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# Pneumothorax as a complication of respiratory failure – two case reports and literature review

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## Abstract

Acute respiratory distress syndrome (ARDS) – severe clinical syndrome with a high mortality rate, is characterized by hypoxemia and respiratory failure secondary to non-cardiogenic pulmonary oedema. The most common cause of ARDS is pneumonia. The cases are presented of 2 male patients: a 28-year-old and a 41-year-old, who were in a severe general condition due to progressive respiratory failure secondary to pneumonia. Despite antibiotic therapy the condition of the patients deteriorated. A differential diagnosis was performed, in which autoimmune causes, tuberculosis and viral infections were excluded. During hospitalization, both men developed pneumothorax requiring urgent drainage. A decline and deterioration of the patients' vital signs was observed and eventually death was pronounced. ARDS is a severe disease that poses a challenge in clinical practice. The course of the disease is difficult to predict, and the patient requires intensive surveillance and treatment.

## Key words

pneumonia, ARDS, Klebsiella pneumoniae, leakage, pneumothorax, VV ECMO

# INTRODUCTION

Acute respiratory distress syndrome (ARDS) is a highly heterogeneous, severe clinical syndrome of acute respiratory failure caused by non-cardiogenic pulmonary oedema, associated with capillary endothelial injury and diffuse alveolar damage [1-3]. ARDS is characterized by inflammation of the pulmonary parenchyma leading to impaired gas exchange accompanied by the release of inflammatory mediators and hypoxaemia [1, 2]. It is a rare but serious life-threatening condition requiring treatment in an intensive care unit (ICU). Scientific research estimates that the incidence of ARDS in the United States ranges from 64.2-78.9 cases per 100,000 people per year [2]. Recently, the significant morbidity and mortality of ARDS have been emphasized by its high incidence in COVID-19 patients [3]. For intensive care units, the incidence of ARDS in seriously ill patients is estimated at about 10% [3].

Currently, the most common causes leading to the development of ARDS include bacterial or viral pneumonia. Less common causes include sepsis, non-cardiogenic shock, aspiration, trauma, contusion, transfusion, pancreatitis and inhalation injuries [1–4]. In all these situations, acute lung injury (ALI) is a consequence of sepsis. Etiological factors lead to pulmonary changes involving a wide spectrum of clinical symptoms of respiratory failure and gasometric and radiological abnormalities. According to Berlin's definition [5]: 'ARDS is the acute onset of impaired oxygenation (within

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one week) and the visualization of bilateral pulmonary parenchymal lesions in chest imaging studies indicating non-cardiogenic pulmonary oedema and  $PaO_2/FiO_2$  ratio of less than 300 mmHg.' The clinical course and prognosis depends on the severity of the course of ARDS, which is determined by the severity of hypoxaemia:

- Mild: 200 mmHg < PaO<sub>2</sub>/FiO<sub>2</sub> ratio ≤ 300 mmHg with positive end-expiratory pressure (PEEP) or continuous positive airway pressure ≥ 5 cm H<sub>2</sub>O;
- Moderate: 100 mmHg < PaO<sub>2</sub>/FiÕ<sub>2</sub> ratio ≤ 200 mm Hg with PEEP ≥ 5 cm H<sub>2</sub>O;
- Severe:  $PaO_2/FiO_2$  ratio  $\leq 100 \text{ mmHg}$  with  $PEEP \geq 5 \text{ cm}$ H<sub>2</sub>O [1].

In January 2024, a proposed new, updated definition of ARDS was published, building on the 2012 Berlin definition. Significant attention was given to four key recommendations, including the use of high-flow nasal oxygen, the identification of hypoxaemia mainly by pulse oximetry, the introduction of ultrasound as a standard chest imaging test, and the treatment of ARDS in resource-limited facilities [6].

Although clinical diagnosis and treatment of ARDS have improved significantly in recent years, it is still the leading cause of death in critically ill patients, with mortality rates of about 30 – 40% [3]. Currently, there is no effective pharmacotherapy for ARDS, and treatment remains primarily supportive, consisting of mechanical ventilation, prevention of stress ulcers and venous thromboembolism, and nutritional support while addressing the underlying etiology [1, 2].

