DOI: 10.7860/JCDR/2026/67908.22249

Original Article

Anaesthesia Section

The Effect of Midazolam Premedication on Intraocular Pressure Following Induction of Different Intravenous Anaesthesia: A Randomised Clinical Trial

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ABSTRACT

Introduction: Thiopentone sodium, ketamine and propofol are commonly used intravenous induction agents for endotracheal intubation with suxamethonium and long-acting muscle relaxants for the maintenance of anaesthesia with air, oxygen and nitrous oxide. Premedication, induction agents, muscle relaxants, inhalational anaesthetics and anaesthetic technique can influence Intraocular Pressure (IOP). These factors are often not considered in anaesthetic practice.

Aim: To compare changes in IOP after various intravenous induction agents with premedication using midazolam or without premedication.

Materials and Methods: This triple-arm randomised, single-blinded clinical trial was conducted at Government Medical College, Latur, Rajasthan, India, from January 2023 to August 2024 included 210 patients of either sex, aged 20-45 years, scheduled for various operative procedures. They were randomly divided into two main groups of 105 each, with subgroups according to the induction agent and whether midazolam premedication was given. Group I-A: thiopentone 5 mg/kg; Group I-B: ketamine 2 mg/kg; Group I-C: propofol 2 mg/kg. Group II-A: thiopentone 5 mg/kg + midazolam 2 mg; Group II-B: ketamine 2 mg/kg + midazolam 2 mg; Group II-C: propofol 2 mg/kg + midazolam 2 mg. Before induction of anaesthesia, all patients were informed and consented for intraocular pressure measurement. The IOP was measured with a Schiotz tonometer at predefined time intervals. Differences in demographic data and baseline values between groups were analysed using one-way Analysis of Variance (ANOVA). For comparisons of observations within and between groups, repeated-measures ANOVA was used.

Results: There were no statistically significant differences among the groups in terms of demographic variables, American Society of Anaesthesiologists (ASA) classification, type of surgery, or preoperative IOP. Intragroup analysis showed a significant increase in IOP in the ketamine group (Group I-B) immediately after induction ($p=0.0231$), whereas propofol (Group I-C) and all Group II subgroups (with midazolam) showed significant decreases in IOP at multiple time points ($p<0.05$). Intergroup comparisons revealed that the addition of midazolam significantly attenuated the rise in IOP across all induction agents, particularly in the early post-induction period ($p<0.05$). No adverse ophthalmic or systemic events were reported during the study.

Conclusion: Midazolam, when combined with intravenous induction agents, significantly lowers IOP, counteracting the rise caused by ketamine and muscle relaxants. Routine intraoperative IOP monitoring is recommended, especially in ophthalmic patients and ketamine should be used cautiously or with sedatives to prevent IOP elevation.

Keywords: Intravenous anaesthetics, Ocular pressure, Schiotz tonometer, Sedative

INTRODUCTION

The Intraocular Pressure (IOP) is a crucial parameter for determining Ocular Perfusion Pressure (OPP) during surgery [1]. An increase in IOP blocks retrograde transport of neutrophilic factors from the brain [2], reduces ocular blood flow [3], leads to optic nerve oedema and ischaemia [4,5] and may result in rare but catastrophic Postoperative Visual Loss (POVL) [6].

In modern anaesthetic practice, premedication is a critical step that enhances patient comfort, safety and overall procedural success. Among the commonly used agents, midazolam, a short-acting benzodiazepine, plays a vital role due to its sedative, anxiolytic, amnestic and muscle-relaxant properties. Its preoperative use is particularly significant when intravenous induction agents such as ketamine, propofol, or thiopentone (thiopental) are administered [7].

Ketamine is a unique anaesthetic with dissociative properties, providing analgesia and amnesia while maintaining spontaneous respiration. However, it is associated with psychotomimetic side-effects such as hallucinations and emergence delirium. Midazolam is commonly used as premedication to counteract these adverse effects. It blunts vivid dreams and agitation that can occur on emergence from ketamine anaesthesia. Additionally, midazolam mitigates the sympathomimetic surge induced by ketamine, resulting in a more haemodynamically stable induction [8].

