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Negative pressure pulmonary edema after emergency craniotomy a case report

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Abstract: *Pulmonary edema following obstruction of upper airway is an uncommon and unpredictable clinical entity. This unusual disease is actually attributed to pulmonary and hemodynamic changes caused by high negative intrathoracic pressures during the state of obstructed respiration, such as laryngospasm, epiglottitis, laryngotracheal neoplasm etc. we report a case of 20 year old healthy male who underwent emergency craniotomy and developed laryngospasm after extubation followed by Negative pressure pulmonary edema. After reintubation and mechanical ventilation in intensive care unit patient was extubated and shifted to neurosurgical ward without anyfurther respiratory problems.*

Keywords: *Laryngospasm,Generalanaesthesia, pulmonary edema.*

I. Introduction

Negative pressure pulmonary edema(NPPE) in adults is an uncommon medical emergencyarising as a consequence of airway obstruction especially during emergence following extubation[1].Forceful inspiration against a closed glottis generates very high negative intrathoracic pressures. This results in clinical picture of pulmuoary edema and very rarely hemorrhage associated with edema[2]. NPPE is classified as two types. Type 1 is secondary to acute obstruction of upper airway and type 2 occurs after surgical correction of airway obstruction. It is common in young healthy males undergoing general anaesthesia[3]

II. Case Report

20 year old male was admitted in emergency department with history of road traffic accidentwith suspected head injury.CT head documented Left parietal brain contusion with thin rim of acute subdural hematoma (Figure 1).On examination patient had GCS 13/15,pupilsnormal size, reacting tolight,heart rate76bpm& blood pressure 130/80mmHg.Physical examination and laboratory investigations were normal.Patient was taken for emergency craniotomy as ASAIIE.General anesthesia was induced with Propofol 2mg/kg besides morphine 0.1mg/kg.Endotracheal intubation was facilitated using atracuriumbesylate 0.5mg/kg. After induction Left radial artery was cannulated for invasive blood pressure monitoring.Anesthesia was maintained with 70%nitrous oxide in oxygen supplemented with 0.5%-1%isoflurane.IntraoperativelyPatient remained hemodynamically stable. Left parietal craniotomy with evacuation of contusion was done. Rest of the brain parenchyma was normal with no evidence of edema. Additional analgesia in the form of Paracetamol 1 gmIV infusion was given towards the end of surgerythat lasted for 3 hours.At the end residual neuromuscular block was reversed and patient was extubated with GCS10/15.Immediately after extubation there was inspiratory stridor consistent with laryngospasm.Patientdeveloped tachypnea,cyanosis and oxygen saturation dropped to 70%. Patient could not be ventilated with bag and mask&was therefore, immediatelyreintubated using 1.5 mg/kg succinylcholine with 100mg propofol. Suctioning of endotracheal tube revealed copious, pink& frothy secretions.ABG showed hypoxia. Auscultation revealed bilateral coarse crepitations in lung bases. Patient was transferred to ICU andput on controlled ventilation with Fio2 of 1& PEEP 10 cmH2o. CXR revealeddiffuse alveolar and interstitial infiltrates,normal sized heart, with no pleural effusion (Figure 2.). The picture was consistent with pulmunaryedema.CVP line was secured to monitor central venous pressure and fluid management.A diagnosis of negative pressure pulmunary edema was made and patient was treated with diuretics & inhalational bronchodilators. Patient was ventilated for 24 hours and his respiratory status continued to improve with peripheral oxygen saturation of >94% on 35% Fio2.Patient was extubated at GCS 15/15.On Istpost operative day, echocardiocardiography was done which revealed normal study.Repeate CXR showed resolution of pulmunary infiltrates (Figure 3), and on 2nd postoperative day patient was shifted to neurosurgical ward with peripheral oxygen saturation of >94% on ambient air.

III. Discussion

Postoperative NPPE typically occurs in response to an upper airway obstruction, where patients can generate high negative intrathoracic pressures, leading to pulmonary edema. The current literature regarding its

epidemiology is sparse. Young, healthy, athletic patients seem to be at risk for this disorder[4] and the prevalence of postoperative NPPE is approximately 0.1%[4,5]. In patients developing acute postoperative upper airway obstruction, NPPE has been reported at an incidence of up to 11%[6]. Typical events leading to acute upper airway obstruction accompanied by perioperative NPPE include laryngospasm and endotracheal tube occlusion by biting. Less typically, NPPE can also occur after foreign body aspiration, oropharyngeal surgery, or postoperative residualcurarisation[7]whichtypically impairs the upper airway dilator muscle strength while preserving inspiratory muscle function[8]. Casereports and retrospective data suggest that the patient characteristics that increase the risk of NPPE seem to include younger patients in American Society of Anesthesiologists physical status categories I and II, who are thought to be most capable of generating highly negative intrathoracic pressures (upto 100 cm of water) during an obstructing event. Procedural characteristics increasing the risk of NPPE may include oropharyngeal surgery (especially for tumors or other potentially obstructing masses) although the true incidence and hazard ratios have not been reported[9].Two different mechanisms may explain the development of pulmonary edema during airway obstruction. The most likely mechanism relates to the observation that high negative intrathoracic pressures cause significant fluid shifts from the microvessels to the perimicrovascularinterstitium, as seen in patients with congestive heart failure or fluid maldistribution states. The second proposed mechanism involves the disruption of the alveolar epithelium and pulmonary microvascular membranes from severe mechanical stress, leading to increased pulmonary capillary permeability and protein-rich pulmonary edema.

