Clinical Question: If ketamine causes clinically important elevations in intraocular pressure, then should it be avoided in patients with ocular pathology?

Background: Ketamine, a widely used agent for emergency department (ED) pediatric procedural sedation, is sympathomimetic and often increases pulse rate and blood pressure. It can also elevate intraocular pressure either as a result of general blood pressure elevation or tension in the extraocular muscles. Two recent ED studies have shown minimal increases in intraocular pressure with ketamine. However, the first was potentially confounded by the midazolam coadministered in the majority of children, and the second obtained baseline intraocular pressure measurements just after (rather than before) ketamine administration. It is known that in healthy eyes, 10 mm Hg increases in intraocular pressure reduces ocular fundus pulsations and decreases choroidal blood flow and that in injured eyes, small pressure increases may lead to choroidal or optic nerve head ischemia. Two previous studies have defined a transient increase of 3 to 5 mm Hg in intraocular pressure as clinically important in previously damaged eyes.

Methods: A prospective study with children aged 8 to 18 years, chosen to receive ketamine sedation in a pediatric emergency department. Tono-Pen measurements were taken before ketamine administration, immediately after, 2 minutes after, and every 5 minutes thereafter for up to 30 minutes. Children with a predisposition to elevations in intraocular pressure (e.g., diabetes mellitus, steroid use, history of previous eye surgery) were excluded. The primary objective was to evaluate the increase in intraocular pressure during pediatric procedural sedation with ketamine. The secondary objectives were to determine the proportion of children whose increase might be clinically important (5 mm Hg), determine the absolute maximum pressures, and contrast clinical features in those with and without clinically important increases.

Results: Of 60 children who received ketamine (median cumulative dose, 1.5 mg/kg), the median increase in intraocular pressure was 3 mm Hg. Fifteen children (25%) had increases ≥5 mm Hg (the predefined threshold for a potentially clinically important increase), which resolved by 15 minutes in all but three. There were no differences in clinical characteristics between children with and without pressure increases ≥5 mm Hg. Mean highest absolute pressure was 19 mm Hg (range, 14–25 mm Hg).

Conclusion: In this study of ketamine sedation in children with healthy eyes, they observed mild increases in intraocular pressure that at times transiently exceeded the bounds for potential clinical importance (5 mm Hg).

Bottom Line: This is consistent with a previous study showing that ketamine caused no significant increase in intraocular pressure (NEJM JW Emerg Med Nov 9 2012). This study reassures us that ketamine should not be avoided for concern about raising intraocular pressure.