Case Report

Pulmonary oedema in a patient undergoing vitreo-retinal surgery under peribulbar block

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ABSTRACT

A 42-year-old diabetic and hypertensive male with good effort tolerance was administered peribulbar block for vitreo-retinal surgery. Ten millilitres of an equal mixture of 2% lignocaine and 0.5% bupivacaine was administered for the block after ascertaining negative aspiration for blood. Inadequate akinesia of the eye necessitated further supplementation with 4 mL of local anaesthetic (LA) mixture. Thirty minutes later, the patient complained of uneasiness, respiratory distress and desaturated despite oxygen supplementation. He was found to be in pulmonary oedema. He subsequently developed a weak thready pulse, became unresponsive, apnoeic and had generalized tonic clonic convulsions. Immediately, atropine 0.6 mg, followed by midazolam, intubation, mechanical ventilation, morphine and furosemide, were administered intravenously. Spontaneous respiration returned in 20 minutes and he started responding to verbal commands 90 minutes later. He was weaned off the ventilator the next morning. There was no evidence of an ischemic myocardial event and non-contrast computerized tomography scan of the head was normal. The reversible cardiorespiratory arrest, associated convulsions and loss of consciousness were suggestive of LA toxicity. Pulmonary oedema manifesting as respiratory distress and desaturation can be the initial manifestation of LA toxicity in patients with pre-existing cardiovascular disease undergoing eye surgery under peribulbar block.

Key words: Complications, local anaesthetic toxicity, peribulbar block, peribulbar eye block, pulmonary oedema, vitreo-retinal surgery

INTRODUCTION

Major complications are rare in patients undergoing ophthalmic surgery under regional eye blocks, with a reported incidence of 0.006% for surgeries under peribulbar block.[1] Life-threatening complications reported are unconsciousness, apnoea, convulsions or cardiovascular depression, occurring within seconds of drug administration to as long as 7 min after the block.[2,3]

There are only two reports in the literature of pulmonary oedema with peribulbar block.[4,5] There are two reports of similar events occurring in patients undergoing eye surgery under retrobulbar block with no known definite aetiology.[6,7] The presentation of pulmonary oedema in this case is suggestive of local anaesthetic toxicity following the peribulbar block and demonstrates that early identification and prompt treatment can be life saving.

CASE REPORT

A 42-year-old, ASA grade II male patient was taken up for vitreo-retinal surgery under peribulbar block. He was a known diabetic for 6 years on metformin (stopped 48 h prior), glibenclamide and pioglitazone, with features of retinopathy and nephropathy (blood urea 60 mg/dL, S Cr 1.5 mg/dL). He was also a hypertensive for 1 year, on treatment with indapamide, triamterene and benzthiazide. He was on regular follow-up and his blood pressure and blood sugar levels were controlled. He had good effort tolerance (4–6 metabolic equivalents) with no history of dyspnoea,

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angina, pedal oedema, palpitations or myocardial infarction.

He was allowed light breakfast (bread and black tea at 6 AM), antihypertensive medications were given and oral hypoglycaemic drugs omitted on the morning of surgery. His morning blood sugar was 130 mg/dL. In the operation theatre, routine monitors (heart rate, electrocardiogram, non-invasive blood pressure and oxygen saturation) were attached. A peribulbar block was administered at the infero-temporal and superior-nasal orbital margin with a 26 G, 13-mm-long needle. Ten millilitres of an equal mixture of 2% lignocaine and 0.5% bupivacaine was administered after confirming negative aspiration of blood. The operative eye was prepped and draped. Oxygen (5 L/min) was insufflated under the drapes. The block was supplemented 5 mins later with 4 mL of local anaesthesia mixture through an infero-temporal injection, as complete akinesia of the eye was not achieved.