The aim of the study is to present the most important problems related to comprehensive diagnostics and treatment of patients with acute respiratory failure due to bacterial pneumonia in an ICU through two clinical case reports.

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# **CASE REPORT 1**

In October 2023, a 28-year-old Caucasian male was admitted in a serious condition to the Anaesthesiology and Intensive Care Unit in University Hospital No. 1 in Lublin, eastern Poland, for acute respiratory failure.

Only 7 days earlier, the patient had presented to his general physycian due to weakness, a cough that had been worsening for several days and a fever (39.5 degrees Celsius for a week). He received naproxen, cough syrup and inhaled budesonide as treatment. Despite treatment, the patient's condition gradually deteriorated. He reported a loss of 7 kg of body weight over 7 days, night sweats, and inability to sleep at night due to dyspnoea.

After one week of outpatient treatment, the patient presented to the emergency department in a moderate condition with preserved logical contact, and was admitted to the hospital's Clinical Department of Internal Medicine, due to increasing dyspnoea, weakness, chest pain, and a single episode of haemoptysis in the course of a lower respiratory tract infection. Physical examination revealed increased respiratory effort with asymmetric chest movements, tachypnoe 40/min, sinus tachycardia 100/min, wheezing and fine-bubble rales at the base of the right lung and auscultatory wheezing at the base of the left lung. A chest X-ray showed changes indicative of lobar right lung inflammation. There was also fluid in both pleural cavities and atelectasisinflammatory changes in the lower lobe of the left lung.

The patient denied chronic diseases and allergies. He was not taking permanent medication, had not been previously hospitalized, and had no treatments performed. He had a history of 20 pack-years and denied other stimulants.

In the Internal Medicine Department, the patient initially received empirical broad-spectrum antibiotic therapy (amoxicillin with clavulanic acid and levofloxacin). Subsequently, due to coagulation disorders and anaemia (haemoglobin level (HGB): 12.5 g/dl), platelet concentrate (PC), red cell concentrate (RCC) and intravenous immunoglobulin (IVIG) were administered. During the second day of hospitalization, a decrease in non-specific inflammatory markers was obtained. In the following days, clinical deterioration was again observed, mainly with an increase in C-Reactive Protein (CRP) and leukocyte levels (Tab. 1).

On day 4 of hospitalization, the patient was in a serious condition and transferred to the Department of Anaesthesiology and Intensive Care Unit. Due to escalating respiratory failure, the patient required intubation and initiation of mechanical ventilation with analgesia and sedation. Microbiological examination of the collected bronchoalveolar lavage (BAL) revealed the presence of Klebsiella ESBL(+). The healthcare team made a therapeutic decision based on the microbiological findings, and meropenem was included. Despite the adjustment of antibiotic therapy and alveolar recruitment manoeuvers under the control of static compliance, no improvement was obtained the patient's clinical condition. In-depth diagnostics for viral infections did not reveal infection with any of the pathogens tested.

On the 8<sup>th</sup> day of hospitalization, following surgical consultation, drainage of the left pleural cavity was performed and 2 litres of dark, bloody fluid was decompressed. During the 9<sup>th</sup> day, there was an abrupt desaturation to 76%. Imaging studies showed a pneumothorax mantle on the left side. Pleural drainage was performed with slight clinical improvement (SaO<sub>2</sub> to 85%). The patient was then transferred to the Second Department of Anaesthesiology and Intensive Care at University Hospital No. 1 in Lublin, where assessment was conducted and veno-venous extracorporeal membrane oxygenation (VV ECMO) therapy initiated. A 25F intake cannula for internal right jugular vein (RIJV) and a 19F donor cannula for internal left jugular vein (LIJV) were used; blood flow 5.5 l/min, sweep gas 6 l. Mechanical ventilation in DuoLevel mode was used with the following ventilator settings: FiO<sub>2</sub> 0.6, inspiratory positive airway pressure (IPAP) 25, expiratory positive airway pressure (EPAP) 10; tidal volume (TV) ~250ml. After the use of alveolar recruitment maneuvers, the static compliance of the lungs (C stat) was 30ml/cm H<sub>2</sub>O. Subsequently, as oxygen saturation dropped to a maximum of 85%, the ventilation mode was switched to pressure regulated volume control (PRVC) with a FiO, of 0.7, tidal volume (TV) of 320ml, positive end-expiratory pressure (PEEP) of 14 cm H<sub>2</sub>O, and peak pressure (Ppeak) of 32 cm H<sub>2</sub>O, resulting in a saturation of 92%.