Propofol is a widely used induction agent known for its rapid onset and smooth recovery profile. However, it may cause pain on injection, hypotension and bradycardia. Premedication with midazolam offers multiple advantages. Firstly, it enhances the sedative effect, allowing a lower dose of propofol and thereby reducing the risk of haemodynamic compromise. Secondly, midazolam's anxiolytic action improves patient cooperation and reduces procedural stress. Lastly, it may provide a synergistic effect that improves the quality of induction and reduces the incidence of intraoperative awareness [8,9].

Thiopentone, a barbiturate, was once a mainstay of induction anaesthesia and is still used in certain settings. While effective, it can cause cardiovascular and respiratory depression, laryngospasm and cerebral excitatory phenomena. Midazolam premedication can reduce excitatory movements, promote smoother induction and provide anterograde amnesia. Like with propofol, midazolam allows

a lower induction dose, which helps minimise side-effects and ensures a more controlled transition to anaesthesia [10].

Premedication, intravenous inducing agents, muscle relaxants, inhalational anaesthetic agents and anaesthetic technique also influence IOP. Many times these aspects are not considered in anaesthetic practice. The literature includes studies [4,7,9] on the effects of different agent combinations and anaesthetic agents on IOP [11]. However, there are no studies specifically addressing agent and premedication combinations that have the least effect on IOP and the haemodynamic response during surgical procedures. This study aimed to compare changes in IOP after various intravenous inducing agents with premedication or without premedication. The primary outcome was change in IOP; the secondary outcome was to assess any serious adverse events.

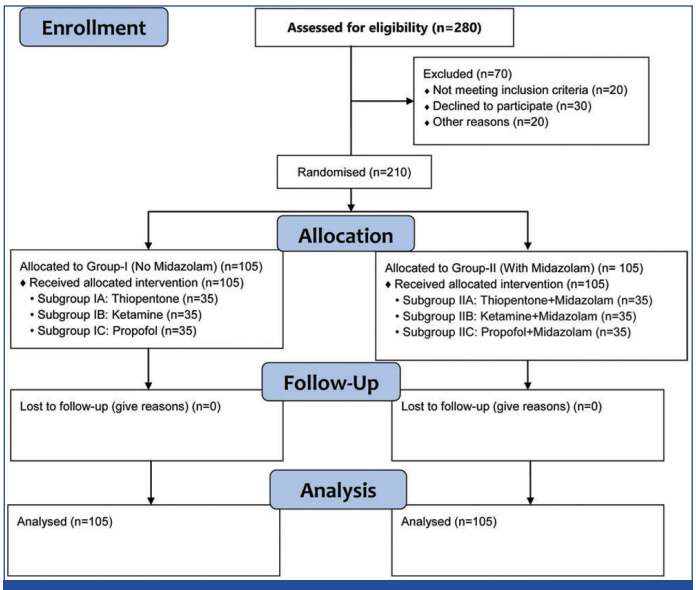
MATERIALS AND METHODS

The present triple-arm randomised, single-blinded clinical trial was conducted at Government Medical College, Latur, Rajasthan, India, from January 2023 to August 2024, after obtaining approval from the Institutional Ethics Committee (IEC/GMC/201/2023). Written informed consent was obtained from all patients prior to the commencement of the study.

Sample size: The sample size was based on a power analysis (α=0.05, β=0.2), which revealed that at least 30 patients should be included in each group.

Inclusion and Exclusion criteria: A total of 210 adult patients of American Society of Anaesthesiologists (ASA) physical status I or II, aged 18-60 years, scheduled for elective non ophthalmic surgeries under general anaesthesia, were included in the study. Exclusion criteria included open-eye injury, glaucoma, intracerebral space-occupying lesions, raised intracranial pressure for various reasons, facial/maxillofacial injuries, cardiovascular diseases (mainly hypertension), hypotension of any cause, low cardiac output states, respiratory compromise, known allergies or contraindications to study drugs, predicted difficulty in intubation, pregnancy and patients receiving any drug known to alter IOP.

Patients were randomly allocated into two main subgroups of 105 patients each based on the use of midazolam as premedication. The two main groups were further subdivided into three subgroups based on the studied drugs: Group I-A - Thiopentone sodium 5 mg/kg; Group I-B - Ketamine hydrochloride 2 mg/kg; Group I-C - Propofol 2 mg/kg; Group II-A - Thiopentone sodium 5 mg/kg with intravenous midazolam 2 mg; Group II-B - Ketamine 2 mg/kg with intravenous midazolam 2 mg; Group II-C - Propofol 2 mg/kg with intravenous midazolam 2 mg [Table/Fig-1].



