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Case Report

Negative Pressure Pulmonary Oedema after Sedation in a Patient Undergoing Pacemaker Implantation

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ABSTRACT

Negative pressure pulmonary oedema (NPPE) is a manifestation of upper airway obstruction and is seen after extubation postoperatively. The large negative intrathoracic pressure generated by forced inspiration against an obstructed airway is thought to be the principal mechanism involved. It is a dangerous and potentially fatal condition with a multifactorial pathogenesis. The Authors report a case of negative pressure pulmonary oedema occurring periprocedurally while undergoing permanent pacemaker implantation under local anaesthesia and light sedation due to upper airway obstruction caused by falling back of the tongue.

Key words: Negative pressure pulmonary oedema, Sedation, permanent pacemaker implantation, Upper airway obstruction.

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INTRODUCTION

Negative pressure pulmonary oedema is a serious and potentially fatal condition with a multifactorial pathogenesis. Frequently, NPPE is a manifestation of upper airway obstruction, the large negative intrathoracic pressure generated by forced inspiration against an obstructed airway is thought to be the principal mechanism involved. This negative pressure leads to an increase in pulmonary vascular volume and pulmonary capillary transmural pressure, creating a risk of disruption of the alveolar – capillary membrane. The early detection of the signs of this syndrome is vital to the treatment and to patient outcome.[1] It is rare and usally occurs in a clinical setting of postextubation, laryngospasm, epiglottits, croup, endotracheal tube obstruction, post tonsillectomy/adenoidectomy and other conditions. The Authors report here a case of negative pressure pulmonary oedema occurring after intravenous sedation in an elderly male undergoing dual chamber permanent pacemaker implantation procedure.

CASE REPORT

An 86 year male with symptomatic Mobitz Type 2 AV block was prepared for creation of a left side pectoral pocket to implant a dual chamber permanent pacemaker under local anaesthesia with strict aseptic precautions in the cath lab. Patient had no past history of cardiac or pulmonary disease in the past and remained asymptomatic except for one episode of syncope coinciding with Mobitz Type 2 AV block one day before being taken up for dual chamber permanent pacemaker implant procedure. Inj. Lignocaine (1 %) 15 ml was infiltrated locally into the skin along the length of the intended incision as well as more deeply and medially with an intent to achieve adequate anaesthesia. As patient did not experience adequate analgesia after additional infiltration of Inj. Lignocaine to a maximum permitted dose of 3mg/kg, patient was administered Inj. Fentanyl 50 mcg intravenously to achieve better analgesia. Incision was placed for creation of pectoral pocket. Soon, it was noticed that the oxygen saturation on pulse oximetry was falling to less than 70 % and lung auscultation revealed bilateral crepitations. The heart rate and blood pressure remained unchanged. The patient was drowsy and sedated and it was noticed that the tongue had fallen back to cause obstruction to the upper airway. Patient underwent immediate endotracheal intubation with artificial ventilation. Pink frothy sputum was noticed in the endotracheal tube. Simultaneously, patient received intravenous Frusemide 40 mg to treat acute pulmonary oedema. The oxygen saturation improved quickly after the above measures and pacemaker implantation procedure was completed successfully. A perioperative chest x-ray confirmed presence of acute pulmonary oedema. (Figure 1). Patient was extubated uneventfully after six hours with no further clinical events and was discharged from the hospital after three days.

DISCUSSION

The first description of the pathophysiological correlation between creation of negative pressure and the development of pulmonary edema was in 1942 by Warren *et al.*[1] The relationship between pulmonary edema and upper airway obstruction in two children, who had croup and epiglottitis was reported by Capitanio *et al.*[2] The first report on the clinical significance of this phenomenon in three adult patients, who experienced onset of pulmonary edema minutes to hours after severe acute upper airway obstruction was by Oswalt *et al.*[3] Negative pressure pulmonary edema (NPPE) is also called post obstruction pulmonary edema (POPE). The presentation of NPPE can be immediate or delayed.[4,5] Negative pressure pulmonary oedema is classified as Type I or Type II.[6,7] Type I NPPE develops immediately after onset of acute airway obstruction and Type II NPPE develops after the relief of chronic upper airway obstruction. As Type I NPPE develops usually with upper airway acute obstruction or after manipulation of the airway surgically, some authors call it laryngeal spasm-induced pulmonary edema.[8] Other factors that increase the risk of Type I NPPE are hanging, strangulation, upper airway tumors, foreign bodies, epiglottitis, croup, chocking, migration of Folly's catheter balloon used to tamponade the nose in epistaxis, near drowning, endotracheal tube (ETT) obstruction, goitre, and mononucleosis. Type II NPPE can result after relief of upper airway obstruction caused by big tonsils, hypertrophic adenoids, or a redundant uvula. According to one study, the incidence of developing Type I NPPE associated with acute postoperative upper airway obstruction is 9.6 – 12%, whereas the incidence of developing Type II NPPE is 44%,[9] In adults about 50% of NPPE occurrences are due to postoperative laryngospasm.[10] NPPE is mainly described in a postoperative clinical situation wherein non-cardiogenic edema results from laryngospasm or other forms of upper airway obstruction following extubation.[4,5] NPPE begins with a significant

