ANAESTHESIA FOR CONGENITAL LOBAR EMPHYSEMA (A Case Report)

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Introduction

Congenital lobar emphysema is a clinico-pathological syndrome characterised by progressive hyperaeration of one or several lobes of lung with resultant compression atelectasis, mediastinal shift, impaired venous return, ventilation perfusion abnormality, secondary hypoxia and hypotension. Anaesthetic manipulations may suddenly aggravate the problems and produce catastrophic results [1].

Case Report

A 4-month-old, female child weighing 5 kg was admitted on 12 Apr 94 with acute respiratory distress with history of breathlessness since 6 April 94. Breathlessness used to increase during coughing, crying and feeding. Physical examination revealed signs suggestive of moderately severe respiratory obstruction. Her heart rate was 136/min, respiratory rate 40/min, intercostal and sub-costal recession was obvious but there was no cyanosis. Examination of respiratory system revealed shift of trachea and apex beat to right, hyper-resonant note with decreased breath sounds over upper left chest. Other systems were normal. Postero-anterior chest radiograph showed a hyperlucent area in left upper and middle zones compressing left lower lobe and also gross mediastinal shift to right (Fig 1). Routine blood examination and urinalysis did not reveal any abnormality.

The clinical diagnosis of congenital lobar emphysema was made and was taken up for emergency surgery after obtaining high risk consent. Breast feed was allowed till 3 hours prior to surgery. Pre-medication consisted of atropine sulphate 0.1 mg orally an hour before surgery. Induction was carried out with halothane (0.5 to 1.5% through Fluotec Mark II vaporiser) in oxygen with total inspiratory gas flow of 4 L/min administered through Ayre's T piece breathing circuit. Surprisingly induction could not be achieved till 20 min. Induction was then

accomplished by administering injection ketamine HCl in the dosage of 3 mg/kg body weight intramuscularly. Endotracheal intubation was achieved with 4 mm uncuffed portex endotracheal tube after ensuring adequate depth of anaesthesia. Intravenous access was obtained by securing left saphenous vein. Right brachial artery was cannulated for invasive arterial blood pressure monitoring. Spontaneous ventilation with occasional breaths assisted manually was maintained till chest was opened. Anaesthesia was maintained with oxygen-isoflurane (0.4-1%) mixture and incremental dosages of atracurium. Ventilation was controlled manually

Fig. 1 : Radiograph chest PA view showing hyperlucent area in left upper zone and mediastinal shift to right.

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thereafter. Maintenance fluid requirement and third space losses were replaced with 5% dextrose at the rate of 10 ml/kg/hour and blood 70 ml by infusion pump.

Intraoperative monitoring included ECG, pulse oximetry, rectal temperature and arterial blood gas analysis. Intraoperative course was smooth. Reversal was achieved with mixture of neostigmine and atropine in appropriate dosages. Extubation and emergence from anaesthesia was uneventful. At operation, left upper lobe was large, emphysematous, non-collapsible. Left lower lobe was small and mediastinum was pushed to right. Left upper lobe was removed and left lower lobe was mobilized, inflated and chest was closed with two drains.

On first post operative day, child became restless with heart rate of 188/min, respiratory rate of 40/min, temperature 38°C and there was abdominal distension. Air entry to both lungs was satisfactory. Child was managed by giving glycerine suppository and N/5 saline (total of 100 ml with 2 mEq of KCl) at the rate of 10 ml/hour. On third post operative day, child developed atelectasis of left lower lobe (Fig 2) manifesting as acute respiratory distress. This complication was managed successfully by postural drainage, physiotherapy and oxygen administration by polymask. She recovered (Fig 3) and was discharged from the hospital on 10 May 94.

Discussion

Congenital lobar emphysema is a crippling, frequently life threatening respiratory disorder. Our patient presented with acute

respiratory distress quite early in infancy. Some even develop symptoms at birth [2]. Clinical diagnosis was confirmed by chest radiograph which is the most important diagnostic tool [3]. Abnormal bronchial cartilage, fibrous dysplasia as alveolar defect or poly-alveolar lobe has been incriminated as aetiological factors in 50% of cases. In our patient, bronchial cartilage dysplasia was demonstrated histopathologically. Mortality with conservative treatment is 50% as against 7% for lobectomy [4]. Our patient underwent emergency left upper lobectomy as treatment of choice.

The specific anaesthetic problems in congenital lobar emphysema related to the administration of nitrous oxide during induction and maintenance of anaesthesia and intermittent positive pressure ventilation (IPPR) [5] were well anticipated. We omitted nitrous oxide altogether and IPPR was not instituted till chest was opened. The problems due to rapid enlargement of bulla giving rise to either pneumothorax or compression of adjoining normal lung tissue or even mediastinal shift resulting into further hypoxaemic

Fig. 2 : Radiograph chest PA view showing atelectasis of left lower lobe and hyperaeration of right lung.

Fig. 3 : Radiograph chest PA view showing disappearance of atelectasis of left lower lobe.

and haemodynamic instability were therefore avoided. However, we did not anticipate prolonged induction of this degree with an inhalational agent. The mechanical factors which can give rise to prolonged induction were excluded. Halothane was within its expiry date and outlet of vaporiser smelled of halothane indicative of its functional state. It was not possible to check accurately percentage of output as adjusted on the vaporiser. There was no leak in the breathing circuit and anaesthetic gases were reaching the patient end.

Exclusion of these factors suggested that cause was lung pathology itself. Prolonged induction in congenital lobar emphysema can be explained on the basis of many factors such as accumulation of large tidal volume in a highly compliant lung, increase in functional residual capacity, decrease in effective ventilation and ventilation-perfusion abnormality [6]. These pathological factors singly or in combination can lead to wasted ventilation. In this patient, large part of tidal volume was wasted out in a highly compliant area and less of pulmonary vascular bed was available for diffusion of anaesthetic gases across alveolar capillary membrane.

In our patient, intra-operative and immediate post-operative period was uneventful. On the following day, patient developed respiratory distress due to abdominal distention and high temperature affecting diaphragmatic excursion and oxygen demand. Symptoms were relieved by conservative treatment without any active respiratory support. On third post-operative day, she developed atelectasis of left lower lobe most probably due to mucus plug but patient also developed hyperaeration of right lung and mediastinal shift to left, all contributing towards acute respiratory distress. This required active respiratory management. Patient responded well clinically and radiologically.

Anaesthetic management of congenital lobar emphysema should consist of premedica-

tion with oral atropine and avoidance of sedatives. Induction with ketamine HCl supplemented with halogenated inhalation agent, maintenance with air-oxygen-halothane or preferably isoflurane mixture. It is not recommended to use only oxygen-isoflurane mixture. Use of oxygen alone can cause absorption atelectasis if anaesthesia time is prolonged. Air oxygen mixture (50/50%) is preferable. It is preferable to maintain spontaneous ventilation till chest is open but some authors have used high frequency positive pressure ventilation [7]. If it is mandatory to use muscle relaxant, transpulmonary pressure (TPP) during IPPR should be similar to that during awake ventilation which can be partly achieved by delivery of tidal volume nearly equal to patient's pre-operative value. Thus pattern of controlled ventilation most appropriate is similar to that used for obstructed lung disease. Post-operatively, patient should be observed carefully for acute respiratory complications. These complications can occur due to either hyperinflation of normal lung which was compressed earlier or mediastinal shift causing acute angulation of trachea.

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