JOURNAL OF SCIENCE & DISEASES

COVID-19 infection in two homozygous sickle cell patients in a reference hospital in Cameroon: Case reports

Infection au COVID-19 chez deux patients atteints de drépanocytose homozygote dans un hôpital de référence au Cameroun : Cas clinique

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Clinical Case

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Key words: COVID-19, sickle cell disease, respiratory distress, acute chest syndrome, Douala-Cameroon.

Mots clés : COVID-19, drépanocytose, détresse respiratoire, syndrome thoracique aigu, Douala-Cameroun.

Date de soumission: 14/08/2025
Date d'acceptation: 15/01/2026

ABSTRACT

We report two cases of COVID-19 infection. The first was a 22-year-old female and the second one a 42-year-old male, were both homozygous sickle cell disease (SCD) patients known since their childhood, but with different medical follow-ups for SCD. Initially, they were admitted to the hospital for a vaso-occlusive crisis. They both subsequently developed respiratory symptoms for which the Initial diagnosis of acute chest syndrome was made. Meanwhile, SARS-COV-2 infection was a potential differential diagnosis due to the prevailing use of COVID-19 pandemic. This infection was subsequently confirmed in these patients, and the common pathophysiological trends described in both conditions were observed, as well as clinical and biological manifestations that could be assimilated to both disease entities. Both patients were managed with the national COVID-19 disease protocol while maintaining specific treatment elements for vaso-occlusive crisis in sickle cell patients. Both had favourable outcomes. In the era of the COVID-19 pandemic, a high index of clinical suspicion of COVID-19 disease is warranted in SCD patients presenting with respiratory symptoms to reduce the morbid mortality of both sickle cell disease and COVID-19.

RESUME

Nous rapportons deux cas d'infection par COVID-19 chez des patients drépanocytaires. Le premier était une femme de 22 ans et le second un homme de 42 ans, tous deux atteints de drépanocytose homozygote. Ils ont développé des symptômes respiratoires pour lesquels un diagnostic initial de syndrome thoracique aigu a été posé. Parallèlement, l'infection par le SARS-COV-2 était un diagnostic différentiel potentiel en raison de la pandémie de COVID-19 qui sévissait. Cette infection a ensuite été confirmée chez ces patients, et les tendances physiopathologiques communes décrites dans les deux affections ont été observées, ainsi que des manifestations cliniques et biologiques pouvant être assimilées aux deux entités pathologiques. Les deux patients ont été pris en charge conformément au protocole national relatif à la COVID-19, tout en conservant les éléments thérapeutiques spécifiques à la crise vaso-occlusive. Les deux ont eu des issues favorables. Dans le contexte de pandémie de COVID-19, il est nécessaire de porter une attention particulière chez le drépanocytaire présentant des symptômes respiratoires afin de réduire la mortalité liée à la fois à la drépanocytose et à la COVID-19.

