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Critical Rounds Newsletter

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Dear Doctor,

Greetings from Micro Labs Limited. We are happy to bring you **"Critical Rounds Newsletter "** which contain latest and interesting news in the field of Critical Care .

This issue contains :

1.Safety and Efficacy of Prostacyclin in COVID-19 Patients with Severe Endotheliopathy.

2.The efficiency of Cell cycle arrest biomarkers for predicting renal recovery in acute kidney injury.

3.Prevalence and Prognosis of respiratory pendelluft in ICU patients with acute respiratory failure on mechanical ventilation.

4.A case of Respiratory failure caused by diaphragm paralysis

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Looking forward to your valuable feedback

Best Regards,
Medical Team, Micro Labs Limited

-- Critical Rounds----------

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1.Safety and Efficacy of Prostacyclin in COVID-19 Patients with Severe Endotheliopathy

Introduction:

Patients with severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) who require mechanical ventilation have a significant mortality rate. Patients with severe COVID-19 have a mortality rate of 30–40%, which is higher in those who require invasive mechanical ventilation.[1] Microvascular thrombosis of the pulmonary vasculature has been seen often in autopsy series. These findings are consistent with endotheliopathy being a prominent feature of the COVID-19 acute respiratory distress syndrome pathogenesis, which has been observed in patients with varying levels of other severe infections.[3], In patients with COVID-19, the level of circulating soluble thrombomodulin (sTM) was associated with mortality.[3] The anticoagulant protein C system includes thrombomodulin, and its cleavage from the endothelium may be involved in the pathogenesis of the prothrombotic phenotype seen in COVID-19 individuals. The use of prostacyclin (PGI2) as a pharmaceutical therapy for patients with primary pulmonary hypertension and critical limb ischemia is based on its paracrine function, which includes dose-dependent vasodilation and platelet inhibition. Multiple beneficial effects of prostacyclin on the endothelium have been described in the new century.[2] The objective of this randomized controlled trial was to evaluate the safety and efficacy of prostacyclin to placebo on days alive without mechanical ventilation in mechanically ventilated COVID-19 patients with documented endotheliopathy, as measured by a circulating sTM level of >4 ng/ml, within 28 days.

Prostacyclin was not associated with a significant reduction in the number of days alive and without mechanical ventilation within 28 days

Methods:

This is a multicenter, blinded, parallel grouped, randomised (1:1, active: placebo) exploratory experiment of low-dose continuous prostacyclin against placebo for 72 hours in mechanically ventilated COVID-19 patientsAdult (>18 yr.) patients with proven SARS-CoV-2 infection who were invasively mechanically ventilated and had a circulating sTM level of.4ng/ml measured by a lateral flow immunoassay were included in the study. Within 28 days following randomization, the primary outcome was the number of days living without mechanical ventilation. Secondary outcomes included 28- and 90-day mortality, mean daily Sequential Organ Failure Assessment (SOFA) score in the ICU up to Day 90, days alive without vasopressor in the ICU between 28 and 90 days, days alive without mechanical ventilation in the ICU between 28 and 90 days, days without renal replacement in the ICU between 28 and 90 days, and days without renal replacement in the ICU between 28 and 90 days, the number of serious adverse reaction and serious adverse events in the first seven days. Prostacyclin (1 ng/kg/min) or placebo was administered as a continuous intravenous infusion for 72 hours in the patients.

Results:

The main outcome was the number of days alive and without mechanical ventilation within 28 days. Key secondary outcomes were 28-day mortality and serious adverse events within 7 days. Eighty patients were randomized (41 prostacyclin and 39 placebo). The median number of days alive without mechanical ventilation at 28 days was 16.0 days (SD, 12) versus 5.0 days (SD, 10) in the prostacyclin and the placebo groups, respectively. The 28-day mortality was 21.9% versus 43.6% in the prostacyclin and the placebo groups, respectively. The incidence of serious adverse events within 7 days was 2.4% versus 12.8% in the prostacyclin and the placebo groups, respectively.

