

## Case Report

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# A case of convulsion resembling masseter muscle spasm (MMS) during caesarean delivery under subarachnoid block

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### Abstract

A 28 yrs old female forty weeks gravida was scheduled for caesarean section for less fetal movement. She did not have any bad obstetric history and any complication during previous operation and anaesthetic procedure. Subarachnoid block was performed at L<sub>3</sub>-L<sub>4</sub> interspace with 2.5ml (12.5mg) 5% bupivacaine heavy. Suddenly the patient became cyanosed and she tried to tell something but could not talk. Then she was given 100% O<sub>2</sub> by face mask but it was not fruitful. Then endotracheal intubation was attempted but failed to achieve due to increased jaw muscle tension and mouth could not be opened like masseter muscle spasm (MMS). At that stage patient became unresponsive and no pulse was palpable, blood pressure was not recordable. Intravenous adrenaline was given immediately and then 100mg of suxamethonium administered intravenously. The jaw relaxed within minutes and tracheal intubation was done. General Anaesthesia was maintained with O<sub>2</sub>/N<sub>2</sub>O, 0.4% halothane and atracurium. The reversal was good enough and the patient was haemodynamically stable. The patient transferred to the recovery room.

**Key words:** subarachnoid block (SAB), masseter muscle spasm (MMS), caesarean section (CS).

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### Introduction

Obstetric anaesthesia is a demanding but gratifying sub speciality of anaesthesiology. The wide spread acceptance and use of regional anesthesia for labour has made obstetric anaesthesia a major part of anaesthetic practice. Spinal or epidural anaesthesia has become the preferred technique because general anaesthesia (GA) has been associated with higher maternal mortality<sup>1</sup>. Other advantages of regional anaesthesia includes, less maternal exposure to potentially depressant drugs, a decreased risk of maternal pulmonary aspiration, an awake mother gets the pleasure of birth of her baby and also the option of using spinal opioid for post operative pain relief<sup>2</sup>. Spinal anaesthesia was first used in 1900, popular in the USA in 1920s. Its popularity increased in UK towards the end of 1900s<sup>3</sup>. The spinal anaesthesia is easier to perform, has more rapid, predictable onset, produces more intense block, and does not have potential for serious systemic drug

toxicity, because of smaller doses of local anaesthetic employed<sup>4</sup>. Though spinal anaesthesia have proved to be extremely safe, but it is not without complications. Complications are related to medications or needle used to perform given the procedure. Adverse reactions and complications range from severe hypotension to permanent neurological deficit and even death. Here we report one such case of convulsion resembling masseter muscle spasm (MMS) during caesarean delivery under spinal anaesthesia.

### Case History

A 28 years old female, forty weeks gravid, was admitted to hospital with the complaints of less fetal movement & scar tenderness. Her weight & height were 55 kg & 5'55" respectively. She has a previous history of caesarean section under spinal anaesthesia & a live female baby. She did not have any bad obstetric history & no history of any complication

during the previous operation & anaesthetic procedure. Her Hb% was 10.6 gm/dl & pre-operative blood pressure was 110/70 mmHg. Her pulse rate was normal. She didn't have any significant respiratory or cardiovascular abnormality with normal body temperature. She was conscious & no neurological abnormality was present.

After an initial assessment, she was preloaded with about 800 ml of Hartmann's solution. Then spinal anaesthesia was administered after proper painting with providone iodine solution and with a 25 gauge Quinke needle in the interspace between the lumbar third & fourth vertebra in a single shot. Then 2.5 ml (12.5 mg) 5% Bupivacaine heavy was administered intrathecally. After 3-5 mins blood pressure was falling rapidly & reached 80/50 mmHg. Then 5mg ephedrine hydrochloride was given intravenously to the patient. Meanwhile the operation was started & incision was made in the lower abdomen & the patient did not complain of any pain.

Suddenly the patient became cyanosed & she tried to tell something but could not speak. Then she was given 100% O<sub>2</sub> by face mask, as there was no improvement of SpO<sub>2</sub> assisted ventilation was started. But it was not fruitful. Then endotracheal intubation was attempted but failed to achieve due to increased jaw muscle tension & mouth could not be opened. At that stage patient became unresponsive, no pulse and blood pressure was recordable. Immediate 1mg of adrenaline was given intravenously when pulse & blood pressure became recordable within 30sec. Then 100mg of suxamethonium was given intravenously and the jaw relaxed within minutes and tracheal intubation was done. General anaesthesia was maintained with O<sub>2</sub>/NO<sub>2</sub>, 0.4% Halothane, and a bolus dose of 25 mg atracurium was given. After 15 mins the patient was spontaneously breathing. Within that period about 2000 ml of Hartman's solution was given to the patient intravenously & the urine output was about 800 ml. The operation was completed within 30 mins & the patient was reversed with 2.5 mg of Neostigmine & 1.2 mg of Atropine. At that time the patient was spontaneously breathing.

