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A case of progressive acute respiratory distress syndrome in a patient with coronavirus disease (COVID-19) and difficulties in its confirmation by instrumental diagnostic methods

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A – research concept and design; B – collection and/or assembly of data; C – data analysis and interpretation; D – writing the article; E – critical revision of the article; F – final approval of the article

Keywords: coronavirus disease, COVID-19, viral infection, acute respiratory distress syndrome, diagnosis.

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The aim of the study is to demonstrate the difficulties in instrumental diagnosis of acute respiratory distress syndrome (ARDS) in a patient with coronavirus disease (COVID-19) in the presence of certain clinical signs of its development and progression.

Results. We present a case of severe COVID-19 in a 79-year-old unvaccinated patient with comorbidities (stage II hypertension and psoriasis). The diagnosis of COVID-19 was confirmed by RNA detection with PCR; treatment was in accordance with the Order of the Ministry of Health of Ukraine No. 762. On the 3rd day of the disease, despite the presence of physical signs such as shortness of breath in the lower parts of the lungs and dry rales, as well as oxygen dependence, no pathology was detected by X-ray. The coronavirus disease course worsened during the period of hospital treatment, which was manifested by an increased supplemental oxygen requirement and the need to enhance the oxygen level and its partial pressure.

On the 9th day of hospitalization, the patient was transferred to the intensive care unit. Instrumental imaging methods (radiography and ultrasound examination of the lungs) were repeated, showing left-sided interstitial edema of the lung parenchyma with the formation of superficial consolidations, that progressively worsened. That same day, acute myocardial infarction was diagnosed. The level of consciousness decreased to 9 points on the Glasgow Coma Scale due to hypoxia, necessitating the patient's transfer to mechanical ventilation. On the 24th day of hospitalization, the patient died due to the development of severe ARDS and acute myocardial infarction.

Conclusions. This clinical observation of an ARDS case in a patient with COVID-19 indicates that the emergence of this complication might occur without the typical radiological and sonographic signs of infiltrative lung injury. Alternatively, this could be attributed to the limitations of the equipment employed, while, conversely, the clinical manifestations might be influenced by the pathophysiological characteristics of ARDS development, prompting researchers to identify its distinct subphenotypes.

Ключові слова: коронавірусна хвороба, COVID-19, вірусна інфекція, гострий респіраторний дистрес-синдром, діагностика.

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Випадок гострого респіраторного дистрес-синдрому, що прогресував, у хворої на коронавірусну хворобу (COVID-19) та труднощі його підтвердження інструментальними методами діагностики

В. В. Черкаський, К. В. Калашник, О. В. Рябоконь

Мета роботи – описати труднощі інструментальної діагностики гострого респіраторного дистрес-синдрому (ГРДС) у пацієнтки з коронавірусною хворобою (COVID-19), у якої були клінічні ознаки його розвитку та прогресування.

Результати. Наведено випадок тяжкого перебігу COVID-19 у 79-річної невакцинованої пацієнтки з коморбідною патологією (гіпертонічна хвороба II стадії та псоріаз). Діагноз COVID-19 підтверджено шляхом виявлення РНК методом полімеразної ланцюгової реакції. Лікування призначено відповідно до Наказу МОЗ України № 762. На третю добу хвороби, незважаючи на наявність таких фізикальних ознак, як ослаблення дихання у нижніх відділах, сухі хрипи та киснева залежність, патологію рентгенологічно не виявлено. Перебіг коронавірусної хвороби за період лікування в стаціонарі погіршувався: посилювалася киснева залежність, пацієнтці необхідне було збільшення кількості кисню та його парціальної частки.

На дев'ятий день госпіталізації пацієнтку перевели до відділення анестезіології та інтенсивної терапії. Здійснили обстеження з повторним застосуванням інструментальних методів візуалізації (рентгенографія та ультразвукове дослідження легень). У результаті виявлено інтерстиціальний набряк паренхіми легені зліва з формуванням поверхневих консолідацій, що надалі поглибилися. Того самого дня в пацієнтки діагностовано гострий інфаркт міокарда. Рівень свідомості знизився до 9 балів за шкалою ком Глазго на фоні гіпоксії, що спричинило переведення пацієнтки на штучну вентиляцію легень. На двадцять четвертий день госпіталізації хвора померла через розвиток ГРДС і гострого інфаркту міокарда.

