NON-INVASIVE VENTILATION AS A DOUBLE-EDGED SWORD IN
CHASING SPO2 IN PATIENTS WITH COVID 19 DISEASE- A CASE SERIES

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Introduction:

A new respiratory virus, subsequently dubbed severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), was discovered to be the cause of the coronavirus illness 2019 in December 2019. It originated in Wuhan (China) (COVID-19). SARS-CoV-2 was proclaimed a pandemic on March 11, 2020, when it was recognised as a serious worldwide public health issue. [1]

WHO describes, “Coronavirus disease (COVID-19) as an infectious disease caused by the SARS-CoV-2 virus, where most people infected with this virus will experience mild to moderate respiratory illness and recover without requiring special treatment. However, some will become seriously ill and require medical attention. Older people and those with underlying medical conditions like cardiovascular disease, diabetes, chronic respiratory disease, or cancer are more likely to develop serious illness. Anyone can get sick with COVID-19 and become seriously ill or die at any age. The virus can spread from an infected person’s mouth or nose in small liquid particles when they cough, sneeze, speak, sing or breathe. These particles range from larger respiratory droplets to smaller aerosols. It is important to practice respiratory etiquette, for example by coughing into a flexed elbow, and to stay home and self-isolate until you recover if you feel unwell”.

During the COVID-19 pandemic, the number of patients who require oxygen (O2) supplementation through face mask, non-rebreathing/reservoir face mask (NRBM), high flow nasal cannula (HFNC), NIV assistance, or invasive
ventilation has increased. These individuals are referred to be "happy hypoxics" and are only intubated if clinical symptoms of increased labour of breathing, desaturation, or developing hypercapnia are present.

HFNC or NIV are now reserved for severe hypoxemia with airborne precautions. [2] Pressure ulcers/necrosis of the nasal bridge, face or ocular abrasions, oronasal mucosal dryness, claustrophobia, anxiety, agitation, abdominal distension, vomiting, aspiration, decreased speech, and reduced nutrition are all common problems linked with NIV.

“Barotrauma, pneumothorax, pneumomediastinum, pneumocephalus, high intracranial pressure, and raised intraocular pressure are all uncommon consequences. In individuals with chronic obstructive pulmonary disease (COPD) and asthma, NIV-induced pneumomediastinum and subcutaneous emphysema (SE) have been documented”. [3]

Here we are presenting a case series of four patients of COVID 19 in our institution who had developed bilateral subcutaneous emphysema while receiving PPV through NIV without pneumothorax and pneumomediastinum having no previous history of respiratory disease.

Out of four, two patients died shortly after developing SE, the other one received invasive ventilation after developing SE but later succumbed to the disease. One patient survived and walked out of ICU after 22 days.

Case # 1.

A 55-year male with underlying hypertension on telmisartan 20mg presented with the complaints of fever and cough for a seven days and shortness of breath for one day duration Rapid Antigen test (RAT) for SARS-COV-2 was POSITIVE. He was allergic to cotrimoxazole. Other medical and surgical history was unremarkable. On admission in covid designated ward of SMHS Hospital, patient was conscious and oriented with heart rate (HR)- 110/min, blood pressure (BP)- 120/70 mm Hg, oxygen saturation (SpO2)- 87% on room air and respiratory rate (RR)- 28/min. Laboratory tests on admission showed Hb 12.3g/dl, TLC 3.5, PLT 146, s.cr 0.83, urea 42, sodium 138, potassium 4.0. Covid markers: Crp 55, IL6 39.3, D-DIMER 4.48, FERRETIN 902, LDH 567.