On the first day of ECMO treatment, the patient's blood gas analysis revealed the following:  $pO_2$  of 39.2 mmHg,  $pCO_2$  of 49.1 mmHg, lactate level (Lac) of 2.3 mmol/l, and pH of 7.55.

During the following days of the hospital stay, VV ECMO therapy and protective lung ventilation under analgosedation



**Figure 1.** X-ray from the 10<sup>th</sup> day of hospitalization showing an enhanced bilateral interstitial pattern with numerous macular, confluent parenchymal thickenings and peripheral translucency in the upper and middle fields of the left lung, with a mantle thickness of up to 21 mm, corresponding to a left-sided pneumothorax.

Table 1. Patient's laboratory results in the first 9 days of hospitalization

Laboratory test	1	2	3	4	5	6	7	8	9	Referential range	Unit
HGB	17.1	9.3	14.1	12.5	11.5	11.8	11.3	11.5	10.9	13–18	g/dl
CRP	321.8	145.6	151.3	219	206.3	240.8	219	317.3	258.6	0–5	mg/l
Prokalcitonin	118.99	75.44	45.63	21.02	10.31	6.06	3.52	3.27	3.37	<0.5	ng/ml
Leukocytes	4.1	9.1	14.9	20.3	18.3	19.7	25.8	29.3	25.9	4–10	G/L
Neutrophils	3.6	8.4	-	13.5	15.6	16.2	22.8	25.7	22.9	2.25-6.8	K/µl

were continued. During the first 6 days of ECMO ventilation, the patient was given rocuronium by intravenous infusion for muscle relaxation. Recruitment manoeuvers were performed regularly under the control of static lung compliance. On the third day of ECMO treatment, the patient was placed in the prone position for 24 hours – significant amounts of bloody secretions were aspirated from the respiratory tract.

A small amount of mucopurulent and purulent-bloody secretions were regularly aspirated from the airways. The patient had periodic fever, reaching up to 38.7 degrees Celsius, despite active cooling of the patient's blood using a heater-cooler unit. The patient's respiratory condition was not improving. There was air leakage from the drain in the left pleural cavity. The patient's water-electrolyte balance disorders were compensated and enteral feeding intervention was provided.

On the 7th day of hospitalization, the patient was mechanically ventilated with DuoLevel mode: EPAP 10/ IPAP 20, FiO<sub>2</sub> 0.6 and ECMO: blood flow 4.0 l/min; sweep gas 5 l. After turning off the sweep gas, the accumulation of  $CO_2>65$  mmHg. A CT scan of the chest showed pleural fluid in the left pleural cavity and a pneumothorax on the right side. Drains were placed into both pleural cavities – into the right to decompress the pneumothorax, and a brownish-grey discharge appeared in the drain on the left side.

After 8 days, the decision to discontinue VV ECMO was made due to improvements in ventilation parameters, but worsening blood morphotic parameters, including a decrease in platelet count. Conventional mechanical ventilation with analgosedation was resumed. The patient remained on intermittent positive pressure ventilation (IPPV) in DuoLevel mode. Due to  $pCO_2$  accumulation, pressure regulated and volume-controlled ventilation (PRVC) was used. The patient was ventilated with VT 440 ml, breathing frequency (f) 26 per minute, FiO<sub>2</sub> 0.6, achieving static lung compliance (C stat) of 50 ml/cm H<sub>2</sub>O and PaO<sub>2</sub>/FiO<sub>2</sub> ratio 115 mmHg. Active drainage of both pleural cavities was maintained. Significant air leakage was found in the drains of both pleural cavities.

In the following days, significant air leakage persisted in drains from both pleural cavities. Auscultatorily, there were numerous crackles.