Висновки. Наведене клінічне спостереження розвитку ГРДС у хворої на COVID-19 показало: це ускладнення може не супроводжуватися класичними рентгенологічними та сонографічними ознаками інфільтративного ураження легень. З одного боку, це можна пояснити недосконалістю використаного обладнання; з іншого боку, такі клінічні прояви можуть бути спричинені патофізіологічними особливостями розвитку ГРДС, і тому науковці розрізняють його субфенотипи.

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The WHO officially announced the end of the novel coronavirus disease (COVID-19) pandemic on 5 May 2023 [1]. In less than 5 years, more than 704 million people have contracted the disease, of whom more than 7 million have died [2]. The clinical manifestations of this disease are quite diverse, and its complicated course can lead to the development of vasculopathies, thrombotic lesions, severe respiratory failure, and death [3,4]. Almost 20 % of patients with COVID-19 developed acute respiratory distress syndrome (ARDS). The majority of patients admitted to anesthesiology and intensive care units (ICUs) demonstrated bilateral lung tissue infiltrates with frosted ground-glass opacity and oxygenation indexes (PaO_2/ FiO_2) of <200 [5].

At the same time, the signs of severe lung damage among patients with COVID-19 exhibited unique characteristics and individual variability [6]. The development of hypoxemia could be accompanied by significant dyspnea and high respiratory rate, or vice versa, with respiratory rate remaining largely unchanged, and hypoxemia described as "quiet" or "happy". Blood gas analysis in some patients revealed a combination of hypoxemia and hypocapnia, and in others, hypercapnia was an integral part of the pathological process [7,8].

Gattinoni L. et al. (2020) were the first to highlight that in certain COVID-19 patients, severe hypoxemia was associated with the presence of only subpleural and peribronchial infiltration, in contrast to patients exhibiting a typical pulmonary parenchymal ground-glass opacification with dorsal consolidation or atelectasis. Based on these and other clinical variables, the authors have identified two heterogeneous phenotypes of ARDS, namely L type and H type, complicating the course of this disease [9,10].

Though the validity of these respiratory phenomena in COVID-19 remains a subject of extensive debate [11], we decided to present a description of the clinical case encountered directly in our clinical practice.

Aim

The aim of the study is to demonstrate the difficulties of instrumental diagnosis of acute respiratory distress syndrome in a patient with coronavirus disease in the presence of certain clinical signs of its development and progression.

Materials and methods

The clinical case of severe ARDS and the possibilities of its diagnosis in a 79-year-old patient with COVID-19, who was treated at the Municipal Non-Profit Enterprise "Zaporizhzhia Regional Infectious Clinical Hospital" of the Zaporizhzhia Regional Council from 30.12.2023 to 25.01.2024, was analyzed. The diagnosis of COVID-19 was confirmed by the isolation of RNA-SARS-CoV-2 from nasopharyngeal mucus by polymerase chain reaction (No. 655033). The examination and treatment of the patient met the requirements of the current protocol on "Provision of medical care for the treatment of coronavirus disease (COVID-19)" approved by the Ministry of Health of Ukraine No. 762 of 02.04.2020, as amended [12].

To assess the severity of infiltrative lung changes in the treatment dynamics, in addition to chest X-ray, lung ultrasound (LUS) was performed using an expert-class ultrasound machine GE LOGIQ P9 (USA) with a modified "Abdominal cavity" preset to better visualize artifacts from the pleura and pulmonary parenchyma. LUS was performed by PhD K. V. Kalashnyk, Associate Professor, at the Department of Infectious Diseases according to the protocol [13].

Case report

A 79-year-old patient B. was admitted to the infectious hospital on 30.12.2023 (the 3rd day of illness) with complaints of fever up to 37.8 °C, general weakness, dry cough and a sensation of breathlessness triggered by physical activity.

Her medical history revealed that she had never been vaccinated against COVID-19. According to the medical record analysis, comorbidities included stage II hypertension and psoriasis.