After 30 mins of uneventful surgery, the patient complained of uneasiness, had difficulty in breathing, started producing copious secretions and desaturated (SaO_2 92–93%). He had a pulse rate 132/min, respiratory rate 24/min and non-invasive blood pressure 120/70 mmHg. Oxygen was supplemented using a facemask and Bain's circuit while the eye was sutured and dressed. Suddenly, his hands became cold, clammy, the pulse thready and he became unresponsive, stopped breathing and had generalized tonic–clonic convulsions. Immediately, bag mask ventilation with 100% oxygen was started. Atropine (0.6 mg IV) was administered following which the pulse rate increased to 120/min and the blood pressure to 90/60 mmHg. Midazolam (2 mg IV) was given for the seizures. On laryngoscopy for tracheal intubation, copious white frothy secretions were seen in the laryngeal inlet and subsequently filled the endotracheal tube. Fine crepitations were heard bilaterally. He showed no response on intubation and remained unresponsive to painful stimuli. Intermittent positive pressure ventilation (IPPV) was continued; 6 mg morphine and 20 mg IV frusemide were administered. Hypoglycaemia and dys-electrolytemia were ruled out. Spontaneous respiration returned after 20 min and he responded to verbal commands only after 90 min.

He was electively ventilated overnight in the Intensive Care Unit (ICU). The chest X-ray was suggestive of pulmonary oedema [Figure 1] and ECG showed no fresh changes (previous T wave inversion in lead I, II, V1-6 persisted). Troponin T test done 6 h later and on two subsequent days was normal. A non-contrast computerized tomography scan of the head was normal. Transthoracic echocardiography showed global hypokinesia with left ventricular ejection fraction of 45%.

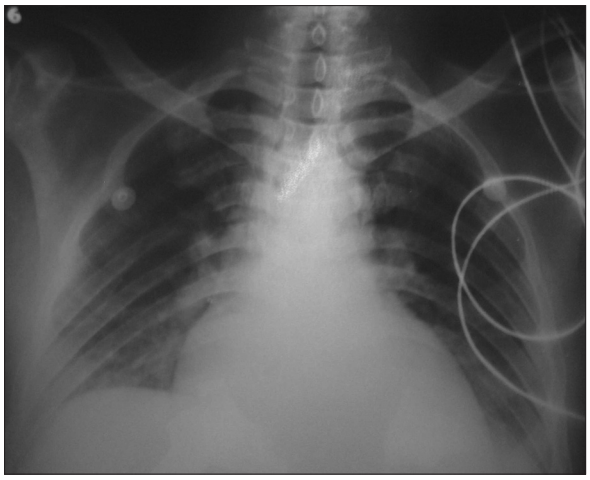

Figure 1: Chest skiagram (AP view) of the patient showing pulmonary oedema

The patient was fast tracked off the ventilator the next day and extubated. He was observed in the ICU for two more days and was discharged home uneventfully on the 5th post-operative day.

DISCUSSION

There are two reports of pulmonary oedema in patients undergoing eye surgery under peribulbar block[4,5] and two similar reports in patients undergoing surgery under retrobulbar block.[6,7] Three of these patients were elderly females (65, 77 and 72 years, respectively) with significant cardiovascular disease.[4-6] However, they were well controlled on medications and stable prior to and following the event.

One of these patients with mitral stenosis, occasional angina, suspected previous myocardial infarction, desaturated 5 min following a peribulbar block and was found to be in pulmonary oedema. She had no associated loss of consciousness, convulsions or haemodynamic instability.[4] Neurogenic pulmonary oedema (NPE), i.e. central nervous system insult resulting in autonomic imbalance, rather than a primary cardiac event was thought to be the cause. Similar NPE was thought to be the cause in the elderly patient undergoing cataract surgery under retrobulbar block as she also desaturated but remained hemodynamically stable.[6] Central spread of local

anaesthetic (LA) resulting in trigeminal block was opined to be the trigger for development of the NPE in this patient.[6]

Another one of these patients (previous congestive cardiac failure, atrial fibrillation and myocardial infarction) developed pulmonary oedema in the recovery area after uneventful cataract surgery under peribulbar block.[5] A transthoracic echocardiogram demonstrated significant deterioration in the left ventricular function as compared to 6 months earlier. An ischaemic cardiac event was ruled out but missing the morning dose of diuretic was thought to be a contributing factor.