During hospitalization, antibiotic therapy with meropenem, tigecycline, amikacin, imipenem and cylastin was administered. Autoimmune diseases and tuberculosis infection were also excluded.



**Figure 3.** X-ray from the 28<sup>th</sup> day of hospitalization show significant bilateral progression of parenchymal thickening throughout the lung fields, obscured hilum and a partially obliterated silhouette of the heart and diaphragm. Bilateral pneumothorax was present: on the right-side mantle thickness up to 9mm, on the left side mantle thickness up to 20 mm. Subcutaneous emphysema at the right lateral chest side also persisted

The patient's condition deteriorated, with the persistence of a large air leak on the left side, drops in saturation, hypotonia and bradycardia despite the use of pressor amines. Despite medical interventions and efforts to stabilize the patient, the condition deteriorated significantly, leading to death on the 30th day of hospitalization. The case report timeline is presented in Figure 4.



Figure 2.1

#### Figure 2.2 – level T4

**Figure 2.1, 2.2** A CT scan on the 23<sup>rd</sup> day of hospitalisation showed bilateral pneumothorax: a right-sided mantle width of up to 22 mm at the base and a left-sided mantle width of up to 23 mm at the base of the lung. In the right lung, there were massive atelectatic and parenchymal lesions in the lower lobe and partially in the middle lobe, and the remaining parenchyma of the middle lobe and upper lobe with numerous cystic air spaces up to 28 mm in size. There was also right-sided subcutaneous emphysema in the anterior, lateral and posterior chest wall. The lower lobe of the left lung showed a heterogeneous picture with air spaces, which raised the suspicion of an abscess. CT scans of the patient were consulted by thoracic surgeons



Figure 4. Case 1 report timeline

### **CASE REPORT 2**

Two months later, a 41-year-old male patient was transferred from the Provincial Hospital to the Intensive Care Unit of University Hospital No. 1 in Lublin due to severe respiratory failure. The patient developed symptoms of respiratory tract infection about one week before hospitalization. The patient was initially hospitalized in the first centre for 6 days. Due to a deterioration in health, he was subsequently transferred to the ICU at University Hospital No. 1. The patient's medical history included 20 years of alcohol abuse, but no other chronic diseases.

At admission, the patient was mechanically ventilated using pressure-controlled volume control/synchronous intermittent mandatory ventilation (PRVC/SIMV) with PEEP 8-12, FiO, 0.6, f 27 per minute, VT 550 ml and Plateau pressure 30 cm H<sub>2</sub>O. Arterial blood gas test revealed compensated respiratory acidosis with pH 7.36,  $pO_2 = 60.9$ mmHg,  $pCO_2 = 68$  mmHg and Lac of 1.3 mmol/l. Lung auscultation revealed massive rhonchus on the left side, muffling over the right upper lobe and bronchial sound below. The radiograph showed an image corresponding to pneumonia and signs of emphysema (Fig. 5). Bronchoscopy with bronchoalveolar lavage was performed. Enteral feeding was started. Laboratory tests revealed elevated CRP and procalcitonin and severe anaemia. Due to low HGB (Tab.2), the patient was given a transfusion of 2 units of packed red blood cells (RBC) and 1 unit of PC.

On the 4<sup>th</sup> day of hospitalization, the patient became oliguric, began to be pyrexial, and laboratory tests revealed an increase of CRP and procalcitonin (Tab. 2). Levofloxacin was therefore empirically added to the antibiotic therapy. Continuous renal replacement therapy (CRRT) was initiated.



Figure 5. X-ray from the admission showed massive consolidations corresponding to pneumonia. No signs of pneumothorax. An oval translucency presented on the border of middle and inferior lobe of the right lung, which may represent emphysema

On the 7<sup>th</sup> day of hospitalization, the patient's condition remained severe. The cardiovascular system was supported with continuous infusion of noradrenaline. Computed Tomography (CT) revealed significant left-side pneumothorax with mantle thickness up to 68 mm and massive emphysema destructing lung tissue, with the biggest air space in the right lung measuring around 90x83 mm. Due to the significant left-sided pneumothorax, a drain was inserted. The patient started to be pyrexial, up to 40.5 degrees Celsius.

