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Epidural Anesthesia for the Patient with Malignant Hyperthermia : A Report of Case

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Abstract Anesthesia for cholecystectomy in a survivor of malignant hyperthermia (MH) was successfully managed with prophylactic administration of dantrolene and epidural lidocaine, the amide type local anesthetic which is still controversial for use in MH susceptible patients (MHSP).

Key Words : Dantrolene, Epidural anesthesia, Lidocaine, Malignant hyperthermia

Malignant hyperthermia (MH) is still one of most serious complications during anesthesia, even though recent advances in prophylaxis and treatment for MH have made it possible to manage MH susceptible patients (MHSP) successfully in the perioperative period. Triggering agents as well as pathogenesis of MH, however, have not been fully understood. We describe the anesthetic management for cholecystectomy in a survivor of MH using epidural lidocaine with the prophylactic administration of dantrolene.

Report of a case

A 53 - year - old male with cholelithiasis presented Tane General Hospital for elective cholecystectomy. Although his family history was non contributory, he had a documented episode of MH crisis[1] during general anesthesia in other hospital 7 years ago.

Previous anesthesia ; The patient was suspected to be lung cancer and was scheduled for mediastinoscopy under general anesthesia with halothane and nitrous oxide. Anesthesia was induced with thiopental 300mg and succinylcholine 60mg iv. Since slight rigidity in masseter muscle and tachycardia (145 bpm) were noted subsequent to endotracheal intubation, volatile anesthetics were avoided and anesthesia was maintained with diazepam 10mg, pentazocine 90mg, and pancuronium 12mg iv and nitrous oxide 60−70 % in oxygen. At emergence following uneventful anesthesia, the patient developed a sudden elevation of body temperature from 37.8℃ to 40.2℃ for 10min, which exceeded criteria for diagnosis of MH, 1℃ per 15min.[2] He was treated with surface cooling, hyperventilation, steroids, sodium bicarbonate, and procainamide. Two months after complete recovery from the MH crisis, the patient underwent radical operation for lung cancer. Dantrolene, in increasing dose as 100, 150, and 200mg, was given orally for 3 days before anesthesia, which was maintained with total spinal block using tetracaine supplemented with droperidol 5mg and diazepam 20mg iv, and nitrous oxide 50% in oxygen. Although

the anesthetic course was uneventful, extubation was followed by elevation of body temperature up to 38.9℃ with shivering, which was suppressed soon after administration of dantrolene 40mg iv. The patient recovered without any sequelae and was discharged 2 months after the operation. He has been healthy until current admission for cholecystectomy to our hospital.

Present anesthesia ; Preoperative laboratory data showed no remarkable abnormalities except slightly increased CPK 104 IU/L (normal value, less than 77 IU/L). An epidural catheter was inserted at the T9-10 interspace one day before the operation. A T6-12 level of analgesia was obtained with 5ml of 2% lidocaine. There was no remarkable change in vital signs after an epidural block until following day of operation. Dantrolene 100mg po and hydroxyzine 75mg im were given 2h and 30min before induction of anesthesia, respectively. In the operating room, infusion of dantrolene 60mg was started at the rate of 2mg/min. A T6-L5 level of analgesia was established with 20ml of 2% lidocaine and endotracheal intubation was facilitated by intravenous administration of thiopental 200mg and pancuronium 4mg. The patient was ventilated with 40-50% oxygen by a ventilator which had never been exposed to inhalation anesthetics. Epidural anesthesia was maintained with total dose of

