OPEN ACCESS

Saudi Journal of Medicine
Abbreviated Key Title: Saudi J Med
ISSN 2518-3389 (Print) | ISSN 2518-3397 (Online)
Scholars Middle East Publishers, Dubai, United Arab Emirates
Journal homepage: https://saudijournals.com

Case Report

Children's Diabetic Ketoacidosis and COVID 19: Two Case Reports

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DOI: 10.36348/sjm.2022.v07i08.009 | **Received:** 19.07.2022 | **Accepted:** 22.08.2022 | **Published:** 26.08.2022

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Abstract

Introduction: The current global pandemic due to SARS-CoV-2 has resulted in a large literature on associated comorbidities, including diabetes. COVID 19 infection in a diabetic child can be complicated by severe ketoacidosis. The aim of this study is to determine the effect of COVID-19 infection on a diabetic child. ***Observations:*** The first case involved a 12-year-old Congolese child from a diabetic mother who was hospitalized for febrile respiratory distress and a consciousness disorder. Biological examinations showed the first diabetic ketoacidosis. Chest radiography showed radiological images suggestive of COVID-19 involvement, which was confirmed by a PCR and a thoracic CT. The evolution was marked by the appearance of a left pleurisy of medium abundance, which regressed after treatment. Second, a 16-month-old infant with no prior history was admitted to the hospital for febrile dehydration with impaired consciousness. Biological tests were consistent with diabetic ketoacidosis. Multiplex PCRs were performed showing infection with SARS-CoV-2 and other viruses (adenovirus, rhinovirus and enterovirus). Chest radiography was normal. The evolution was favorable under treatment with insulin therapy, rehydration, and antibiotic therapy. ***Conclusion:*** After discussing the role of decompensation or the triggering of diabetes in children with SARS-CoV-2 infection, the authors insist on the polymorphism of the clinical presentation and the need to perform a PCR in a diabetic child with ketoacidosis.

Keywords: COVID 19; type 1 diabetes; ketoacidosis, ACE 2.

INTRODUCTION

The new coronavirus, SARS-CoV-2, is at the origin of the current global pandemic.

Its association with diabetes has increased the risk of developing a severe form, including diabetic ketoacidosis, making therapeutic management difficult [1].

In this study, we report 2 pediatric cases of inaugural diabetic ketoacidosis following COVID-19 infection.

CASE PRESENTATION

1st Case: A 12-year-old child, from Congo, whose mother is diabetic, was admitted for respiratory distress and fever at 38°C.

On clinical examination:

The child had kussmaull dyspnea; Glasgow score (GCS) 14, tachycardia 124 bpm/min; blood pressure 11/6 cmhg; oxygen saturation 98% on room air.

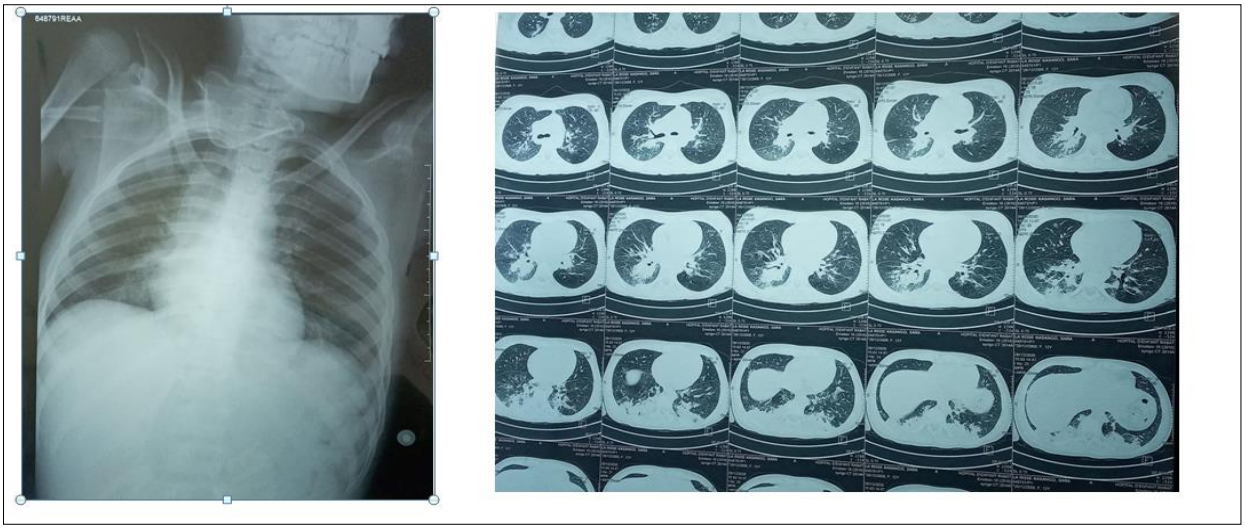
Capillary hyperglycemia at 4 g/l with acetoneuria +++ and glycosuria ++ on urine dipstick were found. Gasometry showed a metabolic acidosis with PH at 6.99 and HCO3-at 4.6.