DOI : https://doi.org/10.64294/jsd.v4i1.264

Citation as **J Sci Dis 2026, Vol 4 (1): 139 - 144**

Introduction

In December 2019, a new strain of coronavirus (SARS-COV-2), emerged in Wuhan, a province in China. It was identified, as the cause of the disease called COVID- 19, characterised by a potentially fatal acute respiratory distress syndrome [1]. COVID-19 is transmitted in humans via inhaled contaminated droplets [2]. Within a few months of its outbreak, COVID-19 was an epidemic on the entire territory of China [3, 4]. By March 2020, COVID-19 had spread worldwide infecting millions of persons and responsible for millions of global deaths, sufficient to be tagged by WHO as one of the world's unprecedented pandemics [5]. The first case of COVID-19 in Cameroon was confirmed on March 6, 2020 [6] and the first case of death due to COVID-19 was on 24 March 2020 [7]. This growing pandemic has had an impact on all sectors of Cameroon, including medical care. Precisely, the need to reorganise general care in this context, to acquire appropriate personal protective equipment, and to manage psychological support for health workers, patients and their families. The care of sickle cell patients during this pandemic has attracted our attention because of their vulnerability to infections in general and lung infections in particular. The mortality rate of COVID-19 in immunosuppressed persons is higher [2, 8]. Given the comorbidities and immune dysfunction due to functional asplenia generally observed in sickle cell disease (SCD) patients, it has been hypothesized that SCD patients may present a particularly high risk of infection susceptibility to COVID-19 [9, 10]. Although COVID-19 is generally more serious and fatal in the elderly, people of any age with underlying health problems may be at increased risk of COVID-19 and have a poorer prognosis [10]. We report here, two cases of COVID-19 infection in homozygous sickle cell patients in a reference hospital in Douala, Cameroon. The aim of this presentation was to highlight the need to pay particular attention to patients with homozygous sickle cell disease during the COVID-19 pandemic, given the pathophysiological similarities that may be observed in acute chest syndrome during vaso-occlusive crises, and which are likely to worsen the prognosis. The ethical clearance N° 2019/0010/HGOPED/DG/CEI was obtained from Douala Gynaeco-obstetric and paediatric hospital institutional ethics committee as well as verbal consent from each patient on the condition that no identification was included in these case reports.

Case 1

This was a poorly managed case of a 22-year-old homozygote SCD (HbSS) female patient who had about five crisis per year on average, and a basal haemoglobin level of about 8g/dL. Prior to the consultation at that time, she had been admitted in another health facility, for vaso-occlusive crisis, and myositis of the right forearm, which was treated surgically. Afterwards, she developed a dry cough, associated with breathlessness and a high-grade fever of 40°C. This prompted the request of a thoracic CT-scan, which revealed bilateral, pulmonary, parenchymal lesions, estimated at 50-60%, suggestive of COVID- 19 pneumopathy (Figure 1). This motivated her transfer to the Douala Gynaeco-Obstetric and Paediatric reference Hospital. On admission, she was ill-looking with conjunctival pallor and mild jaundice. She was in respiratory distress marked by dyspnoea at rest, with a respiratory rate at 52 cycles per minute, and pulse oxygen saturation at 93% in room air. Other vital signs were blood pressure of 102/68 mmHg, pulse 161 beats per minute, and a temperature of 39.7°C. A bilateral Basi thoracic pulmonary consolidation syndrome, a 3/6 heart murmur at the pulmonary focus, and Hackett's stage 2 splenomegaly were notable. A clean healed operative wound from the incised myositis was observed on the right forearm. The diagnosis was COVID-19 pneumonia, complicated by a severe anaemia.

Her continuous treatment for five days in a row included oxygen therapy of 3L/min via nasal prongs, blood transfusion of two units of isorhesus isogroup, packed red blood cells, local protocol COVID-19), hyperhydration with normal saline 0.9% at 40ml/kg/24h intravenously and plain water orally, paracetamol 1g intravenously every 6 hours, tramadol 100mg subcutaneously every 8 hours, enoxaparin 1mg/kg/12h subcutaneously. Clinical outcome between days 3 and 5 of admission was notable for peripheral oxygen desaturation at 80% in room air motivating the administration of oxygen through the "airRASME" CPAP mask at 10 litres of oxygen per minute with a clear improvement in saturation (100%) after 40 minutes. Another panel of blood tests showed moderate anaemia at 8g/dL, normocytic normochromic thrombocytosis at 501G/L, leucocytosis 16G/L and CRP: 36mg/L. On Day 15, she was in apparent good health, asymptomatic with satisfactory biological workups: leucocytosis at 11600 G/L, predominantly neutrophilic, moderate anaemia at 9.3 g /dL and platelets at 426 G/L, CRP: 24mg/L; normal prothrombin time, activated partial thromboplastin time, liver transaminases, serum electrolytes, and renal function test. The patient was discharged from the intensive care unit and transferred to the isolation area for asymptomatic COVID-19 patients. She was discharged on Rivaroxaban 10 mg daily, aspirin 100 mg daily, for the prevention of thromboembolic disease, vitamin supplementation to aid red blood cell production (folic acid 5mg daily and cobalamin 100mcg daily) and then Tanakan (Ginkgo biloba) 40mg twice daily to prevent cognitive disorders with regular follow-