The patient's history, operating room course, and clinical & radiologic findings were most consistent with pulmonary edema due to sudden & high negative intra-thoracic pressure as the most likelycause.However,aspirationpneumonitis (Mendelson-syndrome),Neurogenicpulmunary edema, fluid overload(cardiogenic pulmonary edema), diffuse alveolar hemorrhage resulting from upper airway obstruction[10]etc.were also included in the differential diagnosis.

Aspiration pneumonitis can be of concern in emergency setting but preoperative clinical findings were not consistent with that.In addition,use of cuffed endotracheal tube and the radiologic picture of symmetric bilateral pulmonary interstitial infiltrates would be unusual for aspiration pneumonitis, which typically shows a localized infiltrate. In this patient, intraoperative fluid overload as a cause for pulmonary edema was not considered reasonable because the patient received only 2000 ml isotonic solution intraoperatively over 3 hours and there was no evidence of any pre-existing left ventricular dysfunction. In the immediate setting, we could not rule out acute lung injury or neurogenic pulmonary edema, but the severity of respiratory failure and the time course of clinical and radiologic recovery were not ultimately consistent with this etiology. Coupling these considerations with the clinical picture of laryngospasm, we concluded that the patient's pulmonary edema was likely induced by negative intrathoracic pressure, potentially resulting from strong inspiratory efforts in the setting of laryngospasm.

Although many patients with NPPE recover with conservative management, some patients with severe NPPE (or underlying cardiopulmonary disease) require temporary intubation and mechanical ventilation with positive end-expiratory pressure[11]. Diuretics are often administered but their use is controversial and may evenbe un-necessary[12].Turbelancewithin bronchi, irrespective of the cause, including interstitial edema induced narrowing of bronchial lumina, may account for the development of the clinical symptoms like wheezing. In-vitro and in-vivo studies (both in human and animal models)haveshown that β agonists may increase the rate of alveolar fluid clearance via increased active cation transport[13].

Ultimately, NPPE is generallya benign condition typically resulting in full recovery in 12–48 h when recognized early and necessary supportive treatment is instituted for hypoxemic and/or hypercapnic respiratory failure. Early diagnosis, awareness of the anesthetist, careful surgical manipulation of the upper airway, vigilance of the nurses in the recovery room and in thewards contribute to the successful management& outcome of this syndrome.

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Figure 1

Figure1.CT head showing Left Parietal contusion with thin rim of acute sub dural hematoma

Figure 2.

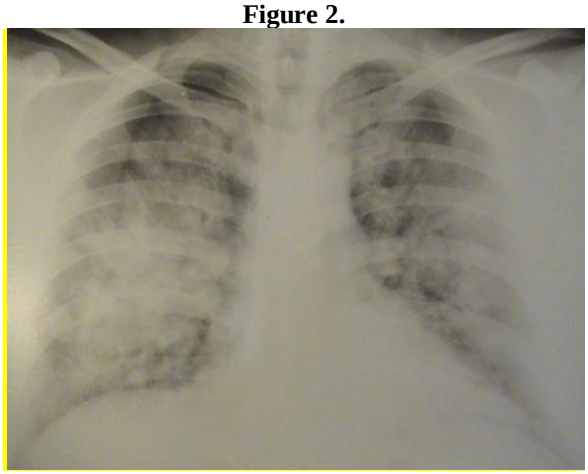

Figure 2. Chest X ray showing bilateral and diffuse alveolar and interstitial infiltrates

Figure 3.

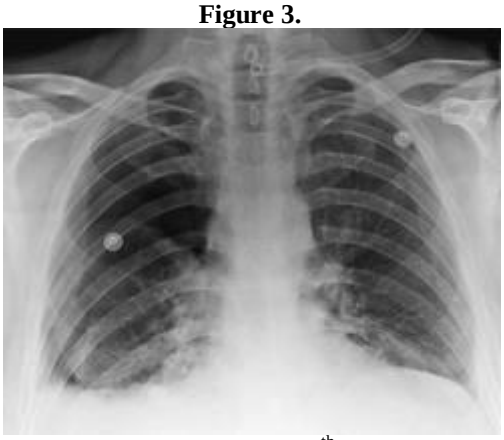

Figure 3. Chest X Ray on 5th post op day.