Nephrol Dial Transplant (2001) 16: 2097–2099

Nephrology
Dialysis
Transplantation

Nephroquiz for the Beginner
(Section Editor: M. G. Zeier)

Supported by an educational grant from AMGEN

Boy with 'rainbow' urine

Keywords: chromium; coloured urine; rainbow urine

Case

A 16-year-old boy, brought by his mother who worked as a chemist, presented with samples of coloured urine that he had passed on different occasions (Figure 1). These samples were coloured violet, pink, yellow, blue and brown. This phenomenon had been occurring episodically for the past 2 years. He was not on any medication during these episodes. There was no history of renal stones. His scholastic performance had been poor and these events corresponded with school examinations. Physical examination was unremarkable. Investigations revealed a normal haemogram, electrolytes, blood urea, serum creatinine, serum uric acid, and a normal routine urine microscopy. Measurements of 24-h urinary delta amino laevulinic acid, and blood lead levels were normal. However, this patient had hyperuricosuria and a non-specific aminoaciduria. The ammonium-chloride loading test showed that he did not acidify urine normally.

The freshly voided urine sample obtained at our laboratory was colourless. Urine microscopy of freshly voided sample was normal; however, the coloured samples showed thick clumps of non-specific pigments.

Questions:

(a) What are the possible diagnoses?
(b) Could this be factitious?
(c) How can one confirm the diagnosis?
(d) What are the characteristics of the chemical that fits in with this clinical presentation?

Fig. 1. Multi-coloured urine samples.

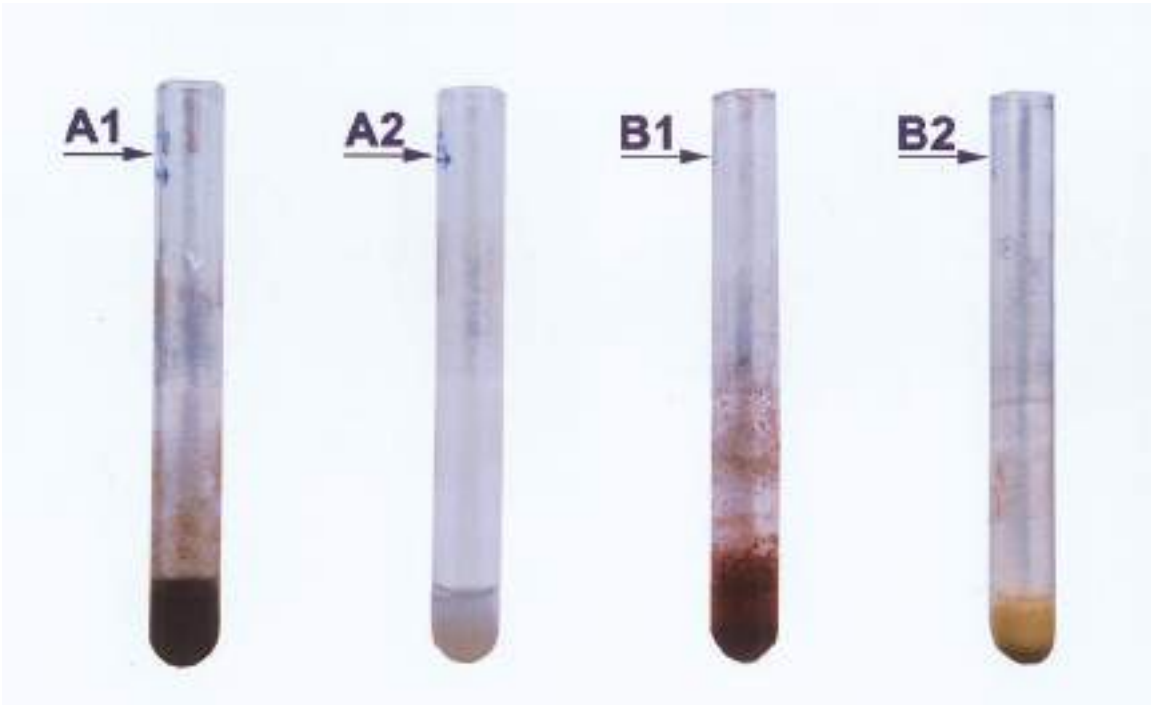

Fig. 2. Tests for chromate: A1, addition of silver nitrate produces a red precipitate; A2, A1 is soluble in aqueous ammonia; B1, A1 is insoluble in dilute acetic acid; B2, addition of barium chloride produces a yellow precipitate insoluble in acetic acid.

