

Labetalol for hypertension after nasal use of lignocaine with epinephrine

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A 42 year, 62 kg, ASA I, male patient was scheduled to undergo endoscopic resection of a nonfunctional pituitary macroadenoma. The patient did not have any cardiorespiratory abnormality and the routine hematological investigations and the hormonal profile were within normal limits. Invasive arterial blood pressure (IBP) was 136/81 mmHg and heart rate (HR) 86/min.

General anesthesia (GA) was induced with fentanyl (125 mcg) and propofol (120 mg) trachea was intubated with 70 mg rocuronium. We maintained GA with sevoflurane and O₂: N₂O (40:60). Ten minutes after intubation, nasal mucosa was infiltrated with lignocaine 2% with epinephrine 1:200,000 and the nasal cavity was packed with epinephrine (1:200,000) soaked gauge. Few minutes after nasal packing,

an increase in IBP to 220/110 and HR to 146 bpm was observed. We administered labetalol (10+10 mg boluses) causing reduction of IBP to 140/90 but the HR dropped to 30/min. The resultant bradycardia responded to inj atropine 0.6 mg IV.

Our patient had a nonfunctional pituitary tumor and he was non hypertensive preoperatively. There were no signs of inadequate anesthesia/analgesia. Hypertension did not occur immediately on upon infiltration of nasal mucosa or nasal packing, was not associated with bradycardia and the surgery had not started. During preparation of surgical site for nasal endoscopic procedures, local anesthetic and adrenaline solution are infiltrated into mucosa and nasal cavity is often packed with epinephrine soaked gauge to reduce venous oozing and facilitate dissection. Hence, the most probable cause of hypertension in our patient was the absorption of epinephrine into systemic circulation from traumatized nasal mucosa.

Labetalol is a non-selective α and β adrenergic receptor antagonist. The β : α ratio of antagonism by labetalol is 7:1 after intravenous administration. It is used in the management of perioperative essential hypertension and

hypertensive crises.¹ Incremental doses of intravenous labetalol have been demonstrated to be safe and effective in acute hypertensive episodes.^{2,3} A continuous infusion with the dose of 2 mg/min, or intermittent intravenous (IV) boluses of 5-10 mg have been recommended upto maximum of 300 mg.³ Normally, epinephrine activates alpha 1, 2 and beta 1, 2 receptors. Though the alpha activation leads to vasoconstriction, there is a balance of beta-2 mediated vasodilation. In our case, probably an unopposed alpha-adrenergic receptor action after labetalol administration caused profound vasoconstriction since labetalol has seven times more affinity for beta-blockade than alpha when given IV.^{4,5} The resulting effects are significant increases in blood pressure and subsequent reflex (vagal nerve mediated) bradycardia.^{4,6} We need to be careful while using vasodilators like sodium nitroprusside or nitroglycerin due to their cerebral vasodilatory property; but we must be cognizant while administering labetalol for transient rise BP due to epinephrine. We recommend use of short acting beta 1 selective blockers like esmolol in titrated doses to alleviate transient hyper tension after epinephrine.

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