Glycated hemoglobin was 18%, which is in favor of an inaugural diabetic ketoacidosis.

A COVID-19 PCR was performed on admission and the result was positive.

Viral and HIV serologies were negative.

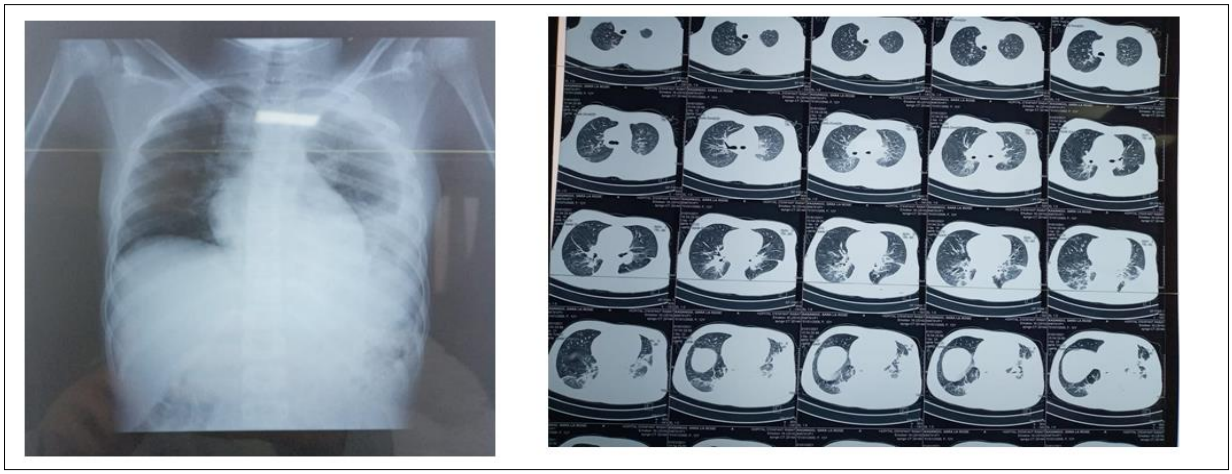
A chest X-ray showed a right paracardiac pneumopathy. It was completed by a thoracic CT scan, which showed an extensive involvement of 25 to 50% of the pulmonary parenchyma.



Figures 1 and 2 show an initial chest x-ray and CT scan

The child was put on chloroquine, azithromycin, insulin therapy, and rehydration with an adapted diet and regular monitoring of blood sugar levels.

On the sixth day of hospitalization, a medium-abundance left pleural effusion appeared which was followed by an increase in CRP to 242.9 mg/l.



Figures 3 and 4: Chest X-ray and CT scan showing left pleural effusion

Pleural puncture yielded an exudative citrine yellow fluid, predominantly lymphocytic. Direct examination and culture as well as the BK test were negative (Table 1).

The anatomopathological examination of the puncture fluid was normal.