	Prostacyclin Group (*n* = 41)	Placebo Group (*n* = 39)	Difference of the Medians, Adjusted Difference of the Means, or Risk Ratio (CI)	*P* Value
Primary endpoint				
Median days alive without mechanical ventilation in the ICU at 28 d (intention-to-treat)	16	5.04	10.96 (−5 to 21)	0.07
Median days alive without mechanical ventilation in the ICU at 28 d (per protocol)	15	3.02	11.98 (−6 to 21)	0.08
Secondary endpoints				
Mortality at 28 d	9 (21.9)	17 (43.6)	0.50 (0.24 to 0.96)	0.06
Mortality at 90 d	13 (31.7)	19 (48.7)	0.65 (0.36 to 1.12)	0.17
Mean daily SOFA score adjusted for baseline values	5.75	6.67	1.1 (0.28 to 1.92)	0.009
Median days alive without vasopressor in the ICU at 28 d	22	13	9 (−1.5 to 18)	0.14
Median days alive without vasopressor in the ICU at 90 d	84	59	25 (−3 to 75.5)	0.16
Median days alive without RRT in the ICU at 28 d	28	21	7 (0 to 12)	0.06
Median days alive without RRT in the ICU at 90 d	90	79	11 (−2.5 to 74)	0.08
Median days alive without mechanical ventilation in the ICU at 90 d	77	13	64 (−6 to 80)	0.10
Serious adverse event(s) within 7 d	1 (2.4)	5 (12.8)	0.19 (0.01 to 1.11)	0.10
Serious adverse reaction(s) within 7 d	0 (0)	0 (0)	—	—

Outcome Measures according to Treatment Assignment

Discussion:

In this multicenter randomised trial, patients with COVID-19 who were given prostacyclin or placebo for 72 hours did not have a statistically significant difference in the number of days alive without mechanical ventilation within 28 days. The point estimate, on the other hand, favoured the prostacyclin group, same as all of the secondary outcomes, the mean daily SOFA scores, which were statistically significantly lower in the prostacyclin group than in the placebo group.Prostacyclin has been shown to have many beneficial effects on the endothelium, including synthesising endothelial glycocalyx constituents , inducing reendothelization of damaged vessels , improving tight junction integrity , and minimising the inflammatory hit on the endothelium . [2]The researchers suspect that the observed results, are due to prostacyclin. Moezinia et al. reported that 5-day continuous infusion of low-dose prostacyclin was associated with reduced oxygen requirements, increased PaO2:FiO2 ratio, and normalisation of heart rate up to 48 hours, suggesting an improvement in vital organ function. [4]The researchers discovered no significant differences in serious adverse events or reactions between the two groups, suggesting that prostacyclin at a dose of 1 ng/kg/min could be safe in critically sick patients with COVID-19 and severe endotheliopathy. Results from randomised clinical trials in patients receiving liver transplantation and those with septic shock back up these findings.[5]

Conclusion:

The study concluded that in mechanically ventilated patients with COVID-19 and severe endotheliopathy who were administered prostacyclin or placebo for 72 hours, there was no statistically significant difference in the number of days alive without mechanical ventilation within 28 days. In all analysis, including mortality, the point estimates favoured the prostacyclin group. Because of the significant unmet medical need, the findings support a major randomised clinical trial of prostacyclin in mechanically ventilated COVID-19 patients with severe endotheliopathy.

