

Case Report

A massive primary spontaneous pneumothorax during the recovery phase of H1N1 pneumonia – An issue of concern

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ABSTRACT

The H1N1 or swine flu outbreak in 2009 was a major pandemic. It is a subtype of influenza A virus that affects humans and has two surface antigens, which led to it being called the novel H1N1 flu. Some patients with swine flu may develop lower respiratory tract infections, which can result in respiratory failure and acute respiratory distress syndrome (ARDS). In this case report, a patient with H1N1 pneumonia developed massive spontaneous pneumothorax during the recovery phase and concludes that, high pressure mechanical ventilation during the management of H1N1 pneumonia and persistent cough could be the reason for spontaneous development of pneumothorax. However, spontaneous development of pneumothorax in patients with swine flu pneumonia is rarely reported. We present an uncommon case of H1N1 pneumonia-induced pneumothorax that occurred during the recovery phase of the patient. This case report highlights the need for clinicians and healthcare teams to be aware of this potential complication during the patient.

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1. Introduction

The H1N1 or swine flu outbreak in 2009 was a major pandemic. It is a subtype of influenza A virus that affects humans and has two surface antigens, which led to it being called the novel H1N1 flu. This virus can potentially cause respiratory distress, primarily affecting the upper and in few cases lower respiratory tract of the human.¹ Some patients with swine flu may develop lower respiratory tract infections, which can result in respiratory failure and acute respiratory distress syndrome (ARDS). ARDS is a condition that occurs suddenly and results in respiratory failure, and can be caused by a variety of factors, including bacterial or viral pneumonia, nonpulmonary sepsis and more. This can be potentially fatal, especially in patients with mechanical ventilators² which

* Corresponding author. E-mail address: mdaamirzuhaib@gmail.com (Mohammed Aamir Zuhaib S). occurs due to the alveolar rupture resulting from elevated transalveolar pressure or barotrauma.³ There is also a high incidence of pneumothorax, which is air trapped outside the lungs but within the pleural cavity or between the parietal and visceral pleura of the thorax, in patients with respiratory failure or ARDS.⁴ However, spontaneous development of pneumothorax in patients with swine flu pneumonia is rarely reported. We present an uncommon case of H1N1 pneumonia-induced pneumothorax that occurred during the recovery phase of the patient. This case report highlights the need for clinicians and healthcare teams to be aware of this potential complication during the post-recovery phase of the patient.

2. Case Report

A 53-year-old male patient presented to the medicine OPD with chief complaints of intermittent fever associated with chills, polyarthralgia associated with myalgia, and persistent

cough with yellowish expectoration for 3 days with nil comorbidities and no significant social habits. On arrival, his vitals were found to be stable except with a temperature of 102 F, respiratory rate of 22 breaths per minute, and oxygen saturation of 92 % on room air. On auscultation, bilateral basal crepitations were heard, and advised for a chest x-ray, which was suggestive of bilateral lower lobe consolidations (Figure 1). The patient's objective evidence was suggestive of leukopenia in view of which the nasal and throat swabs for influenza virus were collected and H1N1 (a subtype of influenza A) infection was confirmed with RT-PCR. Thus, a provisional diagnosis was made as bilateral H1N1 lower lobe pneumonitis with nil co-morbidities, to which the patient was started with an appropriate antiviral agent (oseltamivir- a neuraminidase inhibitor), and supportive management was initiated. During the stay, the patient was shifted to ICU in view of spontaneous progressive desaturation of about 69% on room air associated with severe persistent cough, dyspnoea (Grade 4 - mMRC scale), and intermittent fever spikes. Thus, it was suggestive of ARDS picture or refractory hypoxia. The patient was started with supportive oxygen therapy of 4 liters per hour with intermittent NIV with pressure support of 12/6, steroid therapy, and escalated antibiotic therapy was initiated. Meanwhile, ABG analysis was done which revealed respiratory acidosis with hypernatremia and hypocalcaemia, and also HRCT-thorax was done which made an impression of atypical/viral pneumonia (Figure 2). Patient was continued with intermittent NIV with the same pressure support, oxygen therapy was tapered and all the necessary management was done. Thus the final diagnosis was made as H1N1 pneumonia associated with ARDS. Over the stay, with the view of decreased consolidation on repetitive chest X-rays, the patient was clinically and hemodynamically stable. Hence the patient was discharged with appropriate medication and requested regular followups.

