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CASE REPORT

R. Ewert · F. Buttgereit · M. Prügel · P. Reinke

Intravenous injection of India ink with suicidal intent

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Abstract We describe the case of a 33-year-old man who injected 4 ml of India ink into one of the median cubital veins with suicidal intent. He was hospitalized in good general condition 10 h after the injection. Abnormal laboratory test results were a leukocytosis, an oximetrically determined methemoglobin level of 36.9% (normal range: 1.5%) and a free hemoglobin level of 74 μmol/L (normal range: < 25 μmol/L). Toxicological examination showed the presence of nitrobenzene in blood and urine. Intravenous administration of vitamin C and tolonium chloride plus forced diuresis led to an improvement in cyanosis and a fall in the methemoglobin concentration. Repeated increase in the concentration of aminobenzene were successfully treated by hemodialysis with a high-flux dialyzer.

Key words Suicidal injection · Methemoglobin · Nitrobenzene · Hemodialysis

Introduction

Throughout the world large amounts of nitro and amino derivates of aromatic hydrocabons are used in the chemical industry. Among other uses they serve as intermediary products and solvents in the dyestuff industry. Despite the extensive use of synthetic dyes reports of toxicologically significant acute damage due to them are rare [1, 2]. Their potential for inducing toxic effects in humans is due to the many basic substances, intermediary products, inorganic pigments and solvents that they contain [3, 4]. Most nitro and amino compounds used as solvents for dyestuffs are hemoglobin poisons, therefore the clinical picture that results from their incorporation into the body is marked by methemoglobin formation and direct substrate effects [4–8]. We report here on a case of a patient who gave himself an intravenous injection of India ink with suicidal intent.

R. Ewert (✉)
Deutsches Herzzentrum Berlin, Augustenburger Platz 1,
D-13353 Berlin, Germany
Fax: +49 (30) 45 93 2143

F. Buttgereit · P. Reinke
Department of Internal Medicine, Humboldt University,
Schumannstrasse 20/21, D-10117 Berlin, Germany

M. Prügel
Department of Legal Medicine, Humboldt University,
Schumannstrasse 20/21, D-10117 Berlin, Germany

Case report

A 33-year-old man injected 4 ml of India ink ("Hausleiter-Folientusche", product no. 675, Hausleiter, Munich, Germany) into one of the median cubital veins with suicidal intent. Some hours later he woke up with a headache, noticed that he felt physically weak and 3-4 h later developed a muscular tremor, a feeling of heightened sensory perceptions and a fear of impending death. Around 10 h after the injection he was hospitalized in good general condition (blood pressure 150/90 mmHg, pulse rate 100/min). He was fully orientated and cooperative but had blue-grey cyanosis that was most pronounced in the extremities. Abnormal laboratory test results were a leukocytosis of 16.1×10^9/L (normal range: 3.8–9.8×10^9/L), an oximetrically determined methemoglobin level of 36.9% (normal range: < 1.5%), and a free hemoglobin level of 74 μmol/L (normal range: < 25 μmol/L). Toxicological examination showed the presence of nitrobenzene in blood (1.1 μg/mL) and urine (0.06 μg/mL). Intravenous administration of 1000 mg vitamin C (Ascorvit) and 300 mg tolonium chloride (Toluidinblau) plus forced diuresis (400 mL urine/h) led to an improvement in the cyanosis and a fall in the methemoglobin concentration. Repeated increase in the concentration of aminobenzene was successfully treated by hemodialysis with a high-flux dialyzer (Filtral 16, Hospal). Transient slight rises in the levels of bilirubin, lactate dehydrogenase and free hemoglobin (without evidence of anemia) subsided wthin a few days. After 12 days the patient was discharged to outpatient follow-up.

Methods of aminobenzene and nitrobenzene detection

Aminobenzene and nitrobenzene were measured using a high-pressure liquid chromatography (liquid chromatograph No.6, Shimadzu Comp., Japan with nucleosil-C8-column, 250 × 4 mm, 7 μ). For analysis we used an acetonitrile/phosphate, buffer pH 2,3 (37:63 v/v) with a flow rate of 1 ml/min. The blood samples (500 μl) were extracted with 400 μl methylenchloride.

Discussion

Good cooperation by the patient enabled rapid identification of the toxicologically relevant substances. The India ink product that was injected contains 59.5% nitrobenzene as a solvent. This nitro compound undergoes oxidation to p-nitrophenol and is metabolized by reduction to aminobenzene. Aminobenzene is oxidized by microsomal cytochrome P450-dependent reactions to instable phenylhydroxyamine with is then oxidized in the presence of oxygen to nitrosobenzene. This oxidizes hemoglobin to methemoglobin. Both substances and their metabolites damage erythrocytes by attacking the porphyrin ring and erythrocytic proteins. There may also be direct substrate actions on the CNS manifested as a variety of euphoric to apoplectiform pictures, impairment of consciousness and even convulsions [3–5]. The cyanosis, headache and mental phenomena seen in our patient thus fall within the clinical picture of intoxication. Significant findings are present when 20–40% of the patient's hemoglobin has been altered [9]. Treatment with toluidine blue (rapid reduction of methemoglobin to hemoglobin) is recommended, ascobin acid also being effective as an adjuvant. This regimen was effective in our patient.

Nitrobenzene and its intermediary products are eliminated via the lungs and kidneys partly unchanged and partly after reduction and conjugation [10]. The half-life of these substances is between 2 and 20 days [11] and elimination of aromatic amines can be accelerated by forced diuresis and hemodialysis. We used a dialyzer with an effective surface area of 1.7 m^2 and an ultrafiltration coefficient of 69 mL/h × mm Hg. Dialysis over 3 h brought about a significant reduction in aminobenzene concentration (Fig. 1). Toxicological monitoring permitted appropriate treatment in response to rising aminobenzene levels.

Our search of the literature revealed no other case report of intravenous administration of a dyestuff with a high concentration of nitrobenzene. The only comparable case reports are therefore of intoxication with aromatic nitro compounds incorporated into the body via inhalation, ingestion, or absorption through the skin. The manifestations of such an intoxication are secondary to local irritation at the entry site and to methemoglobin formation [6–8, 12]. The amount of hemolysis is generally clinically insignificant. In the mass intoxication with aminobenzene-denatured rapseed oil that occurred in Spain in 1981 the specific effects of aminobenzene paled into insignificance beside the toxic oil syndrome [13].

As comparably rare as our case of intravenous injection with suicidal intent are e.g. amniotic embolism [14], intoxication with para-chloraniline in premature neonates [15], ingestion of organophosphate [16] and all these cases imply new ideas and arrange special experiences in the daily treatment of patients especially in the emergency room.

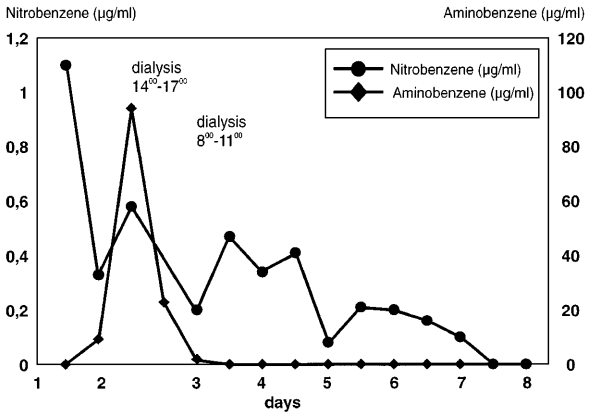

Fig. 1 Concentrations of aminobenzene and nitrobenzene during the first eight days

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