



MENDELSON SYNDROME IN PATIENT POSTED FOR LAPAROSCOPIC CHOLECYSTETOMY

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<p>Article Info</p> <p><i>Received 15/01/2016</i> <i>Revised 27/02/2016</i> <i>Accepted 21/03/2016</i></p> <p>Key words: Aspiration of gastric contents, Mendelson Syndrome</p>	<p>ABSTRACT</p> <p>Mendelson syndrome is characterized by bronchopulmonary reaction following aspiration of gastric contents during general anaesthesia due to abolition of the laryngeal reflexes. The main clinical features are signs of general hypoxia, rales, rhonchi, and tachycardia with a low blood pressure. Decreased arterial oxygen tension is present. Pulmonary edema can cause sudden death. Here we are presenting with a case report of 52 year old male posted for laparoscopic cholecystectomy for empyema of gall bladder Following intubation yellow colored gastric secretions were seen coming from endotracheal tube. On auscultation of chest there were crepitation and wheeze. Respiratory monitor showed increased peak pressure. Patient was manually ventilated using Bain circuit. After creation of Pneumoperitoneum patient's peak pressures increased further up to 60cm of H₂O and ETCO₂ increased up to 70mm of Hg, therefore it was decided to do open cholecystectomy to prevent further CO₂ insufflation. Patient started showing signs of pulmonary edema with pink frothy secretions from endotracheal tube. Patient was managed with intravenous diuretics, steroids, antacids, prokinetics, bronchodilators, nebulisation with steroids and bronchodilators through endotracheal tube. ABG showed severe acidosis with hypoxemia and hypercarbia. Because of empyema of gall bladder patient had increased gastric secretions and unexpectedly developed acid aspiration. With the help of respiratory monitors it was immediately diagnosed and treated accordingly. Thus patient with acid aspiration and pulmonary edema can be managed successfully with prompt diagnosis and treatment.</p>
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INTRODUCTION

Mendelson syndrome is aspiration of gastric contents causing chemical injury with bronchopulmonary reaction during general anaesthesia due to abolition of the laryngeal reflexes. It was first described by Mendelson in 1946. It is a triphasic phenomenon with immediate respiratory distress, cyanosis, dyspnoea, pulmonary wheeze, crepitant rales, rhonchi, tachycardia and hypotension. Decreased arterial oxygen tension is most important and earliest sign of aspiration. Pulmonary edema can cause sudden death or death may occur later from pulmonary complications like septicemia with bacterial infection. This syndrome is due to irritation of the bronchioles by gastric hydrochloric acid. The critical pH value is 2.5 and volume more than 25 ml is more risky

[4,5]. Second phase usually occurs after 6 hours and shows infiltration by neutrophils and inflammation similar to ARDS. Final gradual recovery phase is present. Patients who are high risk for aspiration are female gender, pregnancy, and proper preoperative preparation should prevent most of the cases of aspiration pneumonitis.

CASE REPORT

Here we are presenting with a case report of 52 year old male weighing 70kg posted for laparoscopic cholecystectomy for empyema of gall bladder. He was a known case of diabetes mellitus and hypertension. He was on treatment (OHA and antihypertensive medication) for the same and his blood pressure and sugar levels were



under control. His preoperative investigations and vital parameters were normal. Airway examination revealed adequate mouth opening and MPC grade 2. Patient was taken on OT table. Standard monitors i.e. pulse oximetry, NIBP and ECG were attached. Pulse (80/min), BP(135/85mm of Hg), SpO₂(100%) and ECG were within normal limit. IV line with 20 gauge cannula secured in left upper limb and IVF (Ringer lactate) started. Premedication with Inj. Glycopyrrolate 0.004mg/kg, Ondansetron 0.08mg/kg, Midazolam 0.03mg/kg and Pentazocine 0.5mg/kg were given. Patient was preoxygenated with 100% oxygen using Bain circuit for three minutes. Anaesthesia was induced with Propofol 2mg/kg. Preoxygenation and ventilation with mask was difficult because of heavy jaw which was overcome by oropharyngeal airway no 5. Once the ventilation was confirmed Inj. Succinylcholine 1.5mg/kg was administered and endotracheal intubation was done and Inj. Vecuronium was given for maintenance. Cuff inflated and position of the endotracheal tube in trachea was confirmed with capnometry (ETCO₂=38 mm of Hg). Resistance to ventilation was increased and yellow colored gastric secretions were seen coming out of endotracheal tube [4,5].

On auscultation of chest air entry was reduced bilaterally with bilateral wheeze and crepitation. Immediate ET tube and oropharyngeal suctioning was done. A 30 degree head low position was given and 16 Fr Oro gastric tube inserted to empty the stomach while the patient was being ventilated manually with Bain circuit with 100% oxygen and Sevoflurane. Mechanical ventilation started with low tidal volume (6 ml/kg) and PEEP of 4 cm of H₂O.