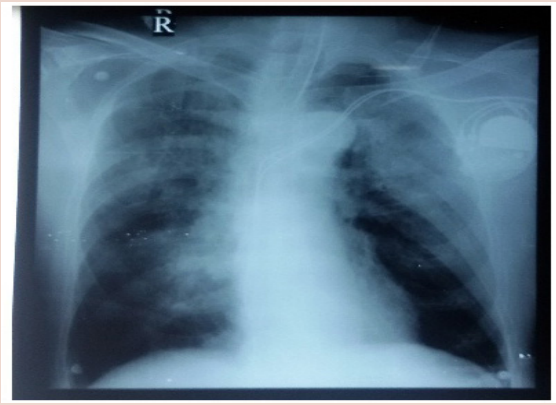

Figure 1: Chest X Ray showing features of pulmonary oedema with Dual chamber pacemaker

upper airway obstruction, inspiratory efforts to overcome the obstruction generate highly negative intrapleural and alveolar pressures, and the high pressure gradient causes fluid to move out of the pulmonary capillaries and into the interstitial and alveolar spaces[11,12] The etiology of negative pressure pulmonary edema is multifactorial, but appears to be related to the generation of marked negative intrathoracic pressure due to forced inspiration against a closed glottis, referred to as a Mueller (or reverse Valsalva) maneuver. As the intrathoracic pressure becomes more negative, blood flow to the right heart increases. This causes the pulmonary vascular bed to dilate, the interstitial pressure around the capillaries to become more negative, and intravascular fluid to be drawn into the interstitial space. This worsens gas exchange and triggers a cascade of hypoxemia, catecholamine release, and systemic and pulmonary hypertension. The result is an acute increase in afterload, which worsens transcapillary fluid efflux and increases interstitial and alveolar edema.[13]

The pathophysiology of NPPE is attributed to four major mechanisms: Disturbances of pulmonary fluid homeostasis can be induced by four pathways that can lead to increased interstitial fluid - increased hydrostatic pressure in the pulmonary capillary bed (or conversely, decreased pressure in the interstitium), decreased osmotic pressure of plasma, increased permeability of the membrane, and decreased return of fluid to the circulation *via* lymphatics.[14,15] In clinical presentation, initial findings usually include decreased oxygen saturation, with pink frothy sputum and chest radiograph abnormalities.[9] Pulmonary hemorrhage and frank hemoptysis have also been reported.[17] Lung auscultation usually reveals crackles and occasionally wheezes. Pulmonary edema causes both impaired diffusion of oxygen and ventilation/perfusion mismatching, leading to sudden and possibly severe hypoxemia. The typical chest radiograph will show diffuse interstitial and alveolar infiltrates. Although the radiographic findings associated with postextubation pulmonary edema have been described, there are minimal data regarding distribution of this postextubation edema within the lungs.[16] Respiratory failure requiring unplanned reintubation in the postoperative period is associated with high morbidity, leading to a longer hospital stay, and increase in 30 - day mortality.[19] Patients usually present with signs of acute upper airway obstruction following extubation and, upon relief of the obstruction, immediately develop dyspnea with pink frothy sputum and bilateral infiltrates on their chest radiograph. Less often, the development of pulmonary edema can be delayed for several hours.[12]

The first treatment priority is relief of the airway obstruction and correction of hypoxemia. The next step is to address the pulmonary edema with a diuretic unless the patient is hypovolemic. Effective airway management and immediate treatment with oxygen and diuretics is sufficient in most cases of NPPE. Persistent airway obstruction may necessitate an artificial airway, and acute respiratory failure would require artificial ventilation with oxygen and appropriate levels of PEEP. Other options include noninvasive ventilation (NIV) and oxygen delivered through high flow nasal cannula (HFNC).[20,21,22]

The Authors here report a case of negative pressure pulmonary oedema occurring in an elderly patient which occurred due to falling back of the tongue after drowsiness and sedation following intravenous analgesia leading to upper airway obstruction. The clinical significance in this case lies in the fact that simple falling back of the tongue with sedation is enough to cause airway obstruction before any procedure. It can lead to serious life threatening complication like NPPE. The Authors place importance on early recognition of this condition with prompt restoration of airway patency. When recognition of negative pressure pulmonary oedema is delayed, NPPE have mortality rates ranging from 11% to 40%,[18] To the best of the Authors' knowledge negative pressure pulmonary oedema occurring due to drowsiness and sedation with falling back of the tongue to cause upper airway obstruction is one of a kind and has not been reported in the literature so far.

CONCLUSION

Negative pressure pulmonary oedema can occur with sedation due to falling back of the tongue and early recognition is life saving.

CONFLICT OF INTEREST

Nil

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Nil

ABBREVIATIONS USED

NPPE: Negative pressure pulmonary oedema; PEEP: Positive end expiratory pressure.

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