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2.The efficiency of Cell cycle arrest biomarkers for predicting renal recovery in acute kidney injury

Introduction:

Acute kidney injury (AKI) is a common ailment in the intensive care unit (ICU) that can lead to chronic kidney disease (CKD) and short- and long-term mortality. Because particular treatment therapies and prevention measures are currently limited, renal recovery post-AKI has become the research focus.[1] Furthermore, alterations in renal functional reserve may have a significant impact on clinical outcomes in AKI patients. Patients with AKI who do not recover renal function have a much higher risk of death than those who recover.[2] As a result, the therapeutic goal of AKI should be to avoid the loss of renal function. Cell cycle arrest of urinary tissue inhibitor of metalloproteinase-2 (TIMP-2) and insulin-like growth factor-binding protein 7 (IGFBP7) are elevated early after AKI onset and have been shown to be superior in the early detection of AKI.[1] Only a few studies have examined at their performance as non-renal recovery prognostic indicators. If we can identify which patients will not recover in early AKI, we can implement effective supportive treatments before permanent recovery occurs, thus preventing future AKI progression and improving clinical prognosis. This study examined and evaluated the efficiency of urine [TIMP-2] × [IGFBP7] for predicting non-recovery in patients who developed AKI after ICU admission by measuring urinary TIMP-2 and IGFBP7 after AKI was identified.

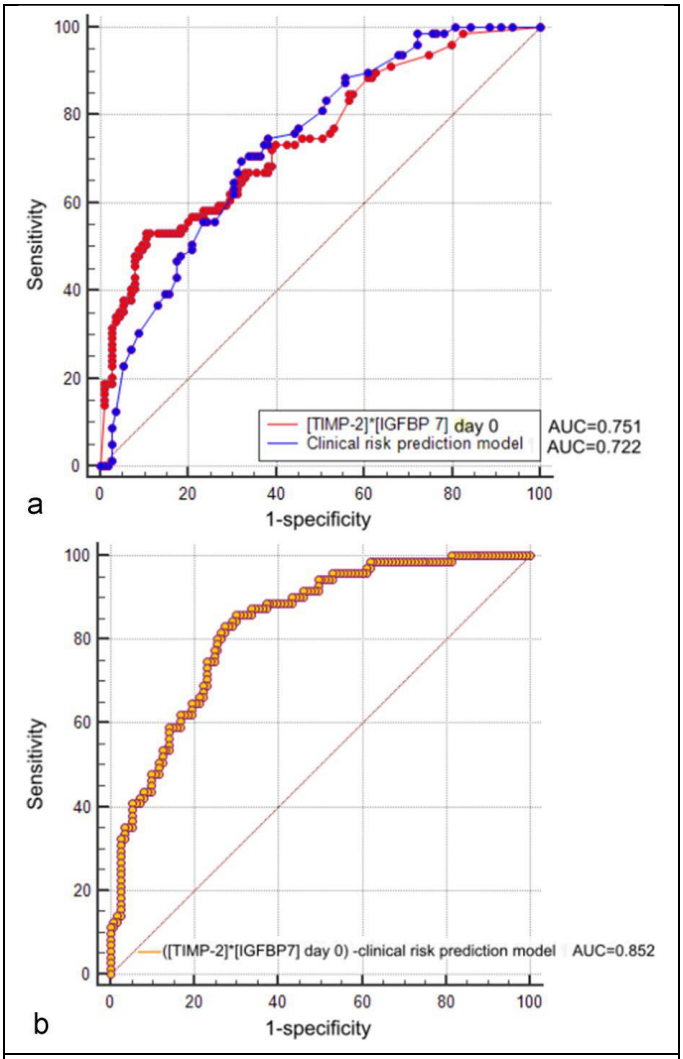
Urinary [TIMP-2]×[IGFBP7] on day 0 showed a fair performance for predicting nonrecovery from Acute Kidney Injury

Methods:

The research was conducted in two hospitals. The study's design, implementation, and reporting followed the standards diagnostic accuracy. Patients who had stayed more than 24 hours in the ICU were critically screened. Patients who had AKI after admission to the ICU were recruited prospectively. All clinical data were obtained prospectively through case report forms. A total of 379 critically ill patients who developed AKI after being admitted to the ICU were separated into two parts: a derivation cohort (194 AKI patients) and a validation cohort (194 AKI patients) (185 AKI patients). The biomarkers of urinary tissue inhibitor of metalloproteinase-2 (TIMP-2) and insulin-like growth factor-binding protein-7 (IGFBP7) were detected at inclusion immediately after AKI diagnosis (day 0) and 24 h later (day 1). In the derivation cohort, the optimum cut-off values for predicting non-recovery were estimated, and their predictive accuracy was evaluated in the validation cohort. Non-recovery from AKI was the primary endpoint (within 7 days).

