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Case Report

Anaesthesia Management for Bronchial Foriegn Body with PDA in CCF.

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ABSTRACT

One of the most challenging situations for anaesthesiologist is airway obstruction, carefull and fast assessment of airway along with planning of technique and co-ordinating with the endoscopist is necessary for a better outcome. We present a case of Patent ductus arteriosis with signs of congestive cardiac failure and foreign body obstruction of airway managed successfully.

1. Introduction

Foreign body (FB) Inhalation is a potentially life threatening event, in the age group of 1 to 3 years. A small reduction in airway radius due to oedema will result in a large increase in resistance to airflow. Organic FB inhalation may result in airway hyper reactivity and mucosal oedema. Hypoxia may rapidly occur because of high oxygen consumption of infants and small children.[1]

Bronchoscopy is done for a wide variety of diagnostic and therapeutic procedures (e.g. assessment of airway obstruction) to the rare (e.g. laser removal of tumours and the insertion of tracheal stents). Anaesthesia for bronchoscopic procedures poses a number of challenges and requires the anaesthetist to be fully familiar with airway management, ventilation and anaesthesia techniques. [2]

Patent ductus arteriosus (PDA) is relatively common vascular lesion, accounts for approximately 10% of congenital heart defects.[3] Forty percent of premature infants weighing ,1750 g and 80% of those ,1200 g have a PDA . PDA in these infants is typically quite large and often results in congestive heart failure because of left-to-right shunting of blood, with pulmonary oedema and decreased systemic perfusion. Small PDAs may close spontaneously or in response to the administration of indomethacin. Alternatively, small lesions may present with a heart murmur in asymptomatic children.[4]

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2.Case Report

We present a case of 6 year old female child, 20 kgs, with Bronchial foreign body as an emergency in congestive cardiac failure. She had cough (productive) , fever and running nose for 7 days.

No other significant past history, on examination she had a heart rate of 160 per minute , a systolic murmur heard at 2nd left parasternal area ,blood Pressure of 90/50 mm Hg, respiratory rate of 72 per minute, saturation of 97 %, generalised oedema, restless and febrile(101 degree Fahrenheit).

Air entry to lungs was decreased on left side; abdominal examination showed Shifting dullness, with hepato splenomegaly. Total protein was 5.2 gram/ decilitre; Serum electrolytes was within normal limits; Chest radiograph showed impression of Left lower lobe consolidation; CT scan confirmed these findings also showed mild pleural effusion on left side. Echo showed a small PDA (gradient 56 mm Hg), Mild PAH, with good Ventricular function. Cardiologist started Tab .Digoxin 0.25 mg loading dose and maintenance of 0.125 mg tid and Tab Lasix 15mg bd.

She was planned for bronchoscopy under General anaesthesia after 24 hours of admission. She was premedicated with Inj Fentanyl 25 micrograms and Inj Glycopyrollate 0.1 mg, induced with Sevoflurane and oxygen, scopy was assisted with Inj Succinyl Choline 40 mg and maintained with Sevoflurane, oxygen and side port of bronchoscope used for ventilation. Inj Hydrocortisone 100 mg and Dexamethasone 4 mg was given. Heart rate, ECG, saturation, Temperature and Blood pressure was monitored. Intraoperative Heart rate was 140-180 per minute, saturation maintained 97-99 %, Isolyte-p fluid was given as maintainance. Broncho scopy revealed oedematous mucosa, secretions and

mucus plug which was removed from left bronchus. Right bronchus was normal. Air entry improved on the left side. Bronchoscope removed and a 5mm size plain oral Endotracheal tube was inserted and the lung was expanded well. Recovery was uneventfull. Chest X-ray showed improved air entry to left side.

3.Discussion

Anaesthesia for paediatric bronchoscopy requires special equipment and a sound knowledge of the anatomy, physiology and pathology of the paediatric airway. Anaesthetist and the endoscopist should plan to ensure that adequate oxygenation is maintained via the shared airway. Examination should focus on the airway and respiratory system. Specific investigations like chest x-ray to localize an inhaled foreign body or a CT-scan is required to evaluate a possible cause for obstruction.

