WHAT HAPPENS TO INTRAOCULAR PRESSURE AFTER PERIBULBAR ANAESTHESIA?

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ABSTRACT

BACKGROUND

Most of intraocular surgeries are done under local anaesthesia. The peribulbar anaesthesia provides adequate anaesthesia and akinesia. There is no reported intraoperative and/or postoperative amaurosis. The peribulbar anaesthesia provides adequate anaesthesia and akinesia. The disadvantages of it are the larger quantity of the aesthetic agent. Increasing the bulk load on the globe and a reported rise of intraocular pressure.

MATERIALS AND METHODS

A study of fifty cases was conducted in patients who received peribulbar anaesthesia undergoing cataract extraction with intraocular lens implantation and their intraocular pressures were noted and studied after giving the peribulbar anaesthesia all given by the same surgeon.

RESULTS

This study did show that the peribulbar anaesthesia increases the intraocular pressure in all the cases. The external ocular compression indeed helps to dissipate the anaesthetic load thereby reducing the enormous rise in IOP, which is only expected if you recollect the fact that the eyeball occupies one sixth of the total volume of the orbit that is 5 mL and 30 mL. The volume of peribulbar anaesthesia (6 mL) does add its effects to increase the IOP. Hence, a properly planned post peribulbar compression helps to minimise the transient rise in IOP.

CONCLUSION

Summarising the study, it is better to give peribulbar injection initially followed by external ocular compression after a delay of at least 2 to 5 minutes. It is also advised that an initial compression maybe given in slightly risk cases, so that the peribulbar-induced rise may not be alarming. The anaesthetic solution maybe fragmented and the second injection maybe delayed by 5 minutes or omitted if good akinesia and anaesthesia are achieved already.

KEYWORDS

Peribulbar Anaesthesia, External Ocular Compression, Intraocular Pressure.

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BACKGROUND

The advent of peribulbar anaesthesia revolutionised the intraocular surgeries. In fact, it had brushed aside the oldest method of local anaesthetic route, i.e. retrobulbar anaesthesia because of its simplicity, safety and less chance of optic nerve injury even in the hands of a novice ophthalmic surgeon.¹

Peribulbar anaesthesia for intraocular anaesthesia for intraocular surgery was introduced by Charles Kelman in 973 and was popularised by Davis and Mandal in 1986.² It has since swept the time tested pair of facial and retrobulbar combination.

The peribulbar anaesthesia provides adequate anaesthesia and akinesia. There is no reported

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intraoperative and/or postoperative amaurosis.^{3,4} The disadvantages of it are the larger quantity of the anaesthetic agent required to produce the desired anaesthetic effects, thereby increasing the bulk load on the globe for a transient period of time. Chemosis and a reported rise of intraocular pressure are the additional drawbacks. Hence, this study was taken to study the pattern of Intraocular Pressure (IOP) changes after peribulbar anaesthesia.^{5,6,7}

Aim and Objectives

It was decided to undertake a study of a rise of intraocular pressure in our setup after peribulbar anaesthesia following or preceding the extraocular compression and assess the pattern.

MATERIALS AND METHODS

A study of fifty cases was conducted in patients who received peribulbar anaesthesia undergoing cataract extraction with intraocular lens implantation.

They were divided into three groups-

Group A consisted of 10 patients.

Group B consisted of 30 patients.

Group C consisted of 10 patients.

MATERIALS AND METHODS

Intraocular pressure was recorded initially in all these cases. Group A received peribulbar and IOP was recorded in the first minute, then every minute for the next 5 minutes. Group A received no ocular compression following peribulbar injection. Group B received peribulbar injection and had external ocular compression. The intraocular pressure was recorded after giving peribulbar injection and at 1 minute and 5 minutes of external ocular compression. Group C received initial external ocular compression followed by peribulbar injection. The IOP was recorded after 1st and 5th minute of external ocular compression and in the 1st and the 5th minute after peribulbar block, which succeeded ocular compression. The results were compared. The study was conducted on fifty patients who were to be operated for cataract with intraocular implantation. The recording of intraocular pressure was done with Schiotz tonometer. All received the same anaesthetic solution of same combination and same quantity and given by the same surgeon. The agents used were Xylocaine 4 mL of 2% with 1:1,000 adrenaline and 0.5% bupivacaine with 150 units of hyaluronidase taking about one minute in the process. 5 mL of this anaesthetic solution was injected through the lower eyelid at the junction of lateral one third and medial two third and 3 mL was injected into the upper eyelid at the supratrochlear notch, i.e. at the junction of the medial third and lateral two third using 26 gauge needle taking about a minute to complete the procedure.

Inclusion Criteria

The criteria for selection of these patients were-Age group - 50 to 70 years. Axial length - 2 to 25 mm.

Exclusion Criteria

None had a history of glaucoma, diabetes mellitus or hypertension. None of the selected cases showed an intraocular pressure of above 20 mm initially.