Results:

During the study period, 3154 critically ill patients who were the ICU for more than 24 hours assessed in two ICUs; 424 (13.4%) of them experienced After excluding the ineligible subjects, 379 people were recruited in the study. Out of patients , with 159 (41.9%) failed to recover from AKI onset, with 79 in the derivation cohort and 80 in the validation cohort. With an area under the receiver operating characteristic curve (AUC) of 0.751 and an optimal cut-off value of 1.05 ((ng/mL)2/1000), urine [TIMP-2] × [IGFBP7] on day 0 had a better prediction ability for non-recovery than TIMP-2 and IGFBP7 alone. The AUC was significantly enhanced to 0.852 when [TIMP-2] × [IGFBP7] combined with the clinical parameters of AKI evaluated by urine output (UO) criteria, AKI 2–3, and non-renal SOFA score predicting non-recovery. On 1, however, urine [TIMP-2] × [IGFBP7], TIMP-2 alone, and IGFBP7 alone did poorly performed in predicting AKI on 1.



Predictive value of biomarkers and the corresponding model. The ROC curves of urinary [TIMP-2]×[IGFBP7] on day 0 and the corresponding model for predicting failure to recover from AKI in the derivation cohort. a The AUCs of urinary [TIMP-2]×[IGFBP7] on day 0 and the clinical risk prediction model. b The AUC of the [TIMP-2]×[IGFBP7] day 0 clinical risk prediction model. *ROC* receiver operating characteristic, *AUC* area under the ROC curve

Discussion:

In critically ill patients, AKI is still a frequent and serious clinical condition. It is generally understood that an episode of AKI can result in a long-term degradation of renal function, with the possibility of progressing to CKD, the use of Renal Replacement Therapy (RRT), and end-stage kidney disease (ESKD) with dialysis dependence, all of which are significantly associated with greater short- and long-term mortality. Urinary [TIMP-2] × [IGFBP7] has been demonstrated to be beneficial in risk stratifying patients at high risk of AKI [1]. The ability of

urine [TIMP-2] × [IGFBP7] to predict failure to recover following AKI development was investigated in this study. Urine TIMP-2 and IGFBP7, two new biomarkers identified in renal tubular cells, induce G1 cell cycle arrest. In cellular and molecular pathways, AKI is associated to inflammation, oxidative stress, and apoptosis. TIMP-2 and IGFBP7 are involved in these pathways and represent early kidney injury. A greater [TIMP-2] × [IGFBP7] level appears to be associated to worse outcomes. Another study found that greater median [TIMP-2] × [IGFBP7] levels were linked to a greater degree of renal damage, with RRT patients having the highest median [TIMP-2] × [IGFBP7] levels. In a study by Dewitte A et al. , urine [TIMP-2] × [IGFBP7] demonstrated a decent ability to predict renal recovery within 48 hours after AKI.[3] The SAPPHIRE trial confirmed the effectiveness of combining TIMP-2 and IGFBP7 with UO in improving risk classification for severe outcomes.[4] As a result, it was reasonable to use the clinical factor of AKI as determined by the UO criteria in the prediction of renal recovery.

Conclusion:

The study concludes that The Urinary [TIMP-2] × [IGFBP7] on day 0 performed fairly well in predicting AKI non-recovery. When urinary [TIMP-2] × [IGFBP7] is combined with clinical factors of AKI determined by the UO criteria, AKI stage 2–3, and non-renal SOFA score, the prediction accuracy improved.

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3.Prevalence and Prognosis of respiratory pendelluft in ICU patients with acute respiratory failure on mechanical ventilation.

