一位慢性腎臟病女患者呈現紫色尿

Purple-colored urine in a woman with chronic kidney disease

施惠瑩[1] 莊美華[1] 陳宜鈞[2]
大林慈濟醫院 藥劑科[1] 腎臟內科[2]

Case Report:

A 70-year-old woman presented with 3 days of fever up to 38˚C, abdominal pain and purple-colored urine (Figure 1). She had a medical history significant for stage 4 chronic kidney disease secondary to diabetes and hypertension that were controlled with insulin and irbesartan (150 mg daily), respectively, as well as neurogenic bladder managed by a urinary catheter for the last 6 months. On examination, blood pressure was 138/70 mm Hg, respiratory rate was 18 breaths/min, and heart rate was 80 beats/min. Abdominal examination indicated a soft abdomen. She reported no previous abdominal surgery, and did not take any medication that may have caused urine discoloration. Plain radiograph of the abdomen revealed no abnormal gas. Blood testing revealed a leukocyte count of 15110/μL with 85% neutrophils, C-reactive protein 6.27 mg/dL (<0.5 mg/dL), blood urea nitrogen 54 mg/dL, serum creatinine 2.5 mg/dL with estimated glomerular filtration rate of 20.23 mL/min/1.73m^2, and normal liver function tests. Urinalysis revealed a pH of 8.0, positive nitrates, marked pyuria and bacteriuria. Urine culture yielded more than 100,000 colony-forming units of *Pseudomonas aeruginosa*, *Morganella morganii*, and *Proteus vulgaris*. The purple urine disappeared after replacement with a new urinary catheter and intravenous administration of ciprofloxacin (250 mg q12h) for 7 days.

PURPLE URINE BAG SYNDROME: A RARE CLINICAL ENTITY

Purple urine bag syndrome (PUBS) is a rare clinical entity. Urinary tract infection is the leading cause of developing PUBS; however, intestinal intussusception has also been reported as a cause. Our case excluded the latter. Several factors have been reported to be involved in the development of PUBS, such as old age, female gender, chronic constipation, chronic urinary catheterizeation, alkaline (common) or acidic (uncommon) urine, and higher bacterial load in urine. The reported pathogenesis of PUBS is illustrated in Figure 2. Dietary tryptophan is metabolized by intestinal bacteria, and then highly water-soluble indoxyl sulphate is excreted into urine. In vitro experiments, urinary indoxyl sulphate has been shown to be converted to indigo and indirubin by indoxyl sulphatase/phosphatase in alkaline or acidic urine. When blue-colored indigo and red-colored indirubin mix together, they become purple. Certain bacteria possess indoxyl sulphatase/phosphatase, such as *Pseudomonas aeruginosa*, *Morganella morganii*, *Proteus vulgaris*, *Escherichia coli* and *Providencia spp*. However, not

all bacteria of the same species can produce these enzymes required for the formation of PUBS. This may explain the rarity of PUBS despite the common occurrence of urinary tract infections in patients with risk factors for PUBS.

■ CHRONIC KIDNEY DISEASE: A POTENTIAL RISK FACTOR FOR PURPLE URINE BAG SYNDROME

Chronic kidney disease (CKD) has been shown to be a risk factor for PUBS in a small cohort study of Taiwanese patients. The serum and urine level of indoxyl sulphate increase markedly in patients with CKD or undergoing dialysis because of impaired renal clearance. Furthermore, indoxyl sulphate, an important constituent of PUBS, is also a cytotoxic material and may increase the rate of renal failure in uremic rats. PUBS is considered a benign condition because purple discoloration of the urine can draw an attention to an underlying urinary tract infection that can be early treated. Therefore, this clinical entity though rare, indicates the need for prompt evaluation and treatment of urinary tract infections in this at risk population, as the outcomes can be fatal if PUBS progresses to septicemia or Fourier’s gangrene.