MYOGLOBINURIA FOLLOWING THE USE OF SUXAMETHONIUM CHLORIDE

A Case Report

BY

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SUMMARY

A case is described of myoglobinuria following general anaesthesia in which suxamethonium chloride was used as a relaxant.

CASE REPORT

A male child aged 7, weighing 70 lb. (32 kg) was admitted to hospital for the removal of tonsils and adenoids. The operation had been advised by the physicians following a severe attack of acute glomerulonephritis sixteen months earlier.

On admission there was nothing in the history obtained from the parents to indicate any previous illness other than the usual childhood ailments. No abnormality could be detected on physical examination. Routine examination of the urine revealed no abnormal constituents.

The premedication consisted of papaveretum 10 mg, hyoscine 0.2 mg, and methylpentynol 750 mg given orally, 1½ hours pre-operatively.

Anaesthesia was induced with thiopentone (2.5 per cent) 75 mg, followed immediately by suxamethonium chloride 50 mg. No muscle fasciculations were observed, and it was noted that there was no relaxation of the jaw musculature, so that oral intubation was not possible. (In the pre-operative examination the child had been able to open his mouth normally.)

A further dose of suxamethonium (50 mg) was given; there were no fasciculations following this dose of suxamethonium; however, the resultant relaxation was just about good enough to allow oral intubation. It was noted at the time of the second injection of suxamethonium chloride that there was a marked generalized myotonia.

Anaesthesia was maintained using nitrous oxide (4 l./min), oxygen (2 l./min) and halothane from a Fluotec vaporizer, through a Jackson Rees modification of the Ayre T-piece circuit. The concentration of halothane used was 1½-2 per cent. There was some delay in the return of spontaneous respiration but it was considered to be adequate after 18 minutes. The concentration of halothane in the anaesthetic mixture was maintained at this relatively high level but, despite this, it was found impossible to obtain sufficient relaxation of the jaw to permit the introduction of the Boyle Davis gag; the operation was therefore abandoned.

Nothing untoward was noted during the recovery phase; the patient's jaw was X-rayed and the radiologist reported normal temperomandibular joints. The myotonia completely disappeared on recovery from the anaesthetic but, some 8 hours later, the child had what appeared to be gross haematuria. All subsequent urine specimens (over a 48-hr period) were examined and found to be completely normal.

RESULTS OF INVESTIGATIONS

First urine sample (8 hours after anaesthesia): red-coloured urine; pH, amphoteric; protein, ++++; reducing substances, nil; ketones, ++; Urobilinogen (Schiesinger's method), weakly positive; porphyrins (Ehrlich's screening test), negative; spectroscopy, absorption bands indicating haemoglobin derivatives; centrifuged deposit, no red cells.

The urine pigment was shown to be composed of myohaemoglobin and haemoglobin.

Spectrophotometry confirmed the presence of myoglobin.

Examination of next specimen of urine (early morning): urine, pale straw; pH, acid to litmus; protein, nil; reducing substances, nil; ketones, nil; urobilinogen, nil; porphyrins, nil; spectroscopy, no absorption bands seen; tests for myoglobin negative; centrifuged deposit, no red blood cells or casts seen.

Blood investigations.

Hb, 86 per cent; 12.5 g/100 ml; W.B.C. 9000 cu. mm; diff. count, polymorphs, 48 per cent; lymphocytes, 49 per cent; monocytes, 3 per cent; reticulocytes, less than 1 per cent.

Film showed normal morphology.

Schumms test for methaemalbumin, negative; blood urea, 21 mg/100 ml; serum bilirubin, less than 0.2.

No evidence of intravascular haemolysis was discovered.

The results show that the period of myoglobinuria was very brief (only detected in the initial urine sample) demonstrating an extremely short period of rhabdomyolysis.

DISCUSSION

Myoglobin is a haemoprotein resembling haemoglobin but has a molecular weight of 17,200, compared with 68,000 for haemoglobin. It is distributed throughout the skeletal musculature, and is also found in the myocardium. Approximately 3 per cent of total muscle protein is myoglobin. It combines rapidly with oxygen at low tensions: at an oxygen tension of 40 mm Hg,
myoglobin is 94 per cent saturated; it will desaturate quickly when the oxygen tension falls below 20 mm Hg. It is not surprising, therefore, that myoglobin should be described as a primitive respiratory pigment (Millikan, 1939).

The role of myoglobin in the body is not fully understood but it is thought to act as a reservoir for the supply of oxygen to muscle groups during brief periods when the local oxygen tension falls. When liberated from the cells into the extravascular compartment, myoglobin is rapidly excreted in the urine. Myoglobinuria has been interpreted as a sign of severe muscle necrosis (Adams, Brown and Pearson, 1962) but the underlying factor is probably a metabolic fault in another vital constituent of the muscle fibre.

The commonest causes of the condition are muscle trauma, e.g. crushing injuries (Biorck, 1949), rare muscle glycogen diseases such as McArdle's disease (McArdle, 1951), and myoglobinuria polymyositis. Myoglobinuria has also been reported following electric shock (Fischler and Rossier, 1947). Haff disease was thought to be endemic toxic myoglobinuria but may be due to a viral infection (Hed, 1955).

It has also been suggested that the muscle fibres could be damaged during the uncoordinated muscle fasciculations produced by suxamethonium chloride depolarization (Paton, 1959) but a microscopic examination of thoracic and abdominal muscles in patients who complained of pain following the use of suxamethonium did not show any histological changes. A remarkable increase in the postoperative levels of serum creatine kinase and myoglobin was found in twenty-four patients who gave no previous history of muscle disease (Airaksinen and Tammisto, 1966). It was pointed out that this level of serum creatine kinase could not occur without damage to muscle cells. It must be assumed, therefore, that there is some protein transport through the apparently normal cell membrane, the damage not being anatomically demonstrable (Bennett, 1965).

Bennike and Jarnum (1964) reported that a single dose of suxamethonium chloride in a patient suffering from idiopathic myoglobinuria induced an attack of myoglobinuria.

It has also been reported that rigidity following the administration of suxamethonium chloride was associated with hyperpyrexia and death of the patient (Thut and Davenport, 1966). Convulsions and myotonia-like rigidity after suxamethonium chloride, followed by ventilatory difficulties and the death of experimental animals, have also been reported (Hall et al., 1966).

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REFERENCES


MYOGLOBINURIE APRES EMPLOI DE CHLORURE DE SUXAMETHONIUM: DESCRIPTION D'UN CAS

SOMMAIRE

Un cas de myoglobinurie, consécutif à une anesthésie générale avec administration de chlorure de suxamethonium comme relâchant, est décrit.

MYOGLOBINURIE IM ANSCHLUSS AN DIE VERWENDUNG VON SUXAMETHONIUM-CHLORID: EINE FALLBESCHREIBUNG

ZUSAMMENFASSUNG

Es wird ein Fall beschrieben, bei dem im Anschluß an eine Narkose, bei der als Relaxans Suxamethoniumchlorid verwendet worden war, eine Myoglobinurie festgestellt wurde.