CASE REPORT

Altered mental status and complete heart block: an unusual presentation of aspirin toxicity

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SUMMARY

Aspirin is one of the most commonly used medications. We report a patient who presented with severe weakness, altered mental status and complete heart block requiring temporary pacing. Despite the patient's family denying that the patient used aspirin, an arterial blood gas that revealed a respiratory alkalosis and metabolic acidosis suggested the diagnosis of salicylate toxicity. The salicylate level was extremely elevated and the patient was successfully treated with haemodialysis. Our case illustrates that salicylate toxicity should be considered in a patient with a combined metabolic acidosis and respiratory alkalosis. A prompt salicylate level should be obtained. This is also the first case of salicylate toxicity causing complete heart block in an adult. The heart block resolved with treatment of the salicylate toxicity.

BACKGROUND

Chronic aspirin toxicity is a significant problem with a mortality as high as 25%.^{1 2} Failure of the patient to provide a history of salicylate use may delay the diagnosis. We report a case of chronic salicylate toxicity that was suggested by the arterial blood gas (ABG) analysis that revealed a metabolic acidosis with a concomitant respiratory alkalosis, which prompted us to obtain a salicylate level.

CASE PRESENTATION

A 77-year-old woman with a history of hypertension presented to our hospital with progressive lethargy and lower extremity weakness, which progressed over a 2-week period. Upon presentation, she appeared extremely somnolent with slurred speech, drooling, lower extremity weakness and difficulty breathing. The emergency department physician suspected an acute stroke, but a CT scan of the head was normal. Neurological consultation suggested myasthenia gravis, but a Tensilon test did not improve her neurological function. An ABG, with the patient breathing spontaneously, revealed pH 7.44, pCO₂ 13 mm Hg, pO₂ 195 mm Hg, HCO₃ 9 mmol/L, base deficit -12.1 mmol/L and lactic acid 0.9 mmol/L. This ABG is consistent with a primary metabolic acidosis and a primary respiratory alkalosis since the respiratory system cannot overcompensate for a metabolic acidosis and raise the pH to greater than 7.38. Critical care consultation suggested the diagnosis of salicylate toxicity, which was confirmed by a salicylate level of 76.1 mg/dL. She went into complete heart block and then developed asystole. She was intubated and the minute ventilation was titrated to maintain a

normal pH. She was resuscitated and a temporary transvenous pacemaker was placed with a ventricular rate of 60 beats/min. She was treated with a bicarbonate infusion with a target urinary pH of 7.8–8.5. Emergency dialysis decreased the salicylate level from 76.1 to 28.4 mg/dL. The bicarbonate infusion was continued, and the salicylate level on day 3 was 0.

On day 4 she was extubated and exhibited a normal mental status without evidence of heart block or weakness. She told us that she had been taking 30–40 tablets of 81 mg of aspirin daily for 2 months to relieve her generalised arthritic pain. She was not aware that aspirin could be toxic. She denied any history of tinnitus, dyspepsia or gastrointestinal distress (GI) distress. There was no history of arrhythmia or cardiac disease. She denied any suicidal ideation. She was discharged without any neurological or cardiac deficits and was counselled on the optimal use of aspirin.

DISCUSSION

Aspirin is one of the most commonly used medications worldwide. The incidence of chronic salicylate toxicity is not well-defined. The manifestations of salicylate toxicity may include tinnitus, mental status changes, tachycardia, tachypnoea, temperature dysregulation and GI irritation. Patients with mental status changes, primary metabolic acidosis and primary respiratory alkalosis should be worked up for possible salicylate toxicity by obtaining a salicylate level.³

One of the principal pathophysiological effects of salicylate toxicity is stimulation of the medullary respiratory centre leading to a respiratory alkalosis. It also causes uncoupling of oxidative phosphorylation that causes increased heat production, lactic acidosis and depletion of hepatic glycogen. Salicylate inhibits Krebs cycle enzymes, which can lead to an increase in organic acid. Salicylates may cause alterations in amino acid and lipid metabolism enhancing metabolic acidosis. Salicylates increase fluid and electrolyte losses leading to sodium and potassium depletion, dehydration and loss of buffer capacity. Salicylates are neurotoxins and can result in white matter damage, cerebral oedema and cerebral haemorrhage.⁴ Thus, the principal manifestations of salicylate toxicity include excessive heat production, respiratory alkalosis, metabolic acidosis, fluid and electrolyte losses and altered glucose levels.⁵

Salicylates can commonly cause tachycardia, but are a very rare cause of conduction abnormalities.⁶ To our knowledge there are only two cases of

To cite: Aggarwal N, Kupfer Y, Chawla K, et al. *BMJ Case Rep* Published online: [please include Day Month Year] doi:10.1136/bcr-2013-010083

conduction abnormalities caused by salicylates in adults and this is the first case, to our knowledge, of complete heart block in an adult.^{6 7} This case emphasises that aggressive treatment of the salicylate toxicity can result in a complete resolution of the conduction abnormality. Treatment primarily consists of hydration, alkalinisation of the urine with target pH between 7.8 and 8.5 and urgent haemodialysis for patients with hypotension, pH <7.2, central nervous system toxicity, pulmonary oedema and those requiring mechanical ventilatory support.⁸

Learning points

- ▶ The presence of a primary metabolic acidosis and primary respiratory alkalosis should prompt one to obtain a salicylate level.
- ▶ Urgent haemodialysis is lifesaving for severe salicylate toxicity.
- ▶ Patients should be informed regarding the safe use of salicylates.
- ▶ Complete heart block is an unusual cardiac manifestation of chronic salicylate toxicity.

Competing interests None.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

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