Non-surgical pneumoperitoneum and pneumoretroperitoneum associated with mechanical ventilation

M Ravi Krishna, [1] Pramod Sood, [2] Parshotam Lal Gautam [3]

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Abstract

We present two rare cases of mechanical ventilation-associated barotrauma presenting with pneumoperitoneum and pneumoretroperitoneum separately. Pneumoperitoneum and pneumoretroperitoneum are not always associated with a hollow viscous perforation and can be seen due to barotrauma as a consequence of the Macklin effect.

Keywords: Pneumothorax, Pneumoperitoneum, Macklin effect

Authors
1. M. Ravi Krishna, MBBS DNB, Senior resident, Department of Critical Care Medicine, Dayanand Medical College & Hospital, Ludhiana, India
2. Pramod Sood, MBBS, MD, EDIC, FNB, Department of Critical Care Medicine, Dayanand medical college & hospital, Ludhiana, India
3. Parshotam Lal Gautam, MBBS MD, Professor and Head of Department, Department of Critical Care Medicine, Dayanand Medical College & Hospital, Ludhiana, India

Corresponding author: m.ravikrishna23@gmail.com
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Introduction

Pneumoperitoneum and pneumoretroperitoneum are abnormal collections of air in the peritoneal cavity and the retroperitoneum. The most common aetiology is hollow viscous perforation. Rarely overdistension of hollow viscera, gas trapped in wounds and Chilaiditi sign can mimic these findings.

Chilaiditi sign also called pseudo-pneumoperitoneum refers to the transposition of the bowel between the liver and diaphragm.[1] The usually interposed bowels are the hepatic flexure and the colon or the proximal transverse colon, while the small bowel is rarely involved.[2]

Case Report 1

A 50 year old female who was a known case of Chronic obstructive pulmonary disease and Obstructive sleep apnea overlap syndrome who was on regular medication with metered dose inhaler (fluticasone + formoterol) came to the emergency with altered mentation and respiratory distress. On evaluation, the patient's vitals were heart rate of 120/min., respiratory rate was 32/min and blood pressure was 100/60 mm Hg. SpO_2 was 82% with oxygen inhalation by venturi mask with FiO_2 of 60%.

Her physical examination showed that she was in mild respiratory distress with the use of accessory muscles of respiration, drowsy but arousable and had a Kelly Matthay score of 4. Her respiratory examination revealed that her lungs had slightly decreased intensity breath sounds with bilateral diffuse expiratory polyphonic wheezes. Her cardiovascular examination showed a regular pulse, and no murmurs, rubs, or gallops. Her abdomen was soft, non-tender. Screening chest sonography was suggestive of sliding lung sign with only 'A' profile and no evidence of any dynamic air sonograms on both sides. In view of altered mentation the patient was immediately intubated with an endotracheal tube of 8 mm size and was ventilated with PC-CMV with adaptive targeting (PC-CMVa) mode with the settings of FiO_2 0.8, tidal volume was set at 400 ml (7ml/ kg of her predicted body weight) with a respiratory rate of 16/min and I:E ratio was set at 1:2.5 and PEEP was set at 6 cmH_2O. Respiratory mechanics evaluation revealed an airway resistance of 23 cmH_2O/l/s and expiratory time constant of 1.6s with peak inspiratory pressure of 40 cmH_2O and plateau pressure of 22 cmH_2O and auto PEEP of 7 cmH_2O. Within a few minutes of mechanical ventilation, the patient developed worsening hypoxia; Post intubation radiograph revealed collapsed right lung with right-sided tension pneumothorax and air under the diaphragm (Figure 1). A few minutes later the patient had a cardiac arrest, Cardiopulmonary resuscitation was initiated, and right Intercostal drain was placed in the 4th Intercostal space immediately. The patient was revived after 6 mins. CT scan revealed right massive pneumothorax, minimal pneumomediastinum with significant pneumoperitoneum and extensive subcutaneous emphysema (Figure 2). The patient was transferred to the ICU after hemodynamic stability and was extubated on day 3. Pneumoperitoneum also resolved and the patient was transferred to a ward on day 6 and the intercostal drain was removed on day 12.