Midazolam can be given 30 min before induction (in older children) ensuring there is no evidence of airway obstruction or respiratory embarrassment. Anticholinergic should be considered, given i.v. at induction, has the dual benefit of preventing bradycardia secondary to airway instrumentation, its antisialogogue effect improves the efficacy of topically applied local anaesthesia and decreases the amount of suctioning during endoscopy. Dexamethasone is given to minimize airway oedema. Sevoflurane can be used for induction and spontaneous respiration maintained via a Jackson Rees T-piece attached to the side port of the bronchoscope. Introduction of the telescope into the bronchoscope seals its distal end, diminishes the cross-sectional area of lumen, significantly increasing the work of breathing and potentially causing hypercarbia. Once the procedure is finished a tracheal tube can be inserted if a full stomach is considered a problem, and the patient woken up and extubated once protective reflexes have returned.

Complications include trauma to lips, teeth, base of tongue, epiglottis and larynx; rare but may include pneumothorax, pneumomediastinum and surgical emphysema. Haemorrhage is usually minor and settles spontaneously. Hypoxia can occur for many reasons (scope is placed in a bronchus, excessive suctioning). Bronchospasm can be secondary to irritation of the tracheobronchial tree. Hypercarbia can occur even when the patient seems to be adequately ventilated, subsequent air trapping can lead to barotrauma, diminished venous return and so reduced cardiac output.

A chest x-ray should be taken after such procedures before leaving the post anaesthetic area. Bradycardia is uncommon but should be assumed to be secondary to hypoxia until proven otherwise.

We should look for signs of stridor secondary to subglottic oedema, nebulized epinephrine , may have to be repeated frequently. I.V. dexamethasone produces more sustained relief of stridor. Re-intubation may be required. Rarer problems include epistaxis when a fibreoptic scope is inserted nasally, and the potential transmission of infection as sterilization of these scopes can prove difficult. [5]

During intrauterine life, 10% of the cardiac output passes through the lungs. The remaining 90% is shunted via the ductus arteriosus (DA) to the aorta and systemic circulation. After birth, most of the right ventricular output should pass through the lungs to facilitate proper gas exchange. In order to make this possible, the ductus undergoes constriction and functional closure soon after birth in term neonates. Eighty percent (80%) of the DA in term infants close by 48 hours and nearly 100% by 96 hours. Shunting of blood from the systemic circulation to the pulmonary circulation results in congestive cardiac failure, which manifests clinically with wide pulse pressure and bounding pulses. Overloading of the pulmonary vasculature leads to pulmonary edema/ hemorrhage which predisposes the neonate to chronic lung disease.

Patients with PDA often have a characteristic heart murmur that can be heard with a stethoscope. Changes may be seen on chest x-rays. The diagnosis is confirmed with an echocardiogram. If the patent ductus is not closed, the infant has a risk of developing heart failure, pulmonary artery hypertension, or infective endocarditis -- an infection of the inner lining of the heart. [6]

Systemic vascular resistance is important as decrease in systemic vascular resistance, increases right to left shunt, decreases pulmonary perfusion and hypoxaemia. Decrease in CVP indicates decrease in Rt. heart filling which in the presence of fixed pulmonary artery hypertension decreases pulmonary perfusion and results in hypoxaemia. Hypotension from any cause can progress to insufficient Right ventricular pressure require to perfuse the hypertensive pulmonary arterial bed and may result in sudden death of the patient. Catecholamine release especially during laryngoscopy and at extubation, causes increase in pulmonary vascular resistance in these patients. IPPV further increases pulmonary artery pressure and also causes fall in blood pressure. The net effect is an increase in the right to left shunt. However, general anaesthesia has been used successfully in these patients.[7]

4.Conclusion

The outcome of airway obstruction cases depend on carefull and quick assessment, planning, timing and co-ordinated decisions with the surgeon. Associated co-morbid conditions can complicate the management and quick retrieval of the foreign body is necessary to reduce the morbidity .

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