Answers to quiz on previous page

(*a*) The intensity of urine colour depends on the urochrome concentration [1]. Abnormal colours may occur in diverse conditions such as haematuria, jaundice, haemoglobinuria, myoglobinuria, porphyria, phenylketonuria, and beetroot intake, or after the ingestion of certain drugs like rifampicin and metronidazole. Urine tested negative for haemosiderin, homogentisic acid, phenylketones, coproporphyrinogen, and porphobilinogen in this patient. One should rule out secondary aetiologies like porphyria, alkaptonuria, lead poisoning, and drug-induced discoloration. Chromium intake can also result in nephrotoxicity, leading to tubular necrosis and chronic tubular dysfunction. The colour produced by these dyes is known to vary with pH. Our patient had renal tubular dysfunction as evidenced by non-specific aminoaciduria and mild renal tubular acidosis.

(*b*) A possibility of factitious coloration of urine should be considered, as there were pigment clumps of multiple colours in different bottles, but in none of those produced under supervision. Chromogens were thought to be present, especially dyes or chemical pigments, since the colour disappeared on the addition of strong sodium hydroxide solution.

(*c*) As chromate pigments are commonly used in preparing water and poster colour for students, the urine samples were tested for chromate [2]. The addition of 0.5 ml of 0.5 g% of silver nitrate solution to 0.5 ml urine resulted in the formation of a red precipitate, soluble in aqueous ammonia but insoluble in dilute acetic acid. When 1 g% of barium chloride solution was added to 0.5 ml urine a yellow precipitate was formed (Figure 2). These tests confirmed the presence of chromate in all the coloured urine samples. The freshly voided colourless urine samples tested negative. It was therefore concluded that this patient used chromate-containing water colours/dyes to produce these bizarre colours in urine. There was a secondary gain of absence from examinations during his visit to our hospital (3000 km away from home). When confronted with proof of chromate contamination, there was strong denial by the mother and the patient. He was then referred to a psychiatrist for further evaluation. Patient and mother were reviewed after a year. The complaint of coloured urine had spontaneously resolved and urine tested negative for chromate compounds.

(*d*) Chromium and chromate compounds change colour as the pH varies. Above a pH of 6, chromium trioxide is yellow, but orange red crystals form when the pH falls below 6. Normally, the urine pH varies from 4 to 8. We postulate, that physiological changes in urine pH in combination with chromate which was added to the urine samples, produced the 'Rainbow urine' in our patient. Renal tubular dysfunction that was demonstrable in this patient probably resulted from chromium intake, which produces tubular necrosis [3] as well as chronic renal tubular dysfunction [4].

We suggest that tests for chromate pigments should be carried out whenever a factitious aetiology is suspected.

References

1. Fogazzi GB, Fenili D. Urine analysis and microscopy. In: Davidson AM, Cameron JS, Grunfeld J, Kerr DNS, Ritz E, Winearls CG, eds. *Oxford Textbook of Nephrology*, 2nd edn Oxford University Press, Oxford, 1998; 21–38
2. Cotton FA, Wilkinson G. Elements of the first transition series, In: Cotton FA, Wilkinson, E, eds. *Advanced Inorganic Chemistry*, 5th edn John Wiley, New York, 1988; 648–775
3. Schamahl FV. Investigations on the nephrotoxicity and hepatotoxicity of trivalent and hexavalent chromium compounds. *Int Arch Occup Environ Health* 1998; 71 [Suppl]: s40–45
4. Wang X, Qin Q, Xu X *et al*. Chromium-induced early changes in renal function among ferrochromium-producing workers. *Toxicology* 1994; 90: 93–101.

Ambika Gopalakrishna Unnikrishnan[1]
Simon Rajaratnam[1]
George Tharayil John[2]
Charles Stephen[3]
[1]*Department of Endocrinology*
[2]*Department of Nephrology*
[3]*Department of Biochemistry*
Christian Medical College and Hospital
Vellore
India