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up by neurocognitive tests. We have combined an anticoagulant acting on activated factor X, which is Rivaroxaban, and an anti-platelet drug, which is aspirin, to guarantee better prevention of both veins and arteries and to control the inflammatory effect of COVID-19. There is a high risk for sickle cell patients with COVID 19 viral infection to have a stroke. At two weeks of follow-up, she had no complaint, a normal physical examination, and a negative repeat RT-PCR with nasopharyngeal swabbing for COVID-19. Thoracic CT scan without injection of contrast agent in a parenchymal window: (a) in axial section and (b) coronal reconstruction showing multiple peri-bronchial alveolar opacities, poorly delimited, confluent in appearance, hyperdensity in "frosted glass" of the lower regions; presented on figure 1.

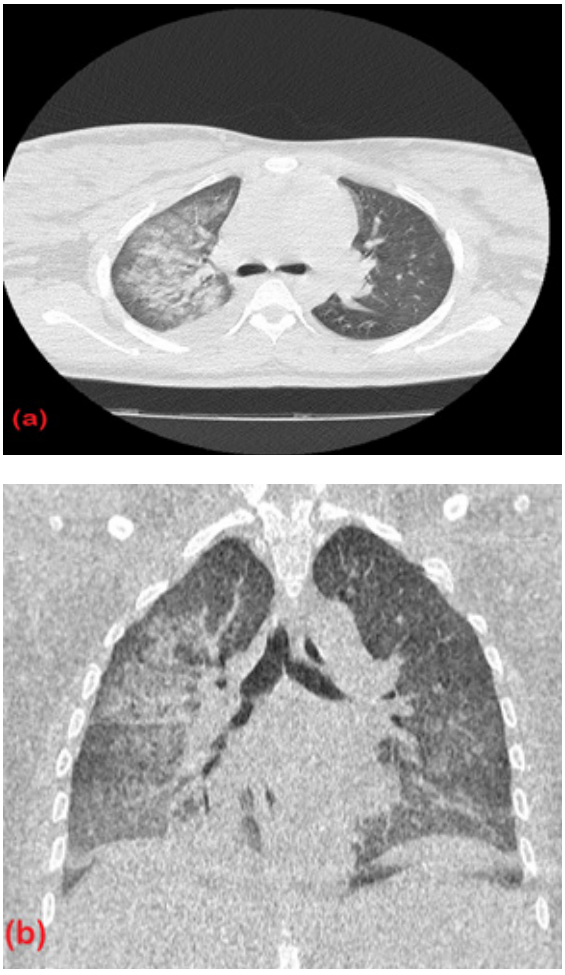

Figure 1: Thoracic CT scan without injection of contrast agent in a parenchymal window: (a) in axial section and (b) coronal reconstruction showing multiple peri-bronchial alveolar opacities, poorly delimited, confluent in appearance, hyperdensity in “frosted glass” of the lower regions.

Case 2

This was 42-years-old patient, who had initially consulted for headache, fever, and asthenia evolving for 24 hours. His clinical examination was notable for an ill-looking general state and an inflammatory syndrome with systemic response (temperature at 40°C, tachycardia at 120 beats per minute). The rest of the physical examination was unremarkable. A rapid diagnostic test for malaria was positive. He was hospitalised for severe malaria (hyperthermia, 40°C and marked prostration) and received treatment with intravenous artesunate, and paracetamol relayed 24 hours later during hospital discharge on an artemisinin-based combination therapy. The outcome on the fifth day after discharge was marked by the persistence of asthenia and the occurrence of anorexia, recrudescence of fever 38-39°C which motivated a second consultation in another health facility where a thick blood film showed 5466 trophozoites of falciparum parasites/mm3 and he was managed for poorly treated severe malaria with quinine infusion.