Table 1: Cytobacteriological results of the puncture

Macroscopic appearance of pleural fluid	**Citrine yellow**
Cytological examination	
White blood cells	250 elements/mm3
Polynuclear cells	10%
Lymphocytes	90%
Red blood cells	1400 elements/mm3
yeasts	absent
Direct exam	Negative
culture	Absence of germ

The patient was put on intravenous protected amoxicillin for 4 weeks with a good evolution: negativity of the COVID-19 PCR as well as regression of the pleural effusion syndrome at the control radiography.

2nd Case: A 16-month-old female infant was admitted for fever, altered consciousness, and dehydration. On clinical examination, the infant was:

Dehydrated with a weight of 7 kg (-2DS); somnolent (GCS 11), febrile at 38.5°, tachycardia at 140 bpm/min; marbled, polypneic at 50 cycles/min;

A capillary hyperglycemia of 6.86 g/l .The urinary strip revealed +++ glucoseuria and +++ acetonuria. A gasometry was performed, showing a metabolic ketoacidosis with a pH of 7 and HCO3-at 5.Glycated hemoglobin was 11%, which is in favor of an inaugural diabetic ketoacidosis.

The blood ionogram and brain CT were unremarkable.

Infectious laboratory tests showed:

CRP was at 37 mg/l with procalcitonin at 41.2 ng/l. Biochemical and cytobacteriological examinations of cerebrospinal fluid as well as cytobacteriological examination of urine were negative. The chest X-ray was normal while COVID-19 PCR was positive and multiplex PCR showed viral infection with adenovirus, enterovirus, and rhinovirus.

The infant received:

vascular filling with 20 ml/kg of SS 9, intravenous insulin therapy at 0.05 IU/kg/h with rehydration, and triple antibiotic therapy with ceftriaxon, gentamicin, and metronidazol with capillary blood glucose monitoring.

The clinical evolution was good: the infant became apyretic, tonic, and reactive with negativity of the COVID-19 PCR on the 7th day.

DISCUSSION

Type 1 diabetes is due to insulin deficiency secondary to the progressive and irreversible destruction of the beta cells of Langerhans. It is most often of autoimmune origin.

Ketoacidosis is defined as hyperglycemia > 2 g/L, pH < 7.30 or alkaline reserve < 15 mmol/L, in the presence of ketonemia (capillary strip) > 3 mmol/L or ketonuria greater than two crosses.

Treatment for diabetic ketoacidosis is based on the principles of blood and fluid restoration, rehydration, correction of insulin insufficiency and hyperglycemia, treatment of acidosis and ketosis, correction of hydroelectrolytic disorders, management of the triggering factor [2, 3].

In our study, type 1 diabetes and SARS-CoV-2 infection were revealed by ketoacidosis, whereas the Congolese child's pleurisy was caused by a viral infection from SARS-CoV-2, which was aided by her diabetic condition, which is thought to have impaired her immune system [4, 5].

It is frequently reported that some viruses might cause autoimmune type 1 diabetes in genetic predispositions person. Serological evidence of infection and viral isolation in the pancreas has been described in a few cases of newly diagnosed diabetes [6, 7].

A severe form of COVID-19 is more likely to occur when SARS-CoV-2 infection is coupled with diabetes. It can be explained by several mechanisms:

The cytokine storm that causes apoptosis of Langerhans beta cells and, as a result, a lack of insulin production.

The presence of angiotensin converting enzyme2 (ACE2) in the endocrine pancreas suggests that SARS CoV 2 uses ACE2 as a receptor to enter the beta islets of Langerhans, which can damage the islets of Langerhans and lead to acute insulin dependency and diabetes.

Hence the interest in prevention by respecting barrier measures, especially in diabetic children, in order to avoid the appearance of serious complications [8, 9].

CONCLUSION

Although pulmonary involvement is common during SARS-CoV-2 infection, COVID-19 is characterized by its clinical diversity (endocrine, cardiovascular involvement, etc.), still the subject of much research and requiring multidisciplinary management [10].

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