Case Report 2

An 8-year-old boy presented with a history of cough and fever for 3 days. On examination, the patient was in respiratory distress and hypoxemia. Chest radiograph revealed bilateral infiltrates (Figure 3). In view of severe hypoxia, the patient was intubated and ventilated with PC-CMV with adaptive targeting (PC-CMVa) mode with the settings of FiO_2 0.8, tidal volume was set at 180 ml (6 ml/ kg of his predicted body weight) with a respiratory rate of 24/min and I:E was set at 1:2 and PEEP was set at 14 cmH_2O after sedating and paralysing with fentanyl, midazolam and cisatracurium. Respiratory mechanics evaluation revealed a plateau pressure of 29 cmH_2O and peak airway pressure of 40 cmH_2O with a driving pressure of 15 cmH_2O. No recruitment manoeuvres were given. An immediate post-intubation radiograph showed pneumomediastinum with infiltrates in bilateral lower zones, but the patient developed abdominal distension after an hour and a repeat chest radiograph revealed pneumomediastinum, pneumoretroperitoneum and extensive subcutaneous emphysema (figure 4). His abdomen was soft, and non-tender, with normal bowel sounds, no guarding, no rigidity, and no rebound tenderness. The patient was managed conservatively without any chest drain insertion and the pneumoretroperitoneum and pneumomediastinum gradually resolved.

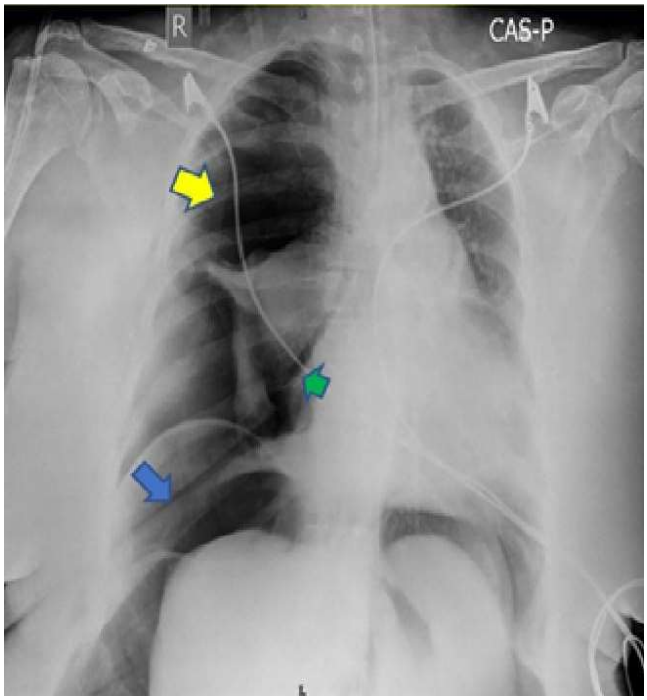

Figure 1: Chest X-ray showing Pneumothorax (yellow arrow), Pneumomediastinum (green arrow), and Pneumoperitoneum (blue arrow)