Introduction:

Asynchronous alveolar breathing causes intrapulmonary gas redistribution, which is known as respiratory pendelluft. [1]This phenomenon is defined as the displacement of gas from a more recruited nondependent (ND) lung region to a less recruited dependent (D) lung region without minimal changes in the ventilator's tidal volume (VT).[2] It can be seen in patients with flail chest, obstructive lung disease, and acute respiratory distress syndrome (ARDS). [1]Pendelluft may be harmful by causing local over distension and tidal recruitment, according to evidence from animal studies.[1,2] As a result, early detection of pendelluft is necessary for appropriate treatment and ventilation strategy adjustments, particularly in severely ill patients. Pendelluft monitoring techniques used in the earlier were not appropriate for critically ill patients. The chest electrical impedance tomography (EIT) is a non-invasive monitoring technology that utilizes bio-impedance changes over consecutive breathing cycles to obtain real-time pictures of regional lung ventilation at the bedside. [1]The use of EIT in the intensive care unit has allowed for the detection of respiratory pendelluft at the bedside (ICU). The key objective of this study was to find out the prevalence of pendelluft is in mechanically ventilated ICU patients with acute respiratory failure (ARF) and how it affects their prognosis.

Respiratory pendelluft occurred at a prevalence of 31% in this study group and it was associated with longer ventilation duration

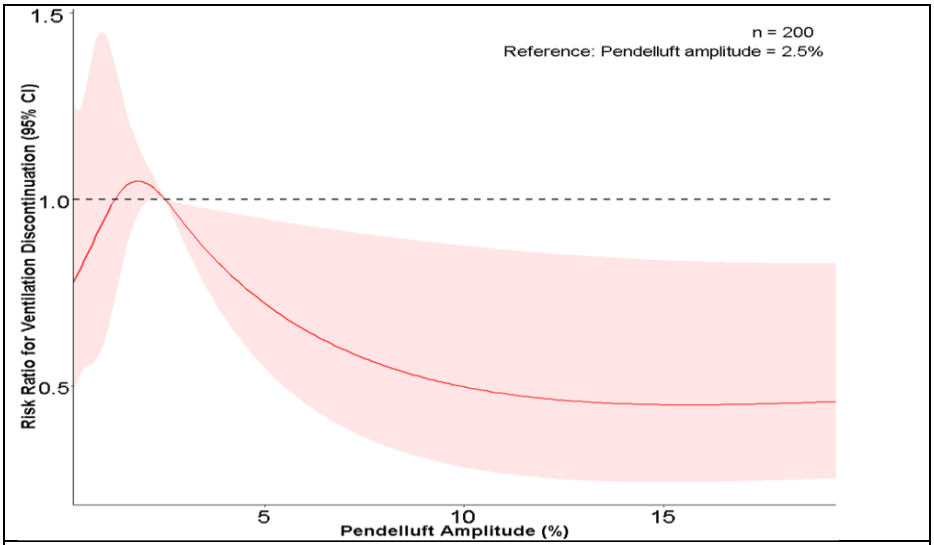
Methods:

This was a retrospective observational study on 200 mechanically ventilated ICU patients with ARF, defined as a partial pressure of arterial oxygen to fraction of inspired oxygen (PaO2/FiO2) ratio of less than 300 mmHg within 48 hours of admission treated in a tertiary care ICU. Electrical impedance tomography (EIT) was used within 48 hours of admission to detect the presence of pendelluft. The difference in impedance between the total of all regional tidal impedance variations and the global tidal impedance variation was used to calculate its amplitude. A value of more than 2.5 percent (the 95th percentile of 30 healthy volunteers) was considered as occurrence of Pendelluft.