[Table/Fig-1]: Consolidated Standards of Reporting Trials (CONSORT) flow diagram.

Randomisation was performed using stratified random sampling based on their allocated sequence in the department's hospital review system software for outpatient data management. The patients were blinded to the studied drugs.

Study Procedure

The procedure was uniform across patients, with preoperative IOP measurement undertaken by the same observer, requiring patient cooperation. General anaesthesia was induced after premedication with atropine 0.01 mg/kg and induction with the allocated intravenous agent according to group assignment. After induction, all patients received suxamethonium 1 mg/kg for endotracheal intubation. All patients were intubated with an appropriately sized endotracheal tube and connected to the anaesthesia machine via a Bain circuit or a closed circuit.

Anaesthesia was maintained with 50% nitrous oxide and 50% oxygen on the Bain circuit or closed circuit with controlled ventilation. After the effect of suxamethonium waned, all patients received a long-acting muscle relaxant (atracurium or vecuronium) intraoperatively. All patients were monitored for pulse rate, blood pressure, oxygen saturation, depth of anaesthesia and muscle relaxation, either clinically or via monitors. At the end of the procedure, reversal was achieved with neostigmine 2.5 mg in combination with atropine 1 mg and after full recovery, extubation was performed. Patients were observed postoperatively for any complications related to the anaesthesia technique or the operative procedure.

Before and after induction of anaesthesia, IOP was measured with a Schiotz tonometer weighing 5.5 g in all patients. Preoperatively, after instillation of local anaesthesia with plain lignocaine hydrochloride 2% in both eyes, IOP was noted. IOP was then measured immediately after loss of the eyelash reflex and after administration of the intravenous induction agent. IOP was measured approximately two minutes after induction, following administration of suxamethonium and endotracheal intubation. A further measurement was taken about five minutes after induction, after stabilisation of the patient with controlled ventilation and after cessation of suxamethonium and administration of a long-acting muscle relaxant. IOP was measured again in the postoperative period after complete recovery from anaesthesia.

STATISTICAL ANALYSIS

Statistical analysis involved comparing demographic and clinical variables across groups using ANOVA for continuous data (age, preoperative IOP) and the Chi-square test (or Fisher's-exact test when appropriate) for categorical data (gender, ASA category, type of surgery). Intragroup changes in IOP at different time intervals were assessed using paired t-tests to compare post-induction and postoperative values against preoperative baselines. Intergroup comparisons of IOP between groups with and without midazolam were performed using independent t-tests. All statistical tests were two-tailed and a p-value <0.05 was considered statistically significant. Analyses were performed using Jamovi Project (2020), Jamovi (Version 2.3).

RESULTS

There were no significant differences among the groups regarding age, gender, ASA category, type of surgery performed, or preoperative IOP [Table/Fig-2]. The IOP values at different time intervals are presented in [Table/Fig-3].

Intragroup comparison of IOP at different time intervals:

- Group I-A (Thiopentone): An insignificant increase in IOP was observed.
- Group I-B (Ketamine): A significant increase in IOP was observed.
- Group I-C (Propofol): A significant decrease in IOP was observed at all time intervals.

Demographic variables	Group I-A	Group I-B	Group I-C	Group II-A	Group II-B	Group II-C	p-value
Mean age (in years) (mean±SD)	34.25±5.40	33.15±3.20	32.10±6.15	35.51±3.20	34.20±5.20	31.05±4.15	0.765*
Gender distribution (Male/Female)	22/13	12/23	16/19	12/23	16/19	20/15	0.568**
ASA distribution (Class I/II)	20/15	22/13	24/11	21/14	23/12	22/13	0.746**
Type of surgery n (%)							
General surgery	19 (54.28%)	23 (65.71%)	24 (68.57%)	24 (68.57%)	19 (54.28%)	1 (2.85%)	0.910**
ENT	13 (37.14%)	8 (22.85%)	8 (22.85%)	10 (28.57%)	14 (40%)	24 (68.57%)	
Orthopaedic	3 (8.57%)	4 (11.42%)	3 (8.57%)	1 (2.55%)	2 (5.71%)	10 (28.57%)	
Pre-op IOP (mean±SD)	15.34±1.60	15.39±1.51	15.12±1.54	15.31±1.52	16.21±2.28	15.41±2.00	0.450*

