ACCIDENTAL SUBCUTANEOUS INJECTION OF VECURONIUM BROMIDE IN A PATIENT WITH BURNS

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ABSTRACT: BACKGROUND AND OBJECTIVES: In patients with burn injury drug pharmacology will be altered and this poses special anaesthetic challenge when an subcutaneous injection of a non-depolarizing muscle relaxant occurs in such a patient. Small studies remain an important source of knowledge and hence this study aims to provide information on the anaesthetic management in a case of accidental subcutaneous injection of vecuronium bromide in a burns patient.

PRESENTATION, DIAGNOSIS & MANAGEMENT: A 22 year young male with 4 days old hot water induced grade 1 burns involving 45% of body surface area was posted for burns dressing. Anaesthesia was induced with propofol and vecuronium bromide through an external jugular vein to aid tracheal intubation. As the patient was not anaesthetised even after 10 minutes routine check lead to the discovery of fullness at the tip of the intravenous catheter indicating an extravasation of the drugs. Hence the other external jugular vein was cannulated and the patient induced and intubated using propofol, sevoflurane, nitrous oxide and oxygen. The action of vecuronium outlasted the duration of surgery. So the patient continued to receive support of mechanical ventilation with nitrous oxide and oxygen. It took 130 minutes for the clinical signs of recovery from the muscle relaxant to manifest. He was then reversed & extubated with subsequent good recovery.

CONCLUSION: Subcutaneous injection of these drugs poses problems of delayed onset of action and prolonged duration of action. In an inadvertent accidental subcutaneous 0.1 mg/kg vecuronium bromide injection in a patient with 4 day old 45% burns showed delayed onset action and prolonged neuromuscular blockade due to subcutaneous deposition of the drug which was managed with mechanical ventilation. The reported resistance to the action of NDMR drugs in patient with burns was not noticed here probably because of the age of the thermal injury.

KEYWORDS: Thermal injury, accidental subcutaneous vecuronium, altered drug pharmacology.

INTRODUCTION: Anesthesia for major burns can be challenging with multiple problems, not the least among them being intravenous access, monitoring difficulty and altered drug pharmacology. Pharmacodynamic explanations as the principal mechanism for resistance to the effects of non-depolarising muscle relaxants are documented by the need to achieve higher drug concentrations to produce given degrees of twitch suppression in burn injured patients compared to non-burn injured patients.[1] We present research findings of a problem we encountered due to accidental subcutaneous deposition of vecuronium bromide in a burns patient.

PRESENTATION, DIAGNOSIS & MANAGEMENT: A 22 yrs male with 4 days old hot water induced superficial burn (scald) of grade 1 involving a total body surface area 45% on the anterior aspect of chest and abdomen and all four limbs was posted electively for burns dressing. He had no significant history of medical illness or drug intake was haemodynamically stable. Routine investigations including Renal function tests and serum proteins were within normal limits.
In the operating room due to paucity of good veins, an intravenous infusion of 0.9% normal saline was started in the left external jugular vein. After pre oxygenation and premedication with fentanyl 2μg/kg, midazolam 1mg and glycopyrrolate 0.2mg intravenously, he was induced with propofol 2mg/kg and 0.1mg/kg of vecuronium bromide.

As the patient was not under anesthesia even after ten minutes the intravenous line was checked only to find a swelling at the tip of the catheter. A diagnosis of subcutaneous deposition of the anaesthetic drugs was made So the right external jugular vein was secured, the patient re induced with 1.5mg/kg of propofol, sevoflurane, nitrous oxide and oxygen and anesthesia maintained with nitrous oxide, oxygen and 2% sevoflurane with controlled ventilation.

Regular neuromuscular monitoring was not possible because of the lack of proper area for electrode attachment. Surgery lasted for 45 minutes at the end of which 1g of paracetamol was injected intravenously. As the patient showed no respiratory efforts at 100 minutes from the start he was maintained on controlled ventilation with nitrous oxide and oxygen. 30mins later, on fulfillment of extubation criteria clinically, he was reversed with neostigmine (2.5mg) and glycopyrrolate (0.5mg). His subsequent recovery was satisfactory.

