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Adverse course of COVID-19 despite the use of molnupiravir: A clinical case

Abstract

Relevance. Patients with type 2 diabetes mellitus are at high risk of severe COVID-19, multiple organ failure, and death. Despite the introduction of direct-acting antiviral agents, including molnupiravir, some patients may still experience an unfavourable outcome despite early initiation of treatment, highlighting the need for further investigation.

Aim. To evaluate the course of COVID-19 in a patient with type 2 diabetes mellitus using a clinical case and to analyse the possible reasons for the unfavourable outcome despite the timely administration of molnupiravir.

Materials and methods. A retrospective descriptive case study was conducted. The course of COVID-19 was analysed in a 56-year-old woman with type 2 diabetes mellitus who received antiviral therapy with molnupiravir from the fourth day of illness. Clinical manifestations, laboratory and instrumental findings, treatment characteristics, and the possible causes of the unfavourable outcome were assessed.

Results. The illness began with moderately severe respiratory symptoms and a low-grade fever. On hospital admission, signs of decompensated diabetes mellitus were identified, including hyperglycaemia (14.7 mmol/L), ketonuria (3+). Also elevated C-reactive protein (CRP) level of 91.5 mg/L and lymphopenia were revealed. Although there were no clinical and radiological signs of pneumonia on admission, the patient's condition deteriorated rapidly, with the development of metabolic acidosis, diabetic ketoacidotic coma, bilateral polysegmental pneumonia, acute respiratory distress syndrome, and multiple organ failure. Early administration of molnupiravir did not prevent a fatal outcome.

Conclusions. This clinical case demonstrates that, in patients with decompensated diabetes mellitus, COVID-19 may progress rapidly to a critical condition even in the absence of signs of pulmonary involvement at the early period of the disease. One of the key factors contributing to the poor prognosis was likely metabolic decompensation associated with ketoacidosis and a systemic inflammatory response. COVID-19 vaccination did not prevent the fatal outcome in this patient. The effectiveness of antiviral therapy for COVID-19 depends not only on the timely initiation of treatment but also on the degree of control of underlying comorbidities and the early correction of metabolic disordes. The clinical effectiveness of molnupiravir may depend on the SARS-CoV-2 variant, the baseline viral load, and individual patient peculiarities. Specialised molecular genetic studies are required to assess the potential role of antiviral resistance.

Keywords: coronavirus disease; SARS-CoV-2; type 2 diabetes mellitus; diabetic ketoacidosis; antiviral therapy; multiple organ failure.

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INTRODUCTION

Despite the end of the pandemic phase of COVID-19, the disease continues to be a significant medical and social problem due to the risk of severe forms and fatal outcomes in patients with underlying conditions. Particular attention is drawn to patients with diabetes mellitus, in whom SARS-CoV-2 infection is significantly more frequently associated with hospitalisation, the development of acute respiratory distress syndrome, multiple organ failure and death. In this context, the search for effective approaches to early antiviral treatment of COVID-19 in high-risk patients remains one of the priority areas in modern medicine. Molnupiravir became the first oral antiviral drug to demonstrate the ability to reduce the risk of hospitalisation and death in patients with COVID-19. In the randomised, placebo-controlled MOVe-OUT trial conducted by A.J. Bernal *et al.* [1], the use of molnupiravir within the first five days of symptom onset in unvaccinated high-risk patients was associated with a reduction in the rate of hospitalisation or death compared with placebo. The authors concluded that early suppression of viral replication may have a positive effect on the clinical course of the infection. A separate analysis of the results of the study by A.J. Bernal *et al*. in patients with immunodeficiency conditions, carried out by D. Focosi *et al.* [2], confirmed the drug's favourable safety profile and its ability to reduce viral load. At the same time, the authors noted that even with timely initiation of antiviral treatment, the risk of adverse outcomes is largely determined by the severity of comorbidities and the patient's baseline condition. Subsequent studies, conducted whilst Omicron variants were circulating and population immunity was high, demonstrated less conclusive results. For instance, in the large-scale randomised PANORAMIC trial by C.C. Butler *et al.* [3], which involved over 25,000 at-risk patients, the addition of molnupiravir to standard therapy did not result in a statistically significant reduction in the rate of hospitalisation or death compared with standard care. However, the authors noted a reduction in the duration of symptoms and rapid clinical recovery in patients receiving antiviral therapy. Commenting on the PANORAMIC results, M.R. Kidd & P.M. Kelly [4] pointed out that the efficacy of molnupiravir may depend on the epidemiological context, the level of population immunity, vaccination coverage and the composition of at-risk groups. According to the authors, the results of early studies cannot always be extrapolated to current conditions where new variants of SARS-CoV-2 are circulating.