[Table/Fig-2]: Demographic parameters.
*ANOVA test ** Mann-Whitney U test; ENT: Ear, nose and throat

Group	Sub group	Pre-op	Immediately after induction	2 minutes after induction	5 minutes after induction	Post-op
I	A	15.34± 1.60	16.34± 1.30	14.74± 0.80	14.44± 0.80	13.95± 1.05
I	B	15.39± 1.51	17.07± 1.36	16.62± 1.32	16.01± 1.15	15.75± 1.19
I	C	15.12± 1.54	13.88± 1.35	13.30± 1.02	13.19± 0.94	13.23± 0.83
II	A	15.31± 1.52	13.62± 1.59	13.31± 1.49	12.93± 1.29	13.76± 1.07
II	B	16.21± 2.38	14.90± 2.17	14.94± 2.27	14.50± 1.93	15.34± 1.86
II	C	15.41± 2.00	12.90± 1.92	12.47± 1.86	12.17± 1.61	13.58± 1.46

[Table/Fig-3]: IOP at different time interval.
Values presented in (mean±SD)

- Group II-A (Thiopentone + Midazolam): IOP showed a significant decrease.
- Group II-B (Ketamine + Midazolam): IOP decreased significantly at all time points.
- Group II-C (Propofol + Midazolam): A significant reduction in IOP was recorded at all time intervals [Table/Fig-4].

Group	Subgroup	Comparison with preoperative values	t-value	p-value
I	A	Immediately after induction	5.69	0.1594
I	A	2 minutes after induction	4.98	0.2942
I	A	5 minutes after induction	4.21	0.1776
I	A	Post-op	8.41	0.1854
I	B	Immediately after induction	-13.605	0.0231
I	B	2 minutes after induction	-8.13	0.1195
I	B	5 minutes after induction	-4.62	0.1379
I	B	Post-op	-2.95	0.1922
I	C	Immediately after induction	13.21	0.0156
I	C	2 minutes after induction	2.99	0.0178
I	C	5 minutes after induction	3.19	0.0238
I	C	Post-op	-3.15	0.0167
II	A	Immediately after induction	13.40	0.0236
II	A	2 minutes after induction	13.96	0.0167
II	A	5 minutes after induction	16.77	0.0193
II	A	Post-op	8.28	0.0102
II	B	Immediately after induction	11.76	0.0373
II	B	2 minutes after induction	4.73	0.0274
II	B	5 minutes after induction	10.57	0.0416
II	B	Post-op	5.31	0.0281
II	C	Immediately after induction	12.63	0.0169
II	C	2 minutes after induction	15.59	0.0128
II	C	5 minutes after induction	16.19	0.0462
II	C	Post-op	9.55	0.0365

[Table/Fig-4]: Intragroup comparison of IOP at different time intervals.
Paired t-tests were used.

Intergroup comparison of IOP at different time intervals: In all three subgroups (Thiopentone, Ketamine and Propofol), the addition of Midazolam led to a consistent and significant reduction in IOP during the immediate and early post-induction period when compared to Group I. This effect was most evident immediately after induction and persisted through the 2- and 5-minute marks. However, preoperative IOP values were similar across groups and by the postoperative phase, differences between the groups were no longer statistically significant. These findings suggest that Midazolam, when combined with standard induction agents, helps attenuate the acute rise in IOP typically seen during anaesthetic induction [Table/Fig-5].

Subgroup	Time point	Group-I (Mean±SD)	Group-II (With Midazolam) (Mean±SD)	t-value	p-value
A (Thiopentone)	Pre-op	15.34±1.60	15.31±1.52	0.07	0.943
A	Immediately after induction	16.34±1.30	13.62±1.59	6.98	<0.0001
A	2 minutes after induction	14.74±0.80	13.31±1.49	4.83	<0.0001
A	5 minutes after induction	14.44±0.80	12.93±1.29	5.14	<0.0001
A	Post-op	13.65±1.05	13.76±1.07	-0.41	0.684
B (Ketamine)	Pre-op	15.39±1.51	16.21±2.38	-1.55	0.126
B	Immediately after induction	17.07±1.36	14.90±2.17	4.55	<0.0001
B	2 minutes after induction	16.62±1.32	14.94±2.27	3.71	<0.001
B	5 minutes after induction	16.01±1.15	14.50±1.93	3.94	<0.001
B	Post-op	15.75±1.19	15.34±1.86	1.02	0.311
C (Propofol)	Pre-op	15.12±1.54	15.41±2.00	-0.63	0.531
C	Immediately after induction	13.88±1.35	12.90±1.92	2.10	0.038
C	2 minutes after induction	13.30±1.02	12.47±1.86	2.15	0.034
C	5 Minutes after induction	13.19±0.94	12.17±1.61	2.91	0.005
C	Post-op	13.23±0.83	13.58±1.46	-1.24	0.221

