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Review

Brainstem anaesthesia following single medial canthal peribulbar block: A case report and review

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ABSTRACT

Brainstem anaesthesia, the most dreaded complication of regional opthalmic anaesthsia has been reported with all routes with the exception of medial canthal peribulbar blocks. We report the first case of brainstem anaesthesia following a single medial canthal peribulbar block in a patient scheduled for cataract surgery and explore the factors that set the stage for this life-threatening complication. These include: A relatively short axial length of the globe, needle trajectory, position of the needle tip in relation to the posterior plane of the globe,and, the optic nerve. We introduce the concept of 'Cornea to Canthus distance' and urge that it is factored in when estimating the depth of needle tip during medial canthal peribulbar approach to avoid inadvertent retrobulbar intraconal injection. This case highlights that the ingress of local anaesthetic into subarachnoid space is unpredictable, and can occur without a direct neural breach. This case, unfortunately, was also complicated with a challenging airway. We reiterate the importance of the immediate availability of advanced resuscitative facilities, and, an experienced anaesthetist for all units providing regional ophthalmic anaesthesia.

Contents

1. Case report

Brainstem anaesthesia due to inadvertent subarachnoid spread (SAS) of local anaesthetic (LA) is a rare complication of ophthalmic blocks. Sharp needle based deep injections such as retrobulbar intraconal blocks are more commonly implicated than extraconal peribulbar blocks. To our knowledge brainstem anaesthesia has never been reported with single medial canthal peribulbar block techniques. We report a case of brainstem anaesthesia following a routine single medial canthal peribulbar block for an elective cataract surgery.

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A 52-year-old woman with bilateral cataracts was scheduled to have cataract surgery to her left eye under local anaesthesia in our institution. Her co-morbidities included systemic hypertension and borderline diet controlled diabetes. She had had one episode of self limiting supraventricular tachycardia seventeen years previously. Her medication included atenolol, bendroflumethiazide and duloxetine. She was known to be allergic to cyclizine. On examination she was obese, had a small mandible, and a short neck. The axial length was 21.82 mm in the right eye and 21.86 mm in the left eye.

Following baseline monitoring of vital signs and topical installation of 0.5% w/v minims proxymetacaine hydrochloride to her left eye, the peri-orbital skin was cleansed with 5% aqueous povidone iodine. The block was performed by an experienced consultant anaesthetist (author HN). A 25 mm 25 gauge sharp bevel needle was inserted at the medial canthus of the left eye to its full depth in a direct vertical direction. No contact with the bone was made and there was no resistance to the needle placement. Following a negative aspiration, a mixture of 3 ml of plain 2% lignocaine and 3 ml of 0.75% levobupivacaine with 300 units of hyaluronidase was slowly injected. A neutral gaze was maintained throughout the procedure. No sedation was used and the 'Tethering test' was not performed. Total akinesia of the left globe and lids ensued rapidly after withdrawal of the needle. Soon after this the patient reported feeling unwell and said that her "head was feeling funny", her speech became slurred and she quickly became unresponsive, and, apnoeic. Manual ventilation of lungs and resuscitation were commenced immediately. Her contralateral pupil was noted to be dilated; the operative side was already dilated due to preoperative mydriatic drops. Endotracheal intubation was attempted twice but was unsuccessful due to an overbite, short neck and small mandible. A supraglottic airway (laryngeal mask, size 4) was eventually inserted and artificial ventilation of the lungs continued.

The patient was noted to have significant supraventricular tachycardia (HR 150 bpm) and hypertension (185/130 mm Hg). There were no skin rashes, erythema or bronchospasm. Intravenous Labetalol (10 mg), Acetazolamide (500 mg) and 50 ml of 20% Mannitol were administered. Her blood sugar was noted to be 5.9 mmols $^{-L}$. Her blood biochemistry and haematology results were unremarkable.

The patient remained unresponsive, areflexic and apnoeic for the best part of the hour, IPPV of the lungs was therefore continued with a mixture of oxygen and air, and the surgery was deferred. She eventually resumed spontaneous ventilation and regained consciousness. Upon recovery her vision had returned to the pre-block level and there were no residual neurological sequelae. The patient had no recollection of the preceding events. She was admitted for overnight observations where she remained stable and was discharged home the following day.

2. Differential diagnosis

Differential diagnoses considered during the period of resuscitation were:

Subarachnoid spread of local anaesthetic, inadvertent intravascular injection, overdose/reaction to LA, medication error and another unrelated intracranial event. Inadvertent intravascular injection was ruled as the test aspiration prior to injection had been negative and there was no periocular haematoma to indicate any vascular breach. There was no obvious injury to the eye. Her cardiovascular picture did not support overdose of local anaesthetic. Throughout the period of resuscitation she did not have any abnormal muscle movements, rashes or convulsions. Her recovery was complete and the lack of residual neurological sequelae ruled out an independent intracranial event.