The results of studies of real-world clinical practice also remain inconclusive. In a study by R. Flisiak *et al*. [5], conducted whilst the Omicron variant was dominant, the use of molnupiravir was associated with a reduction in mortality among hospitalised patients, particularly when treatment was initiated within the first five days of illness. At the same time, the authors emphasised that the drug demonstrated the greatest benefit in patients without decompensated comorbidities. Similar conclusions were reached by W. Abu Ahmad *et al*. [6], who, in a large cohort study, demonstrated that the efficacy of molnupiravir is most significant in older patients and those with risk factors for the progression of COVID-19. However, even in these patient groups, antiviral therapy does not guarantee complete protection against the development of severe disease. Diabetes mellitus plays an important role in determining an unfavourable prognosis. According to studies by S. Lim *et al.* [7], K. Khunti *et al.* [8] and J.E. Nyland *et al*. [9], hyperglycaemia is associated with impaired function of neutrophils, macrophages and T-lymphocytes, increased production of pro-inflammatory cytokines, and the development of endothelial dysfunction and coagulopathy. This is precisely why people with diabetes are considered to be one of the most vulnerable groups of patients with COVID-19. Furthermore, there is a growing body of evidence regarding the mutual exacerbation of COVID-19 and carbohydrate metabolism disorders, whereby the infection may contribute to diabetic decompensation and the development of ketoacidosis even in patients whose diabetes was relatively stable prior to infection [10-11]. There are also reports of a reduction in the clinical efficacy of certain antiviral drugs as SARS-CoV-2 evolves and accumulates mutations, including studies by C.K.H. Wong *et al*. [12], G.S. Tatz *et al.* [13] and Y. Xie *et al*. [14]. Although no conclusive evidence of clinically significant SARS-CoV-2 resistance to molnupiravir has yet been obtained, a number of authors point to the possibility of variations in response to therapy depending on the viral variant, the initial viral load and the characteristics of the patient's immune response [3-4].

Thus, despite the availability of effective antiviral agents, the challenge of predicting treatment outcomes for COVID-19 in high-risk patients remains a pressing issue. Cases of severe or fatal disease progression despite early initiation of etiological therapy are of particular scientific interest, as their analysis allows for a better understanding of the role of comorbidities and other factors that may influence treatment efficacy. It is for this reason that the clinical case presented here warrants detailed analysis and discussion. The aim of this study was to evaluate the course of COVID-19 in a high-risk patient and to analyse the possible reasons for the unfavourable disease outcome despite the timely administration of molnupiravir.

MATERIALS AND METHODS

Study type: retrospective descriptive study as a clinical case. Study design: retrospective analysis of a clinical case of fatal COVID-19. Object: the course of COVID-19 in high-risk patients receiving early antiviral therapy. Subject: factors that may have contributed to the un-

