Other full case

An unusual case of renal tubular acidosis

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A 40-year-old female with a history of glue sniffing and intravenous drug use presented to hospital with a week's history of feeling generally unwell. She had had multiple admissions to hospital with similar presentations in the past. On examination, the only significant clinical finding was that of a reduced level of consciousness. Laboratory investigations revealed a hyperchloremic normal anion gap metabolic acidosis with a positive urine anion gap and a urine pH of 6.5 combined with a severely low hypokalaemia. She was subsequently diagnosed with renal tubular acidosis type 1, secondary to toluene exposure from glue sniffing and was treated with intravenous fluids and potassium replacement and discharged home with oral potassium citrate and advised to stop her inhalant use.

BACKGROUND

Renal tubular acidosis (RTA) is an unusual but an important cause of hyperchloremic normal anion gap metabolic acidosis – an essential diagnosis, which is commonly forgotten as a part of the differential, being a potentially treatable condition. While autoimmune causes of RTA are by far the most common causes, it is important to consider other less common causes such as drug-induced RTA, especially in patients who are known to be substance misusers. While, toluene exposure through glue sniffing is one of the classical causes of RTA 1. It is infrequently seen, but is a cause that can be identified through a thorough complete history and examination, which is seen in a classical presentation such as this.

CASE PRESENTATION

The patient is a 40-year-old lady who presented to the emergency room with a 2-week history of feeling generally unwell with non-specific complaints. She described a 2-week history of feeling generally unwell with progressive fatigue and a productive cough of white phlegm. There was no history of overt muscle weakness or myalgia, haemoptysis, chest pain, fever, night sweats, anorexia or weight loss. There was no history of nausea, vomiting or abdominal pain. Her bowel movements had been normal with no history of diarrhoea.

She also gave a history of frank haematuria during this same period of time with no other urinary symptoms. Prior to this current condition, she had been well with the exception of her previous hospital admissions.

The initiating factor for her presentation that day was an episode of loss of consciousness after admitting to an alcohol binge that morning. Apart from her recent alcohol ingestion, she denied co-ingestion of any other substances including glue sniffing, however, her partner confirmed that she chronically abuses solvents and had indeed been sniffing glue just prior to admission in the hospital. She had presented with similar symptoms, 3 months earlier to hospital, signs and laboratory findings, for which no cause had

been found. She had had multiple hospital admissions for glue sniffing in the past during which a metabolic acidosis had always been present.

On physical examination, she was found to be a thin, malnourished looking lady. Clinically, she was significantly dehydrated with an absent jugular venous pulse. Precordial examination revealed, normal S1/S2 heart sounds with no additional sounds. Examination of the chest revealed, good air entry bilaterally with no additional sounds. Abdominal examination revealed, soft non-tender abdomen with no organomegaly. The kidneys were not palpable. There was no costovertebral tenderness. No vascular bruits were heard on auscultation. Apart from a reduced level of consciousness, no other neurological deficits were found.

Her medical history consisted of alcohol-induced liver cirrhosis with portal hypertension and she was an ex-injecting drug user.

There was no significant family history to note.

At the time of her admission, she was living with her common-law partner and her two children on a native reserve and was unemployed. She was known to abuse alcohol and inhalants (glue) and was a known injecting drug user, even though she consistently denied any inhalant or glue use, or excessive alcohol use.

INVESTIGATIONS

An admission arterial blood gas done on oxygen revealed a metabolic acidosis with a pH of 7.23, PCO₂ 14 mm Hg, pO₂ 100 mm Hg, bicarbonate (HCO₃) of 10 mmol/l and a base excess of -20.9 mmol/l.

A (repeat) renal panel revealed, severe hypokalaemia (1.9 mmol/l) with a normal sodium and magnesium. Creatinine and urea were normal (79 mmol/l and 5.2 mmol/l, respectively). Phosphate was low (0.36 mmol/l). Corrected calcium was normal. Plasma glucose was normal.

The complete blood count revealed a normocytic anaemia (haemoglobin 90 g/dl, mean corpuscular volume 97.1 fl) with a thrombocytopenia (84×10⁹/l). White cell count was normal (6.4×10⁹/l).