[Table/Fig-5]: Intergroup comparison of IOP at different time intervals.

Adverse event: Patients were observed for ophthalmic and other systemic adverse events during the intraoperative and postoperative periods; no adverse events were observed during the study period.

DISCUSSION

The present study was undertaken to assess IOP changes in patients receiving general anaesthesia for operative procedures in various specialties. In a normal eye, IOP is maintained at 12-20 mmHg (±5 mmHg) and reflects a balance between the volume of aqueous humour, vitreous and choroidal vasculature, which exert outward pressure from within the globe and scleral compliance and extraocular muscle tone, which exert inward pressure [9]. Previous studies have measured IOP in patients undergoing non ophthalmic operative procedures under various surgical specialties. We have also studied IOP in patients scheduled for general surgery, orthopaedic, or ENT procedures; in terms of patient selection, the present study coincides with the above authors [12-18].

Sugata A et al., Halstead SM et al., Mirakhur RK et al., Mowafi HA et al., observed a preoperative mean IOP in the range of 15-20 mmHg [15,16,18,19]. The authors observations are consistent with these findings, as normal IOP in individuals is usually in the range of 15-20 mmHg, in accordance with normal physiological readings [4].

After induction of anaesthesia, all readings showed a significant decrease in IOP immediately after induction with propofol and in the groups thiopentone + midazolam, ketamine + midazolam and propofol + midazolam, compared with their preoperative values. There was an insignificant increase in IOP in other groups. Mirakhur RK et al., and Badrinath SK et al., have also observed minimal changes in IOP at 5 minutes after induction, at 10 minutes and postoperatively [18,20]. Halstead SM et al., and Dryana PC et al., have also noted insignificant increases or decreases [16,21].

Halstead SM et al., enrolled 80 patients undergoing procedures requiring Procedural Sedation and Analgesia (PSA) [16]. The mean total ketamine dosage was 1.6 mg/kg (95% CI: 1.4-1.7). The mean initial IOP was 17.5 mmHg (95% CI: 16.4-18.6) and at 2.5 minutes was 18.9 mmHg (95% CI: 17.9-19.9). The mean difference was 1.4 mmHg (95% CI: 0.4-2.4). The authors concluded that ketamine does not significantly increase IOP in pediatric patients without eye injuries.

Induction with thiopentone results in systemic hypotension, primarily due to vasodilatation, decreased venous return, decreased cardiac output, reduced cerebral blood flow and a decrease in intracranial pressure, which results in a decrease in IOP [18]. Propofol induction is followed by sometimes direct myocardial depression and a decrease in cardiac output. Also, decreased venous return reduces cerebral blood flow, thereby decreasing IOP [11]. In the present study, the decreases in IOP observed in the thiopentone- and propofol-based groups can be explained on these grounds. Ketamine induction increases vascular resistance, thereby increasing systemic blood pressure and cardiac output, which increases intracranial pressure and thus can cause an increase in IOP [20,21]. Mirakhur RK et al., assessed IOP during rapid-sequence induction of anaesthesia using thiopentone or propofol as the induction agent and suxamethonium for neuromuscular blockade [18]. IOP in the propofol group was significantly lower than in the thiopentone group, except immediately after induction, when reductions in IOP were similar and significant with both agents. IOP following intubation in patients in whom anaesthesia was induced with thiopentone was not significantly different from baseline values, but showed a significant increase from pre-intubation pressure, which was similar to the present findings. Increases in IOP are counterbalanced by the IOP-lowering action of midazolam. After administration of suxamethonium, IOP increases and is almost completely counterbalanced in combination groups of midazolam with propofol. In the ketamine + midazolam group, after suxamethonium there was an insignificant increase in IOP at the above time intervals; thus the increases in IOP due to suxamethonium are counterbalanced in midazolam combination groups compared with the individual inducing agents.

