Clinical Toxicology

Haemorrhagic pancreatitis – a cause of death in severe potassium permanganate poisoning

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Summary: Severe potassium permanganate poisoning (more than 10 g of potassium permanganate) is invariably associated with massive systemic upset and death. Multiple organ damage has been recognized as an inevitable consequence of such an overdose, although pancreatitis has not been previously reported. Death due to cardiovascular collapse and profound hypotension is a common end point in those who reach hospital, but the pathogenesis is uncertain. We report a case of haemorrhagic pancreatitis following an overdose of potassium permanganate and suggest that this complication may be an unrecognized factor contributing to the extremely high mortality rate associated with this condition.

Introduction

Potassium permanganate has widespread topical use in conditions such as psoriasis and athletes' foot. This makes it readily available as an agent of attempted suicide and a frequent culprit in accidental consumption, typically by children.

When taken orally, potassium permanganate has been found to result in hepatic and renal damage.¹ The gastrointestinal tract is invariably damaged by the caustic nature of potassium permanganate, often resulting in perforation and haemorrhage.² Laryngeal oedema may cause life threatening upper airway obstruction,³ and haematological complications such as methaemoglobinaemia and haemolysis occur.⁴

Pancreatitis has not been previously recorded as a complication of potassium permanganate poisoning. We report a case of acute haemorrhagic pancreatitis and death resulting from ingestion of potassium permanganate.

Case report

A 75 year old man was admitted to hospital following an overdose of approximately 20 g of potassium permanganate, taken 2 hours before admission. The patient was happily married and had no previous psychiatric history.

Maturity onset diabetes mellitus was noted in his past medical history. On admission he was found to

have marked brawny discolouration of the buccal mucosa, and complained of severe dyspepsia, but his blood pressure, pulse and temperature were normal. His general condition at that stage seemed satisfactory. Gastric lavage was not undertaken and no specific therapy was administered. Four hours following admission there was a rise in respiratory rate to 65 cycles per minute, and a sinus tachycardia of 130 beats per minute. These findings were associated with a moderate rise in blood pressure from 140/80 to 170/90 mmHg. Blood gases revealed arterial hypoxaemia with type 2 respiratory failure (PaO_2 8 kPa; $PaCO_2$ 3.4 kPa on 35% O_2).

Intermittent positive pressure ventilation was initiated to secure the airway as laryngeal oedema was a likely complication. Subsequent radiographs of the chest revealed pulmonary infiltration which was compatible with adult respiratory distress syndrome. Parenteral antibiotics, hydrocortisone and histamine type 2 receptor antagonists were started. Haemoglobin, coagulation studies and platelet count remained normal, however a marked leucocytosis of $20 \times 10^9/l$ with a 90% neutrophilia developed. Methaemoglobin was slightly elevated at 4.7% (normal range 1–2%). Serum urea rose to 7.7 mmol/litre, serum osmolality was 330 mosmol/kg with a urine osmolality of 354 mosmol/kg, suggesting renal tubular dysfunction. Potassium and sodium levels were normal throughout. Non-haemolysed blood and protein were present in an acidic urine. Abnormalities of liver function suggesting hepatocellular damage were noted, aspartate transaminase (AST) was elevated at 461 U/l, bilirubin 121 μ mol/l, alkaline phosphatase was

normal. A diagnosis of acute pancreatitis was suspected because of the associated findings of severe abdominal pain, adult respiratory distress syndrome and hyperglycaemia. Serum glucose rose to 17.5 mmol/l and an amylase of 3752 U/l confirmed this diagnosis.

The electrocardiogram revealed first degree heart block and bifascicular block with a ventricular rate of 150 beats per minute. Despite full supportive measures there followed a progressive deterioration with cardiovascular collapse. Asystolic cardiac arrest followed profound refractory hypotension. Post-mortem findings confirmed severe superficial and deep ulceration of the gastrointestinal tract. There was pulmonary oedema and evidence of profound haemorrhagic pancreatitis.

Discussion

As a strong oxidizing agent, potassium permanganate is an intensely irritant substance once inside the gastrointestinal tract.⁵ Oxidant damage of the

intestinal mucosa occurs rapidly, leaving tell-tale brawny discolouration of the damaged tissues. This is followed by ulceration of the mucosa due to the formation of potassium hydroxide.^{6,7}

Widespread visceral damage is also characteristic of potassium permanganate poisoning, suggesting the toxic effects are not confined to corrosion of the gastrointestinal tract.⁸ Renal, hepatic and haematological involvement is well documented, and adult respiratory distress syndrome often develops. Cardiovascular collapse with hypotensive shock is often the mode of death in those who do not die from respiratory obstruction of massive gastrointestinal haemorrhage. The pathogenesis of the cardiovascular collapse associated with potassium permanganate poisoning is uncertain.

We propose that the development of acute haemorrhagic pancreatitis may well be the underlying disorder in these patients who develop such widespread visceral damage. Previous cases have not recorded amylase levels. It is possible that this serious complication of potassium permanganate poisoning has been overlooked.

References

1. Arthur, C. & Huntley, M.D. Oral ingestion of potassium permanganate or aluminium acetate in two patients. *Arch Dermatol* 1984, **120**: 1363–1365.
2. Dreisbach, R.H. *Handbook of Poisoning: Prevention, Diagnosis and Treatment*, 11th edition. Lange Medical Publications, California, 1980, Chap. 29, p. 423.
3. Gilman, A.G., Goodman, L.S. & Gilman, A. (eds) *The Pharmacological Basis of Therapeutics*, 6th edition. Macmillan, New York, 1980, pp. 974–975.
4. Mahomedy, M.C., Mahomedy, Y.H., Canham, P.A.S., Downing, J.W. & Jeal, D.F. Methaemoglobinaemia following treatment dispensed by witch doctors. *Anaesthesia* 1975, **30**: 190–193.
5. Jetter, W.W. & Hunter, F.T. Death from attempted abortion with a potassium permanganate douche. *N Engl Med J* 1949, **240**: 794–798.
6. Willimot, S.G. & Freiman, M. Potassium permanganate poisoning. *Br Med J* 1936, **1**: 58.
7. Johnston, C. Potassium permanganate poisoning: report of a fatal case. *South Med J* 1937, **30**: 1030.
8. Southwood, T., Lamb, C.M. & Freeman, J. Ingestion of potassium permanganate crystals by a three year old boy. *Med J Aust* 1987, **146**: 639–640.