Procedure- The patients were divided into three groups as mentioned. Group A had their initial IOP recording done. Then, the peribulbar injection was administered as described. Following that, external ocular compression was

given for 5 minutes. Their IOP was monitored every minute for 5 minutes. At the end of 5 minutes, external ocular compression was given using balanced weight for 5 minutes with 10 seconds release after every 50 seconds of compression. The IOP was recorded after the external ocular compression.

Group B had their initial IOP measured. They received the peribulbar injection as described. Their post peribulbar block IOP was recorded immediately and ocular compression was given using balanced weight as before. The IOP was recorded at the end of 5 minutes.

Group C patients had their initial IOP recorded. These patients first received external ocular compression for 5 minutes as described above.⁶ Their IOP was recorded after 5 minutes of compression, and at the end of it, the peribulbar block was given. The IOP immediately after giving the block and after 5 minutes of completion of the block were recorded. The results were tabulated and the mean IOP in each group compared using paired t-test and z test.

RESULTS

On analysis of results, all patients in Group A, which did not receive ocular compression did show an immediate and significant rise of IOP (p<0.01). Subsequently, the IOP rapidly began to fall from the second minute onwards, and by the fifth minute, the IOP in all the 10 cases had fallen to initial levels or slightly lower. The mean IOP was 14.4 (S.D. 2.26). After peribulbar injections, it became 24.4 (S.D. 9.35), i.e. a rise of 10 mm (S.D. 8.12) and by the fifth minute, it had fallen to a mean IOP of 11.4 (S.D. 1.8). On adding compression thereafter, the IOP got further reduced by 1.9 mm (<0.01).

All the 30 patients in group B did show a variable and significant IOP rise after the peribulbar injection (P <0.0101). The mean initial IOP was 14.6 mm of Hg (S.D. 2.59). Then, the IOP immediately after the peribulbar block, the mean IOP was 24.7 mmHg (S.D. 7.3) and after the external ocular compression for 5 minutes, the mean IOP was 9.5 mm of Hg (S.D. 2.9).

The following table shows the results at a glance. The pre and post injection mean IOP with standard deviation given in brackets.

Groups	Pre-Injection IOP	Initial Compression	Post Injection IOP	Post Compression	Final IOP	Added Compression
Group A No compression	14.4 (2.26)		24.4 (9.3) P< 0.01		11.4 (1.8)	9.5 (2.03)
Group B with compression	14.6 (2.59)		24.7 (7.3) P <0.001	9.5 (2.1)	9.5	
Group C (initial compression)	16 (2.1)	(1st) 14.4 (2.38) P <0.001 (5th) 13 (2.53) P <0.06	19.1 (8.10) P <0.05		11.4 (2.61) P <0.001	

Table 1. Results at a Glance

This points to the fact that external ocular compression does not seem to alter the final IOP whether it is immediately given or after a time delay following the peribulbar injections.

Group C revealed that the initial mean IOP as 16 mmHg (S.D. 2.71). These cases did receive an initial extraocular compression after the topical Xylocaine. The post compression IOP at first minute after compression as 14.4 (S.D. 2.38) and is significant p <0.001. The mean IOP at the end of 5 minute compression was 13 mmHg (2.52), which showed reduction at 5-minute level (p<0.05). These cases subsequently received peribulbar injection as described. The mean IOP at the end of injection was 19.1 (S.D. 8.10), which fell to a mean IOP of 11.4 mm of Hg (2.61) after 5 minutes of no compression period.

Observations did show 3 cases showing a rise in IOP (P >0.1) following external ocular compression though insignificant poses a further question? Does the compressive devices do themselves cause an elevation of IOP? These finding further questions the use of immediate external ocular compression following the peribulbar injection. Group C cases however showed a significant fall in IOP following initial (1st minute external ocular compression (p<0.001) and at 5 minutes (P 0.05). All the 10 cases again showed a rise of IOP after peribulbar injections (p < 0.05). None of these 50 cases did have any postoperative complications. All did have excellent anaesthesia and akinesia. Those cases who did receive initial compression did not have any problems either during external ocular compression intraoperatively.

DISCUSSION

The main aim of this study is to examine the pressure rise after peribulbar anaesthesia, which was also reported in the literature (-2, 3). It was found that all the 50 cases did show significant elevation after the peribulbar anaesthesia. Some cases (40%) did show a significant and enormous rise of IOP from base levels. Further study in such type of cases needs to be done as to know why it does increase enormously considering that all the parameters were almost alike in these cases. The author also did not only notice that ocular

compression helps to achieve a final IOP levels much lower than those cases, which did not receive external ocular compression,⁷ but also noticed that a delayed compression in these cases can also produce the desired drop in IOP as well as it can eliminate a possible device induced rise in IOP at least initially. The author suggests that a delayed external ocular compression, say 5 minutes after the peribulbar injection may help to avoid the "double effect."

CONCLUSION

Summarising the study, it is better to give peribulbar injection initially followed by external ocular compression after a delay of at least 2 to 5 minutes. It is also advised that an initial compression maybe given in slightly risk cases, so that the peribulbar-induced rise may not be alarming. The anaesthetic solution maybe fragmented⁷ and the second injection maybe delayed by 5 minutes or omitted if good akinesia and anaesthesia are achieved already.

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