Results:

A total of 200 patients (135 men and 65 women) were included in the study; Pendelluft was found in 61 of the 200 ARF patients, while the remaining 139 were not in the pendelluft group, resulting in a prevalence of 31%. Out of 94 patients, 39 were spontaneously breathing (41%), and 22 of 106 patients who were on fully controlled ventilation (21%). The proportion of spontaneous breathing, respiratory rate, and global inhomogeneity index was all higher in patients with pendelluft. Patients with pendelluft stayed in the ICU for longer time and had shorter 14-day ventilator-free days. Only patients with a PaO2/FiO2 ratio of less than 200 mmHg had a real correlation between pendelluft and longer ventilation duration, according to

subgroup survival analysis. There was no difference in ICU mortality between individuals with and without Pendelluft.



Relationship between pendelluft amplitude and risk ratio for ventilation discontinuation in the study population. 2.5% amplitude of pendelluft was set as the reference

Discussion:

The following were the main findings of this study: (1) pendelluft was detected in 31% of 200 ARF patients ventilated in the ICU; (2) higher GI index and the presence of spontaneous breathing were independent factors associated with pendelluft; (3) pendelluft was associated with a longer 14-day ventilation duration among patients with a PaO2/FiO2 ratio less than 200 mmHg. EIT has been increasingly used to detect the pendelluft in critically ill patients which was reported in the previous studies.[1] Sang et al. introduced a method to detect the amplitude of pendelluft by comparing the sum of all pixel TIV (tidal impedance Variation) with the global TIV, and expressed it as percent, where 1% of pendelluft amplitude was equal to 1 mL pendelluft volume per 100 mL t volume was adopted in this study. [3]Previous research has found that when neuromuscular blockers are used in ARDS patients, pendelluft disappears, confirming the link between spontaneous effort and pendelluft. Pendelluft was more likely to occur in patients with spontaneous breathing in this study cohort, but there was no direct assessment of breathing effort. [19, 20] Respiratory rate was an indirect predictor of breathing difficulty.

Conclusion:

In conclusion, pendelluft was found in 31% of ARF patients ventilated in the ICU at a single centre. Pendelluft was more common in patients with spontaneous breathing and higher lung heterogeneity. It was associated with longer ventilation duration in patients with a PaO2/FiO2 ratio of less than 200 mmHg. In the future, patients with severe hypoxia should be monitored carefully and treatments aiming at reducing pendelluft should be evaluated.

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4.A case of Respiratory failure caused by diaphragm paralysis

Introduction:

Mechanics such as stretching or contusion, especially in motor vehicle crashes, cause traumatic brachial plexus injury. [1] When it comes to brachial plexus injuries, traumatic cases, particularly those involving cervical roots avulsions, typically result in severe neurological paralysis and need surgery rather than conservative treatment.[2] Because of the C5-Th1 nerve injury, brachial plexus injuries can cause a variety of symptoms, but diaphragm paralysis is a rare outcome. Diagnosing diaphragmatic paralysis in severely ill patients is difficult. In general, radiography or a fluoroscopic'sniff test' are used to diagnose it. [1] A radiograph, on the other hand, cannot be used to provide a conclusive diagnosis of diaphragmatic paralysis. Furthermore, in critically ill patients, the sniff test is difficult to perform. [1] here is case of patient with respiratory failure caused by diaphragm paralysis and brachial plexus injury was identified rapidly through point-of-care ultrasound (POCUS).

Point-of- care ultrasound provided a clear visualisation and rapid diagnosis for causes of respiratory failure at the bedside.

Case Presentation:

An elderly patient aged 55-years presented to the tertiary medical care centre following a car accident. The patient was in hypovolemic shock and had lost consciousness when arrived. Abdominal pain was the main complaint, which was followed by motor and sensory abnormalities in the right upper extremity. Traumatic subarachnoid haemorrhage, liver injury, and fractures of the rib, right clavicle, right scapula, and right femur were discovered on contrast-enhanced CT. The improved CT's coronal image revealed that the diaphragm's continuity had been maintained. Direct injury did not explain the motor and sensory abnormalities in the right upper extremity. As a result, a brachial plexus injury was suspected. Due to multiple injuries, the patient was intubated after being admitted to the intensive care unit (ICU). Radiographic imaging in the ICU revealed diaphragmatic elevation on occasion and for a short time. As a result, researchers assumed that pleural effusion or atelectasis was causing the diaphragm to elevate. After 6 days of artificial ventilation, the patient had a successful spontaneous breathing trial and was extubated. The patient experienced dyspnoea one day after being extubated. Patient was tachypnoeic and had a respiratory rate of around 50 breaths per minute and was reintubated. Carbon dioxide excretion was reduced because the carbon dioxide concentration remained normal despite

