Respiratory failure due to diaphragm paralysis after brachial plexus injury diagnosed by point-of-care ultrasound

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SUMMARY
A man in his fifties was injured in a traffic accident and diagnosed with traumatic subarachnoid haemorrhage, liver injury, and fractures of the rib, right clavicle, right scapula and right femur. He also presented with motor and sensory disturbances of the right upper extremity and was suspected of having a brachial plexus injury. After undergoing mechanical ventilation due to multiple traumas, he was extubated. However, he developed acute respiratory failure and required reintubation. Respiratory symptoms were not clear until just before reintubation. The diagnosis of right diaphragm paralysis was made using point-of-care ultrasound with no other findings that could cause respiratory failure. MRI led to the diagnosis of brachial plexus injury, which likely caused diaphragm paralysis. Point-of-care ultrasound provided a clear visualisation and rapid bedside diagnosis of diaphragm paralysis, which can be challenging to diagnose while ruling out other causes of respiratory failure.

BACKGROUND
Traumatic brachial plexus injury is caused by mechanisms, such as stretching or contusion, particularly in motor vehicle accidents.

Traumatic cases, especially those involving cervical roots avulsions, tend to cause severe neurological and frequently require surgery instead of conservative treatment among brachial plexus injuries.

Brachial plexus injuries present with a variety of symptoms due to C5-Th1 nerve injury, but complications of diaphragm paralysis have rarely been reported.

Diagnosing critically ill patients with diaphragmatic paralysis is challenging. In general, it is diagnosed using radiography or fluoroscopic 'sniff test'. However, a radiograph alone cannot provide a definitive diagnosis of diaphragmatic paralysis. In addition, the sniff test is difficult to conduct in critically ill patients.

We encountered a case of a patient with respiratory failure due to diaphragm paralysis with brachial plexus injury that was rapidly diagnosed using point-of-care ultrasound (POCUS). The ultrasound findings were also reported in this study.

CASE PRESENTATION
A 55-year-old man presented to our tertiary medical care centre following a motor vehicle accident. On arrival, he was in hypovolaemic shock with impaired consciousness. The chief complaint was abdominal pain associated with motor and sensory disturbances in the right upper extremity. Contrast-enhanced CT revealed traumatic subarachnoid haemorrhage, liver injury and fractures of the rib, right clavicle, right scapula and right femur. The coronal view of the enhanced CT showed that the continuity of the diaphragm was maintained. The motor and sensory deficits in the right upper extremity were not explained by direct injury. Therefore, a brachial plexus injury was considered possible. After being admitted to the intensive care unit (ICU), the patient was intubated due to multiple trauma.

INVESTIGATIONS
In the ICU, radiographic imaging revealed occasional and temporary diaphragmatic elevation (figure 1). Therefore, we considered the elevation of the diaphragm was caused by pleural effusion or atelectasis. The patient had a successful spontaneous breathing trial after undergoing mechanical ventilation for 6 days, and he was extubated.

One day after extubation, the patient suddenly developed dyspnoea. He was tachypnoeic with a respiratory rate of about 50 breaths per min and was reintubated. Arterial blood gas under 3L oxygen via nasal cannula revealed a pH of 7.410, partial pressure of oxygen of 74.4 mm Hg, partial pressure of carbon dioxide of 41.4 mm Hg and bicarbonate level of 25.9 mmol/L.

Since the carbon dioxide concentration was normal despite tachypnoea, the carbon dioxide excretion was decreased. Bedside POCUS revealed that the right diaphragm exhibited significantly less respiratory movement than the left, and the distance of the right diaphragm movement was shortened (video 1).

Therefore, the patient was diagnosed with acute respiratory failure secondary to diaphragm paralysis.

DIFFERENTIAL DIAGNOSIS
POCUS also revealed no signs of pneumothorax, massive pleural effusion and acute heart failure. Chest radiography showed that the right diaphragm was elevated compared with the left. Atelectasis was considered, but there was no clinically significant sputum accumulation on bronchoscopy immediately after reintubation. Contrast-enhanced CT revealed no pulmonary embolism, diaphragm injury or severe pneumonia.
In particular, the diaphragm injury was ruled out due to the lack of segmental diaphragm defects by CT on days 1 and 7. Thereafter, no abnormalities were noted in the follow-up imaging studies.

A neck MRI scan revealed root avulsion injury of C5 and C6, which likely caused diaphragm paralysis (figure 2).

The diagrammatic timeline of diagnosis is shown in figure 3.

**OUTCOME AND FOLLOW-UP**

The patient was extubated on hospital day 15. Although he had dyspnoea for approximately a month after extubation, the arterial blood gas showed no hypoxaemia or carbon dioxide retention. The frequency of dyspnoea decreased over time. The patient was transferred to another hospital for further rehabilitation, and nerve grafting was considered for brachial plexus injury treatment.

