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Death due to multiple organ failure in a patient with severe COVID-19, AIDS, cardiovascular impairment, and previous lung infections

Morte por falência múltipla de órgãos em paciente com COVID-19 grave, AIDS, comprometimento cardiovascular e infecções pulmonares prévias

Muerte por insuficiencia multiorgánica en un paciente con COVID-19 grave, SIDA, deterioro cardiovascular y infecciones pulmonares previas

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RESUMO

Objetivo: Relatar um caso de coinfecção pelo SARS-CoV-2/HIV em um paciente com histórico recente de infecções pulmonares, que teve parada cardiopulmonar no dia de sua internação para tratar da COVID-19, falecendo alguns dias depois de falência múltipla de órgãos. **Detalhamento de caso:** Um homem de 33 anos, imunossuprimido (em abandono de tratamento para o HIV) deu entrada no serviço de pronto-atendimento com sintomas respiratórios e testou positivo para SARS-CoV-2. Durante a internação, o paciente teve parada cardiopulmonar e apresentou intenso perfil inflamatório, infecção fúngica, sepse, acidose metabólica, hipertensão arterial, trombose, hemorragia, além de lesão hepática, muscular e renal. Por fim, o paciente evoluiu para falência de múltiplos órgãos, indo à óbito. **Considerações finais:** A grave imunodeficiência causada pelo HIV, associada a doenças cardiopulmonares de base, contribui para desfechos letais da COVID-19.

Palavras-chave: COVID-19, HIV, Coinfecção, Falência de múltiplos órgãos.

ABSTRACT

Objective: To report a case of SARS-CoV-2/HIV coinfection in a patient with a history of recent lung infections who had a cardiopulmonary arrest on the day of hospitalization for COVID-19 treatment, dying some days after multiple organ failure. **Case detail:** A 33-year-old immunosuppressed male patient (abandoning treatment for HIV) was admitted to the emergency department with respiratory symptoms and tested positive for SARS-CoV-2. During hospitalization, the patient had a cardiorespiratory arrest and presented a robust inflammatory profile, fungal infection, sepsis, metabolic acidosis, arterial hypertension, thrombosis, hemorrhage, and hepatic, muscular, and renal injury. Finally, he evolved with multiple organ failures, leading to death. **Conclusion:** Severe immunosuppression caused by HIV, associated with underlying cardiopulmonary diseases, contributes to lethal outcomes of COVID-19.

Keywords: COVID-19, HIV, coinfection, multiple organ failure.

RESUMEN

Objetivo: Comunicar un caso de coinfección por SARS-CoV-2 en un paciente con antecedentes recientes de infecciones pulmonares, que sufrió una parada cardiopulmonar el día de su hospitalización para tratar el COVID-19, falleciendo pocos días después por fallo multiorgánico. **Detalle del caso:** Un hombre inmunodeprimido de 33 años (que había abandonado el tratamiento contra el VIH) ingresó en el servicio de urgencias con síntomas respiratorios y dio positivo en la prueba del SARS-CoV-2. Durante la hospitalización,

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el paciente sufrió una parada cardiopulmonar y presentó un perfil inflamatorio intenso, infección fúngica, sepsis, acidosis metabólica, hipertensión, trombosis, hemorragia, así como lesiones hepáticas, musculares y renales. Finalmente, el paciente evolucionó a un fallo multiorgánico y falleció. **Conclusión:** La inmunodeficiencia grave causada por el VIH, asociada a enfermedades cardiopulmonares subyacentes, contribuye a los resultados letales del COVID-19.

Palabras clave: COVID-19, HIV, coinfección, fallo multiorgánico**.**

INTRODUCTION

COVID-19 is a multisystem disease caused by the SARS-CoV-2 virus that became a pandemic in 2019 (MIR T, et al., 2021) and led to the death of approximately 7 million people around the world by March 2023 (WHO, 2023). The symptoms of COVID-19 range from mild to severe illness, mainly characterized by fever, dry cough, dyspnea, headache, sore throat and rhinorrhea, and sometimes hemoptysis with often nonspecific symptoms (KADIRVELU B, et al., 2022).