favourable course of the disease despite the early administration of molnupiravir. The study was conducted in accordance with the principles of the World Medical Association's Declaration of Helsinki on the ethical principles of medical research involving human subjects [15] and Order No. 690 of the Ministry of Health of Ukraine [16]. During the preparation of this clinical case report, the confidentiality of the patient's personal data and her anonymity were ensured. Consent to conduct the study and publish its results was obtained from the patient's daughter. A clinical case of an unfavourable course of COVID-19 is presented in a 56-year-old female patient with concomitant type 2 diabetes mellitus who received inpatient treatment at St Michael's Clinical Hospital of Kyiv from 11 September 2025 to 17 September 2025 (medical record No. 9101738). According to her medical history, the patient had received two doses of the COVID-19 vaccine in 2023; no further information regarding the vaccines is available. She had previously contracted COVID-19 in 2021. No antiviral therapy was prescribed before admission. In the hospital, due to possible renal impairment, associated with diabetes mellitus, molnupiravir was preferred for antiviral therapy (administered from the 4th day of illness at a dose of 800 mg twice daily for 5 days). Supportive care involved management of the intoxication syndrome, blood glucose levels and homeostatic parameters. Oxygen support was provided via inhalation of humidified oxygen through a face mask; the patient was subsequently transferred to mechanical ventilation. With the onset and progression of respiratory failure, dexamethasone 8 mg per day intravenously was added to the treatment. To prevent thromboembolic complications, flenox was administered subcutaneously at a dose of 0.4 ml once daily. The diagnosis of COVID-19 was confirmed by SARS-CoV-2 rapid test during the pre-hospital stage and polymerase chain reaction (PCR) in the hospital. Pulmonary ultrasound and chest radiography were used to confirm the presence of pneumonia. The severity of the patient's condition was assessed on the basis of clinical findings, oxygen saturation, and laboratory markers, including blood glucose levels, urinary ketones, arterial blood gas analysis, acid-base balance, C-reactive protein level, and lymphocyte count.

RESULTS AND DISCUSSION

Despite the clinical efficacy of molnupiravir, as demonstrated in numerous studies at the start of the pandemic, even its early administration does not always prevent severe disease progression in patients with underlying chronic conditions. The following case is presented as an example. On day 1 of the illness, the patient complained of a sore throat, a runny nose and mild weakness, with no fever. The following day, her condition worsened: she experienced nausea, repeated vomiting, a headache, increased weakness and a low grade temperature. On the third day of the illness, the patient complained of significant general weakness, a headache, a dry mouth, nausea and vomiting, and sought advice from a general practitioner at a private clinic, who suspected a stroke. This diagnosis was ruled out by a neurosurgeon on the basis of the results of a computed tomography (CT) scan of the brain. The presence of respiratory symptoms and a raised body temperature, as well as the epidemiological situation, formed the basis for conducting a rapid SARS-CoV-2 antigen test. Following a positive result, the patient was admitted to the hospital. At the time of admission (on day 3 of illness) condition of the patient was moderate severe moderately severe due to systemic toxicity: she was lethargic and adynamic. Body temperature 36.50°C, pulse 116 beats per minute, blood pressure 140/90 mm Hg, respiratory rate 22-24 per minute, oxygen saturation 98% whilst breathing room air. There were no clinical and radiological signs of pneumonia. Laboratory test results: complete blood count on 11 September 25 – white blood cells 12.17×10^9/L, metamyelocytes – 1, band cells – 10, segmented neutrophils – 62, eosinophils – 1, monocytes – 11, lymphocytes – 15%, red blood cells – 4.76×10^{12}/L, haemoglobin – 135 g/L, platelets 268×10^9/L, erythrocyte sedimentation rate 37 mm/h. Biochemical blood analysis on 11 September 25 – glucose 14.7 mmol/L, urea – 6.9 mmol/L, creatinine – 97.3 μmol/L, total bilirubin – 3.3 μmol/L, alanine aminotransferase 17.3 U/L, aspartate aminotransferase 15.8 U/L, alpha-amylase 47.5 U/L, C-reactive protein 91.5 mg/L, sodium 141 mmol/L, potassium 5.32 mmol/L, and chloride 103.0 mmol/L. Coagulation studies showed fibrin 240 mg, fibrinogen 6.0 g/L, and prothrombin activity 88%. Urinalysis revealed ketonuria (3+).