The reversal was good & the patient was haemodynamically stable with post operative blood pressure of 120/90 mmHg & pulse rate was 78 beats/min. The patient showed no evidence of further respiratory difficulty & the lung was clear & the

air entry was good. The patient was sent to post anaesthesia care unit. The period was eventless. The patient was stable & sent to the ward next morning.

### Discussion

Masseter muscle spasm (MMS) is a major & serious problem to the attending anaesthetists as it causes clinical problem in opening the mouth in order to achieve tracheal intubation. It has got a strong correlation with Malignant Hyperthermia (MH). The first common use of the term MMS arose when Malignant Hyperthermia reaction subsequently occurred in patient whose mouths had been difficult to open following the use of suxamethonium<sup>5</sup>. This association was apparent in 70% of patients given suxamethonium who went to develop MH. Awareness of the association between MMS & MH led to the referral of many patient who developed MMS for investigation of their MH status. Of those with MMS as the only abnormal feature 28% have been proven to be susceptible to MH. The proportion rises to 57% if there were accompanying metabolic features or to 76% if the MMS was followed by other features of muscle damage such as myoglobinuria or severe incapacity from muscle pains. From this experience which is similar amongst MH investigation centres, it seemed clear that, patients developing MMS were at high risk from MH until proven otherwise & MH is still one of the major potential anaesthetic hazards despite the mortality rate for MH declining from above 70% before 1980 to below 4% over the past five years.

Suxamethonium is a known triggering agent for MH as well as MMS. Suxamethonium is thought to produce a rapid & marked rise in intracellular calcium concentration but its duration of effect is limited. The predominant feature is thus increased muscle activity, evident as rigidity<sup>6</sup>.

The muscle rigidity is sometimes generalized, but may be limited to the jaw muscles. The term MMS is therefore of practical & clinical significant only if its use is restricted to severe & perhaps more prolonged (more than 21 mins) episodes of restricted mouth opening following suxamethonium administration.

Inhalation anaesthetics can also trigger MH as well as MMS. Halothane is the most potent triggering agent. But other inhalation anaesthetics like enflurane, desflurane, isoflurane & sevoflurane can also trigger MH<sup>7,8</sup>.

Patient was well preoperatively. She did not give any history of metabolic disorder or features of muscle damage. Even though when suxamethonium or halothane was administered to facilitate intubation for conversion of spinal anaesthesia to general anaesthesia, her body temperature was quite normal. So in this case there is no apparent relation with prolonged masseter muscle rigidity & MH. Patient received spinal anaesthesia & there was hypotension. But there is no co-relation with MMS & hypotension. The patient experienced a period of hypoxia due to hypotension & ongoing respiratory failure. There is also no evidence of MMS in hypoxemic state. The patient's temperature was quite normal all through the perioperative period & no shivering occurred. So there was no chance of hypothermia induced muscle stiffness.

The local anaesthetic that was used for spinal anaesthesia was 5% bupivacaine heavy. Bupivacaine has got a wide safety margin & even slow intravascular injection of epidural may not precipitate convulsion. Early signs of intravascular injection like numbness of the tongue & circumoral area were absent in our patient<sup>8</sup>.

So, which factors triggered that jaw muscle rigidity was misleading & not clear as an isolated solitary event. The possibilities may be that it was due to toxic effect of local anaesthetic or it was possibly more due to hypoxic convulsion due to acute hypotension from a very high up SAB. Convulsion could not be generalized due to the motor blockage of skeletal muscle by SAB in the lower limb & thoracoabdominal region but leaving the masseter muscle unblocked as it is supplied by cranial nerves mandibular division of trigeminal nerve. However the final cause of masseter muscle rigidity could not be confirmed in this case.

## Conclusion

This was an isolated solitary event. The possibilities may be a toxic effect of local anaesthetic or hypoxic convulsion due to acute hypotension .

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