However, other patterns have already been described, such as severe acute respiratory syndrome, coagulopathies, thromboembolism, intestinal malabsorption, hemorrhagic colitis, kidney failure, myocarditis, kidney failure, heart failure, arrhythmias, ischemia and hemorrhages in the central nervous system, multiple organ failure, among others (MIR T, et al., 2021).

Among the risk groups frequently reported, we highlight those who already have comorbidities such as obesity, hypertension, and diabetes, whose systemic damage can be aggravated by infection (NAVEED M, et al., 2021).

People with immune deficiencies are also at higher risk for acquiring SARS-CoV-2 infection and developing severe COVID-19 since the antiviral response becomes inefficient (BANSAL N, et al., 2021), with Human Immunodeficiency Virus (HIV) being one of the most immunosuppressive pathogens, once HIV leads to depletion of CD4 T-lymphocytes, resulting in a weakened adaptive immune response (MAARTENS G, et al., 2007).

There are striking discrepancies in the literature regarding the difference in the pattern of morbidity and mortality caused by SARS-CoV-2 among people living with HIV and those who are not. Some studies have already pointed out that cases of COVID-19 in HIV patients do not differ significantly from the non-HIV population, especially when individuals are not immunocompromised (KANWUGU ON, ADADI P, 2021; DÍEZ C, et al.,2021). On the other hand, two meta-analyses showed an increased risk of mortality from COVID-19 among people living with HIV (MELLOR MM, et al., 2021; HARIYANTO TI e ROSALIND J, 2021).

In this population, when the CD4+ count < 200 cells/μL, a higher rate of intensive care unit admission, greater use of mechanical ventilation, and decreased survival rate were observed, owing to the impairment of immune response (DANDACHI D, et al., 2021; KARMEN-TUOHY S, et al., 2020).

Furthermore, the depletion of immunity induced by HIV favors the emergence of opportunistic infections, some of which may also affect the lung and intensify the damage caused by SARS-CoV-2, as has already been observed for histoplasmosis (PIPITÒ L, et al., 2023; BASSO RP, et al., 2021, BERTOLINI M, et al., 2020), *Pneumocystis jirovecii* (ALSHARIF N, et al., 2023) and tuberculosis (WHO, 2021). COVID-19 also exacerbates the clinical manifestations of oral candidiasis (HAPID MH e DEWI TS, 2023) and herpes zoster (DAODU J, et al., 2022) in patients with AIDS.

In this context, the present article will report the clinical and laboratory evolution of a case of death due to multiple organ failure resulting from SARS-CoV-2/HIV coinfection in an immunocompromised patient with a history of recent pulmonary infections (tuberculosis and pneumocystosis) and cardiopulmonary arrest that occurred on the day of hospitalization. Our group requested the Research Ethics Committee of the Gaffrée and Guinle University Hospital to use the data available in the internal medical records and the hospital's digital test results system for research purposes. The project was evaluated and approved (CAAE: 52743721.4.0000.5258/Parecer 5.220.441).