On the following day and at two months revisit her best corrected visual acuity was unchanged. Fundus examination, Visual field test, Ishihara colour charts, Magnetic resonance imaging (MRI), Electrodiagnostic tests, Retinal nerve fibre layer scan (Heidelberg Retina Tomogram HRT) and Optic nerve function were performed. They were all found to be normal. This helped to rule out trauma to the globe and the optic nerve.

3. Diagnosis

The clinical course, followed by the full recovery with no residual visual or neurological defect confirmed the working diagnosis of total brainstem anaesthesia. It was unlikely that a direct trauma to the optic nerve sheath or the nerve itself was responsible for the ingress of local anaesthetic (LA) into the subarachnoid space (SAS). The patient had not experienced any sharp or sudden pain during the insertion of the needle and the detailed examination of the optic nerve and its function did not support a direct injury. She was counselled and rescheduled to have surgery at a later date. She eventually underwent a successful operation to her left eye some months later under topical anaesthesia.

4. Literature review and discussion

Subarachnoid spread of local anaesthetic following eye blocks is rare. The overall reported incidence is 0.3%–0.8% [1,2]. Traditional dual peribulbar extraconal blocks have an incidence of 0.02%. Retrobulbar intraconal blocks possibly have much higher incidence. Pressure orbitography reveals it to be 3 in 150 (2%). Literature reports only one case of SAS of LA with sub-Tenon's blocks, it was presumably caused by dural breach from scissors dissection [3]. There are no previous case reports of subarachnoid spread of LA with a single medial canthal peribulbar injection technique.

Subarachnoid spread of local anaesthetic presents in multitudes of clinical features depending on the amount of LA that comes in contact with intracranial structures. Amaurosis, confusion, deafness, dizziness, tinnitus, slurred speech, twitching, shivering and extraocular muscle palsy often herald a more sinister picture. These warning signals may deteriorate to more serious signs such as arrhythmia, hypertensive crisis, convulsions and cardio-respiratory arrest. The onset of may be ultra rapid or may sometimes be delayed [4–10].

Our patient had a rapid onset heralded by marked tachycardia and hypertension which responded well to intravenous labetalol. (Fig. 1). This sympathetic over activity is pathognomonic of cephalic ingress of local anaesthetic [11]. LA entry through this route inhibits

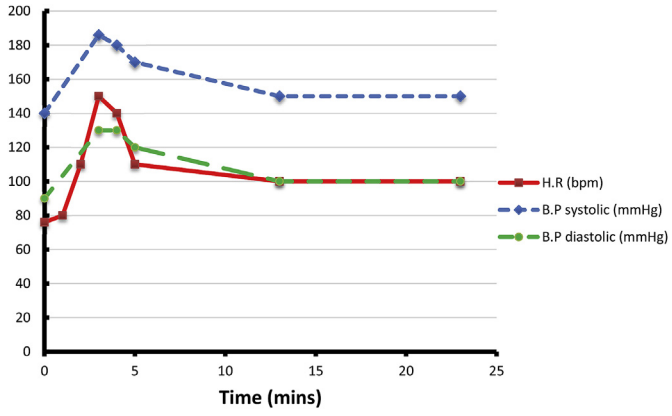

Fig. 1. Haemodynamic observations.

the depressor function (vagus nerve) ahead of excitatory centres. Blockage of glossopharyngeal nerve abolishes carotid sinus reflex resulting in tachycardia in presence of hypertension. This is in contrast to the total spinal block from a misplaced spinal injection where LA moves from caudal to cephalic direction successively blocking thoracic sympathetic nerves, intercostal muscles, diaphragm and central cardio-stimulatory centres manifesting as hypotension, hypopnoea, bradycardia, and finally loss of consciousness if enough LA reaches the brainstem.

It is noteworthy that the local anaesthetic mixture used in this case was a plain solution and did not contain any vasopressor. Therefore the tachycardia and hypertension seen in our patient must have been due to a cephalic ingress of LA into the subarachnoid space.

4.1. Mechanism of subarachnoid spread of LA

It was quite difficult to understand how the LA had spread to the central nervous system from a single medial canthal peribulbar block. It is well known that a direct optic nerve contact with the needle is the commonest reported cause of subarachnoid spread of local anaesthetic [11]. It is more likely to occur during ***Intraconal retrobulbar blocks***, where long needles are inserted into the orbit via inferolateral route. In these techniques, an upwards and medial needle trajectory sets a stage for the neural breach. Atkinson's technique of using long needles and a superomedial gaze (now obsolete) is particularly risky as optic nerve drops down and out into the path of the needle [12].