Examinations upon hospitalization (day 3) revealed a need for supplemental oxygen, namely, blood oxygen saturation was 92 % with a respiratory rate of 18 per minute. Lung auscultation revealed difficulty breathing and moderately diminished breath sounds with scattered dry rales in the lower fields. Ventilatory support at an oxygen flow of up to 10 L/minute by nasal prongs maintained a blood oxygen saturation level (SaO_2) of at least 96–98 %.

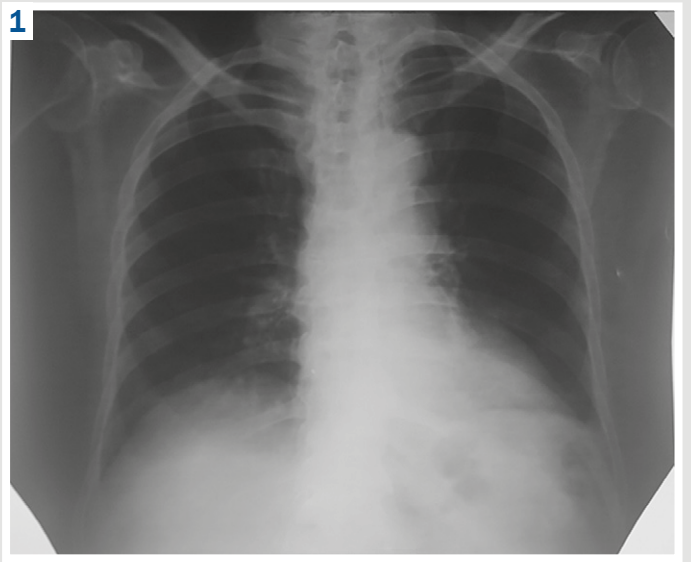
Based on the patient's clinical and anamnestic data, a preliminary diagnosis was made: "Coronavirus disease, moderate severity (Cito Test Covid-19 Ag – positive). Community-acquired pneumonia? Grade II hypertension. Psoriasis". The diagnosis was later confirmed by polymerase chain reaction.

However, chest X-ray showed no infiltrates in the lungs on the day of hospitalization *(Fig. 1)*.

In the specialized infectious department, the patient received therapy in accordance with the current protocol. During the disease course, the patient persistently experienced dyspnea during physical exertion. On the 5th day of hospital treatment, the patient exhibited emotional distress, anxiety, and an agitated behavioral pattern accompanied by delusional thoughts. The sensation of breathlessness intensified. The patient's SaO_2 reached 92 %, with a respiratory rate of 22–24/minute after oxygenation with a fraction of up to 60 %. Lung auscultation revealed rhythmic breathing and moist crackles over the lower pulmonary fields. At the same time, rhythmic heart activity was noted with a heart rate of 69/minute, blood pressure was 140/80 mm Hg.

Ventilatory support was continued through a non-rebreather mask with a reservoir bag at flow rates approaching 10–12 L/minute, which led to an elevation in SaO_2 to 98 %. Nevertheless, the patient's respiratory rate remained in the range of 20–22/minute.

Subsequently, on the 9th day of inpatient treatment, the patient's level of consciousness decreased to 9 points on the Glasgow Coma Scale, necessitating tracheal intubation and the initiation of mechanical ventilation. The O_2 fraction was 95 % on mechanical ventilation, and the patient's SaO_2 was 94–95 %. Lung auscultation revealed diffuse moist rales. On the 9th day of inpatient treatment (the 12th day of illness), diagnostic imaging of the lungs, including chest X-ray and LUS, identified only infiltration in the left lower lobe and superficial consolidation of the lung parenchyma in the left subclavian region, respectively *(Fig. 2)*. Consequently,