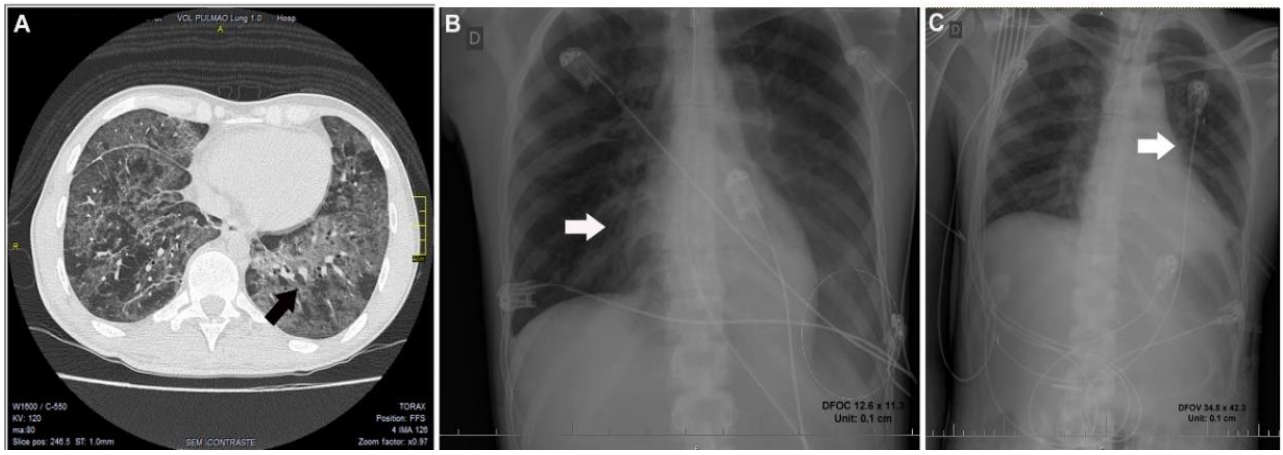
CASE REPORT

An HIV-positive 33-year-old man (poor adherence to treatment), with a history of pulmonary tuberculosis in 2017 and reactivation in 2020, was admitted to the University Hospital's emergency room in November 2021 due to dyspnea and desaturation. On this occasion, computed tomography of the lung revealed a bilateral ground-glass pattern with a distribution suggestive of pneumocystosis (Figure 1A). The patient was then hospitalized and treated with Sulfamethoxazole and Trimethoprim, in addition to corticosteroid therapy with Prednisone. This protocol allowed him to evolve with an improvement of the symptomatology and subsequent discharge from the hospital.

Four months later, the patient returned to the University Hospital's emergency department complaining of cough, fever, and diarrhea for seven days. During this visit to the hospital, he was agitated and tachypneic, with a heart rate of 150 beats per minute and an electrocardiogram profile compatible with sinus tachycardia. After acute pulmonary respiratory failure, oxygen catheter therapy was performed, but without response. Next, an oxygen mask with a reservoir at 15L/min was used, but the patient maintained tachypnea and agitation. To be hospitalized, the patient underwent a qRT-PCR test, and an active SARS-CoV-2 infection was verified. The patient was not yet vaccinated for COVID-19. The X-ray also found a ground-glass opacity pattern and a deep groove on the left side, indicating pneumothorax (Figure 1B).

On the same day, the patient evolved with cardiorespiratory arrest in pulseless electrical activity. The reversal of the picture occurred six minutes later with the use of 2 ampoules of adrenaline. Upon this scenario, the patient was transferred to a COVID-19 dedicated treatment center located in the same hospital with the diagnosis of severe COVID-19.

Figure 1. Thorax imaging exams of patient. Computed tomography with axial sectional plane showing ground-glass opacities (arrow) (A). Chest X-ray on the day of admission showing ground-glass opacities at the arrow and deep groove (circle on the left side) (B). Chest X-ray showing the pneumothorax on the left side (C).



Source: Junior F, et al., 2024.

