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Toxicology ECG

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Case history

A 35-year-old male non-smoker and non-alcoholic presented to the Emergency Department with 3 days h/o bilateral lower limb weakness and paresthesia. His past medical history was unremarkable.

On arrival to the emergency room, the patient was conscious, oriented, and afebrile. His blood pressure was 130/70 mm Hg with a heart rate of 62/min, respiratory rate of 18/min and room air saturation of 98%. His Cardiovascular, respiratory, and Abdomen examination were unremarkable. Neurological examination showed the power of 1/5 in bilateral lower limbs and 4/5 in both upper limbs. Deep tendon reflexes were reduced in all four limbs with intact sensation. His plantar was flexor.

His Initial blood investigations revealed normal hematology profile, renal and liver function. His Sodium, Potassium, Magnesium and Chloride were 132, 1.8, 0.61 and 97, mmol/L respectively., Blood gas showed a pH of 7.33, pCO2 of 40mmHg, HCO3 of 21mmol /L and Lactate of 2.6mmol/L. Blood glucose, Creatine kinase, and myoglobin were within normal limits.

ECG on admission (Figure 1.) showed Sinus rhythm with prolonged PR and QT interval, T- wave inversion, and U- wave in all leads and were attributable to hypokalemia.

Urinalysis showed urine pH as 5.3. The urinarySodium, Potassium, Chloride, and Creatinine were 38, 28, 57, and 14/mmol/L respectively. The Osmolality and UK/UCr of urine was 506 mmol/kg and 2 respectively. The urine osmolality was elevated but negative for ketones and proteins.

Clinical reasoning

- The positive findings were Hypokalemia with normal Anion gap metabolic acidosis and high urine osmolality with Kaliuresis. To identify the inciting pathology behind that, further history was elicited and there were no preceding gastrointestinal losses medication use, diabetes, dialysis, or malnutrition [8-10]. As renal function was normal, uremia could not be the reason for the presentation [8,9].

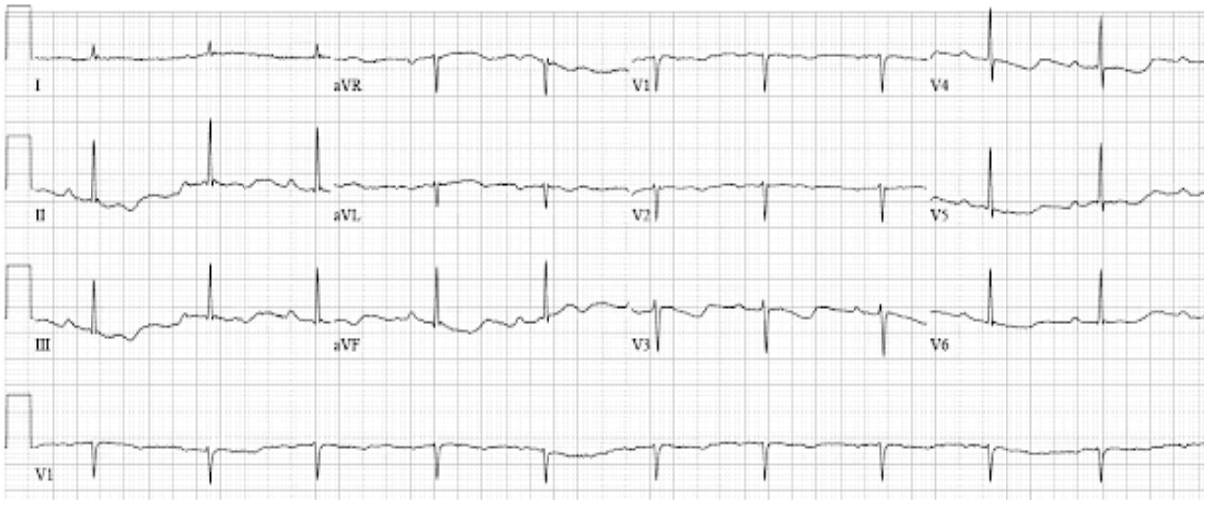

Figure. 1

- From the findings of urinary pH of 5.5 with systemic acidemia[1,10], the possibilities of Distal Renal Tubular acidosis (dRTA) probably due to Toxic ingestion of drugs contributing to exogenous acids [8,9] was considered.
- Methanol, ethylene glycol and, diethylene glycol are volatile alcohols that produce pure high anion gap metabolic acidosis from their metabolism into strong carboxylic acids such as formic acid (from methanol), and a combination of oxalic, glyoxylic, and glycolic acid (from ethylene/diethylene glycol) [4-6,8].
- Isopropanol alcohol poisoning contributes to an osmolar gap, but not high anion gap metabolic acidosis[7,8]. For these reasons, the possibility of Isopropanol alcohol poisoning was ruled out. So the remaining possibility was Hippuric acidosis which presents with a normal plasma anion gap with an elevated urinary osmolar gap due to rapid clearance of hippurate[2,3].

Hippuric acid is the metabolite of toluene. Toluene is a hydrophobic solvent found in paint thinners, cleaning agents, adhesives, and gasoline and is the most commonly abused volatile substance worldwide, owing to its euphoric effects. Absorption occurs from inhalation, ingestion, or transdermal exposure [2,3].

On further interrogation of his occupational history revealed him to be a painter of 5 months duration and used his hands to mix toluene in his paints without ant personal protective equipment.

We thus attributed his hypokalemia with features of acidemia, dRTA, high urinary Osmolality, and normal anion gap to ***chronic transdermal Toluene toxicity.***

References

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Table - 1: Causes of Metabolic acidosis [in this table please revise Note as Renal response and for ketoacidosis in renal response mention as ketones]

Clinical process		**Acid product**	**Note**
Toxic ingestion	Methanol	Formic acid	Metabolites highly toxic, renal excretion
	Ethylene glycol	Glycolic acid, glyoxylate, oxalate	
	Toluene	Hippuric acid	Renal excretion of metabolites
	Salicylate	Endogenous acid	Renal excretion; Urinary Alkalination markedly Increases renal excretion of free salicylate
	Paraldehyde	Acetic acid, chloroacetic acid	Renal excretion of metabolites
	Acetaminophen	5-oxoproline	Possibly due to glutathione Depletion; 5-oxoproline is Renally excreted
Lactic acidosis		Lactic acid (L – or D-)	
Ketoacidosis	Diabetic	Ketoacids, eg., β-hydroxybutyrate	
	Alcoholic	Ketoacids, eg., β-hydroxybutyrate	
	Starvation	Ketoacids, e.g., β-hydroxybutyrate	
Uremia/renal failure		From protein metabolism e.g., sulfate, phosphate, urate	In advanced CKD when glomerular filtration rate (GFR) falls below 15-20 ml/min, acids from protein metabolism are retained.

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