COVID-19 was diagnosed in the patient with decompensated type 2 diabetes mellitus. The diagnosis was confirmed by PCR. The patient was switched to insulin therapy; she was also prescribed Flenox 0.4 ml × once daily subcutaneously, Sterofundin 1,000 ml and sodium bicarbonate solution (Soda-Buffer®) 100 ml intravenously. The following day (day 4 of the illness), her condition deteriorated: weakness increased, dysarthria and a sense of depersonalisation developed, tachycardia increased to 124 beats per minute, and she experienced impaired consciousness resembling stupor; as a result, she was transferred to the infectious diseases intensive care unit. Examination of acid base balance revealed acidosis (pH 7.167). Lagevrio (trade name for molnupiravir) – 800 mg twice daily – was added to her treatment, whilst detoxification therapy and correction of metabolic disturbances were continued. However, the patient's condition progressively deteriorated. On 13 September 25 (on day 5 of illness), vasopressors were added to her treatment to correct haemodynamic disorders. From 14 September 25 (on day 6 of illness), the patient was unconscious; metabolic disturbances (acidosis, acetonuria, hypokalaemia, hypoalbuminaemia)

progressed; blood urea and creatinine levels rose; radiographic signs of bilateral pneumonia appeared, and respiratory failure progressed rapidly. From 16 September 25 (on day 8 of illness), the patient was transferred to invasive artificial ventilation. Despite intensive care, the patient's condition progressively deteriorated, and signs of multiple organ failure increased. On 17 September 25 (on day 9 of illness), the patient died. Post-mortem diagnosis was: COVID-19, bilateral polysegmental pneumonia, acute respiratory distress syndrome. Type 2 diabetes mellitus, diabetic angiopathy, nephropathy. Cerebral oedema with dislocation of the brain. COVID-19 triggered the decompensation of underlying type 2 diabetes mellitus, leading to the development of ketoacidotic coma, electrolyte imbalances and multiple organ failure, which resulted in the rapid progression of the infectious process with a fatal outcome. Antiviral therapy with molnupiravir, initiated at an early stage (on day 4 of illness), failed to prevent an unfavourable outcome of the disease. A timeline of the patient's disease progression and treatment, highlighting key events, is shown in Figure 1.

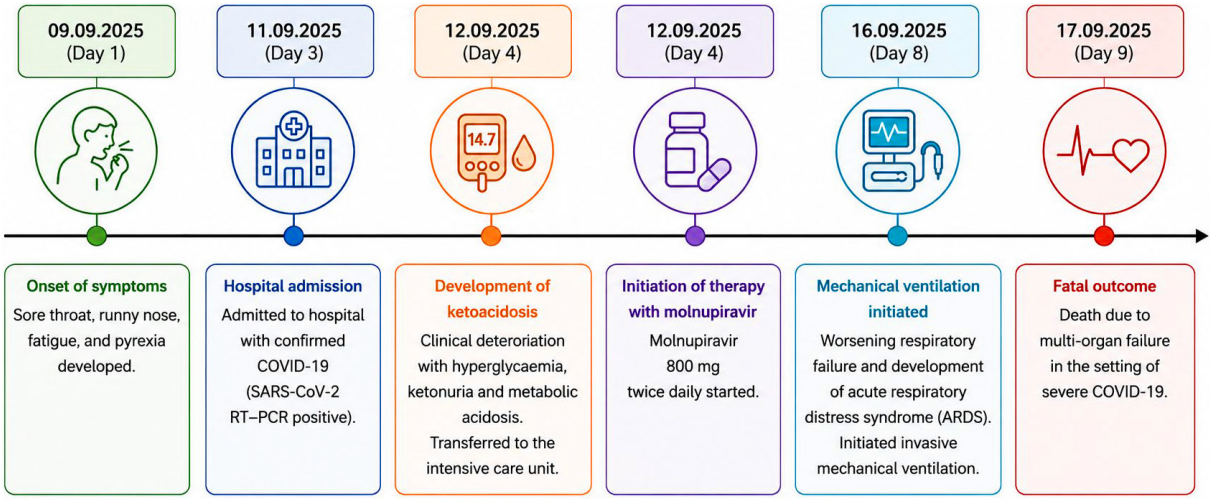

Figure 1. Timeline of clinical course and treatment of a 56-year-old female patient with COVID-19
Note: RT-PCR – reserve transcription polymerase chain reactions; ARDS – acute respiratory distress syndrome
Source: compiled by the authors