Peak inspiratory pressure was increasing from 35cm of H₂O and with simultaneous Hypercarbia with ETCO₂ 50 mm of Hg. ABG was obtained which showed pH=7.15, Pco₂=65 mm of Hg, pO₂=70 mm of Hg and bicarbonate=20mEq/L. Inj. Metoclopramide 10 mg iv, Inj. Hydrocortisone 100mg iv and Inj. Dexamethasone 8mg iv were given. Beta 2 agonist salbutamol administered via endotracheal tube using metered dose inhaler. Simultaneously Pneumoperitoneum was created.

After creation of Pneumoperitoneum patient's peak pressures increased further up to 60cm of H₂O and ETCO₂ increased up to 70mm of Hg, therefore it was decided to do open cholecystectomy to prevent further CO₂ insufflation. Patient was manually ventilated using Bain circuit as ventilation with closed circuit was not possible. Peak pressure and ETCO₂ reduced to pre-Pneumoperitoneum values. Ventilation was difficult because of tight bag. Patient started showing signs of pulmonary edema with pink frothy secretions from endotracheal tube. Patient was catheterized intraoperatively using Foley's catheter given IV furosemide 10mg. Nebulisation with duolin and budesonide was given intermittently via endotracheal tube. Serial ABG samples were taken and intermittent suctioning of endotracheal tube was done. Gastric content was sent for pH estimation and

culture sensitivity. With continuous management clinical condition of patient improved. ETCO₂ and peak inspiratory pressure gradually decreased. Continuous improvement was seen in serial ABG reports. At the end of surgery ETCO₂ was 40 mm of Hg and peak pressure was 30 cm of H₂O. On auscultation of chest both lung fields were clear with bilaterally equal air entry. ABG showed pH=7.3, Pco₂=48 mm of Hg, pO₂=90mm of Hg and bicarbonate=24 mEq/L.

So it was decided to extubate the patient. Patient started breathing spontaneously. Thorough oropharyngeal suctioning was done. Neuromuscular blockade reversed with Inj. Neostigmine 0.05mg/kg and Inj. Glycopyrrolate 0.008mg/kg very slowly to prevent complications of neostigmine causing bronchospasm. Oropharyngeal suctioning repeated. After patient was wide awake with adequate, spontaneous, regular breathing and adequate tone, power, reflexes patient was extubated. Following extubation patient maintained adequate SPO₂ (96%) off mask. Patient was observed in recovery room for one hour. CXR was obtained and it showed mild congestion with bilateral fluffy shadows. Patient was shifted to surgical ICU and observed for 12hrs for any delayed symptoms and signs which are likely to occur after six hours in these patients.

DISCUSSION

Preoperative preparation of the patient is of extreme importance to prevent aspiration pneumonitis which includes anticholinergic agents, antiemetic agents, prokinetics, clear antacids like non-particulate sodium, H₂-Receptor antagonists/Proton pump inhibitors, gastric decompression [3].

Common causes of aspiration pneumonitis are general anaesthesia, obesity, pregnancy, increased intraabdominal pressure, extremes of age, disruption of the gastro esophageal junction.

Diagnosis is usually made with signs and symptoms of the patient, serial CXR and arterial blood gases, increased peak inspiratory pressures, hypoxia, and hypercarbia and if pulmonary edema is present pink frothy secretions will be seen from mouth and nose.

As soon as diagnosis is made 30 degree head down position should be given, oropharyngeal suctioning is carried out with cricoid pressure, endotracheal intubation with immediate inflation of cuff if patient is not maintaining saturation, endotracheal tube suction before administering PPV with oxygen to prevent fluid from going further down in lungs, gastric decompression and administration of bronchodilators is necessary [2,3]. Prophylactic broad spectrum antibiotics should be administered in immunocompromised patients [3]. Immediate corticosteroids should be given to reduce inflammation and stabilize lysosomal membranes [6]. Immediate diagnosis and treatment prevents further development of aspiration pneumonitis into ARDS (Acute Respiratory Distress Syndrome).



CONCLUSION

With empyema of gall bladder patient had increased gastric secretions and unexpectedly developed acid aspiration. With the help of respiratory monitors it was immediately diagnosed and treated accordingly. Thus patient with acid aspiration and pulmonary edema can be managed successfully with prompt diagnosis and treatment.

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CONFLICT OF INTEREST:

The authors declare that they have no conflict of interest.

STATEMENT OF HUMAN AND ANIMAL RIGHTS

All procedures performed in human participants were in accordance with the ethical standards of the institutional research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. This article does not contain any studies with animals performed by any of the authors.

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