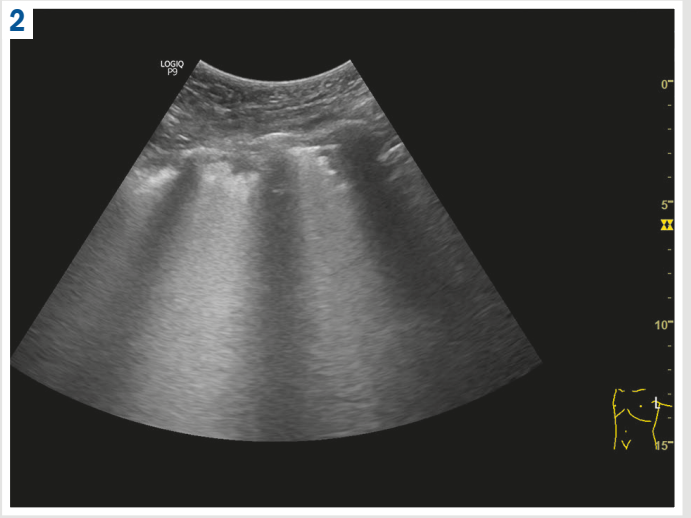


Fig. 1. Chest X-ray of the 79-year-old patient B., on the day of admission to the infectious hospital.

Fig. 2. LUS on day 9 of COVID-19.

given the clinical dissociation between respiratory symptoms and radiological evidence of lung injury, the possibility of pulmonary embolism in the woman was considered.

That same day (day 9 of inpatient treatment), the patient's COVID-19 course was aggravated by the development of acute myocardial infarction affecting the septum and apex of the heart. The electrocardiogram showed sinus tachycardia with a heart rate reaching 106 bpm and signs of focal injury to the septum and apex of the heart; troponin I level was 2.48 ng/ml. Anticoagulant therapy was adjusted according to the relevant protocol.

Nevertheless, the patient's condition continued to deteriorate. The oxygen fraction administered to the woman on mechanical ventilation varied dynamically, reaching 90–100 % in the final 7 days of her life *(Table 1)*.

Despite the progression of acute respiratory failure signs. on the 19th day of hospital treatment (the 22nd day of illness), no corresponding significant worsening was observed on imaging in the patient with COVID-19. Thus, according to the chest radiography results, only signs of left-sided multisegmental pneumonia remained *(Fig. 3A)*, and the LUS results indicated increased left-sided infiltrative alterations in the lower-basal regions of the pulmonary fields, with the development of significant consolidation and the presence of pleural effusion *(Fig. 3B)*.

On the 24th day of inpatient treatment (the 27th day of illness), amid progression of cardiovascular and respiratory failure with a radiological picture revealing only congestion in the pulmonary circulation and severe consolidation in the lower lobe of the left lung *(Fig. 4)*, the patient died.

A pathological examination was conducted to determine the underlying cause of the patient's death. The analysis identified a progressive exacerbation of respiratory failure, accompanied by clinical manifestations consistent with a progressive increase in ARDS. Post-mortem examination revealed the following macroscopic findings: bilateral subtotal viral large-focal consolidating pneumonia affecting segments III–X of the right lung and segments IV–X of the left lung, with features indicative of adult ARDS as well as acute transmural anterior septal infarction of the left ventricular myocardium, 3.5 × 2.0 × 2.0 cm in size. No morphological evidence of pulmonary embolism was identified during the autopsy.

Discussion

This study presents the clinical case of the 79-year-old female patient diagnosed with COVID-19, admitted to the infectious hospital on the 3rd day of illness. The patient presented with symptoms including hyperthermia (up to 37.8 °C), general weakness, dry cough, and exertional dyspnea. Notably, a dissociation between clinical symptoms and radiological findings was observed from the moment of hospital admission, with no pathological changes detected on chest radiography despite reduced oxygen saturation (SaO_2 92 %) and reported shortness of breath (the respiratory rate was 18/minute).

It is known that the severity of clinical manifestations associated with COVID-19 is influenced by a combination of disease-specific progression patterns and individual patient characteristics, including age, pre-existing comorbidities, and other factors. The infection caused by the SARS-CoV-2 induces distinctive pathophysiological alterations, which establish novel interaction paradigms among gas exchange, pulmonary mechanics, clinical symptoms, and findings of instrumental imaging techniques [8,9].

In May 2020, L. Gattinoni et al. theoretically identified two phenotypes (L and H) of lung damage that occurred among patients with COVID-19 in cases of hypoxemic respiratory failure. The L-type was characterized by focal subpleural and peribronchial opacities detected by computed tomography, indicative of high lung compliance, reduced elasticity, a low ventilation-perfusion ratio, and limited responsiveness to recruitment maneuvers. The features of H-type were characterized by changes in classical ARDS, namely, signs of diffuse pulmonary ground-glass opacification with dorsal consolidation/atelectasis, severe hypoxemia, a significant right-to-left shunt, low compliance, and good lung recruitment capacity as assessed by computed tomography [6,9].

Based on the 2012 Berlin Definition, the internationally recognized diagnostic criteria for ARDS in adults include the

following: an early onset of respiratory system impairment symptoms, occurring within 7 days from the initiation of the underlying condition; the presence of bilateral lung infiltrates on radiographic imaging, characterized by a "ground glass" appearance, which cannot be attributed to acute left ventricular failure or other concomitant pathology; the persistence of hypoxic changes refractory to oxygen therapy, as evidenced by an oxygenation index (PaO_2/ FiO_2) of less than 300 mm Hg [14].

In conditions of limited resource availability, lung imaging for oxygen-dependent patients with COVID-19 primarily relies on chest radiography conducted using portable X-ray equipment and LUS, the latter being recommended by the WHO during the novel COVID-19 pandemic [15,16,17].

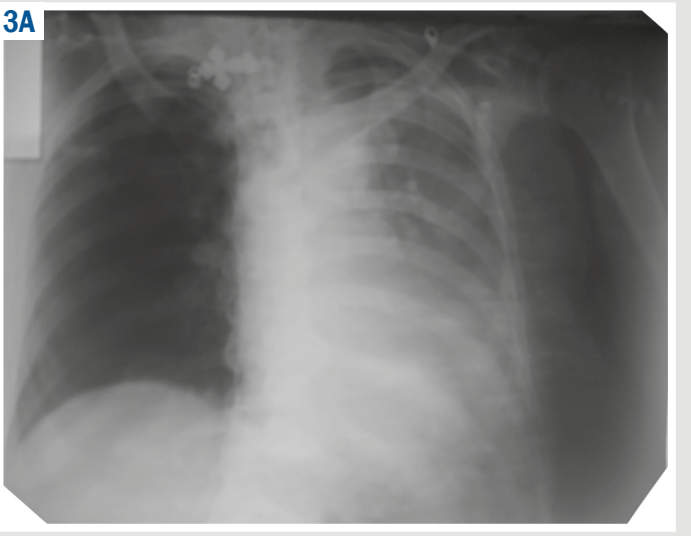
Nevertheless, studies have shown limited sensitivity of chest radiography in detecting mild cases of COVID-19 or its early developmental stages. Lung X-ray images frequently fail to reveal infiltrative alterations, whereas computed tomography scans are capable of identifying pathological changes [18,19]. A comparative analysis of the sensitivity of lung imaging modalities, including ultrasound and radiography, among patients with acute respiratory failure highlights the superior efficacy of LUS, particularly in evaluating interstitial pathology, opacities, and consolidations [18,20]. The precise capabilities of lung ultrasound in the management of COVID-19 have yet to be comprehensively defined [18]. Comparable to chest radiography, LUS is limited to evaluating the condition of the lung parenchyma and lacks the ability to assess the vascular structures within the pulmonary microcirculation [21].

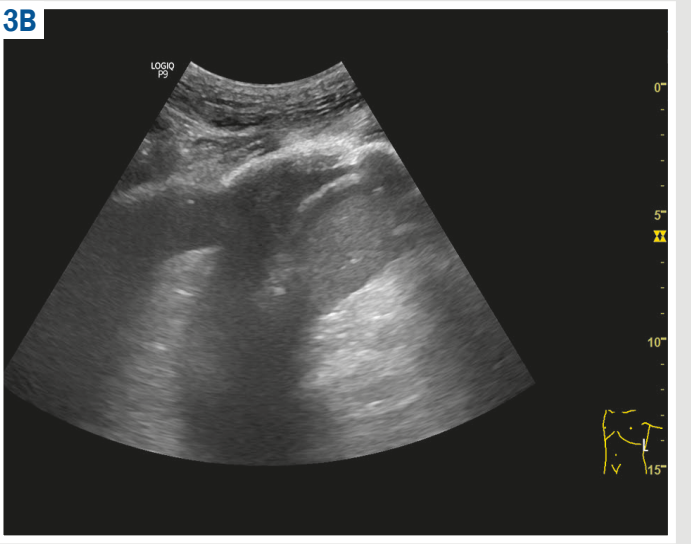
The novel coronavirus disease in oxygen-dependent patients is accompanied by endothelial injury of the pulmonary microcirculation vessels and the development of *in situ* thrombosis. Thrombotic damage to the pulmonary microcirculatory bed contributes to an increased dead space effect, which, in conjunction with the development of interstitial pulmonary edema and pathological right-to-left blood shunting, results in severe hypoxemia in affected patients [21].

Also, for a number of reasons, the assessment of hypoxemia severity and its response to oxygen therapy in patients not requiring mechanical ventilation is controversial. For example, the calculation of such an indicator as the oxygenation index is based on accurate measurements of the oxygen partial pressure in arterial blood – PaO_2 and the oxygen fraction in a breathing mixture – FiO_2. In spontaneously breathing patients, FiO_2 is estimated using predictive algorithms of oxygen delivery devices. Numerous studies indicate that non-rebreather masks can deliver a FiO_2 ranging from 60 % to 90 %, whereas standard face masks with a rebreather mechanism provide a FiO_2 of approximately 40 % to 60 % [22]. It is believed that an oxygen flow rate of 10 L/minute results in an approximate 4 % increase in FiO_2 for each additional liter of oxygen delivered [23]. Alternatively, FiO_2 can be calculated by the formula:

estimated FiO_2 = FiO_2 of atmospheric air (e. g., 0.21) + + 0.03 × O_2 flow rate (L/min) [24].

In resource-limited settings, it is also problematic to determine arterial PaO_2, without which it is impossible to calculate the oxygenation index as defined by the 2012 Berlin criteria for ARDS. In this regard, a modified version





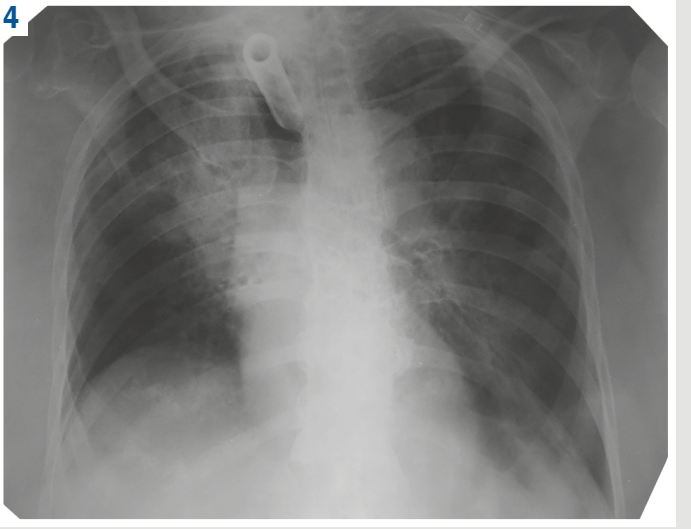


Fig. 3. Chest X-ray **(A)** and LUS **(B)** of the 79-year-old patient B. on day 19 of inpatient treatment.

Fig. 4. Chest X-ray of the 79-year-old patient B. on the 24th day of inpatient treatment (the 27th day of illness).

Table 1. Parameters of blood oxygen transport function in the 79-year-old patient B. and instrumental alterations in her lungs throughout the disease progression

Methods of examination	Day of inpatient treatment	Findings	SaO_2, %	FiO_2, %	SaO_2 / FiO_2, %
Chest X-ray	1	No pathology	97	51	190
Chest X-ray	9	Left-sided lower lobe pneumonia	95	100	95
LUS	9	Superficial consolidation in the axillary region on the left			
Chest X-ray	13	Left-sided multisegmental pneumonia	97	40	243
LUS	13	Deep consolidation along the middle axillary line on the left*			
Chest X-ray	19	Left-sided multisegmental pneumonia	93	90	103
Chest X-ray	25	Left-sided lower lobe pneumonia. Pulmonary congestion	88	100	88

*: limited examination due to the severity of the patient's condition.

of the ARDS diagnostic criteria for countries with limited resources was proposed in 2016 [25], which was subsequently approved at a consensus conference comprising experts in resuscitation and ARDS in 2023 [24]. According to these consensus guidelines [24], SaO_2 can be used to determine the oxygenation index in spontaneously breathing patients if its value does not exceed 97 %. That is, it is currently recognized that a SaO_2 / FiO_2 ratio of ≤315, with SaO_2 ≤97 %, can be used as a valid diagnostic criterion for ARDS in adults.

In the case of our clinical observation, considering the technical characteristics of the oxygen concentrator and other equipment used to provide oxygen support to the patient, the FiO_2 ranged from 50 % to 100 %, and the oxygenation index ranged from 190 % on the day of hospital admission to 88 % during the final days of the patient's life. Notably, neither chest radiography nor LUS revealed characteristic features indicative of ARDS throughout the hospital stay. That is why, given the dissociation between the clinical symptoms of respiratory impairment and the findings from instrumental lung imaging techniques, the patient was diagnosed with a possible pulmonary embolism, which was not substantiated by subsequent pathological examination.

According to existing literature, in oxygen-dependent patients diagnosed with COVID-19, pulmonary embolism is identified in approximately 50 % of cases solely through postmortem pathological examination [26]. Pulmonary thromboembolism in patients with COVID-19 has no specific clinical symptoms that could be used to diagnose its development. The clinical presentation of this complication, on the one hand, may be asymptomatic or manifested only by moderate dyspnea, but on the other hand, it may be accompanied by obstructive shock and severe hypoxemia [27]. Furthermore, the common clinical symptoms of pulmonary embolism and ARDS in oxygen-dependent patients with COVID-19 exclude clinical criteria from the algorithm for the differential diagnosis between these conditions [28]. In addition, there are currently no diagnostic criteria for pulmonary embolism based on the analysis of hemostatic parameters or other biological markers [27,28]. It is widely considered that the best option for antemortem diagnosis of pulmonary embolism is to perform multidetector computed tomographic pulmonary angiography in patients with suspected pulmonary embolism [27]. This imaging modality, when applied to COVID-19 patients, is capable of detecting not only pulmonary embolism but also pulmonary perfusion defects consistent with in situ thrombosis [27].

In the context of the presented clinical case, a post-mortem pathological examination of the deceased female patient revealed an absence of morphological evidence indicative of pulmonary embolism. The primary cause of her death was determined to be the progressive course of COVID-19, exacerbated by escalating respiratory insufficiency and adult ARDS (macroscopic signs of bilateral subtotal viral large-focal pneumonia affecting segments III–X of the right lung and segments IV–X of the left lung).

The observed discrepancies between clinical manifestations and the diagnostic efficacy of two imaging modalities, chest radiography and LUS, can be found in scientific literature. Research suggests that in oxygen-dependent patients with COVID-19, pulmonary thrombosis may develop concurrently with the suppression of the compensatory Euler-Liljestrand reflex, reactive hyperemia, and a redistribution of pulmonary blood flow toward non-ventilated or inadequately ventilated lung segments [29,30]. The predominance of such pathogenetic mechanisms leads to severe hypoxemia, the development of which is accompanied by the appearance of only focal subpleural or peribronchial ground-glass opacity detectable exclusively via computed tomography. Furthermore, defects in pulmonary perfusion can be identified only through multidetector computed tomographic pulmonary angiography.

Conclusions

1. This clinical observation demonstrates the diagnostic challenges associated with acute respiratory distress syndrome in patients with COVID-19 in low-resource settings, where the clinical presentation may not fully align with established diagnostic criteria.

2. The development of acute respiratory distress syndrome may not be accompanied by classical radiological and sonographic signs of infiltrative lung disease. Such a pathological pulmonary condition can be detected in vivo only by computed tomography and multidetector computed tomographic pulmonary angiography or be confirmed only after autopsy, as demonstrated in the presented case report.

Prospects for further research. Further research should focus on the possibilities of visualizing ARDS manifestations using instrumental methods of examination (LUS) at the patient's bedside.

Ethical approval
According to the conclusion of the Bioethics Commission of Zaporizhzhia State Medical and Pharmaceutical University

(Protocol No. 5 dated April 24, 2025), the article complies with all applicable requirements for research involving human participants, as established by international regulations and the current legislation of Ukraine.

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