The occurrence of a dry cough without expectoration as well as breathlessness and a stab-like Basi thoracic pain on the second day of this new hospitalisation prompted the realisation of a thoracic CT scan which revealed bilateral pneumopathy lesions compatible with a COVID-19 attack estimated at 10 to 15% (Figure 2). A RT-PCR on the nasopharyngeal swab in search of COVID-19 carried out revealed the presence of SARS-COV-2 RNA. This led to the patient transfer through our referral health centre for management. On his admission, his general state was altered by asthenia, there were also signs of respiratory distress (dyspnoea at rest, respiratory frequency at 42 cycles per minute, and saturation at 91% in the room air), heart rate at 121 beats per minute, blood pressure at 110/60 mmHg. His conjunctivae were pale and the sclera icteric. In addition, low-grade fever (38.2°C), pulmonary condensation syndrome, 2/6th heart murmur at the mitral site, Hackett's stage 3 splenomegaly. The diagnosis of COVID-19 pneumopathy associated with malaria in a sickle cell patient was retained. As a history, he was homozygous SCD on folic acid, with an average of four vaso-occlusive crises per year and a usual haemoglobin level of 7g/dL. He has never been transfused since birth but had to receive erythropoietin twice during two episodes of severe anaemia at 5g/dL. He is monitored for his disease by a haematologist and systematically receives an annual check-up. A complete blood count showed hypochromic normocytic anaemia at 6.3g/dL, hyperleukocytosis at 18.9G/L, predominantly neutrophilic with monocytosis, and thrombocytosis at 875G/L. CRP was positive at 16.35mg/L, Gamma-GT 373.12 IU/L or 7.4 times higher normal lactate dehydrogenase (LDH) 1551 IU/L (normal 230-460). Aspartate aminotransaminase (ASAT) 240.07 IU/L or 5 times above normal, Alanine aminotransaminase (ALAT): 58.47 IU/L or 1.4 times above normal, serum urea/serum creatinine: 0.60 g/L/ 9.3mg/L, fasting blood glucose 0.78g/L, Alkaline aminophosphatase (PAL): 269 IU/L or 2.1 times above normal, D-dimers: 2434ng/mL (normal less than 500). Thoracic CT scan without contrast injection in the parenchymal window:

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(a) in the axial section and (b) reconstruction, showing multiple ground-glass opacities hyperdensity ranges of mixed topography, poorly defined, especially right apical and bilateral postero-basal; presented on figure 2. He was placed on oxygen (with nasal prongs at 2 litres per minute), and the local protocol of COVID-19 was initiated without hydroxychloroquine initially. The quinine infusion was continued until the fifth day and hydroxychloroquine was started afterwards and administered 5 days consecutively. The patient's outcome was good, and he was discharged from the hospital after 10 days of treatment to continue his isolation at home. There was also a clear improvement in the biological situation with a tendency to normalise the initially disrupted values. Haemoglobin level at 7.1g/dL, leucocytosis at 10.5G/L, thick drop of negative control, D-dimer at 569 ng/L, LDH at 551UI/L, Gamma-GT at 110 IU/L, SGOT(ASAT) 70.2UI, SGPT (ALAT): 28.4UI/L, PAL: 120 IU/L. RT-PCR with nasopharyngeal swabbing for COVID-19 carried out 21 days after his discharge from hospital noted the absence of SARS-COV-2 RNA.

