Sodium Nitroprusside Toxicity

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Summary

A young patient who suffered visceral leishmaniasis and received treatment with sodium stibogluconate had to have a mastoid operation seven years later. A hypotensive technique employing sodium nitroprusside had to be stopped due to excessive hypotension and tachyarrythmias.

Introduction

A healthy 30 year old man weighing 60 kilos had suffered from chronic left ear discharge and was persuaded to undergo a mastoidectomy. He gave a history of having recovered from a bad bout of visceral leishmaniasis seven years previously. During the general anaesthesia preparation haemoglobin, blood urea, serum electrolytes were normal. A chest X-Ray and ECG were all within normal limits.

Premedication with Atropine 0.6mg, fentanyl 0.1mg and droperidol 5mg was given intravenously twenty minutes before the start of the operation through the infusion tubing of a Ringer lactate solution.

The operation

When placed on the operating table the blood pressure read 130/90 torr and the pulse was steady at 60 per minute. A calculated dose of 300mg Thiopentone sodium and 60mg Suxamethonium chloride was slowly infused while preoxygenation was performed. Following lignocaine 4% spray to the pharynx and vocal cords intubation was performed without any detectable change in the E.C.G.

The patient was connected to a supply of Oxygen and Nitrous oxide and Halothane 2% and kept on manual ventilation till the suxamethonium effect was reversed.

When the patient was breathing on his own an infusion of 5% dextrose with sodium nitroprusside 1.5 mgn/kg/per minute[1] was connected to the previous infusion through a Y piece.

After 50 centilitres of the second solution had run in, cyanosis was observed, extreme tachyarrythmias appeared on ECG and systolic blood pressure was hardly 60 torr. No obstacles in air passages were observed. Immediately the infusion of sodium nitroprusside was stopped. Halothane was shut off[2]. The haemodynamic values returned to normal. Pancuronium was now given 0.1mg/kg and the lungs ventilated artificially with 50% nitrious oxide in oxygen. Analgesia was maintained with doses of Fentanyl. Thirty minutes later, to lower bleeding in operating field, sodium nitroprusside was again connected. Cyanosis and haemodynamic changes mentioned above started again. After this event sodium nitroprusside was abolished and operation finished without any problems.

Discussion

Sodium nitroprusside Na_2 (Fe/CN/$_5$NO). $2H_20$ is a powerful hypotensive agent[3][4][5]. Its mechanism of action probably depends on the "FeNO" group, but during his subsequent breakdown significant quantities of cyanide appear in the blood[6]. One of the free CN^- ions joins the methaemoglobin to become cyanmethamoglobin. The remaining four CN^- groups are eventually detoxified into thiocyanate. This reaction requires a liver enzyme a sulfuryl transferase termed rhodanese and a sulfur donor, often thiosulfate[7] . There is ample evidence that rhodanese is present in abundance and that sufur donor availability limits the rate of cynide detoxification. The resultant compound, thiocyanate, is much less toxic than cyanide. The activity of cytochrome B, one of the enzyme necessary for 0_2 transport of cellular levels is impaired and may progress to severe cellular damage manifested as metabolic acidosis, cardiac arrest is possible if this condition is not recognised[8].

Sodium stibogluconate, a non essential metal, includes antimony compound which probably acts

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by inhibiting the enzyme in parasites. They combine to form a ring with thio-SH groups of essential enzymes. 73% of total single does is excreted in four weeks. One year after the treatment the levels of stibogluconate remain increased. There is a rise in blood 6.7 ug/1 and in urine 27.6 ug/1. Levels in controls not so treated are 3.4 and 6.2 ug/1. The drug is mainly found in tissues of liver, kidney and lungs. Liver function test and ECG pattern remain effected for a long time[9].

References

1. **D. Oliviera M., Sykes M.K., Chakrabarti M., Kaslin J. and Orchard C.** Depression of hypoxic pulmonary casoconstriction by sodium nitroprusside and nitroglycerine.
British Journal of Anaesthesia 1981, 53:11
2. **Burch G.E.** Cardiomyopathy. Philadelphia 1972.
3. **Bisset W.J.K., Butler A.R. Glidewell C. and Reglinski J.** Sodium nitroprusside and cyanide release: reason for re-appraisal.
British Journal of Anaesthesia 1981, 53: 1015-1019.
4. **Gustafson C.** The central and perifpheral circulation during and after sodium nitroprusside induced hypotension in the rat.
British Journal of Anaesthesia 1984, 56:1029.
5. **Meyer L.** Side effect of drugs. Excerpta medicl fundation. 1964 p. 111.
6. **Laurence D. R.** Clinical Pharmacology. London 1973 p.31.
7. **Bridges J.W.,** Chasseaud 1. F. Progress in Drugs Metabolism. London 1976.
8. **Tinker J., Rapin M.,** Care of the Critically ill patient. London 1983.
9. **Friberg L., Wordberg G.F., Vonk V.B.** Handbook of the toxicology of Metal.