The next day (day 8), antibiotic therapy was adjusted, and the patient was given 600 mg of linezolid intravenously twice (i.v.)

Laboratory test	1	2	3	4	5	6	7	Referential range	Unit
HGB	7.7/8.8*	8.9	9.6	9.4	9.5	9.1	8.4	13–18	g/dl
CRP	36.3	48.8	287.6	407.4	510.8	537.6	405.4	0–5	mg/l
Prokalcitonin	4.77	3.39	31.69	48.61	51.49	69.70	53.38	<0.5	ng/ml
Leukocytes	7.43	7.08	11.83	6.84	7.90	12.58	12.32	4–10	G/L
Neutrophils	6.86	6.25	9.90	5.95	6.55	10.57	10.59	2.25-6.8	K/µl

Table 2. Patient's laboratory results during the 7 days of hospitalization

CRP-C – Reactive Protein; HGB – haemoglobin level; \*haemoglobin level after transfusion

a day. The cardiovascular system continued to be supported by catecholamines. CT scan showed a pneumothorax on the right side – active drainage was placed. Left pleural cavity drainage was continued. A big air leak was reported. Clinical examination showed peripheral oedema. Cultures were obtained from BAL, urine, nasopharynx, and anus. However, the results did not reveal any pathogens responsible for pneumonia. This suggests that despite thorough testing, a specific microbial cause for the pneumonia had not been identified at this point.

During the following days, the patient remained in a critical condition. Peripheral oedema persisted. Drainage of both pleural cavities was continued, as well as CRRT. An air leak on the left side was continuously observed. Due to the worsening condition of the patient, the enteral feeding was stopped, and glucose infusion was administered.

After 2 weeks of hospitalization, control CT (Fig. 6.1 and 6.2) revealed massive consolidations of inflamed and collapsed alveoli in both lungs, and clustered cystic air spaces corresponding to pulmonary fibrosis. A thoracic surgery consultation was ordered.

Later that day, a bloody discharge appeared from the patient's respiratory tract, ventilation deteriorated rapidly, and the patient died.

The case report timeline is presented in Figure 7.

### DISCUSSION

ARDS is defined as hypoxaemia and respiratory failure secondary to non-cardiogenic pulmonary oedema [3,7]. Studies have shown that pneumonia, ALI and ARDS account for a large proportion of global morbidity and mortality, with pneumonia itself being the most common cause of ARDS [8,9].

The presented cases indicate that acute respiratory failure due to bacterial pneumonia was the immediate cause of death in both patients. Despite treatment efforts, radiological examinations revealed progressive interstitial changes in the lung lobes. According to various data, and despite the many diagnostic tests available, identification of the pathogen is only 36.5%-38%. In addition, the presence of drug-resistant strains can complicate antibiotic therapy [10]. Carbapenemresistant Klebsiella pneumoniae, ESBL-positive, detected during bronchoscopy, is among the concerning pathogens. The risk of mortality for individuals infected with it escalates with age [11]. In our patient, based on the antibiogram, targeted meropenem was used for treatment.

In addition to bacterial infections, viral infections contribute to a significant proportion of ARDS cases, ranging from 20% to more than 30%, depending on the data source [12]. Among patients hospitalized for COVID-19 infections, approximately one-third met criteria for ARDS [13]. In the described cases, laboratory diagnosis for viral infections was made, after which special attention should be focused on the possibility of viral infection in patients with bilateral opacities on chest x-ray without clinically present cardiogenic pulmonary oedema and volume overload [12]. COVID-19 infection typically presents with specific radiological changes in the lungs, including frosted glass opacities, a peripheral distribution of opacification, and vascular thickening or dilatation, but these changes were not present in the described cases [14]. The clinical picture of COVID-19 infection is highly heterogeneous with variations in disease severity and



**Figure 6.1, 6.2** A CT scan on the 7<sup>th</sup> day of hospitalization showed significant left side pneumothorax with mantle thickness up to 63 mm. Massive consolidations of lung tissue are visible and most intensified in the superior, middle and posterior parts of the lungs. Advanced clustered cystic air spaces described as honeycombing and corresponding to pulmonary fibrosis. Enlargement of the air spaces with the biggest air space measuring up to 86×96 cm.



