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Adult Bradycardia during Laryngoscopy: A Case Report

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Abstract: ***Haemodynamic changes during laryngoscopy vary among patients and may be exaggerated in certain populations. The major manifestation of laryngoscopy in adult patients is usually hypertension and tachycardia however, bradycardia is rarely reported. Bradycardia following laryngoscopy is commonly observed in pediatric patients, especially in patients with hypoxia. Here, we have reported such an event in haemodynamically stable adult thalassemia patient undergoing nephrectomy.***

Keywords: Laryngoscopy, Bradycardia

1. Introduction

Direct laryngoscopy and passage of endotracheal tube through the larynx is a noxious stimulus, which can provoke untoward response in the cardiovascular, respiratory and other physiological systems.[1]Haemodynamic changes during laryngoscopy vary among patients and may be exaggerated in certain populations. Although healthy and young patients generally tolerate these responses well, patients with limited coronary reserve may experience myocardial ischaemia, acute heart failure or serious arrhythmia.[2] The major manifestation of laryngoscopy in adult patients is usually hypertension and tachycardia however, bradycardia is rarely reported.[3] Bradycardia following laryngoscopy is commonly observed in pediatric patients, especially in patients with hypoxia.[4] Here, we have reported such an event in a haemodynamically stable adult thalassemic patient undergoing nephrectomy.

2. Case Report

A 45 year old patient was scheduled for elective right sided radical nephrectomy for renal trasistional cell carcinoma. He was a known case of thalassemia and required 1-3 times blood transfusion every month since 20 years. There was no positive surgical history. No significant family history. He was non- alcoholic and non- smoker. He had a weight of 50 Kg and height of 5 feet. On general physical examination, patient was conscious and oriented with heart rate of 94 beats per minute and blood pressure of 126/70 mm of Hg. Patient was pale and mildly icteric. Systemic examination was within normal limits. On airway assessment, he had an inter-incisor distance of 3.5cm, bulky tongue, Mallampati grade of III and moderate reduction in extension of head. His pre-op investigations were: Hb- 9.8 mg/dl, BT-2'14, CT- 5'12, MCV- 66.4fl, MCH- 15.8pg,, MCHC- 23.8g/dl, PBS- Microcytic Hypochromic red cells, HPLC- HbF (.6%), HbA2 (≥3.5%), PT- 19.2 secs, INR- 1.4, PTTK- 30.3 secs, Ratio- 1.08,BU- 44, S. creatinine - 1, S. uric acid- 5.1, S.E- WNL, RBS- 80mg/dl, LFTs- WNL,ECG- LAD, LVH changes, ECHO- CMP, Moderate LV dysfunction, trace MR, trace TR, EF-35%, Chest Xray- normal, Thyroid profile- WNL and HHH- Negative. On arrival of the patient in the operating room, routine monitors were applied and baseline vitals were observed to be within normal limits. Difficulty in ventilation and intubation was anticipated and difficult airway cart was kept ready. General anaesthesia was induced using glycopyrrolate 0.2mg, fentanyl 100µg and etomidate 12mg and succinylcholine 100mg intravenously to facilitate intubation and anesthesia was deepened with sevoflurane in oxygen. When the laryngoscope blade was introduced to perform tracheal intubation, sudden severe bradycardia (<35/min) was observed, at once the laryngoscope blade was removed, the heart rate slowly reverted back to normal over 10-15 seconds without any pharmacological intervention. The patient was then mask ventilated with 100 percent oxygen and was successfully intubated on 3rd attempt of direct laryngoscopy with the help of stylet and no significant hemodynamic changes were observed in further 2 attempts. Anesthesia was maintained with sevoflurane in oxygen nitrous oxide mixture (40:60) and intermittent boluses of fentanyl and atracurium as and when required. The remainder of the intraoperative course was uneventful. The trachea was extubated when the patient was fully conscious and following commands.

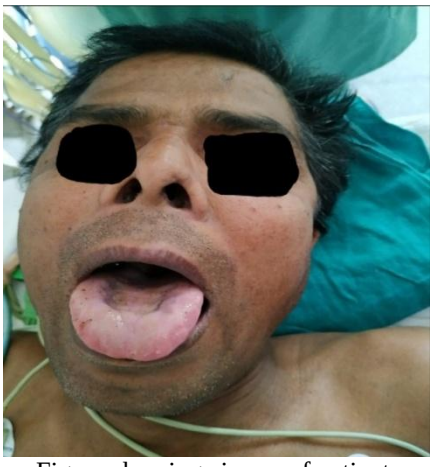

Figure showing airway of patient

3. Discussion

Possible causes of bradycardia in our patient are hypoxic vagal stimulation, hypotension, drugs and the patient's

cardiac status. We ruled out hypoxia and hypotension-induced bradycardia by pulse oximetry and NIBP (the oxygen saturation was (100%).[5,6] Patient's preoperative cardiac status can be excluded because our patient did not have any bradycardiac rhythm disturbance in his history and had not taken any medication which might have enhanced the bradycardia response to inducing agents up to surgery. Fentanyl causes activation of central vagal tone and it was documented in animal experiments even with a small doses, therefore combination with another vagomimetic agent may lead to enhancement of the bradycardia effects of the latter.[3] Succinylcholine is associated with cardiac dysrhythmias (i.e. junctional rhythm and sinus bradycardia) by its muscarinic activity at the sinoatrial node.[3] Etomidate has been reported to cause variable but minimal heart rate changes. In fact, in animal experiments etomidate occasionally increases central vagal activity and usually lacks vagolytic action. It is therefore reasonable to assume that the vagomimetic action of fentanyl is not attenuated and may occasionally even be enhanced by etomidate as a result, the addition of another potentially vagomimetic agent such as suxamethonium may lead fairly frequently to severe bradycardia or asystole.[3,4,5] Therefore, a plausible explanation of bradycardia in our patient may be related to the sequence of administering IV fentanyl, IV etomidate and IV suchcinylcholine that may have resulted in summation of individual vagotonic effects resulting in profound bradycardia.[3] Etomidate, fentanyl and succinylcholine are commonly used drugs that can independently cause bradycardia therefore the combination should be used cautiously. Also continuous and meticulous monitoring of the ECG and pulse waveform during induction and intubation should be done so that hemodynamic changes can be recognized easily and managed effectively.

Conflicts of interest: Nil

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