This clinical case demonstrates the rapid progression of COVID-19 in a female patient with decompensated type 2 diabetes mellitus, despite the absence of signs of pneumonia and respiratory failure at the time of admission and the early initiation of antiviral therapy with molnupiravir. Most likely, the unfavourable outcome followed several reasons. First and foremost, the presence of decompensated diabetes mellitus with hyperglycaemia and acetonuria at the time of admission is noteworthy, indicating significant metabolic disorders even before the development of severe lung involvement. Hyperglycaemia is associated with impaired innate and adaptive immune responses, increased systemic inflammation and endothelial dysfunction [17-18]. It also increases the risk of thrombotic complications, which may contribute to the rapid progression of COVID-19 [19-20]. The patient presented with signs of severe systemic disorders, including progressive intoxication, impaired consciousness and metabolic acidosis, despite the early (on day 4 from the onset of the illness) administration of molnupiravir. This suggests that critical pathophysiological processes associated with the decompensation of diabetes mellitus and a systemic inflammatory response had already been triggered before the start of aetiological treatment. Under these circumstances, the suppression of viral replication may have proved insufficient to prevent the subsequent development of multiple organ failure.

Other factors capable of influencing the efficacy of antiviral therapy cannot be ruled out, in particular a high initial viral load, individual variations in the drug's pharmacokinetics against a background of severe metabolic disorders, or infection with a SARS-CoV-2 variant exhibiting reduced sensitivity to the drug due to the possible development of resistance to molnupiravir. At the same time, it is not possible to conclude, on the basis of a single clinical case, that molnupiravir is ineffective as a drug in general. The choice of antiviral therapy for high-risk patients remains an important clinical issue. Given the high likelihood of diabetes decompensation against the background of an infectious process, it might be more effective to initiate antiviral therapy before the development of severe metabolic disordes – on days 1-2 of COVID-19. The possibility of using alternative antiviral drugs or combined approaches in patients with severe comorbidities also requires further investigation. The case presented here highlights the need for particular vigilance regarding patients with diabetes mellitus, even in the absence of severe lung

involvement, and the importance of early detection of metabolic decompensation as one of the key predictors of an unfavourable course of COVID-19.

Molnupiravir remains one of the recommended oral antiviral drugs for the treatment of patients with COVID-19 who are at high risk of disease progression. The results of early randomised trials have demonstrated its ability to reduce the risk of hospitalisation and death when administered within the first five days of symptom onset. At the same time, data from C.C. Butler *et al*. [3] and W. Abu Ahmad *et al.* [6], collected during the circulation of later SARS-CoV-2 variants, indicated a more moderate clinical effect of the drug and a significant dependence of treatment outcomes on patients' age, comorbidities, vaccination status and the timing of treatment initiation. The clinical case presented here demonstrates an unfavourable course of COVID-19 in a high-risk patient. Early administration of molnupiravir was unable to prevent the development of severe metabolic disorders associated with decompensated diabetes mellitus, which may have contributed to subsequent clinical deterioration and the development of multiple organ failure. The findings are partly consistent with current literature data on the efficacy of molnupiravir. In a study by A.J. Bernal *et al.* [1], early administration of the drug to patients with risk factors for COVID-19 progression was associated with a reduced risk of hospitalisation or death compared with placebo. At the same time, further studies of real-world clinical practice and the results of the large-scale randomised PANORAMIC trial have demonstrated that, in the context of circulating new variants of SARS-CoV-2 and against a background of prior vaccination of the population, the effect of molnupiravir on the rate of hospitalisations and mortality is less pronounced, although the drug promotes faster clinical recovery and viral clearance [3]. Data from the meta-analyses by Y.M. Mesfin *et al*. [21] and S.H. Lin *et al*. [22] also demonstrated that the clinical effect of molnupiravir varies depending on patients' age, the presence of comorbidities and the timing of treatment initiation.