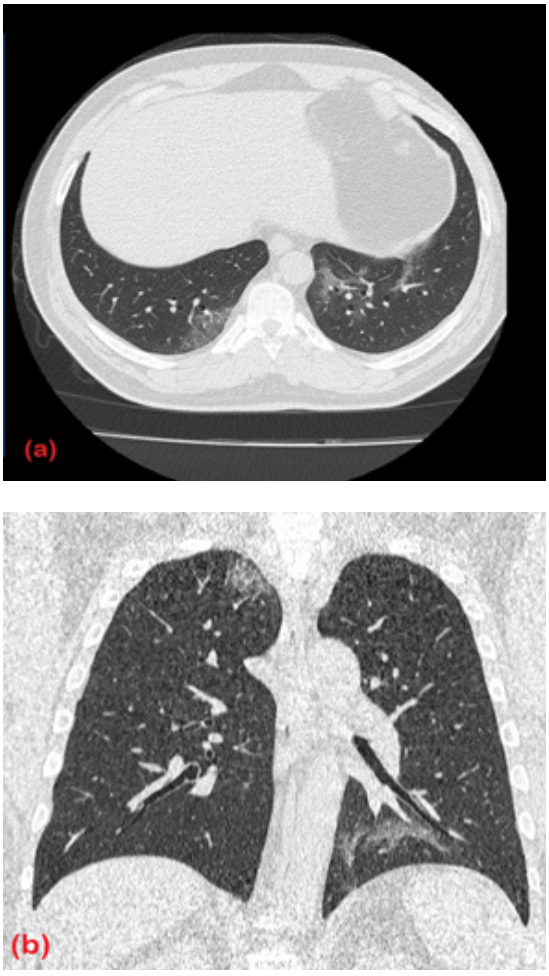

Figure 2: Thoracic CT scan without contrast injection in the parenchymal window: (a) in the axial section and (b) reconstruction, showing multiple ground-glass opacities hyperdensity ranges of mixed topography, poorly defined, especially right apical and bilateral postero-basal.

Discussion

We report two cases of sickle cell patients, infected with COVID-19. Both patients were of different gender, with different medical follow-up of SCD and presenting a favourable outcome, after adequate and meticulous medical management in our setting, while being aware of the vicious circle involving COVID-19 and sickle cell disease. The pathophysiological elements of COVID-19 that are commonly found in sickle cell disease such as inflammation, pulmonary involvement, thromboembolic events (the source of vascular obstructions) can generate a vicious circle but often leads to rapidly severe manifestations. The diagnostic difficulty lies in being able to differentiate between an acute chest syndrome or vaso-occlusive crisis in sickle cell patients, and a simple COVID-19 infection in sickle cell patients which, given the pathophysiology could also be the cause of a vaso-occlusive crisis. The treatments initiated to address these pathophysiological elements in both cases, could provide good responses to the severe manifestations.

The pulmonary damage was important in our patient, in the first case an acute respiratory distress syndrome and in the second case a picture of bilateral pneumopathy. On the one hand, this could be explained by the fact that in sickle cell patients, haemoglobin S induces polymerisation of haemoglobin in red blood cells, which leads to their sickle deformity in hypoxemic conditions [11]. This deformation, initially reversible in an oxygenated environment, becomes permanent and leads to intravascular haemolysis and vaso-occlusive phenomena [12]. Occlusion of the pulmonary capillaries leads to a cascade of local inflammation, intravascular coagulation, and thrombi formation with pulmonary micro-infarction, which leads to acute thoracic syndrome [13]. If the damage to the alveolar epithelium and vascular endothelium is sufficiently severe, capillary leakage with inflammation leads to a constituted acute respiratory distress syndrome [14]. Some authors have suggested that in addition to polymerisation of haemoglobin S in red blood cells, an adhesion phenomenon and a deficit in endogenous vasodilators such as nitric oxide (NO) could be incriminated [15, 16].

The biological inflammatory syndrome was encountered in both cases, indeed, as high levels of circulating cytokines have been reported in patients with severe COVID-19 (IL2, IL6, IL7, IL10, GSCF, IP10, MCP1, MIP1A, and TNFα) [17]. Several chemokines are also hyper-produced and may explain the inflammatory lung infiltration observed in infected patients, among them CXCL17 (capable of recruiting alveolar macrophages), CCL2 and CCL8 (associated with recruitment of neutrophils), CCL7 (recruiting monocytes) and CXCL9/CXCL16

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(recruiting T and NK lymphocytes) [18, 19].

