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CASE REPORT

HALOTHANE INDUCED REVERSIBLE MUSCLE RIGIDITY -- A PRECURSOR OF MALIGNANT HYPERTHERMIA?

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ABSTRACT

A case report of muscular rigidity triggered by halothane is presented. It was observed in a young patient with multiple drug allergies. The rigidity disappeared on switching off halothane. It is concluded that this muscular rigidity was probably an early stage of malignant hyperthermia [MH]. But it came up gradually, was clearly linked to halothane use and finished slowly when the causative agent was discontinued.

INTRODUCTION

Malignant hyperthermia is a nightmare for an anaesthesiologist. Proper preanaesthetic assessment is mandatory to get some relevant clues regarding the probability of this syndrome. The practicing anaesthesiologist must depend heavily upon his clinical judgement in the absence of sophisticated laboratory investigations as well as advanced monitoring facilities in our developing country. We present a case of a young civilian patient who developed gradual muscular rigidity of almost all of his skeletal muscles following induction by inj ketamine and midazolam and exposure to halothane vapours. The rigidity was gone after we switched off the halothane and recurred as the halothane use was restarted. This clearly was a case of reversible muscle rigidity and could have followed by overt MH, had we used succinyl choline.

CASE REPORT

A 30 years old young patient reported for pre-anaesthetic assessment. He had to undergo herniorrhaphy. He gave history of multiple adverse drug reactions including aspirin, sulphonamides, penicillin, aminoglycoside anatibiotics and so on. He had a prolonged history of allergic diathesis with episodes of bronchospasm. On general physical examination, he was a young man of good muscular build, BP 110/70 mm Hg and pulse 70/min. His chest had no physical abnormality, had normal vesicular breathing with no added sound. His cardiovascular examination did not reveal any abnormality.

The surgeon was confident to finish the procedure within ten to fifteen minutes so a choice of general anaesthesia by inhalation through face mask was made. He was induced with inj. midazolam 2 mg IV and inj. ketamine 100 mg IV. Halothane 2% was administered in oxygen and nitrous oxide mixture through mask and he was allowed to breathe spontaneously. No parenteral analgesics were administered. The surgeon was asked to proceed with the surgery. About 2-3 minutes later, a stiffness of his jaw muscles was noticed. The surgeon complained of increased tightness of his abdominal muscles. His limb muscles also felt taught. The possibility of malignant hyperthermia flashed in the minds of the anaesthesiologist and the surgeon simultaneously.

The halothane was switched off and the patient was administered 100% oxygen. The surgeon was asked to finish the surgery. The patient started showing gradual relaxation of his musculature. Inj. Ketamine was given again in booster dose. The surgery was allowed to commence, and completed uneventfully, with the patient breathing 100% oxygen.

At the end, halothane was restarted in 1% concentration for a few seconds. The muscles of the patient restarted getting tight and his jaw muscles felt firm and contracted. Halothane was switched off and the gradual onset of muscle relaxation was noticed a second time. The patient was allowed to recover slowly on 100% oxygen. No after effects including myalgia, awareness or headache were complained. The changes in pulse rate and blood pressure were only mild to moderate, but no change in body temperature was noticed.

DISCUSSION

MH ,as a distinct familial condition associated withunexpected near-death under anaesthesia, was first

describedin an Australian family by Denborough and Lovell in 1960 (1). In UK, about 0.5 severe reactions per million of the population each year has been reported. A number of anaesthetic agents have been shown to produce masseteric spasm and even overt malignant hyperthermia. The combination of succinyl choline(2) and halothane has been the most notorious one, although other anaesthetic agents including isoflurane, methoxy flurane, sevoflurane or enflurane have induced malignant hyperthermia. Thiopental, atracurium and pancuronium appear to be protective. Atracurium has even been shown to be able to reverse succinyl choline induced masseter spasm. Masseteric spasm is probably the first indication of onset of malignant hyperthermia, although incomplete muscular relaxation and myotonia must be ruled out. The incidence os this spasm on succinyl choline administration may be higher than 1% at some paediatric centers. Although many of these patients will not develop classic signs of MH, about 50% may prove to be MH-susceptible by muscle biopsy.

In our case, the patient was not administered succinyl choline. Probably this was the factor which prevented acute onset of MH. But the generallized muscle rigidity including that of masseters, was clearly more than to be labelled as only a case of masseteric spasm or trismus. Further the reversal of this rigidity on cessation of halothane linked it with out doubt to halothane. In the end it was re-confirmed by administering halothane a second time, with a full knowledge of reversibility of the procedure the precipitation of an acute episode of MH. The acute onset of MH is manifested by sudden rise of blood pressure, tachycardia, muscular rigidity tachypnoea, cyanosis, and in most cased an elevation of body temperature and mottling of skin etc. There is doubling or tripling of end-tidal carbon dioxide on capnometer(3). The capnometer was not available in our setup. However, there was no evidence of cyanosis. This might be due to brief episode of the exposure and 100% oxygenation of the patient.

Further, MH is associated with hyperkalaemia, myoglobinuria, decreased mixed venous oxygen tension, hypercalcaemia and elevated serum creatine kinase levels. We acknowledge that these facilities were not available in that hospital. The patient was not investigated further for two reasons. First he was a civilian non-entitled patient of a very poor family background, and could not pay for costly out of the station laboratory investigations. Secondly the facilities of these investigations were not available at that city.

The onset of MH has been postulated to be due to irreversible release of Ca^{++} from sarcoplasmic reticulum, resulting in impaired excitation-contraction coupling. The rigidity observed is said to be irreversible, even persisting after death.The gradual onset and setting down of muscular rigidity in our case is in contrast to this belief, if it is accepted that the case was of MH. Further, it can be justifiably concluded that the onset of MH is not necessarily an acute one in case of exposure to ketamine and halothane, without the use of succinyl choline. (Ketamine has been shown to be a weak trigger for MH).

CONCLUSION

Masseteric spasm is associated in more than 50% cases with MH. It may be induced by halothane, in the absence of the use of succinyl choline. The muscle rigidity, seen after halothane use is gradual in onset and can be terminated with cessation of halothane and administering 100% oxygen. However, every patient must be thoroughly investigated to confirm MH or otherwise as far as the facilities available locally may allow.

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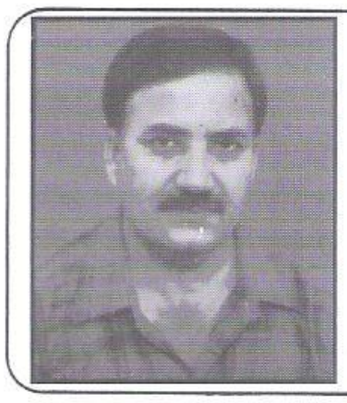

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