**DISCUSSION**

In our case, the brachial plexus avulsion injury was associated with diaphragm paralysis, and POCUS aided in the diagnosis.

In general, diaphragmatic dysfunction is difficult to diagnose with certainty without direct observation, either by laparoscopy or laparotomy. For example, diaphragmatic injury is one of the differential causes of diaphragmatic dysfunction and requires special attention because it may be asymptomatic immediately after injury. Chest X-ray fails to reveal abnormalities in 20%–50% of patients with diaphragmatic injury.

The gold standard for diagnosis is an examination by CT, which has been reported to have high diagnostic accuracy with a sensitivity of 61%–87% and a specificity of 72%–100%. A segmental diaphragmatic defect is a common finding, with a reported sensitivity of 95.7%, and accuracy is further improved by combining sagittal and coronal views.

However, slight abnormalities in diaphragm thickness or small diaphragmatic defects cannot be noted, especially when a major distracting abnormality is nearby. Furthermore, in ventilated patients, positive pressure may mask diaphragm elevation, which can make diagnosis difficult. In our case, the initial CT and follow-up CT images over time did not show any findings suspicious of diaphragmatic injury.

The diaphragm is innervated by the phrenic nerve, which originates from the C3 to C5 nerve roots. In most cases, the C4 root is integral to diaphragmatic function. The brachial plexus is composed of the C5-Th1 nerve roots, and the C4 and C5 roots are fibrously connected (figure 4). When the C5 root is avulsed and damaged, C4 is also damaged, resulting in diaphragmatic dysfunction.

Phrenic nerve injury has reportedly occurred in 10%–20% of brachial plexus injuries. However, to the best of our knowledge, only Franko et al have reported a case of diaphragm paralysis following brachial plexus injury who developed acute respiratory failure. Although chest radiography revealed unilateral diaphragm elevation on hospital day 5, pleural effusion was primarily suspected. The diagnosis of diaphragmatic paralysis was confirmed by CT.
was not made until an ultrasound was performed to evaluate the pleural effusion.

In our case, the diagnosis of diaphragmatic paralysis was also difficult because the radiologic finding of diaphragm elevation was not always apparent. The reason for the rarity of similar case reports may suggest that diaphragm paralysis may have been overlooked because of the difficulty of diagnosis.

The sniff test on fluoroscopy and X-ray was reportedly helpful in diagnosing unilateral diaphragm paralysis. However, its application to critically ill patients remains controversial. The sniff test requires the patient to control spontaneous breathing and move to the examination room. Unilateral diaphragm elevation on radiography is a suggestive finding, but it has low specificity (sensitivity: 0.90, specificity: 0.44). In our case, unilateral diaphragm elevation was occasionally seen on radiography. However, the respiratory symptoms had only occurred just before the patient required reintubation. Although diaphragm paralysis lacks specific findings, it is essential to diagnose it immediately because it has serious consequences.

Point-of-care diaphragmatic ultrasound was reportedly useful in evaluating critically ill patients. Ultrasonography is noninvasive and highly reproducible, allowing bedside assessment within approximately 5–15 min, which can simultaneously assess findings of abnormalities in the thorax that may cause respiratory failure. The ultrasound findings of diaphragm paralysis are documented as a loss of diaphragmatic excursion, paradoxical excursion during deep breathing and sniffing, and a respiratory variation of diaphragm thickness of <20%. In this patient, the right diaphragm motion was significantly less than that of the left diaphragm, and the right diaphragm excursion disappeared (video 1), which led to the rapid diagnosis of diaphragm paralysis. Video 1 shows that there was little respiratory variability on the right diaphragm despite tachypnoea, while the left diaphragm moves along with the breathing. Moreover, no other abnormal findings could have caused the respiratory failure. Therefore, even a rare disease like this case could be diagnosed easily. This case highlighted that POCUS plays an important role in diagnosing the cause of severe respiratory failure.

### Learning points

- **Diaphragm paralysis associated with brachial plexus injury is rare but can cause severe respiratory failure.**
- **The specificity of hemidiaphragmatic elevation on X-ray is low, and diaphragm paralysis may be overlooked and mistaken for pleural effusion or atelectasis.**
- **Point-of-care ultrasound provided a clear visualisation and rapid diagnosis for causes of respiratory failure at the bedside.**

### Contributors

WF and SY equally contributed to the literature review, the study concept and the drafting of the manuscript. All other authors provided critical feedback and contributed to the formation of this study and manuscript. WY and TK obtained the consent for publishing this study from the patient and family. MU approved the final manuscript.

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Case reports provide a valuable learning resource for the scientific community and can indicate areas of interest for future research. They should not be used in isolation to guide treatment choices or public health policy.

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