Chest X-ray showed Bilateral infiltrates involving middle and lower lobes more marked on Left side (fig 1). He was started on supportive treatment including injectable steroids and injection remdisivir with O2 supplementation @ 8L/min.
initially via a face mask and later 15L/min via a NRBM. Patient was managed in covid 19 ward for 3days and then shifted to COVID ICU in view of Tachypnea (R/R = 30-32), fall in spO2 (82%) and Arterial blood gas suggestive of Mild Hypoxemia (PO2 50) not improving on NRBM. Patient was put on High flow nasal cannula (HFNC) with O2 @ 40L/min initially with subsequent increase to 60L/min over 7days. With further worsening of symptoms , Non resolving fever, increase work of breathing and worsening covid markers the patient was put on NIV with inspiratory positive airway pressure 16cmH2O, expiratory positive airway pressure 12 cmH2O and a fraction of inspired oxygen (FiO2). Patient was also advised Awake proning for 12 hrs a day. After 5 days of NIV, the patient complained of pain and swelling on the neck. On examination, his neck was uniformly enlarged, and crepitus could be felt in the bilateral supraclavicular fossa, which extended down both sides of his neck and into his upper chest. An x-ray of the chest and neck revealed increased lucency along the subcutaneous tissue of the neck in both directions, indicating subcutaneous emphysema but no signs of pneumothorax (fig 2). A bedside chest ultrasonography revealed that there was no pneumothorax. Other hemodynamics were within normal limits with oxygen saturation of 92% with persistent hypoxemia on ABG, patient was continued on NIV with inspiratory positive airway pressure 12cmH2O, expiratory positive airway pressure 8cmH2O and a fraction of inspired oxygen (FiO2) 1 and intermittent HFNC + NRBM trials for next 5 days. Subcutaneous emphysema was clinically non progressive and started showing gradual resolution on subsequent serial chest x-rays. with resolution of the covid markers the fever subsided and patient showed clinical improvement and gradual correction of hypoxemia

Patient was put on full time HFNC with gradual reduction in oxygen flow from 60L/min to 30L/min. With further improvement in hypoxemia and gradual reduction of oxygen dependency patient was finally discharged from ICU on Nasal prongs with oxygen flow @ 6L/min after 22days.
Case # 2

65 year old female hypertensive, hypothyroid and on permanent pacemaker for underlying complete heart block presented with fever and body aches for 5 days and breathlessness for 1 day. RAPID ANTIGEN TEST was POSITIVE in this patient. Vitals on admission in covid designated ward of SMHS hospital was as
follows: GCS 15/15, heart rate (HR)-100, blood pressure (BP) 130/90, saturation (SPO2) on room air 84%, respiratory rate (RR)-30. Covid markers = CRP-26, IL6-39, D-dimer-0.6. Chest X-ray showed bilateral lower and middle lobe consolidation (fig 3). Based on clinical symptoms, examination and lab parameters patient was diagnosed as a case of covid pneumonia and admitted in covid designated ward of SMHS hospital. She was started on supportive treatment including injectable steroids and Injection REMDISIVIR, O2 supplementation @15L/min via a NRBM. Patient was managed in covid 19 ward for 1 day. In view of worsening of symptoms and high oxygen demand patient was shifted to covid ICU for further management. Patient was put on High flow nasal cannula (HFNC) with O2 @ 60L/min, increased dose of steroids and subsequent adjustment according to covid markers and clinical symptoms. Awake proning for more than 12 hrs a day with intermittent change in posture 2 hourly and other supportive management. Vitals on HFNC with O2 at 60lts/min, Heart rate (HR) -110, blood pressure (BP) -136/82, saturation (SPO2)- 85%, respiratory rate (RR) -36. Patient continued to be on HFNC for 3 days. Further worsening with high grade fever, increase work of breathing, worsening of hypoxia, rising trend in covid markers = CRP-126, IL6-560, D-dimer-2.1. Patient was put on NIV with inspiratory positive airway pressure 18cmH2O, expiratory positive airway pressure 14 cmH2O and a fraction of inspired oxygen (FiO2) 1. Pulse therapy of steroids (methyleprednisolone 125mg bd for 5 days with constant check on kft). Inj Tocilizumab could not be added to the patient for her raised procalcitonin levels. After 3 days on NIV, “patient’s neck appeared to be swollen and crepitus was palpable in the bilateral supraclavicular fossa extending on to both sides of the neck and upper chest. Bed side chest xray was S/O increased lucency along subcutaneous tissue of neck bilaterally diagnostic of subcutaneous emphysema without any evidence of pneumothorax” (FIG-4). Bed-side chest ultrasonography was negative for pneumothorax but with oxygen saturation of 78-82%, persistent hypoxemia on ABG patient was continued on NIV with high pressures to maintain spo2 as the attendants were reluctant for intubation and mechanical ventilation. Further progression of SE was seen involving chest, abdomen and upper limbs and then further airway was compromised with fall in spo2 followed by hypoxia, cardiac arrest and death.
Case 3:

50 year female with underlying type2diabetes on oral hypoglycemics since 5 years developed fever and cough. she took symptomatic treatment at home for 3 days. On worsening of symptoms i:e non resolving fever and cough, she underwent rapid antigen test for SARS-COV2 which came out to be positive, patient was advised symptomatic treatment and remained in home isolation. On further worsening of (breathlessness and fall in saturation -82% on room air) she was then shifted to covid designated ward of SMHS hospital. Vitals on admission :GCS15/15,Heart Rate (HR)-102,Blood Pressure (BP)120/90 Oxygen saturation (SPO2) 84% on room air, Respiratory Rate(RR)-30. 