Particular attention is drawn to the role of diabetes mellitus as one of the leading predictors of an unfavourable prognosis in COVID-19. According to cohort studies, the risk of hospitalisation, the development of acute respiratory distress syndrome and death in patients with uncontrolled diabetes mellitus is significantly higher than the corresponding figures in individuals without carbohydrate metabolism disorders. S. Lim *et al.* [7] and R. Pal *et al*. [10] have shown that the presence of diabetic ketoacidosis or severe hyperglycaemia at the time of hospitalisation is an independent predictor of a poor prognosis, regardless of the severity of lung involvement. In the present case, hyperglycaemia, acetonuria and laboratory signs of systemic inflammation were already present on admission, supporting the assumption that metabolic decompensation may have contributed to the subsequent severe course of COVID-19. It is evident that antiviral drugs are most effective early in the disease course, when active viral replication plays a leading role. Once a systemic inflammatory response, metabolic disorders and organ dysfunction have developed, the course of the disease is largely determined by immunopathological mechanisms. Thus, the lack of clinical response to molnupiravir in this patient does not necessarily indicate the drug's ineffectiveness, but may reflect the severity of the underlying condition and the already established cascade of pathophysiological changes at the time treatment began. A distinctive feature of this case was the absence of signs of pneumonia and respiratory failure on admission, despite the concurrent presence of marked metabolic disturbances. Hyperglycaemia, acetonuria and elevated C-reactive protein levels were observed as early as the first few days of the illness, indicating the development of a systemic inflammatory response and decompensation of diabetes mellitus. Type 2 diabetes remains one of the most significant risk factors for severe COVID-19. Hyperglycaemia contributes to impaired functional activity of neutrophils and lymphocytes, increased production of pro-inflammatory cytokines, and the development of endothelial dysfunction and coagulopathy. Furthermore, SARS-CoV-2 infection can impair glycaemic control and trigger the development of diabetic ketoacidosis, even in patients whose diabetes had previously been relatively stable. In the present case, it was the decompensation of diabetes mellitus that likely constituted one of the key mechanisms underlying the progression of the disease and the development of multiple organ failure.

Despite the initiation of antiviral therapy within the recommended timeframe (on day 4 of illness), by the time molnupiravir was prescribed, the patient was already exhibiting signs of severe systemic complications, including metabolic acidosis, progressive intoxication and impaired consciousness. This suggests that the pathological cascade associated with diabetic decompensation and systemic inflammation had already been triggered before the start of aetiological treatment. Since molnupiravir's mechanism of action involves inhibiting viral replication, its clinical efficacy is predictably reduced in situations where metabolic and immuno-inflammatory disorders already play a leading role in the pathogenesis. It is also worth noting that recent studies demonstrate heterogeneity in the clinical efficacy of molnupiravir. In some high-risk patients, particularly those with severe comorbidities, the drug does not fully prevent hospitalisation, the development of respiratory failure or fatal outcomes. The influence of factors such as a high initial viral load, characteristics of the immune response, the drug's pharmacokinetics in the context of severe metabolic disorders, or individual variability in the virus's sensitivity to therapy cannot be ruled out.

From a clinical perspective, this case highlights the need for the earliest possible identification of patients with decompensated diabetes mellitus and the timely initiation of antiviral therapy before the development of severe metabolic disorders. Equally important are aggressive correction of hyperglycaemia, careful monitoring of acid-base balance, and early diagnosis of diabetic ketoacidosis. Further research is needed to evaluate the efficacy of alternative antiviral drugs and personalised approaches to the treatment of patients with COVID-19 and severe comorbid conditions. Thus, the presented case demonstrates that even early administration of molnupiravir does not always prevent an unfavourable course of COVID-19 in patients with severe metabolic disorders. It should be noted that vaccination against COVID-19 did not prevent a fatal outcome. Not only early antiviral therapy, but also the degree of control of the underlying condition, the timeliness of correction of metabolic disorders, and individual characteristics of the course of the infectious process may be of decisive importance for the prognosis.