Similarly, Dryana PC et al., prospectively enrolled 25 children without ocular abnormalities undergoing procedural sedation that included ketamine for non periorbital injuries [21]. The authors reported that the largest predicted difference from baseline IOP occurred at 15 minutes, with an estimated change of 1.09 mmHg (95% CI: -0.37 to 2.55). The association between ketamine dose and mean IOP was not statistically significant or clinically meaningful (95% CI: -1.71 to 1.95). There were no clinically meaningful elevations in mean IOP reached at any time point.

In a systematic review, Chang CY et al., and Mirakhur RK et al., have explained reductions in IOP with thiopentone and propofol on the above grounds [11,18]. These mechanisms can be applicable to the present study observations: midazolam, administered intravenously, has a sedative action and can potentiate the effects of inducing agents such as thiopentone and propofol. Midazolam, by its sedative effect and tranquilising action, reduces stress and anxiety that could otherwise increase IOP; thus, this counteracts potential rises in IOP. Midazolam in combination with ketamine decreases the cardiovascular effects of ketamine, thereby reducing intracranial pressure and IOP [20,22].

Sugata A et al., Halstead SM et al., Mirakhur RK et al., and Mowafi HA et al., reported that induction of anaesthesia with either thiopentone or propofol results in a decrease in IOP immediately after induction in their patients [15,16,18,19]. This decrease in IOP with various induction agents is important for counteracting increases in IOP after suxamethonium and endotracheal intubation [20]. It is documented that increases in IOP after suxamethonium may be secondary to a sudden rise in arterial pressure, straining, or reflex venospasm. However, contraction of extraocular muscles and dilation of choroidal vessels may also play important roles in the increase of IOP after suxamethonium [18-22].

In individual groups of thiopentone, ketamine and propofol, IOP at two minutes and five minutes after induction and also postoperatively remained unchanged from preoperative readings. There was an insignificant decrease in the thiopentone and propofol groups at two and five minutes after induction and postoperatively when compared with preoperative readings. Here, a decrease in IOP with thiopentone and propofol is counterbalanced by an increase in IOP with suxamethonium. Hence, IOP remains somewhat unchanged at these time intervals when compared with preoperative readings. Postoperatively, one may encounter technical complications of tonometry such as conjunctivitis, corneal abrasion, corneal opacity, etc. None of the above authors observed any complication related to measurement of IOP with the tonometer. The lower incidence of technique-related complications may be explained by the soothing effect of local anaesthesia.

Limitation(s)

The present study was single-centre study, which limits generalisability. Use of tonometry to measure IOP is subject to several sources of error, including improper positioning on the eye, high variability compared with other devices and measurements influenced by individual ocular rigidity, although efforts were made to minimise error.

CONCLUSION(S)

The combination of midazolam with intravenous induction agents significantly reduces IOP, making it a beneficial adjunct in general anaesthesia, particularly during laryngoscopy and endotracheal intubation.While thiopentone and propofol independently decrease IOP due to their effects on intracranial tension, ketamine alone increases IOP, which can be counteracted by adding a sedative like midazolam. No complications were observed from the use of the Schiotz tonometer or the anaesthetic technique.Given the potential impact of anaesthetic agents and muscle relaxants on IOP, routine intraoperative IOP monitoring should be considered, especially in patients with ophthalmic concerns. Ketamine should be used cautiously and preferably in combination with a sedative to mitigate its hypertensive ocular effects.

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PLAGIARISM CHECKING METHODS: [Jain H et al.]
- Plagiarism X-checker: Oct 07, 2023
- Manual Googling: Sep 09, 2025
- iThenticate Software: Sep 11, 2025 (12%)

ETYMOLOGY: Author Origin

EMENDATIONS: 8

AUTHOR DECLARATION:
- Financial or Other Competing Interests: None
- Was Ethics Committee Approval obtained for this study? Yes
- Was informed consent obtained from the subjects involved in the study? Yes
- For any images presented appropriate consent has been obtained from the subjects. NA

Date of Submission: **Oct 06, 2023**
Date of Peer Review: **Oct 31, 2023**
Date of Acceptance: **Sep 13, 2025**
Date of Publishing: **Jan 01, 2026**