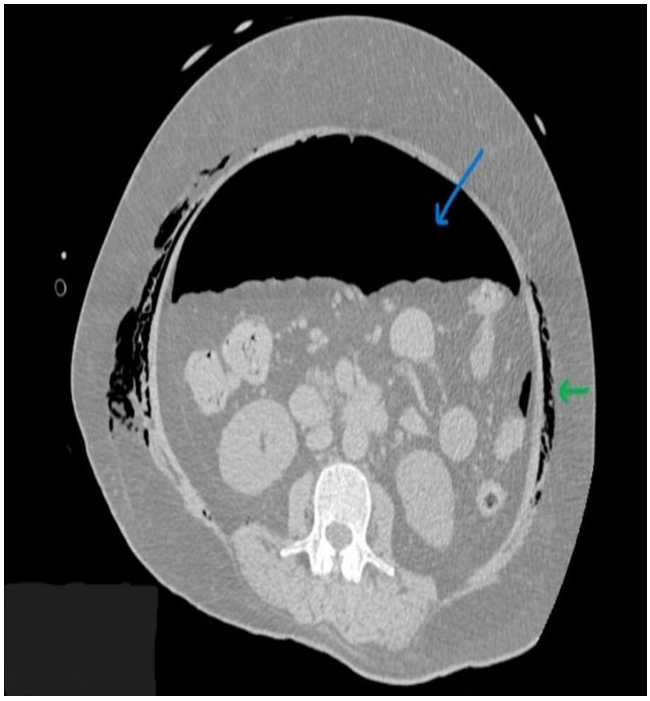

Figure 2: CT scan of the abdomwn showing Pneumoperitoneum (blue arrow) and subcutaneous emphysema (green arrow)

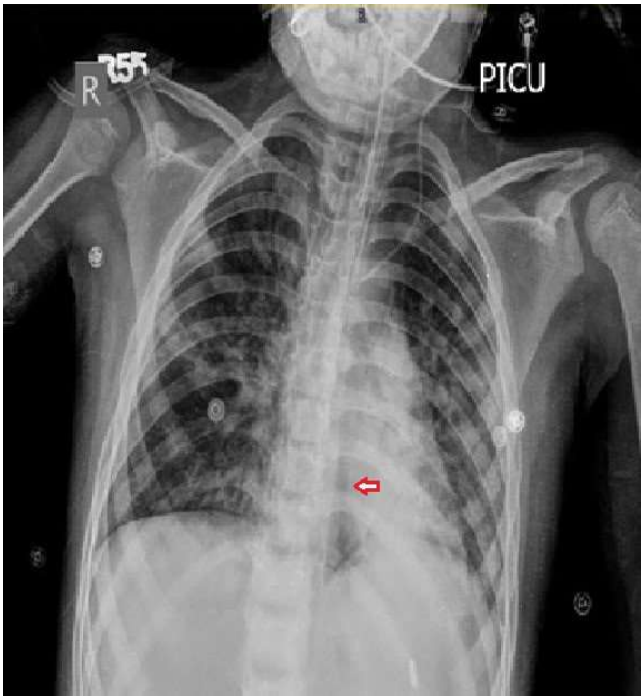

Figure 3: Chest X-ray showing the presence of Pneumomediastinum (red arrow)

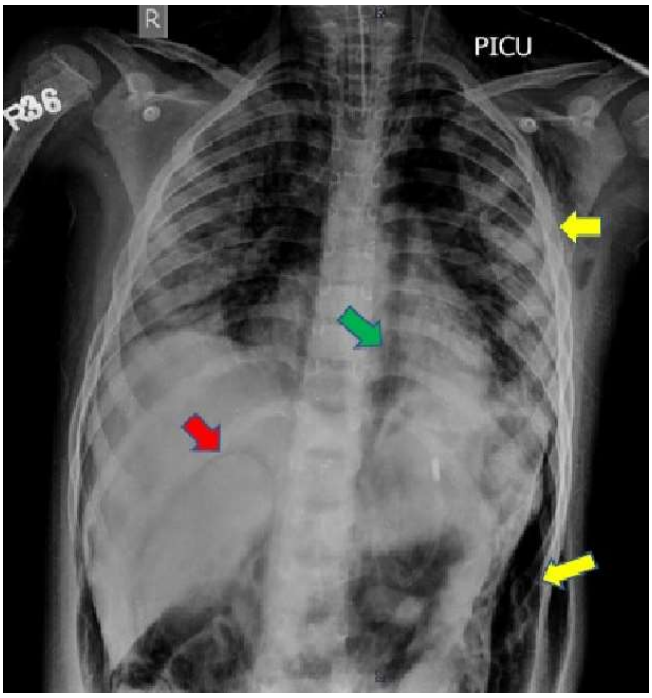

Figure 4: CXR showing the presence of Pneumomediastinum (green arrow), Perinephric air/Pneumoperitoneum (red arrow) and extensive subcutaneous emphysema (yellow arrow)