Inferolateral peribulbar extraconal blocks pose slightly less but similar types of perils as retrobulbar intraconal ones. Edge and Davis described a case of brainstem anaesthesia following a dual extraconal block with a 37 mm needle. The first injection was inferolateral and second superomedial [13]. Another case following an ***extraconal retrobulbar*** block with a 25 mm needle [14] suggests that the angle and the trajectory of the needle have a more important role to play than its absolute length. ***Single inferolateral extraconal*** blocks are also not entirely safe as the needle needs to negotiate the orbital ridge and then travel along the floor of the orbit. In doing so it changes its trajectory along the way. ***Inferonasal extraconal*** block has not been exempt either [9]. In this case, a delayed brainstem anaesthesia following a single, inferonasal injection using a standard 25G, 25 mm needle was described. It couldn't be ascertained if it was due to an unrecognised intravascular injection, or a direct neural contact.

'Single' Medial canthal peribulbar blocks do not call for the change in the direction of the needle and are deemed much safer than other needle blocks, especially in axial myopes [15]. Introduced by Hustead and Hamilton as a part of a dual injection technique the medial canthal peribulbar extraconal block is performed with the eye in the neutral gaze. In this technique a 25 mm 25G needle is inserted at the medial canthus and advanced directly backwards parallel to the medial wall of the orbit. This block has evolved successfully and has been adapted as a single injection technique [15, 16,17]. The needle tip isn't meant to go beyond the posterior plane of the globe. As the optic nerve lies in the middle of the orbit in the neutral gaze it is well protected from the advancing needle. A change in the direction of the gaze, or a 'Tethering test' may however put the nerve at a risk of trauma. In out-gaze, direct up-gaze, up and out gaze, and down and out gaze, the optic nerve moves medially thus coming in the path of the needle [18]. The block in our patient was performed with the eye in the neutral gaze; there was no deliberate misdirection of the needle and no bony contact. Sedation was not used. The 'Tethering test' was not performed. Hence it became quite difficult to understand how LA had managed to reach the subarachnoid space of this patient.

4.2. 'Cornea to canthus' distance and needle depth

One unrecognised fact with respect to the medial canthal peribulbar blocks is the location of the medial canthus in relation to the anterior surface of the cornea (the reference point from where the axial length is measured). The plane of the medial canthus is usually located 5–7 mm back from this surface. This means that if a 25 mm long needle is inserted up to its hub at the medial canthus, it would have its tip much further back in relation to the posterior plane of a normal globe. Any indentation of the soft tissues during procedure would push the needle tip even deeper into the orbit thus risking a neural injury.

The distance between the anterior surfaces of the cornea to the medial canthus in our patient turned out to be 7 mm (Fig. 2). The axial length of the globe was only 21.86 mm. The block had been performed with a 25 mm long needle inserted all the way to its hub. Thus mathematically speaking it appears that the tip of the needle in this patient would have been approximately **10.14 mm (1.014 cm) behind** the posterior pole of the globe and in all likelihood in the posterior intraconal space. The ultra rapid onset of the

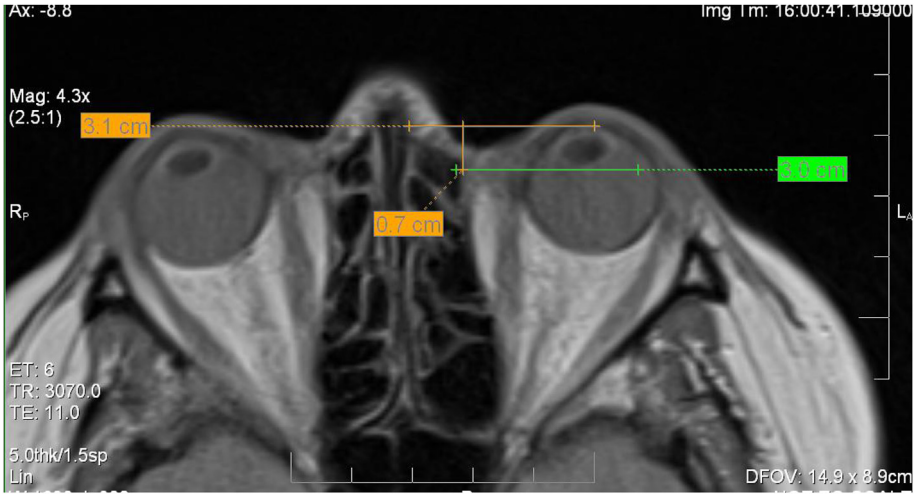

Fig. 2. Cornea to medial canthus distance, left eye.