CONCLUSIONS

1. In patients with type 2 diabetes mellitus, COVID-19 can rapidly progress to a critical condition even in the absence of signs of pneumonia in the early stage of the disease.

2. Decompensation of diabetes mellitus with the development of metabolic acidosis may be one of the key factors contributing to an unfavourable prognosis of COVID-19.

3. In the presented clinical case, early administration of molnupiravir (on day 4 of illness) didn't prevent disease progression and a fatal outcome. This may have been associated with severe metabolic disorders in the context of decompensated diabetes mellitus.

4. The clinical efficacy of molnupiravir may depend on the SARS-CoV-2 variant, the baseline viral load and the patient's individual peculiarities. Specialised molecular genetic studies are required to assess the role of possible antiviral resistance.

5. Further studies are warranted to determine how concomitant metabolic disorders influence the response to antiviral therapy and to optimise treatment strategies for patients with COVID-19 and diabetes mellitus.

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CONFLICT OF INTEREST

None.

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Несприятливий перебіг COVID-19 із застосуванням молнупіравіру: клінічний випадок

Анотація

Актуальність. Пацієнти з цукровим діабетом належать до групи високого ризику тяжкого перебігу COVID-19, розвитку поліорганної недостатності та летального наслідку. Незважаючи на впровадження противірусних препаратів прямої дії, зокрема молнупіравіру, окремі хворі навіть за умов раннього початку лікування можуть мати несприятливий наслідок хвороби, що потребує подальшого вивчення.

Ціль. На прикладі клінічного випадку оцінити перебіг COVID-19 у пацієнтки з цукровим діабетом II типу та проаналізувати можливі причини несприятливого наслідку захворювання попри своєчасне призначення молнупіравіру.

Матеріали та методи. Проведено ретроспективне описове дослідження у форматі клінічного випадку. Проаналізовано перебіг COVID-19 у пацієнтки 56 років із цукровим діабетом II типу, яка отримувала противірусну терапію молнупіравіром з 4-го дня захворювання. Оцінювали клінічні прояви, результати лабораторних та інструментальних досліджень, особливості лікування та причини несприятливого наслідку.

Результати. Захворювання розпочалося з помірно виражених респіраторних симптомів та субфебрильної температури тіла. На момент госпіталізації виявлено ознаки декомпенсації цукрового діабету: гіперглікемію (14,7 ммоль/л), ацетонурію (3+), а також підвищення рівня С-реактивного білка до 91,5 мг/л та лімфопенію. За відсутності клініко-рентгенологічних ознак пневмонії при поступленні у стаціонар, стан пацієнтки швидко погіршувався з розвитком метаболічного ацидозу, кетоацидотичної коми, двобічної полісегментарної пневмонії, гострого респіраторного дистрес-синдрому та поліорганної недостатності. Раннє призначення молнупіравіру не запобігло летальному наслідку.

Висновки. Представлений клінічний випадок демонструє, що у пацієнтів із декомпенсованим цукровим діабетом COVID-19 може швидко прогресувати до критичного стану навіть за відсутності ознак ураження легень на початку захворювання. Одним із ключових чинників несприятливого прогнозу, ймовірно, була метаболічна декомпенсація з розвитком кетоацидозу та системної запальної відповіді. Щеплення від COVID-19 не попередило летальний наслідок хвороби у пацієнтки. Ефективність противірусної терапії при COVID-19 залежить не лише від своєчасності її призначення, а й від ступеня контролю супутньої патології та ранньої корекції метаболічних порушень. Клінічна ефективність молнупіравіру може залежати від варіанта SARS-CoV-2, вихідного вірусного навантаження та індивідуальних особливостей пацієнта. Для оцінки ролі можливої противірусної резистентності необхідні спеціальні молекулярно-генетичні дослідження.

Ключові слова: коронавірусна хвороба; SARS-CoV-2; цукровий діабет II типу; діабетичний кетоацидоз; противірусна терапія; поліорганна недостатність.