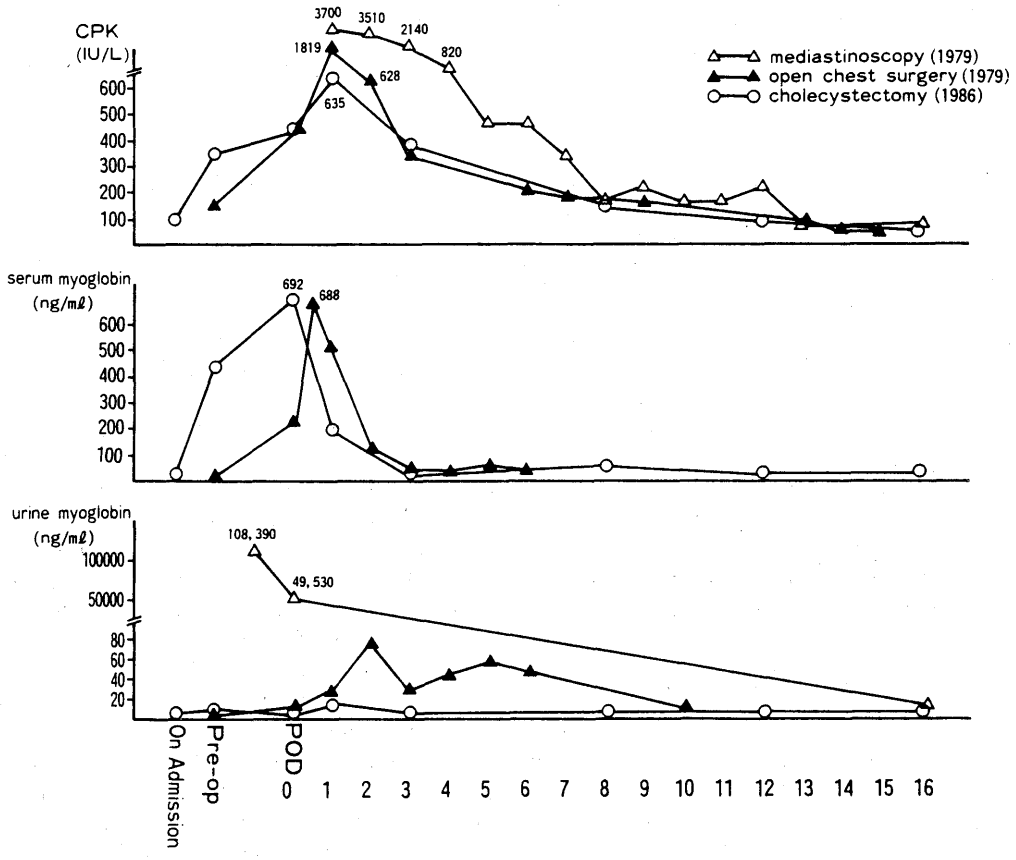

Fig. 1 Serum CPK, myoglobin, and urine myoglobin in the perioperative course of 3 successive operations with and without an episode of MH.
open triangles ; general anesthesia with MH
closed triangles ; total spinal block without MH
open circles ; epidural lidocaine without MH

lidocaine 750mg, supplemented with flunitrazepam 2mg iv. No inhalation agent including nitrous oxide was used. Blood gas values determined during anesthesia were within normal range and serum lidocaine concentration, 4.9 to 6.7μg/ml. The rectal and esophageal temperatures were monitored continuously and maintained at 37.0-37.2℃. No signs to suspect a MH crisis were observed during anesthesia. After operation he was extubated and transferred to ICU, where his respiratory and circulatory condition was satisfactory except a moderate increase of the rectal temperature up to 38.3℃ which subsided the next day without treatment. Serum CPK and myoglobin and urine myoglobin were determined at postoperative day (POD) 1, 3, 7, 10, and 14 (figure 1). The patient was discharged without any problems 1 month after the operation.

Discussion

In MH, acute and unexpected increase of skeletal muscle in oxygen consumption and lactate production, resulting in greater heat production, respiratory and metabolic acidosis, muscle regidity, sympathetic stimulation[2]. Halothane, succinylcholine and ketamine are known as triggers of MH[2]. As local anesthetics are concerned, it has been believed that the amide type is best avoided[2]. In fact, there are a few experimental results supporting those principles for choice of local anesthetics.

Britt[3] proposed that the amide type local anesthetics are contraindicated in MHSP because they stimulated calcium release from sarcoplasmic reticulum in vitro. Bianchi[4] demonstrated that lidocaine potentiated the caffeine-induced contracture of frog sartorius muscle. Since the impairement in control of intracellular calcium is thought to be one of possible mechanisms in pathogenesis of MH[2], the previous reports suggest to avoid the amides when local anesthetics are used in MHSP.

However, the use of local anesthetics for MHSP is controversial. MH crisis is unlikely to occur solely by use of the amide type local anesthetics without epinephrine[5]. Furthermore, there is no fatal case even if it is developed[6]. There are several reports in MH susceptible swine supporting those clinical facts. Lidocaine for iv, did not trigger[7] and for epidural anesthesia, was effective for block porcine MH[8]. As Gronert mentioned in his review[2], we must be cautious to evaluate the results *in vitro* that the caffeine-induced contracture of the frog muscle is potentiated by extremely high concentration of lidocaine (3.67mM=1060μg/ml) which is more than 100 times of therapeutic level.

The anesthetic management of our patient was satisfactory. Although preoperative serum myoglobin level (428ng/ml) is similar to the level immediately after previous lung surgery (470ng/ml) with a violent shivering. But there were no clinical symptoms related to the increased serum myoglobin in the perioperative course of cholecystectomy.

Some investigators proposed central and peripheral neural machanisms for the onset of MH. It has been reported emotional stress or excitement is one of triggers of MH for susceptible swine and human[2]. Since postoperative delirium is not uncommon in anesthesia practice, postanesthtic excitement may be also regarded as a trigger of MH. In fact, some MHSP develop crisis not in induction but at emergence, even after extubation. The local neural mechanisms also appears to play an important role in the pathogenesis of MH. Kerr[8] demonstrated that complete epidural anesthesia blocked MH susceptible swine to develop MH and incomplete anesthesia did not. He suggested that intact pathways between the spinal cord and skeletal muscles are necessary for development of MH. Thus, the blocking of the stress response, not the type of local anestheic, is perhaps the important factor in avoiding MH[6].

The first, perioperative mental status of our patient was satisfactory. He was not noted psychological disturbances in the operating room and ICU, only with minimal clinical doses of preoperative hydroxyzine im and intraoperative flunitrazepam iv. The second, thoracic epidural anesthesia in this case was considered "complete" to prevent the onset of MH because the patient showed no evident

signs including cardiovascular responses to incomplete blockade of noxious stimuli. The pretreatment with dantrolene might be, however, rather unsatisfactory compared with the recommended dose for both therapy and prophylaxis of MH[9]. MH did not develop despite use of lidocaine probably because epidural anesthesia might block local neural pathways essential for development of MH. We should be prudent, however, to conclude that lidocaine is a safe anesthetic for MHSP because of the fluctuation in the ease of initiation of MH. More clinical evidences are necessary to draw definite conclusions.

In summary, our case supports the hypothesis that lidocaine is unlikely to be a trigger for MH. But it may not deny the possibility that pretreatment with dantrolene reduce the triggering property of lidocaine for the disease.

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