Discussion

Both of our cases are interesting as both of our patients had no evidence of hollow viscous perforation or a diaphragm defect, but they had developed a pneumoperitoneum and pneumoretroperitoneum respectively as a consequence of positive pressure ventilation.

In most instances, Pneumothorax leading to the development of pneumoperitoneum is reported in the presence of a diaphragmatic hernia.[3,4] Likewise development of pneumothorax after laparoscopic surgeries where pneumoperitoneum is induced was also reported. [5]

In up to 90% of the cases of pneumoperitoneum bowel perforation is the usual cause. [6] The other 5% of cases are usually related to gynaecologic, thoracic, abdominal, postoperative, nonsurgical, or idiopathic causes.[6] It has also been reported after cardiopulmonary resuscitation and due to mechanical ventilation. The true incidence of pneumoperitoneum associated with mechanical ventilation varies from rare to up to 7%. [7]

The thoracic causes of pneumoperitoneum are mostly associated with mechanical ventilation due to the Macklin effect. [8] It occurs in 3 steps: alveolar rupture due to high pressure, air dissection along the Bronchovascular sheaths and finally spreading of this pulmonary interstitial emphysema into the surrounding structures like skin, mediastinum, pleural cavity. The Macklin effect is mainly associated with pneumomediastinum and is seen mostly in trauma, respiratory infections, diving accidents.

The other pathway through which air can enter the abdominal compartment is through the pleural and diaphragmatic defects. [9]

It is also important to note that a simple pneumothorax cannot cause a pneumoperitoneum even in the presence of a diaphragm defect as the intrabdominal pressure normally exceeds intrapleural pressure both in inspiration and expiration during normal breathing.

In tension pneumothorax or during mechanical ventilation, the inspiratory pressure and expiratory pressure (PEEP) might exceed the intraabdominal pressure and air can freely enter the peritoneal cavity through the diaphragm defect or can dissect along the mediastinal planes to the retroperitoneum and rupture into peritoneal cavity leading to pneumoperitoneum. [10]

Pneumoretroperitoneum is defined as the presence of gas within retroperitoneal space. Perforation of the retroperitoneal portions of the intestines, such as the duodenum, ascending and descending colon, and rectum are the usual causes. Air can also track down through various posterior and midline openings in the diaphragm which provide a route for communication between the mediastinum and the retroperitoneal space. [11]

Mechanical ventilation per se represents a rare aetiology of pneumoretroperitoneum. As retroperitoneal gas is bound by fascial planes, it usually collects linearly along the margins of the kidney and psoas muscles. The air is most commonly seen surrounding the right kidney and in the left upper quadrants of the abdomen. [12]
When air is visible around the right kidney like in our patient it can be described as a veiled right kidney sign. [13]

Once a diagnosis of pneumoperitoneum and pneumoretroperitoneum had been made, it is important to identify the cause. If the only finding is pneumoperitoneum and pneumoretroperitoneum and there are no abdominal symptoms or significant physical findings like tenderness or rebound tenderness, guarding and rigidity in addition to the absence of free peritoneal fluid and mesenteric or bowel wall thickening a diagnosis of non-surgical pneumoperitoneum and pneumoretroperitoneum can be made. In these patients, a careful wait and watch approach can be followed.

In conclusion, non-surgical pneumoperitoneum and pneumoretroperitoneum may be uncommon complications of mechanical ventilation. Early recognition of nonsurgical causes by both surgeons and intensivists helps in avoiding unnecessary surgical procedures.

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