Like the above patients, our patient underwent eye surgery under a peribulbar block. He was younger and more active but was a diabetic, hypertensive with retinopathy and nephropathy suggestive of significant atherosclerotic disease. Similar to the above patients, he complained of difficulty in respiration, desaturated and developed frank pulmonary oedema. Unlike the others, he had momentary cardiorespiratory arrest, convulsions and a reversible loss of consciousness lasting for 90 min. At no time were ventricular arrhythmias observed. Like one of the reported patients,[5] pulmonary oedema occurred 30 min after the block.

The presentation in our patient was suggestive of LA toxicity. Systemic absorption of the LA and direct cardiotoxicity is unlikely with the small doses and volumes of drugs used. However, due to the anatomic proximity between the eye and the central nervous system, LAs may track along the optic nerve sheath or through a dural cuff breach and reach the brain stem directly after an eye block.[2,3,8,9] The resulting presentation is variable depending on the amount of drug reaching the medulla and the exact areas affected.[8,10]

Thomas *et al*. showed that injection of LAs directly into the cardioactive areas in the ventral medulla (C1 region, intermedullary columns, nucleus tractus solitarius) in rats resulted in significant hypotension, bradycardia and ventricular arrhythmias.[10] They further demonstrated that bupivacaine-induced cardiac arrhythmias were more resistant to treatment and more likely to be fatal (50%) as compared with those caused by lignocaine.[10] However, only 55% animals with injection in nucleus tractus solitarius developed ventricular arrhythmias. This has also been observed in reports of systemic bupivacaine toxicity in humans.[11]

The delayed manifestations of LA toxicity in our patient as well as other reported cases of delayed brainstem toxicity with eye blocks[8,9] could be explained by the slow diffusion constants of lignocaine and bupivacaine as demonstrated in the study by Thomas *et al* (the LAs diffuse through 1 mm^3 of brain tissue in 16 mins when small quantities are applied to the medulla).[10]

A retrobulbar block would be more likely to be associated with brain stem toxicity because of the use of a longer needle and more chances of needle injury. However, Ripart *et al*. have demonstrated in a cadaver study that spread of dye in the orbital cavity is almost similar after retrobulbar or peribulbar block.[12] In addition, there are other reports of brainstem anaesthesia with peribulbar block, probably caused by inadvertent injection of the LA through a breach in the dural sheath surrounding the optic nerve.[2,3]

An inadvertent intravascular injection seems unlikely in this patient in view of the slow manifestation of the symptoms.

A primary cardiovascular event could be a possibility. However, the reversible nature of cardiorespiratory arrest, associated loss of consciousness, convulsions combined with the absence of post-operative cardiovascular sequelae are all suggestive of LA toxicity as the more likely cause.

To conclude, pulmonary oedema can be the initial manifestation of toxicity with LAs in patients undergoing eye surgery under regional blocks. Patients with pre-existing cardiovascular disease have a lower threshold for this presentation because they are more likely to have suboptimal left ventricular function.[13] The pulmonary oedema may or may not be accompanied by the classical features of LA toxicity. The condition is reversible, constant monitoring and presence of an anaesthesiologist in the operation room is of utmost importance for the successful management of this event.

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Announcement

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Family Benevolent Fund (FBF) is one of the welfare programs of Indian Society of Anaesthesiologists (ISA). It is registered under the Societies Registration Act. Please visit the website www.isafbf.com. Membership is limited only to ISA members and President and Secretary are in the executive body of FBF. ISA member can be a member of FBF by paying the Membership fee depending on the age of members.

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Age proof is required, the membership fee increased from April 2010. Immediate settlement of Fraternity amount to the nominee, incase of death of a member. So far 14 members were supported with an amount of Rs.18 Lakhs.

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