**DISCUSSION:** Vecuronium is an amino steroid non-depolarizing neuromuscular blocking drug with an onset of action in 2mins and recovery time of about 14-30mins after an initial bolus dose of 0.08-0.1mg/kg.[1][a] Thermal injury causes resistance to the effect of non-depolarizers, occurs approximately 10 days post injury, peaks at about 40 days and declines after approximately 60 days.[2] The reason for it is multifactorial but is mostly related to changes in acetylcholine receptors which have an altered affinity for these drugs.[3] This resistance manifests as a slower onset of paralysis, inadequate paralysis or faster recovery when normal doses are administered to these patients.[4]

Non-depolarizing blockers like d-tubocurare, and metacurine are seen to be less effective according to studies involving burns patients, the requirement of metacurine being as much as three times the dose for normal patient for an equivalent degree of blockade.[5] A study employing atracurium in burns patients investigated the relationship between the response to atracurium with the magnitude and age of burn injury.

The conclusion of the study was that a normal response to the intubating dose of atracurium is obtained in burns patients with less than 33% total body surface area burns and also in any sized burns patients of less than 6 days post injury. However with increased area of burns (>33%) increased resistance to the drug is noticed being maximum at 15-40th day.[5]

Reports regarding accidental subcutaneous injection of non-depolarizing muscle relaxants even in non-burn patients are rare. In one such report by Tarmey et al, the effects of subcutaneous vecuronium (0.16mg/kg) given in the forearm lasted for 4hrs.[6] In another report, it took 270 minutes for a patient given subcutaneous vecuronium (0.1mg/kg) via a displaced ante-cubital cannula to start spontaneous respiratory effort.[7] However in the latter study, succinylcholine was used to facilitate tracheal intubation after discovering the accidental injection of the non-depolarizer given earlier.

Similar effects have been reported with pancuronium when a subcutaneous injection resulted in delayed block with unpredictable duration of action. In a burns patient accidental subcutaneous pancuronium at the ankle showed prolonged duration of action and required 5 hrs 30min for train of four value of 0.46.[8]
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Studies involving subcutaneous vs. intravenous pancuronium have demonstrated that onset of neuromuscular blockade occurred relatively slowly after subcutaneous administration with a delayed onset probably the result of slow systemic absorption of drug from subcutaneous site to the plasma. The rate of recovery from the blockade is governed largely by the rate of fall in the plasma concentration of the drug. In our patient the subcutaneous injection of a normal dose of vecuronium resulted in a delayed onset and prolonged blockade for approximately 130 minutes. The usual resistance reported of non-depolarizing drugs in burns patients was not observed here probably because of the age of the burns in our patient i.e. less than 6 days.

The fact that vecuronium was injected subcutaneously in the richly vascular neck region could have been the reason for the relatively earlier recovery in our patient compared to Tramay or Pradhan instances (injection in the upper limb) because there is a regional difference in skin blood flow at various sites of the body as studied by use of xenon 133 with a tendency for the flow to decrease gradually from the upper part of the body to the lower. Skin blood flow of the face and anterior chest are significantly greater than that of the deltoid region.

As muscle relaxants are usually preceded by iv induction agents one should also be skilled in the management of such situation of subcutaneous deposition of drugs. Along with vecuronium there was extravasation of propofol in our patient. There have been isolated reports documenting tissue necrosis after the use of propofol. However other clinical reports and animal studies have shown that propofol extravasation does not cause serious clinical consequences. The above does not usually lead to tissue necrosis due to the favorable chemical properties, including a neutral pH and is otonicity of propofol. In our case we followed up our patient for fifteen days only to find that he did not show any signs of tissue necrosis.

CONCLUSION: In summary management of burns patients poses problems of intravenous access, monitoring and altered drug pharmacology. An inadvertant subcutaneous vecuronium injection 0.1 mg/kg in our patient with 4 day old 45% burns showed no resistance to neuromuscular blockade probably because of the age of the burn but resulted in a delayed onset and prolonged blockade implicating the subcutaneous site of injection as the cause for the above delay. There was no adverse event because of the subcutaneous deposition of propofol made earlier. In conclusion, we recommend vigilance when injecting muscle relaxants. When administered subcutaneously in a burns patient special attention should be paid to the age and degree of the burn injury and the altered drug pharmacology with regard to neuromuscular blockade, attention should also be directed towards recognizing misplacement of induction agents outside the venous system.

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