Liver aminotransferases showed a raised aspartate aminotransferase (263 U/l) but normal alanine transaminase (89 U/l). Alkaline phosphatase was high (529 U/l), total bilirubin was 39 mcg/mol/dl and direct bilirubin was 30 mg/dl. Amylase was 55 U/l. Lactate dehydrogenase was 415 U/l, uric acid was 400 mg/dl. Troponin was slightly elevated (0.06 mcg/l). Creatine kinase was elevated (4871 U/l). Hepatitis screen for B and C was negative. HIV screen was negative.

A toxicology screen which included paracetamol and aspirin was also negative.

Urinalysis revealed a large amount of blood, and a small amount of protein (1+). The urine pH was 6.5. Urine glucose was negative. Urine microscopy was normal.

Urinary electrolytes were done which showed sodium of 42 mmol/l, potassium of 17.6 mmol/l, chloride of 44 mmol/l and urea of <16 mmol/l. Urine glucose could not be calculated due to limitations in the regional laboratory testing which prevented the urine osmolar gap from being calculated; however, the measured urine osmolality was 227. The calculated urine anion gap was positive (+15.6).

An ECG done on admission revealed a prolonged QTc only. The chest x-ray revealed an old T4 fracture and healed right clavicular fracture. CT imaging of her brain was normal, and a CT of her chest and abdomen only revealed the presence of varices and splenomegaly consistent with portal hypertension.

DIFFERENTIAL DIAGNOSIS

The list of differential diagnoses included:

- (1) Normal anion gap metabolic acidosis secondary to gastrointestinal losses, co-drug ingestions and uretral diversion.
- (2) Primary RTAs (1 and 2).
- (3) RTAs secondary to substance abuse/medication/nephrocalcinosis or autoimmune causes.

TREATMENT

On admission, she was fluid resuscitated with intravenous fluids, sodium bicarbonate infusion and potassium replacement in the form of potassium phosphate to replace the phosphate and potassium.

The ultimate goal for treatment for her type 1 RTA secondary to glue sniffing was for her to stop glue sniffing, which would normally resolve the RTA – however, this was not a viable option for this patient through her constant denial of glue sniffing. For patients who continue to sniff glue then the long-term therapy involves the continuous neutralisation of the retained renal acids using an alkali supplement such as potassium citrate, which would concomitantly replace the potassium while delivering sufficient alkali to neutralise the excess acid.

OUTCOME AND FOLLOW-UP

She consistently denied inhalant use despite her diagnosis and the need to stop. Before we had the opportunity to

perform further investigations regarding her RTA which would have included urinary fractional excretion of bicarbonate, she self-discharged home on potassium citrate replacement.

DISCUSSION

RTA 1 is due to a primary defect in the distal acidification of urine compared to the other forms of RTA, and can be distinguished from the other types by having a urine pH of greater than 5 which is usually associated with a severe hypokalaemia. Although it is not a common presentation, it is essential to consider it in the differential of any hyperchloremic normal anion gap metabolic acidosis as it is a treatable condition.

Case reports of patients presenting with toluene inhalant abuse most commonly present either with electrolyte abnormalities such as hypokalaemia¹ or hypophosphatemia as the presenting complaint, thus commonly mimicking other electrolyte disorders such as periodic hypokalaemic paralysis.¹ However, the presenting features are not limited to the above – one study of 16 patients presenting with toluene exposure had multiple presenting features including proteinuria and similar to our case, haematuria² and reported metabolic complications similar to that in our patient. There are no documented case reports reporting such a classical presentation as ours in a middle-aged lady presenting (recurrently) with a normal anion gap metabolic acidosis with a history of glue sniffing.

The metabolic acidosis that occurs may be as a consequence of the overproduction of hippuric acid³ and thus the acidosis should normally resolve with the cessation of toluene exposure (glue sniffing), however, if complications occur such as RTA 1, then this may indicate a long-term complication.

Learning points

- Consider toluene solvent abuse in a young person presenting with unexplained hyperchloremic normal anion gap metabolic acidosis
- Always consider RTA as a cause of a normal anion gap metabolic acidosis, especially if chronic in nature and when no obvious aetiology has been recognised
- A urine pH greater than 5 and hypokalaemia can help to distinguish between the different types of RTA 1.

Competing interests None.

Patient consent Obtained.

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