Figure 7. Case 2 report timeline

intensity from case to case, and the pneumonia occurring in its course is associated with a higher incidence of emphysema and thrombotic disease, compared to other viral pneumonias [13, 15].

ARDS caused by direct lung injury poses a significant challenge for clinicians, primarily because of the difficult-to-predict course of the disease. Several factors contribute to this unpredictability, and these factors are interconnected, involving the patient's clinical condition, type of pathogen involved, and environmental factors. [3, 9, 10].

The medical documentation of the presented patients indicates the presence of several risk factors associated with ARDS, such as pneumonia, smoking, hypoalbuminaemia, male gender, and blood transfusion. The absence of information on comorbidities in both cases made it challenging to differentiate ARDS from other conditions that may present similarly, such as vasculitis or diffuse alveolar haemorrhage. It is difficult to identify environmental risk factors associated with toxic exposures in the workplace – both men worked in the construction industry [7].

Pneumothorax is a serious, potentially fatal complication of mechanical ventilation in patients with ARDS. The development of pneumothorax is a direct result of lung damage during ARDS. Urgent drainage is necessary for treatment. In addition, other forms of air leak, such as subcutaneous emphysema, pericardial or peritoneal emphysema, can occur [16]. Among the complications specific to severe ARDS, widespread pneumothorax was identified in both patients, and one patient additionally had widespread subcutaneous emphysema.

During hospitalization of patients with similar clinical manifestations, extensive differential diagnosis should be considered. One of the causes of serious respiratory diseases is alfa-1 antitrypsin deficiency (AADT), the cause of which is mainly attributed to the PI\*ZZ genotype in SERPINA1. These patients are more susceptible to complications related to the use of a respirator, oxygen therapy, cachexia, and secondary melanoma. In addition, they may develop abscess, pneumothorax, which was observed in the patients in both case reports [17, 18]. AATD increases susceptibility to lung damage, especially in the context of bacterial infections, which may contribute to the severity of respiratory complications. [19]. Hypoxaemia, abnormal alveolar-arterial O<sub>2</sub> gradient, and features of intrapulmonary leakage, are characteristic of hepatorenal syndrome [20]. In the patients in both case reports, the presence of liver problems that could lead to acute pulmonary complications were excluded.

One of the patients described had an elevated bilirubin level, which increased during hospitalization (highest level – 4.33 mg/dl) and an elevated AST level. Cirrhosis, in addition to liver failure, is associated with decreased immunity. There is abnormal functioning of T lymphocytes, B lymphocytes and neutrophils, defect in the reticuloendothelial system, impaired phagocytosis and uncontrolled secretion of cytokines, which is associated with increased susceptibility to infection [21, 22].

Pulmonary inflammation and excess inflammatory factors lead to further impairment of liver function and further immune decline [21]. In patients with cirrhosis, pneumonia was associated with increased mortality (the highest risk of

mortality among infectious diseases) as influenced by the patient's age, bacteraemia, bilirubin levels, leukocytosis, and inadequate antibiotic therapy or use of antibiotic prophylaxis [21–23]. In addition, increased mortality has been associated with the development of acute-on-chronic liver failure [21]. A good predictor of bacterial infection among patients with cirrhosis is the lymphocyte-to-monocyte ratio [24]. A study among patients with cirrhosis and pneumonia indicates that the Model of End Stage Liver Disease Plus (MELD-P) scale is a good diagnostic tool for assessing the risk of in-hospital and short-term mortality [25]. In addition, increased mortality has been associated with the development of acute-onchronic liver failure [21]. Therefore, in the presented patient in whom cirrhosis could not be conclusively established, other liver disorders, such as drug-induced liver injury (DILI), chronic liver disease (CLD) and acute liver failure (ALF), should also have been included in the differential diagnosis.