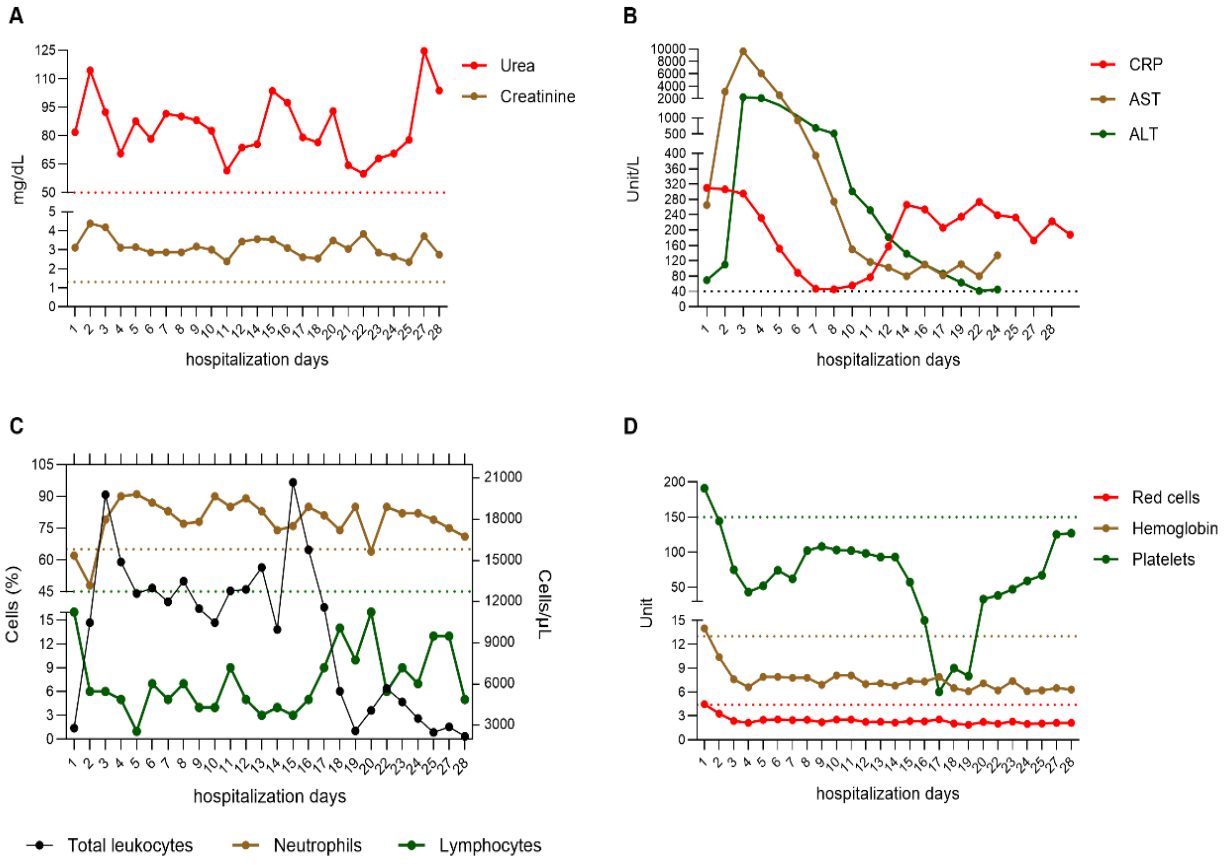
The patient, at the time of hospital admission, was severely ill, sedated (Richmond Agitation-Sedation Scale -4), normotensive in use of amine (129/66 mmHg), tachycardic (134 beats per minute), tachypneic (25 respiratory incursions per minute), with myotic pupils and hemodynamically unstable. In the clinical evaluation, fungal lesions were found throughout the chest length and cyanotic right hand. During his stay in a few hours, it evolved to RASS -5 and became febrile (39°C), which motivated the opening of a sepsis protocol with 1L of crystalloid, in addition to treatment with Rocefin and Caritheromycin, thinking about a probable pulmonary focus.

At this moment, arterial blood gas analysis showed metabolic acidosis, with a pH of 6.97, pCO_2 of 37mmHg, HCO_3 of 7mmHg, and lactate of 5mmol/L. Additional laboratory investigations were performed. The results also showed changes in urea and creatinine that were maintained throughout the hospitalization period (Figure 2A). The patient was also oliguric and had amber urine staining. Due to the picture suggestive of renal damage, the nephrology team started renal replacement therapy. On the third day after admission, an X-ray confirmed

pneumothorax (Figure 1C), and an ultrasound showed the absence of pleural slippage. As a conduct, surgery for closed pleural drainage was adopted. On the same day, a routine laboratory examination detected creatine phosphokinase (CK) of 5,185 IU/mL (reference value: 0 to 170 IU/mL) and creatine phosphokinase-MB (CK-MB) of 155 IU/L (reference value: 0 to 25 IU/L). On his fourth day, the CK value rose to 6,876IU/mL. Simultaneously, liver damage was observed through aspartate aminotransferase (AST) and alanine aminotransferase (ALT) parameters, as well as the pro-inflammatory state through ultrasensitive C-reactive protein (CRP) (reference values of 10 to 40mg/dL, 0.7 to 1.3mg/dL, and 0 to 5 mg/L, respectively) (Figure 2B).

