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 kussmaul 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 acetonuria +++ 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.
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.

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

<table>
<thead>
<tr>
<th>Macroscopic appearance of pleural fluid</th>
<th>Citrine yellow</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cytological examination</td>
<td>250 elements/mm3</td>
</tr>
<tr>
<td>White blood cells</td>
<td>10%</td>
</tr>
<tr>
<td>Polynuclear cells</td>
<td>90%</td>
</tr>
<tr>
<td>Lymphocytes</td>
<td>1400 elements/mm3</td>
</tr>
<tr>
<td>Red blood cells</td>
<td>absent</td>
</tr>
<tr>
<td>yeasts</td>
<td></td>
</tr>
<tr>
<td>Direct exam</td>
<td>Negative</td>
</tr>
<tr>
<td>culture</td>
<td>Absence of germ</td>
</tr>
</tbody>
</table>
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 metabolic ketoacidosis with a pH of 7 and HCO3-

The infant received:
- vascular filling with 20 ml/kg of SS 9,
- intravenous insulin therapy at 0.05 IU/kg/h with rehydration, and 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 enzyme (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].

REFERENCES