Features of interstitial pneumonia, especially with negative culture results, may raise suspicion of connective tissue group diseases. In the diagnostic process, attention is focused on the performance of a panel of autoantibodies [26–28]; for example, one cause of autoimmune vesicular bleeding may be vasculitis associated with the presence of ANCA antibodies. The clinical signs may include blood-coloured sputum, a feature observed in one of the described patients. [29].

ANCA antibody-associated vasculitis may account for the development of interstitial lung disease (ILD). The risk is higher when vasculitis is accompanied by MPO-ANCA positivity or microscopic vasculitis is present [30-33]. There are 2 categories of patients with ILD and ANCA antibodies: patients with overt systemic vasculitis (AAV-ILD), or patients without clinical features of systemic vasculitis (ANCA-ILD) [30]. For this reason, ANCA screening should be routinely performed in patients with newly-diagnosed ILD. In the case of a positive ANCA result, close monitoring for progression to systemic vasculitis is necessary in the absence of AAV [30, 31]. Triggers of AAV include smoking, genetic factors, exposure to drugs, solvents and environmental particles [30,32]. In patients with newly-diagnosed AAV and respiratory symptoms, it is worthwhile performing a chest radiography, and high resolution computed tomography (HRTC) is preferred [30, 31]. Nearly 80% of patients had pulmonary lesions at the time of diagnosis [31]. Nodular opacity was most commonly observed on CT. Other changes described in patients with vasculitis include a matte-glass pattern, honeycomb signs, thickening of the interlobular septum, atelectasis, interstitial pneumonia, pulmonary venous stasis and pleural effusion [31, 32, 34].

Patients with (MPO)-ANCA antibodies were found to have a more frequent incidence of interstitial lung disease, pulmonary haemorrhage, and severe lung involvement with severe respiratory failure and the need for intensive care [31]. Honeycomb sign on HRCT, elevated CRP and erythrocyte sedimentation rate are adverse prognostic factors for all AAV-ILD [32]. Guidelines on treatment strategies for AAV-ILD are not available. If AAV is present, the therapeutic option is immunosuppressive therapy alone or in combination with antifibrotic therapy, while in the absence of vasculitis, treatment is recommended for progressive-fibrosing interstitial lung diseases (PF-ILD), non-specific interstitial pneumonia (NSIP), or organizing pneumonia (OP) [32,34].

One patient underwent the VV ECMO procedure, which is one of the methods used to treat patients with ARDS.

The specialized invasive method is used only in selected centres and in patients who meet certain criteria. It is a lifesaving method, particularly in cases of severe ARDS where conventional ventilation is insufficient. However, it carries a high risk of long-term complications, and death, and its use is carefully considered based on specific criteria [35–37]. After 8 days of applied ECMO, it was decided to discontinue the procedure due to the improvement of ventilation parameters while morphotic parameters deteriorated. In patients with ARDS, accompanied by septic shock, the use of the VV ECMO procedure is not always possible, and sometimes there may be a need to consider switching to venoarterial extracorporeal membrane oxygenation (VA ECMO). Data on patients with COVID-19 show high mortality associated with extracorporeal membrane oxygenation [11].

The use of VV ECMO for very severe ARDS often also carries the possibility of multiple complications. Anticoagulant treatment in the form of unfractionated heparin (UFH) may have been an additional factor in the complication of airway haemorrhage in patient Case Report 2. A retrospective study by Diaz et al. of 22 ARDS patients on COVID-19 requiring VV ECMO showed a higher risk of bleeding, thrombotic events and mortality in the group of patients who received UFH as treatment, compared with the group where bivalirudin was used [38]. The Vajter et al. study highlighted the benefit of reduced bleeding as a postoperative complication in patients who received lower doses of UFH as thromboprophylaxis when using ECMO in lung transplantation [39].

The clinical presentations of the presented cases underscore the potential complexity of symptoms and underlying causes culminating in acute respiratory failure and eventual demise. In the Discussion, the aim was to emphasize the necessity for a comprehensive diagnostic approach, including the exclusion of multiple causative factors that may exhibit similar or identical clinical manifestations. Despite the patients' differing ages, they both presented with comparable symptoms, which were found intriguing given their simultaneous arrival at the Anaesthesiology Clinic.

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