Case Report

Adrenaline-induced pulmonary edema in a pediatric patient

Asma Rahat, Sayeedur Rehman, Sarfaraz Ahmad, Bhawna Singh

Department of Anaesthesiology, Rohilkhand Medical College and Hospital, Bareilly, Uttar Pradesh, India.

Corresponding Author:
Dr.Asma Rahat, Assistant professor, Anaesthesiology, Rohilkhand Medical College and Hospital, Bareilly, Uttar Pradesh, India.
Email: Asma11rahat.ar@gmail.com

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We present a case report of 7 months old 10 kg child being operated for ankyloglossis who developed pulmonary edema after inadvertent injection of adrenaline. The patient was successfully treated with positive pressure ventilation (PPV), furosemide, morphine, and steroids. Patient was extubated after 4 h of PPV and shifted to high-dependency unit and later discharged after 24 h of monitoring.

KEY WORDS: Adrenaline, Pulmonary edema, Intermittent positive pressure pulmonary edema

INTRODUCTION

Ankyloglossis (also known as "tongue-tie") is a congenital anomaly in which there is presence of abnormally short lingual frenum, which restricts tongue tip mobility.[1]

The procedure "frenotomy" means the cutting of the frenum. This procedure can be conducted with or without local anesthesia and with very little discomfort to the patient.[2]

Usually, this can be performed under local anesthesia with mild sedation. However, in this case, as the frenum was v. tight and the child was only 7 months old, case was taken up under sevoflurane anesthesia with face mask. While taking the suture after division of frenum, child went into laryngospasm and saturation dropped very fast to 10% and heart rate to 46/min. For this atropine was advised and patient was intubated. Instead of atropine, nursing staff gave him adrenaline that too without dilution and in much higher dose. Patient developed pulmonary edema for which positive pressure ventilation (PPV) was given. Pre-operative laryngospasm is an emergency which may leads to significant morbidity and mortality (specially in pediatric patients).[3]

We present here a case report of pulmonary edema developed after inadvertent injection of adrenaline which was managed successfully and extubated.

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CASE REPORT

Laryngospasm is an exaggeration of a protective reflex that prevents aspiration of foreign objects into the lower airway (e.g., during swallowing). This may result in complete or partial glottic closure, or there may occur complete obstruction of airflow to the airway passage (trachea and lungs). The resulting hypoxia sometimes by itself may break a laryngospasm; but, if this continues without relief, it may lead to pulmonary edema, cardiac dysrhythmias, cardiac arrest, and ultimately death.[4]

The causes of laryngospasm are many like presence of local, chemical, thermal, or mechanical stimuli, which ascend signals through sensory

fibers of superior laryngeal nerve. Superior laryngeal nerve supplies the supraglottic region while inferior or recurrent laryngeal nerve gives sensory innervation below vocal cords.

Inadequate plane of anesthesia, presence of blood or secretions in oral cavity, stimulation by nasogastric tube or suction catheter, newborn or v. young child, and upper airway surgery may be the risk factors for developing laryngospasm.

In this patient, laryngospasm developed while surgeon was taking suture after frenutomy, which may be due inadequate anesthesia or analgesia. Saturation dropped to 10% and e.cg. showed bradycardia with pulse falling from 134 to 87 to 58 to 46. For this atropine was advised to nursing staff. By this time, there was a tremendous decrease in pulmonary compliance. Hence, patient was taken on 100% oxygen and intubated. Controlled ventilation was given, but on auscultation of chest the etiology of this decreased compliance could not be revealed. Immediately after atropine injection, patient's heart rate was increased to 186, which further increased to 234/min. Blood pressure was 124/88 mmHg. After few minutes of intubation saturation was again 100%. Patient was ventilated with 100 O_2 on volume-controlled ventilation mode with tidal volume of 100 mL, RR of 22, and positive end-expiratory pressure of 8 cm of H_2O. Child whole body showed flushing and petichae. When atropine ampule was examined, it was found to be of adrenaline. Instead of atropine nursing staff had given injection of adrenaline that too without dilution and in v. high dose (1 mg). Hence, adrenaline overdose was suspected. After half an hour on auscultation of chest B/L crepts were found and pink frothy sputum was seen in endotracheal tube. Patient has developed pulmonary edema which was most probably due to adrenaline toxicity. Patient was propped up, urinary bladder was catherized, injection furosemide 5 mg was given, dexamethasone 2 mg and injection hydrocortisone 20 mg were given. Furthermore, morphine in a dose 100 mcg/kg, that is, 1 mg was given. Patient was extubated when chest was clear of all secretions after 4 h of PPV inside o.t and then shifted to high-dependency unit for further monitoring.

DISCUSSION

Although certainly avoidable, administration of an incorrect drug or dose is one of the common errors in clinical practice. Immediate treatment is required for the salvage of patients who have received large doses of sympathimometic drugs. Adrenaline has a long period of action both endogenously or exogenously. Overdoses of adrenaline have been described many times in the past and pulmonary edema following such an overdose is well-documented.[5-8]

Various mechanisms have been described for pulmonary edema due to high blood levels of adrenaline; out of which four commonly accepted postulates are;

1. Cardiac failure from: (a) High peripheral resistance, (b) tachycardia and resultant diminished diastolic filling time this all leading to back pressure in pulmonary circulation
2. Pulmonary vasoconstriction and increase in the left atrial pressure, resulting in significant rise in pulmonary arterial pressure. This results in disturbance of the hydrostatic and oncotic pressure relationships in the pulmonary capillary bed
3. There occurs shift of blood volume from peripheral (which are highly responsive to vasoconstrictors), to pulmonary vascular beds (which are less vasoactive)
4. Furthermore, there may be some central nervous system mechanisms which play role in the production of pulmonary edema from massive doses of adrenaline.

Adrenaline has an alpha and beta stimulatory action in peripheral vessels and beta stimulatory action on the myocardium.[9] Hypertension after adrenaline injection is due to beta stimulation on the myocardium and alpha stimulation of blood vessel walls.

Sarnoff and Berglund[10] observed sympathetic stimulation of the peripheral vascular bed results in gross increase in the pulmonary blood volume. Thus, as stated earlier in postulate 3 above, an increase in peripheral resistance may result in the shifting of blood to the less vasoactive pulmonary bed.

CONCLUSION

Adrenaline administration is the corner stone for managing anaphylaxis in the pediatric patients; however, it needs judicious administration and its effect should be assessed to ensure correct dosing. In cases of adrenaline induced pulmonary edema, PPV should be given immediately. Early diagnosis and treatment is the key to success in such cases.

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