Clinical Toxicology

Haemolytic anaemia associated with ingestion of naphthalene-containing anointing oil

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Summary: We present a patient who developed a severe haemolytic anaemia as a result of ingestion of a naphthalene-containing anointing oil. Previous reports of naphthalene-induced haemolytic anaemia are reviewed. Predisposing factors to haemolysis are outlined and the variability in the haematological response to naphthalene is discussed. It is clear that ingestion of anointing oil is dangerous; even topically, significant absorption of naphthalene may occur especially in infants, as it is oil-based. Because of these dangers, the use of naphthalene-containing anointing oils should be strongly condemned.

Introduction

Haemolytic anaemia secondary to naphthalene toxicity is well documented, but its use has largely been abandoned. The more common source is mothballs which are either ingested, 1-4 absorbed through the skin or inhaled from stored clothes. 5, 6 Other sources described include lavatory bowl cleaners and nappy deodorizers. 7, 8 The original reported cases of naphthalene intoxication were secondary to treatment either of typhoid fever or helminthic infection following its introduction for treatment of these conditions in the 1840s. 9-12 We present a patient who developed a haemolytic anaemia following the non-accidental ingestion of a naphthalene-containing oil, usually used for anointing, acquired from a Nigerian church.

Case report

A 30 year old Nigerian housewife was brought to casualty having fainted in the street. She had returned from a 3 month holiday in Nigeria 6 days previously. Four days before admission she had drunk approximately 50 ml of an anointing oil that she had acquired from an Aladura church in Nigeria. Her sister had drunk a slightly smaller quantity of the oil. Six hours after ingestion the patient developed central colicky abdominal pain and profuse diarrhoea which lasted 2 days. The day before admission she had noticed a red discoloration of her urine and had felt generally weak. She gave no family history of thalassaemia or sickle cell disease. Her sister had also developed abdominal pain and diarrhoea after drinking the oil, which resolved within 24 hours with no other symptoms.

Examination of the patient revealed an anaemic, pyrexial (37.5°C) but otherwise well-looking Nigerian woman, with no jaundice, lymphadenopathy or hepatosplenomegaly. Stick testing of urine was negative to urobilinogen and positive to blood but microscopy showed no red blood cells.

Investigations on admission revealed a haemoglobin of 8.4 g/dl, MCV 91 fl, white cell count of 10.4 x 10⁹/l, platelets 282 x 10⁹/l and a reticulocyte life count of 11%. No Heinz bodies were demonstrated. The blood film showed irregularly contracted red blood cells and polychromasia with no malarial parasites on thick films. Further investigations revealed sickle cell trait (haemoglobin A + S) and evidence of intravascular haemolysis. Serum haptoglobin was undetectable and Schumm's test was positive. Glucose phosphate dehydrogenase levels were normal and both the direct antiglobulin test and isopropanol stability test were negative.

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The patient's sister's blood picture 5 days after drinking the oil showed a haemoglobin of 11.5 g/dl, MCV of 87 fl, white cell count of 5.4 x 10^9/l and reticulocyte count of 4%. The film showed polychromasia and occasional irregularly contracted red cells.

Analysis of the oil by ultraviolet spectrophotometry at New Cross Poisons Unit showed it to contain a high concentration of naphthalene.

During the patient's 5-day admission her pyrexia resolved. Her haemoglobin increased to 9.2 g/dl and her reticulocyte count to 15%. She was discharged and did not attend her follow-up appointment.

Discussion

We believe that this is the first reported case of haemolytic anaemia occurring after the non-accidental ingestion of an anointing oil containing naphthalene.

Naphthalene is well absorbed following oral exposure; it can also be absorbed through the skin or following inhalation. It is metabolized in the liver to alpha and beta naphth and alpha and beta naphtholoquinone. Alpha-naphth possesses potent haemolytic properties unlike the parent compound. Naphthalene was introduced by Rossbach as an internal antiseptic for typhoid fever in 1841 and as an antihelminthic in 1842 and the majority of early cases were the result of intoxication secondary to this. Nowadays mothballs are most commonly incriminated and acute toxicity has been reported after accidental ingestion in children suicide attempts and even in a case of murder. Infants' clothes and blankets stored in mothballs are another well described source of naphthalene that has resulted in haemolytic anaemia in babies.

In the patient we report the source of naphthalene was an anointing oil. This had been acquired from the Church of the Lord, one of the 960 denominations found in Nigeria. The patient and her sister had been advised to drink the substance by a relative, though she agreed in retrospect that the oil was probably for anointing rather than ingestion.

This patient shows the typical clinical and haematological features found in naphthalene intoxication. After ingestion, vomiting, diarrhoea and fever appear in 1 to 2 days followed on the third to fifth day by an acute haemolytic crisis as evidenced by pallor, mild jaundice and pigmented urine. Five to six days after ingestion the haemolytic process ends and if the patient survives this stage, then recovery is rapid.

Haematological changes occurring as early as one day after initial exposure in severe cases lead to a Heinz-body haemolytic anaemia and a sharp fall in haemoglobin. There is often a concurrent leucocytosis. Reticulocytosis follows with subsequent gradual restoration of the normal blood values except in the most severe cases. Haemoglobinuria and methaemoglobinaemia may also occur.

Although naphthalene is a known cause of haemolytic anaemia, the factors that predispose patients to the serious effects of toxicity are less clear. Newborns have thinner skins and are often subject to the application of baby oil which promotes dermal absorption. In addition they are unable to conjugate naphthalene metabolites effectively and are therefore more susceptible to haemolysis. Patients with glucose-6-phosphate dehydrogenase deficiency are also more susceptible to haemolysis. Valaes et al. in 1963 reported 21 neonates who developed haemolysis after exposure to naphthalene, 12 of whom were found to have glucose-6-phosphate dehydrogenase deficiency.

Besides these variables, there are also unexplained differences in the haematological response to naphthalene exposure; Gidron & Leuren reported two cases, one of whom ingested 6 g leading to significant toxicity, while the other who ingested 10 g had no symptoms. This appears to have been the case in the two sisters reported here and may partly be explained by differing absorption, the presence of a fatty meal significantly increasing absorption because naphthalene is lipophilic or differing efficiency of detoxification in the liver.

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References

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