Supraventricular tachycardia after an intercostal nerve block with bupivacaine treated with 10% intralipid

Sir,

The use of 20% intralipid as a rescue drug for bupivacaine cardiotoxicity has been reported. There are laid down guidelines to treat severe toxicity, but there are conflicting reports on the success of 10% intralipid to treat it.[1] We report a case of severe cardiotoxicity by racemic bupivacaine treated successfully by 10% intralipid.

A 26-year-old, 75 kg, male with fractured 9th thoracic vertebra and fractured 8th, 9th, 10th, and 11th right-sided ribs, with paraparesis, underwent transthoracic fixation of affected dorsal vertebra under general anesthesia with one lung ventilation. Toward the end of surgery, intercostal nerve block at three levels was planned for postoperative analgesia and to facilitate early tracheal extubation. The patient developed supraventricular tachycardia soon after the administration of the first injection of 5 ml of 0.5% racemic bupivacaine. The heart rate peaked to 244–250/minute and invasive arterial pressures came down to 50–56/30–36 mmHg. Further injection of local anesthetic was stopped and 10% intralipid, which was available at that time, was started through the right internal jugular vein (canulated at the beginning of the surgery). Figure 1 shows the photograph of the monitor taken five minutes after starting treatment. Sinus rhythm returned after 150 ml of intralipid was injected over 15 min. The hemodynamics returned to normal and the patient remained stable in postoperative period and did not need vasopressors or antiarrhythmics. The patient was electively ventilated and weaned off successfully the next day. There was no evidence of any cardiac or neurological sequelae in the follow-up period. The blood levels of bupivacaine could not be measured due to nonavailability of the required facility, though clinically it is beyond doubt that bupivacaine was the cause of tachyarrhythmia.

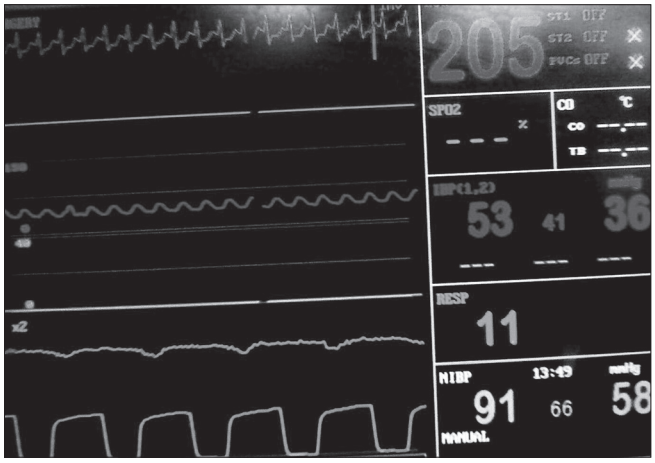

Figure 1: Photograph of monitor 5 mins after administration of 10% intralipid

Intercostal nerve blocks are associated with a higher incidence of local anesthetic toxicity because of increased vascularity. Injection of a small amount of drug can cause toxic symptoms if injected rapidly. Bupivacaine usually slows down ventricular conduction because of its effect on sodium channels. At higher plasma drug concentration, bupivacaine acts on calcium and potassium channels and causes slowing of conduction with decreased contractility and certain metabolic changes. Supraventricular arrhythmias are sometimes due to reentry of an impulse around an arc of functional conduction block.[2]

The lipid sink theory[3] describes how intralipid forms a lipid phase inside plasma into which bupivacaine is extracted from the cardiac tissues and thus reduces the amount of free bupivacaine in the aqueous phase of plasma. Cardiotoxicity with bupivacaine is known but what was remarkable in this case is that it occurred almost immediately with a small volume of injected drug and presented as supraventricular tachycardia with severe hypotension. The electrocardiographic changes reverted completely with 2 ml/kg of 10% intralipid within 15 min. We were successful using 10% intralipid in the same volume and in a short time possibly because we had the central venous catheter in place or 10% intralipid was effective. Adult cardiac life support guidelines should be followed simultaneously in all cases where required.

Weinberg *et al.*[4] have suggested that propofol should not be used for treating cardiotoxicity though it contains 10% intralipid because propofol itself may be detrimental in such situation. The authors suggest that if 20% intralipid is unavailable, 10% intralipid may be used. It is strongly recommended that intralipid should be kept available in the operation theaters where nerve blocks are practiced routinely so that immediate action may be taken in case of local anesthetic toxicity.

Rakhee Goyal, R N Shukla, Gaurav Kumar[1], M Tandon

Department of Anesthesia and Critical Care, Armed Forces Medical College, Pune and Command Hospital (SC), [1]Department of Cardiothoracic Surgery, Military Hospital, Cardiothoracic Centre, Pune, India

Address for correspondence: Dr. Rakhee Goyal,
NP-5 Officers Project Quarters, MH, CTC, Pune - 411 040, India.
E-mail: rakhee_goyal@yahoo.co.in

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