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Neurological Complications Following a Retrobulbar Block. Is There a Role for Intravenous Lipid Emulsion (ILE)? A Case Report and Literature Review

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Abstract

Regional anaesthesia in eye surgery can lead to life-threatening complications such retrobulbar haemorrhage, local anaesthetic systemic toxicity (LAST), due to intra-arterial injection, and brain stem anaesthesia (BSA). Intravenous lipid emulsion (ILE) has been shown to reverse cardiac arrest induced by LAST but there are also reports that suggest its role in reversing neurological complications due to local anaesthetic (LA). Here it is presented a case of a woman undergone to retrobulbar block for a vitrectomy who developed neurological complications, maybe related to BSA, in whom ILE was also administered, with a successful recover of pre-block neurological status.

Keywords: Intravenous lipid emulsion; Retrobulbar block; Brain stem anaesthesia; Local anaesthetic systemic toxicity

Introduction

Ophthalmic surgery can be performed under general, regional or topical anaesthesia (GA, RA or TA). Among RA techniques, peribulbar and retrobulbar blocks are the most commonly used, and those mostly associated with complications. In fact due to the particular anatomy of the eye, life-threatening complications such as retrobulbar haemorrhage, intra-arterial injection, with ensuing "local anaesthetic systemic toxicity" (LAST), and brain stem anaesthesia (BSA) are possible [1]. The first one is characterized both by neurological involvement, leading up to a coma, and by cardiovascular effects that can conduct towards a cardiac arrest. The second complication, described in 0.13-0.79 % of all retrobulbar blocks [2], starts with drowsiness, headache and can progress eventually towards respiratory arrest and hemodynamic instability [3]. Intravenous lipid emulsion (ILE) has been shown to reverse cardiac arrest induced by LAST [4] and there are only a few reports about its use in neurological complications due to LA [5]. We present here a case of retrobulbar block complicated by systemic adverse effects, where ILE was also administered. A mixture of ropivacaine and mepivacaine was used for the block. Informed consent to publish the case was obtained from the patient.

Case Presentation

A 70 year-old female patient had to undergo vitrectomy for macular hole and pucker in retrobulbar block. Her past medical history included hypertension, non insulin dependent type II diabetes mellitus, hypercholesterolemia, past appendectomy, hysterectomy for uterine fibroid, oophorectomy for cancer, thyroidectomy for Basedow disease. Vitrectomy on the other eye for retinal detachment had been previously performed under GA without any consequences. She also had been treated with a PTCA with and drug eluting stent for coronary artery disease 9 months earlier. Before carrying out the block SpO_2 was 100% in room air oxygen, non invasive blood pressure (NIBP) 158/76 mmHg, HR 70 bpm rhythmic and her respiratory rate (RR) 15/min. Retrobulbar block was carried out by the surgeon with a mixture of 5 ml ropivacaine 1% and 5 ml mepivacaine 1%. The anaesthetic mixture was injected through an Atkinson needle 25 G diameter and 22 mm length. Two minutes after the block the patient developed sinus tachycardia with HR 113 bpm and severe hypertension with NIBP 224/113 mmHg; headache and dizziness were also reported by the patient. After Urapidil 15 mg IV NIBP dropped to 160/74 mmHg and HR to 90 bpm. Five minutes after the completion of the block, the patient did not respond (GCS 3) and respiratory arrest followed. Hemodynamic stability was documented by a NIBP of 155/70 mmHg and HR 85 bpm. She was promptly intubated and mechanical ventilation was started. Assuming that the clinical picture was

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due to LAST caused by inadvertent intra-arterial injection or to BSA, ILE (20% Intralipid®, B Braun Melsungen AG, Melsungen, Germany) was administered to the patient in order to prevent the progress of toxicity. The dose was 1.5 mL*kg-1 of total body weight (TBW) bolus followed by an infusion of 0.25 mL*kg-1*min-1.