Apparently, a month later, the post-discharge patient had reported to the Emergency room with chief complaints of persistent cough for 15 days, dyspnoea (Grade 4 mMRC scale) for 2 days, and follow-up incompliance. On arrival oxygen saturation was 81% on room air and there was a decreased air entry in the right lung (anteriorly and posteriorly) associated with right lower lobe Ronchi on auscultation. In view of this chest x-ray was done and notified as the right massive pneumothorax. Thus the patient was taken up for the emergency interventional chest thoracotomy procedure, where an intercostal drainage (ICD) tube of 20 Fr size was inserted between the right-sided 4th - 5th intercostal space in the anterior axillary line under local anaesthesia with high-flow oxygen support (4-6 litres per hour) and patient was shifted to ICU where broad spectrum antibiotic and steroid therapy were initiated. During the stay, approximately 300ml of drainage was

collected over 5 hours. On post-operative day 1 patient complained of chest pain and on examination, the barrel chest was seen and repositioning of the chest tube was done. On a post-operative day, 3 chest x-rays was repeated which showed complete resolution of pneumothorax, thus the patient was discharged in healthy condition on day 4 of hospital admission with long-term follow-ups.



Figure 1: HRCT-thorax- Bilateral Atypical/Viral Pneumonia

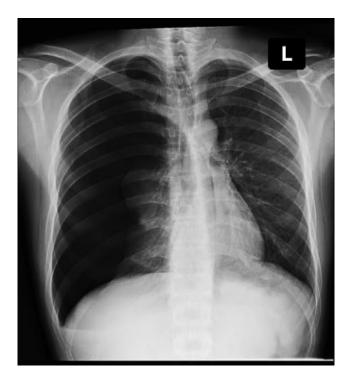


Figure 2: chest x-ray (PA) right massive pneumothorax

3. Discussion

In this case, a patient with H1N1 pneumonia developed massive spontaneous pneumothorax during the recovery phase, which is rarely reported. The development of pneumothorax or air leak was presumed to be occurred during management of pneumonia using mechanical ventilator causing alveolar damage or baro trauma. The majority of patients' gross and microscopic results are reflected in "diffuse alveolar damage," which initially appeared in early descriptions of the pathological outcomes in patients with acute respiratory distress syndrome.^{5,6} There are studies which explains underlying histologic abnormalities that cause clinical lung barotrauma are intraprenchymal pseudocysts, bronchiolar dilatation, pleural cysts, and alveolar overdistension. It also states that mechanical ventilation can exacerbate the condition, especially when the ventilator supplies high peak airway pressures and huge tidal volumes.⁷ In the above case, the patient was initially admitted and managed with intermittent NIV supplying high pressures and was discharged with a follow-up request. Eventually patient developed a dry cough which was managed on an OPD basis with oral anti-tussives. Over time patient symptoms aggravated and readmitted for spontaneous massive pneumothorax. Discussion arrived on the cause of spontaneous pneumothorax which was presumed likely due to the mechanical ventilation support during previous admission and negligence of dry cough. During mechanical ventilation, high peak airway pressures and huge tidal volumes can cause pneumothorax, intraparenchymal pseudocysts, and alveolar overdistension. High incidence of bronchial injury suggests that mechanical ventilation with high pressure played a role in its pathogenesis. It's significant to note that patients suffering from acute respiratory distress syndrome (ARDS) are susceptible to developing a range of complications, including subcutaneous emphysema, pneumomediastinum, pneumopericardium, and pneumoperitoneum. While there have been recent reports of spontaneous pneumomediastinum and pneumothorax, there is still much to be learned about the precise mechanisms that lead to their development.^{8,9} However, coughing may induce a sudden increase in intra-alveolar pressure, ultimately leading to the rupture of subpleural alveoli and distal bronchus. This occurrence can result in the spontaneous development of pneumothorax and subpleural bullae.¹⁰ However, there is a possibility that spontaneous pneumothorax could be caused by the application of high-pressure ventilation in association with persistent dry cough, which may result in the occurrence of barotrauma.

4. Conclusion

Our case report concludes that, high pressure mechanical ventilation during the management of H1N1 pneumonia

and persistent cough could be the reason for spontaneous development of pneumothorax. Hence, it is imperative to recognize the potential consequences of coughing and its hindrance on recovery process and pulmonary health. It may be prudent to consider alternative ventilation strategies that could mitigate these risks and promote better patient outcomes. Thus, the close monitoring of patients for any such indications of potential health conditions is of utmost importance. It is vital to take swift actions to address any identified issues to ensure that the patient receives the best possible care and optimum chances of recovery. Therefore, healthcare professionals must remain vigilant and proactive in their approach towards monitoring and managing their patients' health and promote the well-being of those under our care and prevent needless suffering.

5. Source of Funding

None.

6. Conflict of Interest

The authors declare no conflict of interest.

7. Acknowledgement

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