Covid markers=CRP-30,IL6-51,D-dimer-0.5. She was started on oxygen therapy via non rebreather mask(NRBM) with oxygen flow at 15lts/min and other supportive treatment including injectable steroids and Injection REMDISIVIR. Patient was managed in covid 19 ward for 2 days. In view of worsening of symptoms (SPO2-60%), persistent hypoxemia on ABG and high oxygen demand patient was shifted to covid designated ICU of SMHS hospital for further management. Patient was put on High flow nasal cannula (HFNC) with O2 @ 60L/min, pulse therapy of steroids(with constant check on kft) and subsequent adjustment according to covid markers and clinical symptoms. Awake proning for more than 12 hrs a day with intermittent change in posture 2 hourly and other supportive management for next two days patient was maintaining saturation of 72 -80% on HFNC. In view of increased work of breathing and worsening hypoxemia patient was put on Non Invasive Ventilation (NIV) with inspiratory positive pressure of 12cm H2O and expiratory positive pressure of 8cmH2O (which subsequently increased to ipap =18 and epap=14 over next two days) and fraction of inspired oxygen. After 2 days, patient developed pain and swelling in neck. On examination crepitus was palpable on both sides of neck and upper chest. Xray(FIG-5) chest was s/o subcutaneous emphysema without any evidence of pneumothorax. Bed-side chest ultrasonography was negative for pneumothorax. Since there was persistent worsening of symptoms and expanding subcutaneous emphysema on NIV patient was intubated and put on mechanical ventilation, patient was on mechanical ventilation for 2 days and died of cardio respiratory arrest due to hypoxemia.
Discussion:
The frequency of instances of SARS COV2 and patients presenting with ARDS requiring ventilatory support has increased. “NIV has been recommended in severe situations [4,5]. NIV is simple to use, the patient may be self-prone [6],
and it can be used in wards as well. After starting NIV, our patient developed subcutaneous emphysema. Although NIV-induced barotrauma is uncommon, it can cause pneumothorax, pneumomediastinum, and subcutaneous emphysema. Spontaneous SE and pneumomediastinum have been reported as a result of a sudden rise in intra alveolar pressure, resulting in an imbalance in capillary and lung pressure, rupture of marginal alveoli, and subsequent tracking of air into the mediastinum (Macklin effect), where it dissects through the soft tissue planes of the neck, causing subcutaneous emphysema [7,8].

The pressure differential between the alveoli and the interstitial space is increased by NIV, which might lead to alveolar rupture. NIV-induced pneumothorax has been described in individuals with neuromuscular illness, pleural blebs, cystic fibrosis, asthma, and a history of trauma [9,10,11]. Pneumomediastinum is thought to be caused by increased intrathoracic pressure from coughing during NIV, increased air trapping areas (emphysema) from alveolar septa rupture, and increased traction on stiffness of small airways (peribronchial fibrosis) from bronchoalveolar junction discontinuation [12]. Gonzalez and colleagues (2016) described “a case of NIV-induced air leak exacerbation in a patient with asthma, which resulted in severe cervical subcutaneous emphysema and pneumomediastinum [3]. In individuals with cavitary pulmonary TB, SE can arise without a pneumothorax or pneumomediastinum [13]. In a patient with H1N1 pneumonia, Singh and colleagues described a case of spontaneous SE with pneumomediastinum”[14].

COVID-19 pneumonia has been demonstrated to induce severe diffuse alveolar damage (DAD) when pneumocytes type I and II are infected directly. COVID-19 virus enters target cells, including surfactant-producing type II pneumocytes, via the angiotensin converting enzyme-2 (ACE-2) receptor, causing cellular injury and surfactant production dysregulation, which contributes to the development of SE and pneumomediastinum due to impaired lung compliance [15,16]”. SE can be caused by trauma, surgery, infections, or it can happen on its own. Dental treatments, drug misuse, adenoid-tonsillectomies, intestinal perforation, arthroscopy, and strangling of the neck from hanging have all been linked to SE. In our case, no precipitating risk factor was discovered. Serial chest x-ray data indicated that NIV was the most probable contributing cause (Fig. 1aed). The possibility of spontaneous bullae rupture, on the other hand, cannot be ruled out. A CT scan of the thorax would have shed further light on the etiopathogenesis, however it was not done due to logistical concerns.
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

REFERENCES:


