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Acute Pancreatitis as an Extrapulmonary Manifestation and Pulmonary Embolism as a Complication of COVID-19: A Case Report

Ljiljana Milić[1,2]*, Dragana Arbutina[1], Dragica Mitrović[3], Marko Šurlan[1], Aleksandar Karamarković[1,2]

[1]Nikola Spasic Clinic for Surgery University Clinical Hospital Center Zvezdara Belgrade, Republic of Serbia
[2]Faculty of Medicine, University of Belgrade, Belgrade, Republic of Serbia
[3]Department of physical medicine and rehabilitation, University Clinical Hospital Center Zvezdara, Belgrade, Serbia

***Corresponding author:**
Ljiljana Milić, MD, PhD
Nikola Spasic Clinic for Surgery
University Clinical Hospital Center Zvezdara
Dimitrija Tucovi a 161, 11000 Belgrade
Republic of Serbia
E-mail: ljilja.milic@gmail.com

ID ORCID:
Ljiljanja Milić: 0000-0002-3327-2841
Dragana Arbutina: 0000-0003-4334-0470
Dragica Mitrović: 0000-0002-7672-3403
Marko Šurlan: 0000-0001-7863-7130
Aleksandar Karamarković: 0000-0001-5716-2009

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Rezumat

Pancreatita acută ca manifestare extrapulmonară și embolia pulmonară ca o complicație a COVID-19: prezentare de caz

Introducere: Boala coronavirus 2019 (COVID-19), cauzată de SARS-CoV-2, a apărut pentru prima dată în Wuhan, China, la sfârşitul anului 2019. Pe lângă afectarea respiratorie, COVID-19 poate determina tulburări de coagulare, conducând la evenimente tromboembolice. De asemenea, forme uşoare de pancreatită acută au fost raportate la pacienții cu pneumonie COVID-19. Scopul acestui raport de caz este de a evidenția manifestările extrapulmonare rare ale infecției cu SARS-CoV-2, cu accent pe afectarea pancreatică.
Prezentare de caz: Un pacient de sex masculin a fost internat pentru durere în gât, tuse seacă şi febră de până la 38,0 °C. Acesta primise prima doză de vaccin Sinopharm cu trei săptămâni înainte şi prezenta obezitate severă (IMC 44,7 kg/m²). La internare a dezvoltat tromboembolism pulmonar. Ecografia Doppler color a membrelor inferioare nu a evidențiat tromboză venoasă superficială sau profundă. În a unsprezecea zi de tratament s-au constatat creşteri ale amilazei, lipazei şi leucocitelor. Ecografia abdominală a arătat un pancreas hiperecogen, nehipertrofiat, sugestiv pentru pancreatită acută uşoară. Având în vedere distribuția largă a celulelor endoteliale, infecția cu SARS-CoV-2 poate afecta multiple organe extrapulmonare, inclusiv sistemul nervos central, cardiovascular, rinichii, pancreasul, ficatul şi tractul gastrointestinal.
Concluzii: Infecția cu SARS-CoV-2 poate fi asociată nu doar cu pneumonie, ci şi cu complicații tromboembolice şi pancreatită acută uşoară. Recunoaşterea acestor manifestări rare este importantă pentru diagnosticul şi managementul adecvat al pacienților.

Cuvinte cheie: COVID-19, pneumonie, embolie pulmonară, pancreatită acută, vaccinare

Abstract

Introduction: Coronavirus disease 2019 (COVID-19), caused by SARS-CoV-2,

first emerged in Wuhan, China, in late 2019. Besides respiratory involvement, COVID-19 may cause coagulation abnormalities, leading to thromboembolic events. Mild forms of acute pancreatitis have also been reported in patients with COVID-19 pneumonia. The aim of this case report is to highlight rare extrapulmonary manifestations of SARS-CoV-2 infection, with emphasis on pancreatic involvement.

Case Report: A male patient was admitted with sore throat, dry cough, and fever up to 38.0°C. He had received the first dose of the Sinopharm COVID-19 vaccine three weeks prior and was obese (BMI 44.7 kg/m²). On admission, he developed pulmonary thromboembolism. Color Doppler ultrasonography of the lower extremities showed no signs of superficial or deep vein thrombosis. On the eleventh day of treatment, laboratory tests revealed elevated serum amylase, lipase, and leukocyte count. Abdominal ultrasonography demonstrated a hyperechoic, non-enlarged pancreas, consistent with mild acute pancreatitis. Given the widespread presence of endothelial cells, SARS-CoV-2 infection may affect multiple extrapulmonary organs, including the CNS, cardiovascular system, kidneys, pancreas, liver, and gastrointestinal tract.

Conclusion: SARS-CoV-2 infection may lead not only to pneumonia but also to thromboembolic complications and mild acute pancreatitis. Awareness of such rare extrapulmonary manifestations is important for timely diagnosis and management.

Keywords: COVID-19, pneumonia, pulmonary embolism, acute pancreatitis, vaccination

INTRODUCTION

An epidemic caused by the Coronavirus (COVID-19) first appeared in Wuhan, China in late 2019. year. The most common symptoms of pneumonia caused by a viral infection were: cough, sore throat, fever, headache (1). The epidemic rapidly spread to all 193 countries around the world reaching the scale of a pandemic. In addition to respiratory problems, the infection also leads to coagulation disorders in a significant number of patients, which results in the occurrence of thromboembolic disorders (2). On the other hand, in patients with COVID-19 pneumonia, mild forms of acute pancreatitis have been proven, which are a consequence of the direct influence of the virus on the pancreatic tissue. Acute pancreatitis caused by COVID-19 infection does not show a serious clinical form and is also not a common manifestation of COVID-19 infection (3). Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), the virus responsible for COVID-19, primarily infects the respiratory epithelium by binding to angiotensin-converting enzyme 2 (ACE2) and utilizing the serine protease TMPRSS2 as a cofactor for cell entry (4). The highest expression of ACE2 within the respiratory tract is found in alveolar type II pneumocytes, which explains the predominant clinical presentation of pneumonia and acute respiratory distress syndrome (ARDS) (4,5). Nevertheless, numerous clinical and pathological findings indicate that COVID-19 represents a systemic disease. One of the most important mechanisms is so-called thromboinflammation, where a synergy of inflammatory and coagulation pathways can lead to microthrombosis and pulmonary embolism (PE) (6,7). The underlying pathophysiological mechanisms include the following (7-9):

- Endothelial dysfunction caused by direct injury to the vascular endothelial barrier;
- Cytokine storm, with elevated levels of pro-inflammatory cytokines such as IL-6 and TNF-α;
- Coagulation activation, with increased D-dimer, fibrin formation, and inhibited fibrinolysis;
- In situ thrombosis in pulmonary vessels, independent of classical venous thromboembolism (8,9).

These changes may result in the clinical presentation of pulmonary embolism, especially in patients with severe forms of COVID-19. PE further compromises ventilation-perfusion matching in the lungs and can cause acute right ventricular strain, significantly impacting disease outcomes (9,10).

In addition to the pulmonary and vascular systems, increasing evidence suggests that the pancreas may also be a target of SARS-CoV-2 infection. Studies have shown that pancreatic acinar cells and islets of Langerhans express both ACE2 and TMPRSS2, enabling direct viral invasion (11,12).

The proposed mechanisms of pancreatic injury in the context of COVID 19 include:

- Direct cytotoxic effects of the virus, resulting in stress and necrosis of pancreatic cells;
- Systemic inflammation and cytokine-mediated injury, acting at a distance;
- Lipotoxicity, through the release of free fatty acids from peripancreatic adipose tissue, further exacerbating inflammation;
- Hypoxia, microthrombosis, and drug-induced injury as potential cofactors contributing to the

development of pancreatitis (3,13,14).

Although acute pancreatitis (AP) is a rare complication of COVID-19 (13).

The aim of this case report is to present a patient in whom acute pancreatitis occurred as an extrapulmonary manifestation of SARS-CoV-2 infection, contributing to a better understanding of the broad clinical spectrum of this disease.

Case Report

A 52-year-old male patient was admitted to Clinical Hospital Center Zvezdara due to a sore throat, dry cough and fever up to 38.0 °C. Epidemiological screening was performed upon admission, including clinical assessment and PCR testing for SARS-CoV-2, which confirmed COVID-19 infection. Symptoms had started four days prior.

The patient was vaccinated with the first dose of "Sinofarm" vaccine three weeks before, and obese (BMI 44.7%). The patient does not consume cigarettes and alcohol. An X-ray of the heart and lungs was done and showed basally reduced transparency on both sides. Laboratory analyzes: Leucocytes 5.6x10^9 /l; D-dimer 2.27 mg/l; CRP 12.5. mg/l.

Therapy was initiated with intravenous Levofloxacin 500mg twice daily, Metronidazole 500mg three times daily, Paracetamol 1g twice daily, intramuscular Diclofenac 75mg twice daily, 2.5 liters of fluids, Methylprednisolone according to the scheme 80mg twice daily for next 3 days, without oxygen was prescribed.

Empirical antibiotic therapy was initiated due to the presence of fever, elevated inflammatory markers, and radiographic findings suggestive of possible bacterial superinfection at admission." "The patient was managed in a multidisciplinary setting, including consultation with an infectious disease specialist, in accordance with institutional COVID-19 treatment protocols.

The next day, there was a worsening of respiration and a decrease in oxygen saturation (SpO2 91%) when MDCT pulmoangiography is performed: The pulmonary vascular net is of normal ramification, in the basal segmental branches bilateral-CT signs of acute, non-occlusive lobar and segmental pulmonary thromboembolism. No signs of pleural effusion. Bilateral diffuse, multiple zone ground glass attenuation and spotty parenchyma consolidation: CO-RADS 6, Total CT Severity Score: 12. (*Fig. 1*). Lower extremity CDS: no signs of superficial and deep vein thrombosis.

Therapeutic values of low molecular weight heparin 0.6 ml twice daily and O2 mask were introduced. No major bleeding complications were observed during anticoagulant therapy. Given the markedly elevated D-dimer levels and the presence of major risk factors such as morbid obesity (BMI 44.7), the patient was classified as high risk for thromboembolic events, and therapeutic anticoagulation with low molecular weight heparin was promptly initiated according to clinical indications.

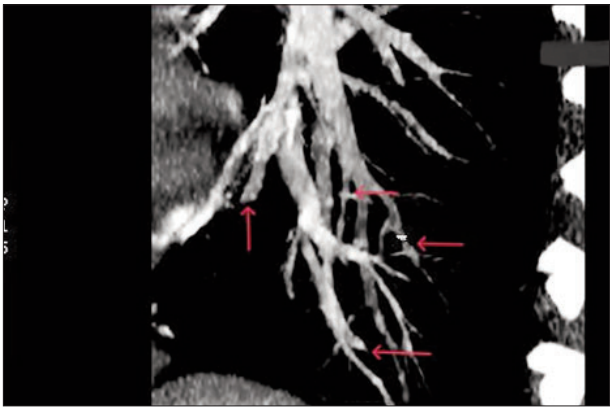

Figure 1. MDCT pulmoangiography: signs of acute, non-occlusive lobar and segmental pulmonary thromboembolism.

For the next few days, the patient was in respiratory deterioration and had a fever, so he was transferred from the O2 mask to HF 60 l/min, FiO2 100%, to which oxygen saturation is maintained (SpO2 90%). X-ray findings in the lungs in deterioration (*Fig. 2*), and since there is a decrease in O2 saturation (SpO2 77%) the patient is transferred to the Intensive Care Unit tahidispnoic in respiratory distress and placed on NIV, FiO2 100%, peep 8. There was an increase in oxygen saturation (SpO2 91%). Elevated values of D-dimer 3.75 mg/l and CRP 91.7 mg/l were observed. Levofloxacin was excluded from therapy and Meropenem 1g three times a day was introduced. The administration of Methyl-

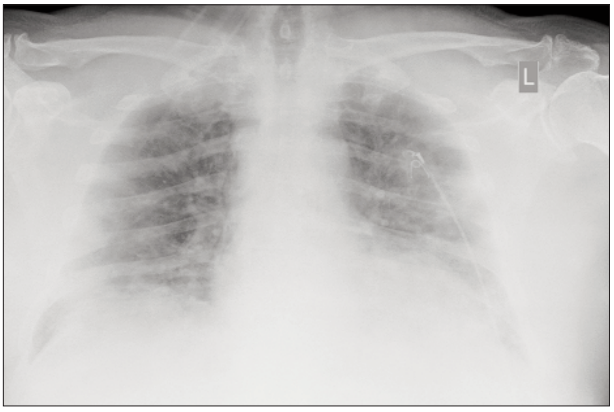

Figure 2. Lung and heart X-ray: Bilateral signs of pneumonia

prednisolone according to the protocol was also continued 60 mg and 40 mg for 3 days, then 40 mg twice daily for 3 days, then transferred to oral Methylprednisolone therapy 20 mg twice daily.

Clinical radiographic and laboratory improvement occurs on the given therapy, so that the patient is switched to HF. On the eleventh day of treatment, amylase increased (1350.0 U/L) as well as lipase (2722.63 U/l) and Le (23.5 x 10^9 ???/l) while CRP values decreased (0.8 mg/l), abdominal ultrasonography showed hyperechoic pancreas that was not enlarged. The patient had no abdominal pain. Other findings in order. Oral administration was discontinued and intravenous fluoroquinolone therapy with Ciprofloxacin 200 mg twice daily was initiated. The next day there was a drop in lipase (761 U/l) and amylase (711 U/l), the patient was transferred to an O2 mask. During further treatment, the patient improves his general condition on a daily basis. After three weeks of treatment, the patient was discharged home in good general condition, exhausted.

Discussion

Vaccination in the Republic of Serbia began on December 24, 2020. Our patient received the first dose of the Sinopharm vaccine, but three weeks later around the time he was scheduled to receive the second dose, he developed complications. Most likely due to an insufficient immune response, the patient exhibited a clinical picture consistent with COVID-19 pneumonia and was hospitalized at the Clinical Hospital Center Zvezdara. In this context, it is important to note that the patient had received only the first dose of the Sinopharm (BBIBP-CorV) vaccine three weeks prior to symptom onset. Data from clinical studies of inactivated SARS-CoV-2 vaccines indicate that, during this period, a fully developed protective immune response is not yet expected. In phase 1 and 2 clinical trials of the BBIBP-CorV vaccine, it was observed that the humoral response after the first dose is limited, whereas significant levels of neutralizing antibodies develop only after the second dose, with peak titers occurring 14–21 days following the booster dose (15). Similarly, in a phase 3 randomized clinical trial, vaccine efficacy was assessed only after completion of the two-dose regimen, demonstrating that a single dose does not provide sufficient protection against symptomatic COVID-19 (16).

This finding is consistent with recommendations from the World Health Organization (WHO) and the SAGE expert group, which state that an adequate level of protection with inactivated vaccines, including BBIBP-CorV, is expected only after the second dose (17). Real-world studies conducted in Qatar, the UAE, and Bahrain further confirm that protection after the first dose is low, while a significant increase in efficacy is achieved only after completion of the two-dose regimen. Chemaitelly and colleagues, in a population of over one million individuals, reported that the risk of infection and hospitalization after a single dose of BBIBP-CorV remains high, with a clear increase in protection only after the second dose (18). Similar trends have been observed in studies from the UAE and Bahrain, where seroconversion and the presence of neutralizing antibodies were markedly more frequent after completion of the full vaccination schedule (19,20).

Therefore, it is likely that our patient was in a phase of partial or suboptimal immune response at the time of infection. SARS-CoV-2 infection three weeks after the first dose of the Sinopharm vaccine is fully consistent with the immunobiology of inactivated vaccines and does not imply vaccine inefficacy, but rather indicates that the disease developed before full protective immunity was established. This timing also explains why the patient could develop a more severe clinical course of COVID-19, including pneumonia and thromboembolic complications, despite vaccination. These circumstances further emphasize the importance of completing the two-dose regimen and allowing sufficient time for the development of full immune protection.

The most common symptoms of COVID-19 infection include cough, sore throat, fever, and headache. The viral infection can lead to pneumonia and, in more severe cases, to ARDS (15). In our case, empirical antibiotic therapy was initiated at admission due to the inability to exclude bacterial superinfection, which is consistent with clinical practice in patients presenting with moderate to severe COVID-19 and elevated inflammatory markers.

The SARS-CoV-2 virus primarily enters cells via ACE2 receptors, which are expressed on endothelial cells throughout the body. The spike (S) protein of the virus binds to these receptors, facilitating viral entry and triggering an inflammatory cascade that can result in endothelial dysfunction, vascular leakage, and thrombus formation (2). In our patient, pulmonary embolism affecting the basal segmental branches likely developed through this mechanism, especially considering the concurrent COVID-19 pneumonia. Respiratory deterioration followed, but the patient responded well to treatment, which included non-invasive respiratory support and low-molecular-weight heparin, as recommended in similar cases.

COVID-19 is also known for its hyperinflammatory state, which contributes to a prothrombotic condition.

Deep vein thrombosis (DVT) and pulmonary embolism (PE) are well documented complications, often attributed to systemic inflammation, hypoxia, prolonged immobilization, and direct viral damage to the endothelium (16).

Autopsy reports of deceased COVID-19 patients have revealed pulmonary endothelialitis, widespread microthrombosis, and abnormal neovascularization, suggesting direct vascular injury and endothelial dysfunction (5). These findings are consistent with the clinical picture observed in our patient, where pulmonary embolism developed despite early hospitalization. A meta-analysis by Suh et al. indicated that pulmonary embolism can be present in up to 23% of hospitalized COVID-19 patients, with increased prevalence in those requiring intensive care (6).

Coagulopathy is a hallmark of severe COVID-19 and is associated with elevated D-dimer levels and poor prognosis (7). Moreover, some studies have shown a high incidence of thromboembolic events even in patients on therapeutic anticoagulation (8), reinforcing the hypothesis of endothelial-driven hypercoagulability and calling for vigilant monitoring. In addition, in patients with severe COVID-19 and obesity, individualized therapeutic anticoagulation should be considered due to the high risk of thromboembolic complications.

It is important to emphasize that endothelial cells are not only present in the lungs but are widely distributed throughout the body, including the central nervous system, cardiovascular system, kidneys, pancreas, liver, and gastrointestinal tract (2). This explains the broad spectrum of extrapulmonary manifestations of COVID-19.

Although our patient did not complain of abdominal pain, laboratory analyses showed increased levels of amylase and lipase, and an abdominal ultrasound suggested acute pancreatitis. While pancreatic involvement in COVID-19 is relatively rare, recent literature describes cases of acute pancreatitis in patients with SARS-CoV-2 infection, even in the absence of classic symptoms. The pathogenesis is not fully understood but is believed to involve direct viral invasion of pancreatic tissue via ACE2 receptors, as well as systemic inflammatory effects (11).

In addition to the aforementioned mechanisms, recent studies have expanded our understanding of the virus's interaction with host systems. SARS-CoV-2 enters host cells via ACE2 and TMPRSS2, both of which are co-expressed in several organ systems, including the lungs, heart, pancreas, kidneys, and gastrointestinal tract (4). This explains the systemic nature of COVID-19, especially in patients with a poor initial immune response, such as those vaccinated with inactivated virus vaccines like Sinopharm, which may elicit a less robust T-cell-mediated immunity.

The pancreas, though less frequently affected, has also been identified as a target organ. Wang et al. reported pancreatic injury patterns in COVID-19 patients, and Liu et al. demonstrated that ACE2 is highly expressed in pancreatic islets and exocrine tissue (9,10). In a systematic review by Karimzadeh et al., cases of acute pancreatitis in COVID-19 patients were documented even in the absence of typical triggers such as alcohol or gallstones, implicating a possible viral etiology (17).

The diagnosis of acute pancreatitis is based on at least two of the following three criteria: 1) characteristic epigastric pain; 2) serum amylase and/or lipase levels more than three times the upper limit of normal; 3) imaging findings consistent with inflammation of the pancreas (18). In this case, the diagnosis was made based on elevated enzyme levels and ultrasound findings, despite the absence of pain. The clinical course was mild, and the patient recovered with supportive therapy.

Several studies have also suggested that the severity of pancreatic involvement in COVID-19 does not always correlate with the severity of the respiratory illness, meaning that even patients with mild or moderate COVID-19 can experience pancreatic complications (13). Therefore, elevated pancreatic enzymes should not be dismissed as incidental findings, es an interesting observation in our case is the absence of abdominal pain despite laboratory evidence of pancreatic injury. Although pain is a typical and almost universal symptom of acute pancreatitis according to the revised Atlanta classification, accumulating evidence suggests that pancreatic involvement in the context of COVID-19 often follows a mild and subclinical course, without the development of classic clinical manifestations. Wang et al. reported that 17% of patients with COVID-19 had elevated pancreatic enzymes, with none exhibiting abdominal pain or clinically severe pancreatitis (3). Similar findings were described by Liu et al., who also observed a 17% incidence of pancreatic injury, again without abdominal pain or radiologic evidence of necrosis (11).

A possible explanation for the absence of pain lies in the underlying pathophysiology of SARS-CoV-2 infection. The virus uses ACE-2 receptors for cellular entry, and these receptors are highly expressed in the exocrine pancreas and pancreatic islet cells (3,21). This suggests that SARS-CoV-2 may cause direct cytopathic damage to pancreatic cells with minimal accompanying local inflammation. Unlike classical acute pancreatitis - where inflammation and edema are responsible for the characteristic severe abdominal pain - cytopathic injury

may generate an insufficient inflammatory response to produce clinical symptoms, including pain.

This mechanism provides a plausible explanation for why COVID-19 patients may exhibit laboratory abnormalities suggestive of pancreatic injury without corresponding clinical signs. Therefore, current literature indicates that SARS-CoV-2 can cause mild, subclinical pancreatic injury without abdominal pain, either through direct viral effects on pancreatic tissue or as part of a broader systemic response to infection. The absence of pain in our case is therefore not an exception but rather consistent with patterns already described in emerging studies.

Conclusion

This case illustrates the potential for multiple systemic complications of COVID-19, including pulmonary embolism and acute pancreatitis, likely driven by endothelial dysfunction and systemic viral spread. It also emphasizes the importance of careful monitoring for extra-pulmonary manifestations, especially in partially immunized individuals or those with atypical presentations. Even in the absence of classic symptoms, biochemical and imaging findings should be interpreted with a high index of suspicion during the COVID-19 pandemic.

Conflicts of Interest

There is no conflicts of interest.

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There is no financial support and sponsorship.

Informed Consent

In the form, the patient gave consent for his photographs and other clinical information to be published in the journal. The patient understands that his name will not be published and that every effort will be made to conceal his identity.

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