On the following days of his admission, in addition to mechanical ventilation and hemodialysis, the patient began to need sodium nitroprusside to control the evolution of hypertension; his D-dimer dosage reached 10,000ng/mL (reference value: 500ng/mL) and returned to febrile peaks. On the eleventh day, a bronchoscopy was performed, in which an abundant amount of diffuse purulent secretion, reddish lesions with small whiteish blisters at the entrance of the lower lobes, and some areas of white lesion adhered diffusely to the mucosa were observed. When the examination was performed with entry through the right nostril, similar lesions were identified at the base of the tongue, epiglottis, and hypopharynx. Bronchoalveolar lavage culture identified the growth of *Escherichia coli* sensitive to Meropenem, so treatment with this antibiotic was initiated. The condition remained severe with evolutionary worsening, need for amines, fever, maintenance of elevated inflammatory parameters, metabolic acidosis, and anuresis. The bilirubin dosage (which until then was within the normal range) reached a value of 4.04mg/dL, increasing daily until reaching 8.80mg/dL seven days later (reference value: less than 1.30mg/dL). The blood count showed a high global count and percentage of neutrophils and lymphopenia (Figure 2C).

Figure 2. Biochemical and hematological parameters of the patient. Classic renal biomarkers (A), liver injury biomarkers and nonspecific markers of systemic inflammation (B), leukocyte variation (C), and red blood cell parameters. The dotted line represents exam reference values. CRP = C-reactive protein; AST = aspartate aminotransferase; ALT = alanine aminotransferase.



Source: Junior F, et al., 2024.

On his 16th day of hospitalization, the patient had spontaneous bleeding from the nostril. Simultaneously, a drop in platelet count, global red blood cell count, and hemoglobin levels were observed (Figure 2D), constituting anemia of hemorrhagic origin. In addition, the patient progressed from hyper to hypotension, requiring suspension of sodium nitroprusside and administration of noradrenaline. Cyanosis, which was already occurring in the hands, also began in the feet. On the same day, the result of the CD4+ cell count was also released, which was only four cells/mm³ (severe immunodepression; AIDS).

On the following day, the 17th after the admission, the vascular surgery team evaluated cyanosis of the upper and lower limbs, defining the need to wait for the delimitation of the lesion for amputation.

After twenty-six days of hospitalization, the patient was already diagnosed with multiple organ failure and, upon realizing that he was severely hypotensive (60 x 20mmHg), the doses of amine were quickly increased, and the Propofol used for sedation was stopped. However, when performing this procedure, the patient evolved with a disorganized rhythm on the monitor and absence of pulse on palpation, configuring cardiac arrest in electrical activity without a pulse. Cardiac resuscitation measures were initiated, but without success, thus verifying the death of the patient.

DISCUSSION

This case report was intended to discuss the coinfections of HIV/AIDS and COVID-19 of a single patient with a recent history of pulmonary tuberculosis and pneumocystosis. In this case study, the patient was admitted and was observed during 26 days of hospital stay. Although COVID-19 affects all types of individuals, patients with HIV and multiple comorbidities, including advanced chronic obstructive lung disease, are reported to be at risk of the worst outcome (YANG et al., 2020).

COVID-19 is a contagious disease with recurrent and severe pulmonary involvement, which can lead to viral pneumonia, hospitalizations, and deaths. According to the literature, SARS-CoV-2 recognizes ACE-2 receptors for entry into target cells, and depletion of these molecules, especially in the lung, stimulates increased production of angiotensin II, which in turn stimulates angiotensin II receptor type 1a. This cascade causes increased vascular permeability in the lung and causes acute lesions (HOFFMANN M, et al., 2020) that, on imaging tests, can be visualized as ground-glass opacities (Figure 1). This finding represented 90.95% of the elemental lesions observed in a study of tomographic aspects of pneumopathies associated with COVID-19 (TIEMTORE-KAMBOU BM et al., 2022). In general, the patient's symptomatology at the time of admission is characteristic of severe cases of COVID-19 (KADIRVELU B, et al., 2022), and recent previous damage caused by tuberculosis and pneumocystosis may have aggravated pulmonary manifestations. A non-fatal case of spontaneous pneumothorax in a patient with pulmonary infections, AIDS, and COVID-19 had already been reported (PHILIP et al., 2023). Additionally, angiotensin II is vasoconstrictive (BUDHIRAJA R, et al., 2004), which partly explains the blood pressure changes observed in the patient.