Transfer to the ICU was planned and 20 minutes after the admission (50 min after the block) the patient had completely recovered her pre-block neurological status and she was promptly extubated. A CT scan of the head, performed before extubation, did not show any neurological abnormality. No sedation had been given during that period. ILE therapy was interrupted after the extubation. A total amount of 580 mL, 10 mL*kg-1 of ILE was administered, as suggested [6]. The patient was monitored for the following 12 hours in the ICU and then discharged to the ward. No blood test abnormalities were registered and no further complications developed caused by ILE. Vitrectomy was rescheduled and performed 2 months later under GA without any complication.

Discussion

Neurological complications due to retrobulbar block are unpredictable in terms of severity and clinical presentation. The broad spectrum of clinical scenarios that can occur [7,8] depend on local anaesthetic volume and its concentration, depth of the needle and spread to the cerebral area [9]. Intra-arterial injections have been reported even following a negative–aspiration test [10]. However, the development of severe hypertension and sinus tachycardia two minutes after the block makes this hypothesis unlikely in our patient. Vagus and glossopharyngeal nerve involvement resulting in parasympathetic blockade and abolition of carotid sinus reflex, respectively, appears more likely [11]. In fact it is more feasible that local anaesthetic (LA) spread had had an intra-cerebral or subarachnoid involvement through the optic nerve sheath leading to BSA. Usually these two potential mechanisms occur when needle tip perforates the sheath and the injected volume can spread in the subdural or in the subarachnoid space respectively. In this case report, the needle utilized was shorter than those commonly used to perform this block but local anaesthetic volume was 10 mL, greater than the usual 5-6 mL, and this could explain why neurological complications appeared. Another relevant consideration is that neither paralysis nor seizure activity developed, because the patient had a prodromic headache and dizziness, then a sudden loss of consciousness and respiratory arrest ensued.

ILE has been applied to reverse local anaesthetic cardiac toxicity [4,12] but there is also an increasing interest in its use to reverse neurological complications. Spence reported a case of inadvertent intravenous bupivacaine injection with central nervous system toxicity after epidural analgesia for labour. In that case the patient was treated with 20% lipid emulsion bolus of 100 ml followed by a continuous infusion of the drug. No cardiovascular toxicity developed and no eventful post-operative course was reported [5]. Another case of LAST after a lumbar plexus block with neurological symptoms, hypotension and ECG abnormalities was successfully dealt with 20% Intralipid bolus with a rapid normalization of ECG and a complete reverse of neurological status [13]. McCutchen and Gerancher [14] reported about a patient who had had a combined femoral and sciatic block and developed general seizures and ventricular tachycardia. He was treated with a bolus of 100 mL of 20% lipid with an additional infusion of 400 mL of the drug in the following 15 minutes, preventing cardiac arrest and obtaining a complete reverse of neurological symptoms within two hours. Lange et al. [15] have described a case of a patient who developed systemic neurological complications from lidocaine skin infiltration. In this case the patient was treated with a bolus of 1.5 mL/kg of 20% lipid emulsion not followed by a continuous infusion of the drug with improvement of neurological status and complete recovery within 12 hours.

It seems that ILE rapidly decreases tissue and plasma concentrations of LA [4] and potentially hastens the disappearance of neurological symptoms [13], possibly due to a “lipid-sink” mechanism, where infused lipids bind local anaesthetic/s lowering their /its plasma and tissue concentration with a partitioning effect [16]. It is also reasonable that ILE could act with a “channel” mechanism, where lipids reduce sodium channel inhibition by LA [17].

When BSA occurs a full recover it is difficult to be predicted in terms of time for the different spread of LA in the single cases and for metabolism of LA in itself [1,7]. In this situation we immediately started with a lipid bolus followed by an infusion with the main aim to avoid further cardiovascular toxicity. A full recovery of neurological status was obtained within 50 minutes but we don’t know how much ILE has contributed to speed up it. It is necessary to have more studies to prove the efficacy of ILE in neuronal cells but lipid infusion is mandatory to be considered in life-threatening conditions related to LAs in order to avoid worsening and progressing of their complications.

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Author’s Contribution

I.C., C.A., G.P. Patient recruitment, data collection and writing up of the first draft of the paper.

S.F., A.R. DG Scientific contribution to discussion of the case report.

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