In our case, the D-dimers were very high (1200 ng/mL in the first case and 2434 ng/mL in the second) compared to the study by Guan et al., who found an increase in D-dimers in 46.4% (260/560) of cases, and up to 59.6% (65/109) in the case of severe forms. This elevation seems to be predictive of mortality particularly when it exceeds one milligram per litre in the study by Zhou et al. (81% versus 24% between survivors and non-survivors) [20]. Nevertheless, it is important to note that a recent study found no significant difference in the rate of D-dimers between COVID-19 and non-COVID-19 pneumonia [21].

Some of the COVID-19 coagulopathies may be due to interactions between coagulation activation and inflammation during sepsis. This is the concept of thrombo-inflammation [22]. One of the main triggering factors is the secretion of proinflammatory cytokines (TNF-α, IL-1, and IL-6). This results in an activation of coagulation, mainly due to the release of tissue factors by mononuclear cells (favouring the generation of thrombin), as well as the activation of platelets and their interaction with the activated endothelium. In our case, we did not measure the proinflammatory cytokines, which limits the demonstration of the thrombo-inflammation concept. However, we did find a biological inflammatory syndrome. It should be noted that this interaction between IL-6 secretion and the pro-coagulant phenotype of patients suffering from CoV-2 SARS is notably described by an Italian study with a correlation between the fibrinogen level and that of IL-6 [23]. The activation of coagulation is spread by the combination of an inhibition of natural anticoagulant factors (antithrombin III, proteins C and S) and the suppression of fibrinolysis by the release of the plasminogen activator inhibitor type I (PAI-1). It should be noted that in our case, we were unable to measure antithrombin III, protein C and protein S. One of the main signalling pathways promoting thrombo-inflammation is the NF-B pathway. This signalling pathway induces the expression of genes favouring a pro-coagulant phenotype of the endothelium, stimulates the synthesis of PAI-1, and promotes the release of tissue factors and the release of pro-coagulant DNA via NETose from neutrophils [24].

Conclusion

Sickle cell disease patients, like other patients weakened by chronic pathologies, deserve special attention during the period of the COVID-19 pandemic. The comorbidities are usually less marked, or little observed in these patients and could present in their most severe forms. It would, therefore, be important for practitioners in charge of sickle cell disease patients in this context, to integrate an approach that does not aim to remain conventional and to look for all points of similarity that could be observed in their pathophysiology's and manifestations that have been elucidated to date. Primarily, inflammatory and thromboembolic events, followed by radiological findings such as multiple ground-glass opacities hyperdensity ranges of mixed topography, to detect and effectively manage this association when it occurs.

Acknowledgments: The authors would like to thank all staff of the intensive care unit for their dedication to the management of these patients during their admission to the intensive care unit. We would also like to thank the general practitioners of the emergency department of the Douala Gynaecological-Obstetric and Paediatric Hospital, who played an important role in organising the multidisciplinary management of the patients from the outset.

Conflict of interest: All authors of this piece of work declare having no conflict of interest in its writing up and publication.

Ethics approval: Ethical approval is not required at our institution to publish anonymous case report.

Consent: A Written informed consent was obtained from patient to publish this report in accordance with the journal's patient consent policy. They agree to their case being presented anonymously.

Author's contributions: Dominique Djomo Tamchom: Conceptualization, Data curation, Formal analysis, Funding acquisition, Investigation, Methodology, Project administration, Resources, Software, Supervision, Validation, Visualization, Writing - original draft, Writing - review & editing. Eric Bila Lamou: Formal analysis, Funding acquisition, Validation. Berinyuy Nyuydzefon: Formal analysis, Software, Visualization. Bertolt Brecht Kouam: Data curation, Software. Verla Siysi: Supervision, Validation, Visualization, Writing - original draft

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Citation as **J Sci Dis 2026, Vol 4 (1): 139 - 144**