It is important to note that it is already well-described that COVID-19 is a highly inflammatory disease that can affect several organs (MIR T, et al., 2021). In the case described, C-reactive protein changes show this intense and constant inflammation, which is also a consequence of uncontrolled HIV infection (PENG X, et al., 2020). It is also possible to observe liver damage (by increasing AST and ALT) to skeletal and cardiac striated muscles (by increasing total CK and CK-MB) and severe renal impairment (by alteration of urea, creatinine, and need for hemodialysis). At the end of the interaction period, multiple organ failure had already been observed.

These conditions are rare and mainly affect individuals with comorbidities. In this reported case, the low CD4+ cell count allowed a favorable environment for the proliferation and action of the virus without an adequate immune response. This reduction is characteristic of both untreated HIV infection and severe cases of COVID-19 (BO XU, et al., 2020). In addition to tuberculosis that was not fully resolved, the patient had already developed pneumocystosis four months earlier, a classic opportunistic disease among people with HIV in the framework of AIDS (DE FIGUEIREDO IR, et al., 2019). During his stay, the patient also presented fungal lesions on the chest and presented sepsis at different times along the days of hospitalization.

The high values of D-Dimer reflect a context of coagulation imbalance that, associated with reduced platelet count, suggests intense thrombus formation. It has already been reported that severe cases of COVID-19 have a pro-thrombotic profile. In a study conducted with 150 COVID-19 patients admitted to intensive care units, 64 (42.6%) had significant thrombotic complications associated with virus infection (HELMS J, et al., 2020). These thrombi may have been the cause of cyanosis with subsequent necrosis of the extremities reported in this study, as well as the drop in platelet count. On the other hand, thrombocytopenia caused hemorrhagic episodes, which culminated in anemic conditions and increased absolute and relative neutrophil counts. The increase in these cells is evidence of the progression to sepsis, which could contribute to establishing biomarkers for clinical usage (LEITE RO, et al., 2023). Decreased lymphocyte counts are joint in severe cases of COVID-19 and are usually associated with a worse prognosis (PENG X, et al., 2020).

Finally, it is worth highlighting that the overlapping damage caused by infections and low immunological levels are added to the impact of cardiopulmonary impairment occurring after successive cardiorespiratory arrests, which, it themselves, already have a solid potential to damage the cardiovascular, neurological, pulmonary, renal, and metabolic systems (RAVETTI CG, et al., 2009). The severe manifestations of COVID-19 can be prevented through vaccination. Studies already show that people living with HIV can present similar protection results from the vaccine when compared to individuals without HIV (PLUMMER MM e PAVIA CS, 2021). Vaccination is still highly recommended, especially when the individual already has AIDS (CDC, 2023). However, a systematic review with meta-analysis showed that vaccine acceptance among people living with HIV is still less than 70%. Among the main factors associated with this resistance would be higher monthly income, history of chronic disease, being non-homosexual, COVID-19-related medical mistrust, not knowing anyone who died of COVID-19, general vaccine refusal, believing oneself to be immune to COVID- 19, negative attitude to the vaccine, safety, and side effects, concerns about efficacy, distrust in familiar sources of vaccine-related information and using social media as a source of information on COVID-19 (EJAMO JY, et al., 2023).

At the time of the patient's death, vaccines against COVID-19 had already been available in the Unified Health System for a few months. Still, the patient was not immunized. Considering that he had also abandoned treatment for HIV, the success of protection against COVID-19 among people living with HIV must begin by combating the natural resistance that a large part of this population has against the vaccine and raising awareness about the importance of immunization, especially among those who already evolved to AIDS.

This reported case highlights the understanding of the relationship between HIV and SARS-CoV-2 and its importance in public health implications. This fatal case due to multiple organ failure showed the importance of treatment of HIV infection and vaccination against SARS-CoV-2 to reduce the chances of evolution to severe and fatal COVID-19. It also underlines the main severe manifestations in a single patient, who simultaneously presented an inflammatory solid, thrombotic, hemorrhagic profile with cardiovascular dysregulation and pulmonary, hepatic, muscular, and renal lesions.

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