Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

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Alveolar air leakage in COVID-19 patients: Pneumomediastinum and/or pneumopericardium

We read with great interest the recent article by Sahu k et al., which presents their case report of pneumopericardium in COVID-19 patient.[1] We congratulate the authors for their good observation and addressing this clinical problem. However, we have some concerns about the report and we would like to share our insights about air leakage conditions in COVID-19 patients.

In isolated pneumopericardium, the air is confined to the limits of pericardial reflection on the proximal great vessels and this constitutes the basis for diagnosis. However, the differentiation between pneumopericardium and pneumomediastinum (PM) may not be possible in plain chest x-ray; especially when both conditions are present simultaneously. Accordingly, further work up may be needed to support proper diagnosis. In decubitus film, Pericardial (but not mediastinal) air will shift to the nondependent side. Additionally, in upright lateral film, the normally invisible ventral part of the left hemidiaphragm will be clearly outlined between the air in the pericardium and the gastric air bubbles; this is called the continuous left hemidiaphragm sign.[2] Needless to say, that chest computed tomography (CT) scan delineates the distribution and extent of the air in the mediastinum more clearly.[3]

Moreover, in pneumopericardium, the electrocardiogram may detect nonspecific ST segment, T-wave changes, and low voltage. Also, echocardiography may be technically difficult due to the presence of air: however, the detection of air gap sign (which denotes loss of the image of the heart during the systolic phase of each cardiac cycle in both M-mode and two-dimensional views) supports the diagnosis.[4]

Unfortunately, the authors depended in their diagnosis only on one chest x-ray view; we wish if they could have supported their diagnosis with any of the aforementioned modalities.

The pathophysiology of PM can be explained by Macklin effect; with alveolar rupture, the released air dissects through the perivascular and peribronchial sheathes to the mediastinum. Thereafter, the air may find its way to other spaces including the pericardium. Air enters the pericardium along the pulmonary veins or through pericardial defects whether congenital or induced by trauma.[2,4]

Multiple studies have observed an increased number of air leakages in COVID-19 patients producing PM with or without pneumothorax.[5,6] The cytokine storm involved in the pathophysiology of COVID−19 induces diffuse alveolar injury which makes alveoli more liable to rupture. Alveolar rupture can be induced by either strenuous cough in spontaneously breathing patients or by barotraumas in mechanically ventilated patients due to frequent use of high positive end expiratory pressure.[7]

Pneumomediastinum is usually self-limiting, and requires no specific intervention. However, for cases on mechanical ventilation the air leakage can be so massive that it may disturb the airway, venous return, and possibly produce cardiac tamponade when there is communication with the pericardium. Adjustment of ventilator settings to be less injurious to the lung and treatment of the underlying lung disease are the mainstay of management of PM.

Regarding the leaked air itself, authors deal with this problem differently. Wali et al., followed an aggressive approach of bilateral intra−pleural and subcutaneous chest drains insertion for decompression of severe PM.[8] They also advised the use of the technique described by Byun et al., which entails the application of vacuum-assisted closure therapy (originally used for wound management) via a small infra-clavicular skin incision that extends deeper to the pectoral fascia.[9]

On the other hand, Volpi et al., successfully managed PM in two COVID-19 patients conservatively without any intervention.[10]

We followed a less interventional approach. Our policy in absence of any evidence of pneumothorax is to insert a unilateral intrapleural chest drain to protect at least one side against potential pneumothorax. We applied this policy in five COVID-19 cases. The justification for this approach is that, the development of pneumothorax (which could be tension or bilateral) in these patients with relatively low pO_2 can be life threatening and needs immediate intervention; the time delay needed to call thoracic surgeon may be hazardous Fig. 1.

At the time of writing this letter, three patients were successfully weaned from mechanical ventilation, one patient is still on ventilator through a tracheostomy tube and another patient died of multi-organ failure 16 days after chest drain insertion. None of our patients developed pneumothorax on the un-drained side. Mediastinal and subcutaneous emphysema resolved in 3 patients, and significantly diminished in the others.

In summary, our management strategy of pneumomediastinum in COVID-19 patients is to insert a prophylactic unilateral intrapleural chest drain and then intervene accordingly.

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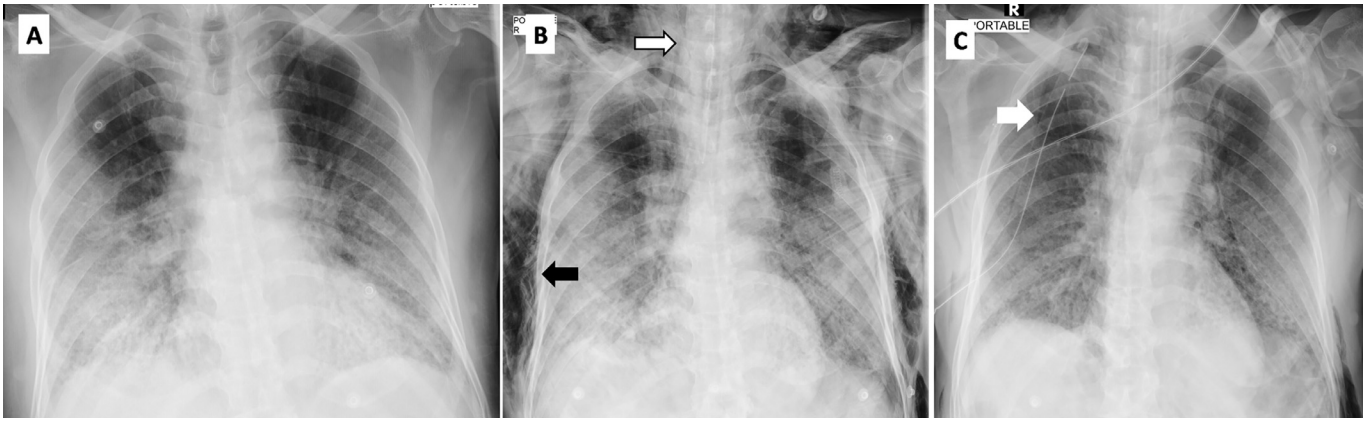

Fig. 1. Serial chest x-rays in a patient with covid-19 infection demonstrating clinical course of mediastinal emphysema.
A: a chest X-ray on admission, showing bilateral heterogonous opacities.
B: a chest X-ray two days after intubation, showing development of mediastinal emphysema (white arrow) extending to subcutaneous tissue (black arrow). Note, the air outlines the borders of the heart extending along the base of great vessels; however a firm diagnosis of pneumopericardium cannot be proposed.
C: a Chest X-ray seven days later, showing chest drain in place (white arrow) with marked diminution of surgical emphysema.

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Declaration of Competing Interest

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