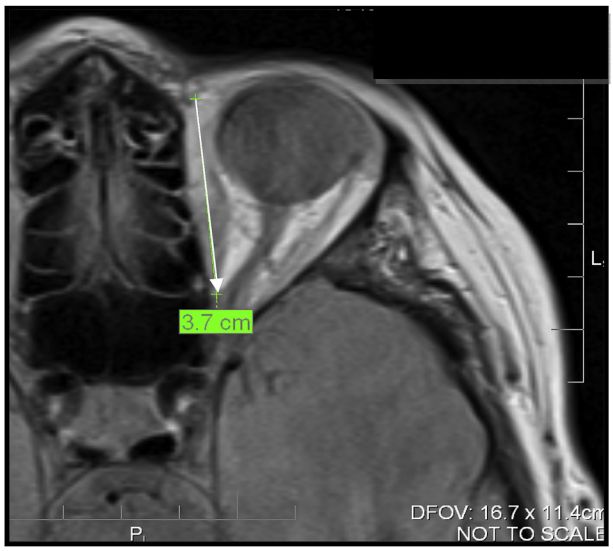

Fig. 3. Medial canthus to optic nerve distance at apex, left eye.

globe and lid akinesia suggests an inadvertent intraconal injection of local anaesthetic in this case as a true peribulbar extraconal block would have taken much longer to be effective.

The MRI of the orbit of our patient showed that the distance from the medial canthus to the optic nerve at the apex was 37 mm (3.7 cm) (Fig. 3) Anatomical studies have shown that the distance from the lachrymal crest to the medial edge of the optic foramen is 42 mm in adult males [19]. Even when allowing for gender differences in the bony anatomy, going by the clinical presentation in this case we don't think that the needle had actually approached the optic foramen itself. It does however appear that on this occasion the needle tip had ended up very close to the optic nerve, and, being at 32 mm depth it was just short of piercing it. It is noteworthy that the apex of the orbit is very crowded. The optic nerve is generally 3.5 mm thick and anatomically it presents itself proximal to the bony foramen. It is entirely plausible that in our patient a high concentration and the large volume (6 mls) of LA deposited in such close proximity of the nerve at the tight apex had led to a subarachnoid spread of LA even though there was no evidence of direct physical trauma to the nerve itself.

4.3. Mechanisms of intraneural transgress of LA

A direct neural breach of the optic nerve, commonly seen during long needle intraconal retrobulbar blocks, is the most obvious aetiology of SAS of LA [20]. Clinical and radiological pictures in our patient did not demonstrate any direct dural breach. Any inadvertent intravascular injection within the nasal cavity or orbit was ruled out as initial test aspiration prior to injection was negative, there was no periosteal contact during needle placement, and, no orbital haematoma either. It was therefore difficult to understand the mechanism of the intracranial spread of LA.

One possibility lay in the microanatomy of the optic nerve. The optic nerve is actually a direct extension of diencephalon. It has meningeal dura, arachnoid and pia mater. The periosteal dura is attached to the optic foramen. Anatomical studies show that the optic nerve is rich in arachnoid villi. Most of these villi lie well within the meningeal dural sheath, some however protrude through it. These exposed villi form conduits though which the local anaesthetic can get absorbed and spread to the contiguous subarachnoid spaces reaching the chiasmal cistern, contralateral optic nerve, interpeduncular structures and finally, the medullary centres [21].

We suspect that the exposed arachnoid villi within the intraconal space may well have formed the route through which LA gained access to the CSF in this patient.

4.4. Laterality of the block

The left eye is damaged more frequently during eye blocks than the right as most people are right handed [22]. The procedure to this patient's left eye was performed from the head end by a right handed operator (author HN). A limited clearance due to the large nasal bridge perhaps had a role to play in a lateral misdirection of the needle towards the globe resulting in an inadvertent ***intraconal*** placement of the needle tip.

4.5. Airway management

The airway access was unduly difficult in this patient. Two attempts at the endotracheal intubation had failed. A laryngeal mask airway fortunately provided a clear airway. It was just as well that the patient did not have a full stomach otherwise the clinical course might have been very different.

5. Conclusion

This case reiterates that no block is entirely safe and well intentioned extraconal placements of the needle may indeed become intraconal. With respect to medial canthal peribulbar blocks, ignoring the Cornea to Canthus distance will lead to placement of the needle tip deeper than intended. LA placed at the apex of the orbit risks its central spread as exposed arachnoid villi of the optic nerve form a route for absorption even when the nerve is not breached as such. Insertion of needles in the left eye by a right handed person standing behind the head end may lead to misdirection of the tip toward the globe due to prominent brows or a broad nasal bridge.

Resuscitation facilities as well as the immediate availability of experienced help are absolutely essential wherever eye blocks are performed.

Conflict of interest

"The authors declare that they have no conflict of interest."

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