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Lesson from Clinical Practice

Non-invasive ventilation (NIV) induced bilateral subcutaneous emphysema in SARS COV-2 patient: A rare occurrence

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ABSTRACT

A patient with coronavirus disease-2019 (COVID-19) developed bilateral subcutaneous emphysema involving the neck and upper chest following the institution of non-invasive ventilation (NIV) for worsening hypoxia. We discuss the various causes, differentials and successful management of this patient.

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1. Introduction

The COVID-19 pandemic, has seen an increase in the number of patients suffering from varying degrees of breathing difficulties requiring oxygen (O₂) supplementation via face mask, non-rebreathing/reservoir face mask (NRBM), high flow nasal cannula (HFNC), NIV support or invasive ventilation. These patients are considered “happy hypoxics” and intubated only when there are clinical signs of increased work of breathing, desaturation, evolving hypercapnia, worsening mental status, hemodynamic instability or multiorgan failure. Currently, the use of HFNC or NIV is reserved for mild hypoxemia, with airborne precautions [1]. Common complications associated with NIV are pressure ulcers/necrosis of nasal bridge, facial or ocular abrasions, oronasal mucosal dryness, claustrophobia, anxiety, agitation, aerophagia, abdominal distension, vomiting, aspiration, hemodynamic instability, impaired communication and impaired nutrition. Rare complications are barotrauma, pneumothorax, pneumomediastinum, pneumocephalus, raised intracranial pressure and raised intraocular pressure. Cases of NIV induced pneumomediastinum and subcutaneous emphysema (SE) have been reported in patients with chronic obstructive pulmonary disease (COPD) and asthma [2]. Recently a few authors have also reported this in Covid-19 patients [3,4]. In this communication we report an occurrence of bilateral subcutaneous emphysema in a patient of COVID-19 pneumonia without

pneumothorax and pneumomediastinum having no previous history of respiratory disease.

2. Case report

A 34 year male patient presented with the complains of fever and cough for 6 days and shortness of breath for 1 day duration. He was a known case of type 2 diabetes mellitus since 5 years, well controlled on oral anti-diabetic agents. Other medical and surgical history was unremarkable. On examination, patient was conscious and oriented with heart rate (HR)- 110/min, blood pressure (BP)- 115/91 mm Hg, oxygen saturation (SpO₂)- 92% on room air and respiratory rate (RR)- 22/min. As he tested positive for SARS COV-2 he was shifted to the COVID- 19 care isolation ward and was started on supportive treatment as per our institutional protocol. O₂ was supplemented initially via a face mask and later via a NRBM and he was advised awake proning. His chest x-ray showed bilateral lower lobe infiltrates (Fig. 1a). On the next day of admission he became tachypnoeic (RR 30–34/min), SpO₂- 88%, Arterial blood gas (ABG) showed mild hypoxemia, not improving with NRBM. The patient was put on NIV with inspiratory positive airway pressure 12 cmH₂O, expiratory positive airway pressure 8 cmH₂O and a fraction of inspired oxygen (FiO₂) 0.6. After a day of NIV he complained of pain and swelling in the neck. On examination, his neck appeared uniformly swollen and crepitus was palpable in the bilateral supraclavicular fossa extending on to both sides of the neck and upper chest. A bed-side chest ultrasonography was negative for pneumothorax. An x-ray of the chest and neck showed increased

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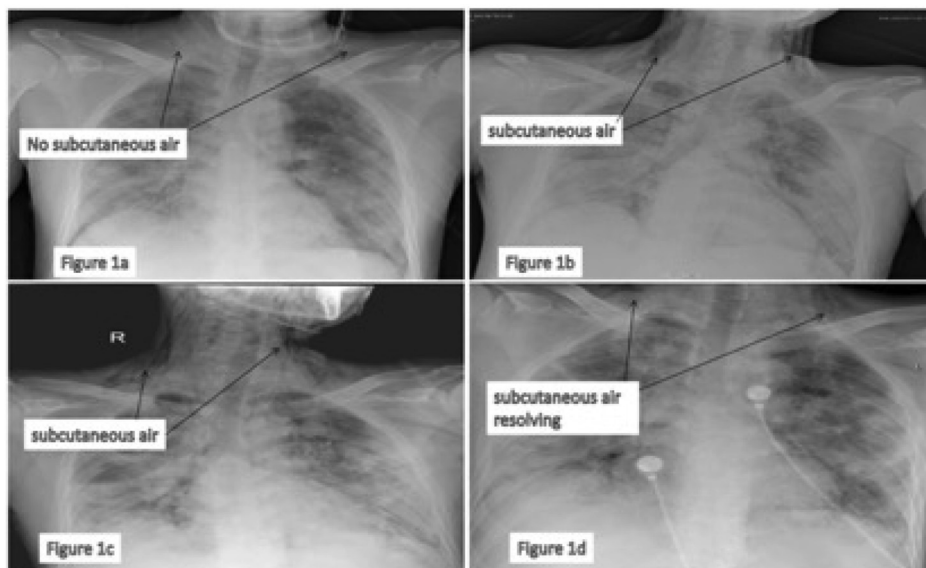