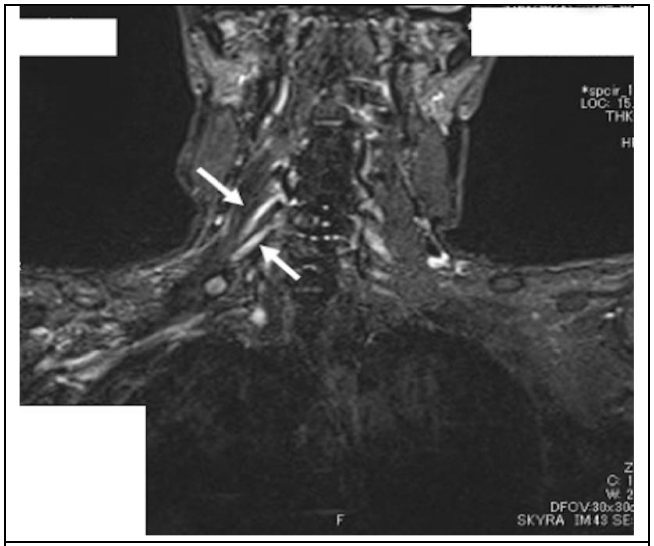

Figure 2 MRI scan on STIR sequence: White arrows show the injury to the right C5 and C6 nerve roots. STIR, short TI inversion recovery

tachypnoea. The right diaphragm exhibited considerably less respiratory movement than the left, and the right diaphragm movement distance was shortened, according to bedside POCUS.

The lack of segmental diaphragm abnormalities on CT on days 1 and 7 ruled out the possibility of a diaphragm injury. An MRI examination of the neck indicated C5 and C6 root avulsion injuries (Figure 1), which likely caused diaphragm paralysis. As a result of the diaphragm paralysis, the patient was diagnosed with acute respiratory failure. On day 15, the patient was taken off the ventilator. Despite the fact that patient had dyspnea for about a month following extubation, the arterial blood gas revealed no hypoxemia or carbon dioxide retention. Dyspnoea became less frequent as days progressed. The patient was transported to another facility for additional rehabilitation, and brachial plexus injury was treated by nerve grafting.

Discussion:

POCUS assisted in the diagnosis of a brachial plexus avulsion injury with diaphragm paralysis in this instance. Without direct observation, such as laparoscopy or laparotomy, diaphragmatic dysfunction is difficult to diagnose with accuracy. The use of point-of-care diaphragmatic ultrasonography in critically ill patients has been reported to be beneficial. Ultrasonography is noninvasive and highly reproducible, allowing bedside assessment in 5–15 minutes [3,4] and can assess findings of abnormalities in the thorax that could induce respiratory failure at the same time. Loss of diaphragmatic excursion, paradoxical excursion during deep breathing and sniffing, and a respiratory variation of diaphragm thickness of 20% can be found out using ultrasonography in diaphragm paralysis.[4] The importance of POCUS in detecting the cause of acute respiratory failure was demonstrated in this case.

Conclusion:

The study concludes that while diaphragm paralysis caused by brachial plexus injury is rare but it can result in severe respiratory failure. Hemidiaphragmatic elevation on X-ray has a limited specificity, and diaphragm paralysis can be mistaken for pleural effusion or atelectasis hence, clinicians should be aware of this while diagnosing. Point-of-care ultrasound allows for a clear visualisation and rapid detection of causes of respiratory failure.

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