Sodium nitrite: not your cure-all remedy

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Background

Sodium nitrite has a variety of uses, its main use is in the treatment of cyanide poisoning in conjunction with sodium thiosulfate. In cyanide poisoning, sodium nitrite reacts with haemoglobin to form methaemoglobin which then protects cytochrome oxidase activity from cyanide ions.[1, 2] Sodium nitrite is also used as a food additive in cured meats, primarily to prevent the growth of *Clostridium botulinium.*[3] It can easily be purchased online.

Objective

To describe an admission following a suicide attempt with an intentional ingestion of sodium nitrite and to highlight a pharmacists role in providing specialist medication knowledge in a complex case with acute kidney injury (AKI).

Clinical features

A 52-year-old Caucasian male was transported to hospital via ambulance after being found at home by family, unconscious and slumped awkwardly for approximately 24 hours.

Observations on admission

BP 140/109, HR 109,
GCS 11, Temp 37.3, CK 62734,
Troponin 2739, creatinine 299, eGFR 20

Past medical history

Schizophrenia, previously treated with Electro Convulsive Therapy (ECT)
Obsessive compulsive disorder
Depression
Hypertension
Bells Palsy

Medications on admission

Amisulpride 400mg nocte
Paroxetine 20mg mane
Perindopril 10mg nocte
Clonazepam 1.5mg nocte

On day 2 the patient divulged purchasing sodium nitrite online and self-administering after reading a website dedicated to euthanasia. Psychiatry reviewed and withheld his regular psychiatric medications. Methaemaglobin was found to be 2.5% (reference range 0%-1%). Toxicology was consulted and felt that sodium nitrite was not the cause of his organ dysfunction.

New onset chest pain and further troponin rise (from 2739 to 21337) with ECG showed T wave inversion. An echo and Sestambi study performed the following day showed evidence of a prior infarction. Cardiology reviewed and recommended to treat with dual antiplatelets, clopidogrel and aspirin, and to commence a statin on discharge.

Creatinine was continuing to rise and he became anuric despite fluids and high doses of frusemide.

Haemodialysis was started on day 4 to treat his acute kidney injury (AKI) from haem pigment nephropathy.cause by his long lie prior to admission.

Figure 1. Photograph of sodium nitrite purchased by patient.

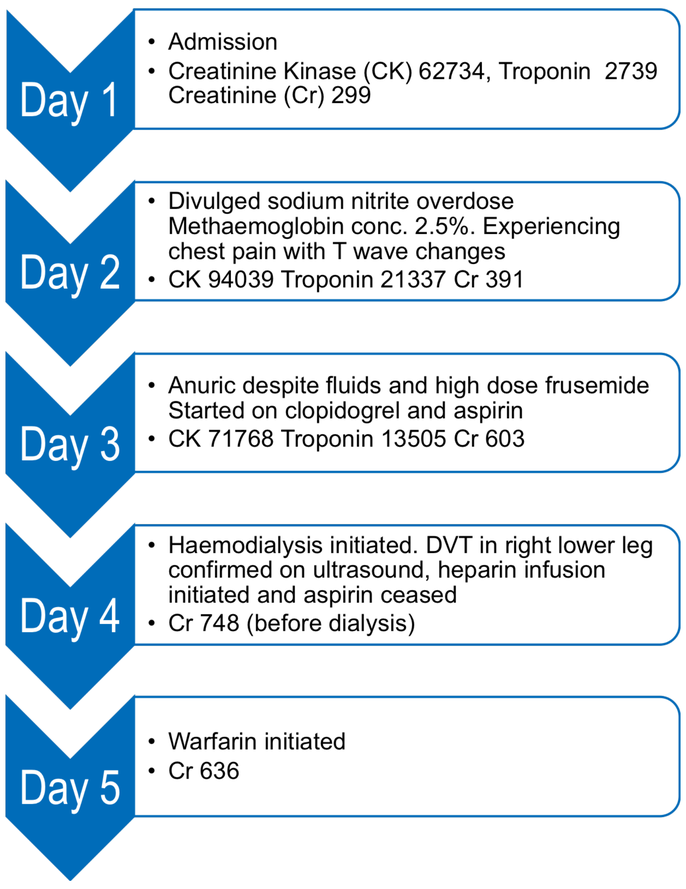

Figure 2. Flowchart of patient's progress during the early stages of admission.

Clinical features (cont)

On day 4, a deep vein thrombosis (DVT) was confirmed on ultrasound and a heparin infusion was commenced. It was decided to cease aspirin and treat with clopidogrel and warfarin.

The pharmacist was consulted to advise on dosing of psychiatric medications and provide warfarin counselling. In the context of AKI and haemodialysis it was recommended to initiate medications one at a time. Clonazepam was initiated first due to agitation at a reduced dose of 500microg nocte. Amisulpride is poorly dialysed and eliminated unchanged in the urine. Minimum dosages are recommended in haemodialysis.[4] Due to this it was recommended to restart at amisulpride 100mg nocte. Paroxetine is not dialysed and recommended dosing is 20mg daily with review.[4] Due to this paroxetine was dosed very conservatively and restarted at 10mg mane and patient closely monitored.

On day 17 the patient was unable to retain warfarin counselling information on sequential days. A Montreal Cognitive Assessment (MoCA) was performed, scoring 13/30, with poor executive, visuospatial and verbal fluency. It was unclear if this was premorbid, but it was felt past history of ECT could have contributed.

Clinical features (cont)

The pharmacist raised new considerations to the psychiatry and renal teams of concern with understanding and ability to safely manage warfarin, and risks if another suicide attempt was to occur.

The patient had a long inpatient stay including rehabilitation, remaining on warfarin while an inpatient and the risks of harm were low. The warfarin was eventually ceased prior to discharge home.

Discussion

In sodium nitrite overdose, methaemoglobin causes an acute impairment in oxygen delivery (as ferric iron in red blood cells is oxidised to the ferric form) to tissues that does not allow sufficient time for compensatory mechanisms to take place.[5]

Early symptoms of an overdose include cyanosis with pale, grey or blue coloured skin, lips and nail beds, lightheadedness, headache, tachycardia, fatigue, dyspnoea and lethargy. Higher methaemoglobin levels (methaemoglobin concentration >30%) causes respiratory depression, altered sensorium, coma, shock, seizure and death.[5]

Initial treatment is with oxygen and intravenous fluids, whilst symptomatic patients with methaemoglobin concentration >20% and asymptomatic patients with methaemoglobin > 30% are treated with methylene blue.[6]

Haem pigment nephropathy is a type of AKI that can occur in patients with rhabdomyolysis after myoglobin is released from muscle. The myoglobin is then filtered in the glomerulus where the haem pigment is released. Haem pigment may injure the kidney by tubular obstruction, direct proximal tubular endothelial cell injury or by vasoconstriction.[7]

Conclusion

Sodium nitrite is easily obtained and ingestion can result in tissue hypoxia, which in this patient indirectly resulted in AKI. Pharmacist input can ensure medications are dosed appropriately and safely in patients with AKI.

Acknowledgement

Case presented with permission of
Dr Emmanuel D'Almeida, Renal Physician, John Hunter Hospital

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