Fig. 1. a: Chest X-ray on admission. b: Chest X-ray on day 1 of NIV showing subcutaneous emphysema at bilateral supraclavicular and neck area. c: Chest X-ray on day 2 after discontinuation of NIV showing subcutaneous emphysema at bilateral supraclavicular and neck area. d: Chest X-ray on day 4 after discontinuation of NIV showing resolving subcutaneous emphysema at bilateral supraclavicular region.

lucency along subcutaneous tissue of neck bilaterally diagnostic of subcutaneous emphysema without pneumothorax (Fig. 1b). He was hemodynamically stable with SpO₂ 97% and normal blood gases and hence planned for conservative management. O₂ was provided via NRBM under continuous monitoring for respiratory parameters and emphysema progression. HFNC was kept on standby as a rescue measure if the patient deteriorated further. HFNC provides improved oxygenation, decreased anatomical dead space, decreased metabolic demand of breathing, decreased production of carbon dioxide, superior comfort, improved work of breathing, better compliance, positive nasopharyngeal and tracheal airway pressure, better secretion clearance and has lesser risk of barotrauma. The patient remained stable with NRBM and maintained SpO₂ above 95%. Subcutaneous emphysema was clinically non progressive and started showing gradual resolution on subsequent serial chest x-rays (Fig. 1b–d). O₂ requirement gradually decreased and he started maintaining saturation above 96% on room air and crepitations resolved. The patient was discharged home in a stable condition on the 15th day post admission when he became negative for SARS COV-2.

3. Discussion

There has been an increase in the number of cases of SARS COV-2 and patients presenting with ARDS requiring ventilatory support. In early mild cases NIV has been advocated [5,6]. NIV is easy to apply, the patient can be self prone [7] and can also be used in the wards. Our patient developed subcutaneous emphysema after he was put on NIV. NIV induced barotrauma though rare, may present with pneumothorax, pneumomediastinum with subcutaneous emphysema. Spontaneous SE and pneumomediastinum has been reported due to a sudden rise in intra alveolar pressure resulting in imbalance in capillary and lung pressure and rupture of marginal alveoli and subsequent tracking of air along bronchi, interstitial and vascular support tissues into the mediastinum (Macklin effect) and then dissects through the soft tissue planes of the neck, producing the subcutaneous emphysema [8,9]. End expiratory pressure provided by NIV increases the pressure gradient between alveoli and the interstitial space and may cause alveolar rupture.

Pneumothorax with NIV has previously been reported in patients having neuro-muscular disease with pleural blebs, cystic fibrosis, asthma and previous history of trauma [10–12]. Pneumomediastinum is likely to be related to rapidly increased intrathoracic pressure due to coughing during NIV, increasing air trapping areas (emphysema) with alveolar septa rupture and enhancing traction on stiffness of small airways (peribronchial fibrosis) causing the discontinuation of bronchoalveolar junction [13]. Gonzalez and colleagues (2016) reported a case of NIV induced exacerbation of air leak resulting in intense cervical subcutaneous emphysema and pneumomediastinum in a patient with asthma [2]. SE can occur without pneumothorax or pneumomediastinum in patients with cavitary pulmonary tuberculosis [14]. Singh and colleagues reported a case of spontaneous SE and pneumomediastinum in a patient with H1N1 pneumonia [15]. COVID-19 pneumonia has been shown to cause severe diffuse alveolar damage (DAD) secondary to direct infection of pneumocytes type I and II. Studies have demonstrated COVID-19 viral entry via angiotensin converting enzyme-2 (ACE-2) receptor into target cells including surfactant-producing type II pneumocytes causing cellular injury and dysregulation of surfactant production contributing to the development of SE and pneumomediastinum from impaired lung compliance [16,17]. SE can be due to trauma, surgery, infections or spontaneous origin. SE has also been reported due to dental procedures, drug abuse, adenoid-tonsillectomies, bowel perforation, arthroscopy, strangulation of the neck from hanging. No precipitating risk factor was found in our patient. NIV was the most likely contributory factor as supported by serial chest x-ray findings (Fig. 1a–d). However, the probability of spontaneous bullae rupture cannot be denied. CT thorax would have thrown more light into the etiopathogenesis, but it was not performed due to logistical issues and also because the patient improved with conservative treatment measures. The possibility of pneumothorax was ruled out with bedside ultrasonography which showed normally sliding pleura over all of the chest quadrants and absent barcode or stratosphere sign on M-mode scan. Ultrasound has a sensitivity of 58.9%–95.3% and a specificity of 91.1%–99.6% for detecting pneumothorax [18].

4. Conclusion

An increased number of COVID-19 patients receive NIV for the management of acute hypoxemic respiratory failure. Subcutaneous emphysema, though rare may occur in these patients. Timely recognition, intervention and monitoring may help reduce progression to dangerous complications.

Author's contribution

1. Dr Sunil Routaray, MD - Substantial contributions to conception and design and drafting of the article.

2. Dr Chandrakant Prasad, MD- Drafting of the article and critical revision for important intellectual content.

3. Dr Vanitha Rajagopalan, MD, DM - Drafting of the article and critical revision for important intellectual content.

Final approval of the version to be published.

Agreement to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the article are appropriately investigated and resolved.

Declaration of competing interest

The authors have no conflict of interest to declare.

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