, and then reaches the portal circulation where it is converted to 3-hydroxyindole by cytochrome P450.4,5

Subsequently, a sulfonation process mediated by the SULT1A1 enzyme takes place, which transforms 3-hydroxyindole into indoxyl sulfate6.

This compound at the level of the urinary tract and due to the influence of bacterial enzymes such as sulfatases, phosphatases and an alkaline environment end up forming indirubin (red color) and indigo (blue color).4

This clinical entity is a challenge for the doctor in the search to avoid the inappropriate use of antibiotics and a correct diagnosis, as well as not treating this entity as something benign and transitory that could trigger sepsis of urinary origin and later antibiotic-resistant septic shock.7

CASE PRESENTATION

The patient is a 77-year-old male patient, alcoholic, he drinks approximately 6 to 8 beers every 8 to 15 days without becoming intoxicated. He has a history of Benign Prostatic Hyperplasia with permanent Foley catheter, Type 2 Diabetes Mellitus and Systemic Arterial Hypertension. He has a Barthel scale of 25 points, with Severe Dependence.

At the time of direct questioning, a patient was reported with an alteration in the color of the urine, which had been purple for two days, accompanied by diarrhea on five occasions. At the time of questioning, the patient was stable as well as 200 ml in a purple urine collection bag without urinary sediment in a Foley catheter (Fig. 1).
In the analytical analysis, Urea 41 Cr 0.9 Ca 9.3 Cl 103 K 4.2 Mg 1.5 Na 140 was found, normal liver biochemistry. In the blood count Hb 14.2 Hto 41.9 Leu 14.1 Plaq General examination of the urine Purple color, cloudy. Leukocyte Esterase 500, Abundant Bacteria Leukocytes 100 - 150 p/field Erythrocytes (-).Ciprofloxacin 400 mg IV every 12 h was started during the hospital stay; after taking the urine culture to avoid alterations in its result. Due to improvement of the clinical picture, but without changes in the purple coloration, it was decided to discharge him to continue with outpatient management of urinary tract infection with Ciprofloxacin 500 mg tablet every 12 hours for 10 days. Once the result of the urine culture was obtained, a positive culture for multiple strains of E. coli was obtained, the patient was informed and the antibiotic regimen was adjusted with nitrofurantoin 100 mg every 8 hours for 7 days. A notable clinical improvement was observed from the 4th day of treatment, turning the color of the urine to a completely amber color. Completely remitting the urinary tract infection.

DISCUSSION
Purple urine bag syndrome is a benign entity that does not usually compromise the patient’s life and does not require aggressive treatment in most cases; Normally, the urine clears and regains its color when the bacteriuria resolves, once the infection is controlled and the urine regains its physiological acidity. Antibiotic treatment will depend on the sensitivity of the uropathogens involved in the infection at the time of urine culture. It is recommended to start empirical antibiotic treatment with a third-generation cephalosporin or a fluoroquinolone of choice, depending on the reported local sensitivity profile. Emphasizing that a urine culture should be previously performed to subsequently adjust the antibiotic therapy, if necessary and if the pathology requires it.

CONCLUSION
The identification of risk factors as well as the early initiation of antibiotic therapy together with obtaining an early urine culture is a challenge for health professionals who seek to treat it definitively, making this pathology a diagnostic and therapeutic challenge. However, if it is controlled correctly, complications such as sepsis of urinary origin could be avoided. The expansion of knowledge of this pathology is key to its correct treatment.

REFERENCES