



Diabetes Keto-acidosis in Children: A Report of Two Cases and Literature Review

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Authors' contributions

This work was carried out in collaboration among all authors. Author BGM designed the study, performed the statistical analysis, wrote the protocol and wrote the first draft of the manuscript. Authors ASB and OJA managed the analyses of the study. Author DNN managed the literature searches. All authors read and approved the final manuscript.

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Case Study

ABSTRACT

Aims: To present features of two cases of diabetes keto-acidosis observed in the Nouveau Village de Pédiatrie, Democratic Republic of Congo (DRC), between 2014 and 2018.

Cases Presentation: The first case was a male patient, 13 years-old, who arrived with fever, vomiting, polyuria, unconsciousness, respiratory distress and coma. His fasting plasma glucose was 570 mg/dl. Urea nitrogen 56,4 mg/dl; Creatinine 2,1 mg/dl. C reactive protein was 27 mg/l. The treatment of diabetes ketoacidosis (DKA) was based on insulin, fluids and antibiotics. Despite glycemic normalization, he died with cerebral edema and sepsis. The second case was a female child of 6 years, received with fever, polydipsia, asthenia, polyuria, a familial history of diabetes. Random plasma glucose was 500 mg/dl and C reactive protein 10 mg/l. Despite insulin and

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antibiotics, her clinical state worsened by a pyelonephritis and pulmonary edema and, probably tuberculosis. She also died.

Conclusion: Diabetes mellitus type 1 in children, complicated with DKA and sepsis, have worse prognosis. More children death would be avoidable by correct *global* treatment including insulin and hydro-electrolytic balance to prevent cerebral or pulmonary edema. Children with tuberculosis should realize routine screening for diabetes mellitus and inversely.

Keywords: Children; diabetes keto-acidosis; tuberculosis; mortality.

1. INTRODUCTION

Diabetes is one of the most common chronic medical disorder in children. Most children with diabetes have type 1. In low-income countries diabetes type 1 is insufficiently diagnosed and its mortality high, due to insufficient access to healthcare. Diabetic ketoacidosis (DKA) is the most frequent discovery syndrome. It has significant morbidity and mortality [1-4]. The diabetes burden is growing in all countries including Sub-Saharan Africa (SSA). Due to economic, demographic (population expansion, urban migration), epidemiological and nutrition transitions in SSA, the growing prevalence of diabetes appears to be related to obesogenic lifestyles, the declining physical activity, and dietary factors. The organization of diabetes care is poorly coordinated, leading to poor outcomes [5-7].

This study describes clinical and biological features of two children hospitalized for diabetes mellitus in the Nouveau Village de Pédiatrie between June 2014 and June 2018. The main purpose is to make physicians of DRC aware of this disease, and the challenging management of its complications.

2. PRESENTATION OF THE CASES

The first case was a male patient, 13 years-old, who arrived at hospital with fever, vomiting, polyuria. The axillary temperature was 39°C. He had Kussmaul's respiration type. Neurologic exam showed no focal sign. Glasgow's coma scale was 6. His BMI was 14,8 (2nd percentile, WHO BMI chart for age and sex). The fasting plasma glucose (Contour®, Bayer) was 570 mg/dl, urea nitrogen 56,4 mg/dl; Creatinine 2,1 mg/dl, hemoglobin (Hemocue® 301) 13 g/dl and C reactive protein (QuickReadGo® CRP, Orion) 27 mg/l. Thick blood smear and rapid diagnostic test for malaria (SD Bioline®) were negative.

Physicians diagnosed sepsis and DKA and gave alternatively infusions of lactate ringer or saline

isotonic, estimating quantity on the basis of daily water requirements and gradient between all water intake and loss. Hydro-electrolytic assessment was not available.

Rapid insulin was given (1,5 IU/kg/day) intravenously after each hour. He received also cefotaxime (Claforan®), associated to gentamycine. On day 2, C reactive protein was 26 mg/l and pediatricians replaced gentamycin by amikacin. Dietetic care was based on local food with low glycemic index in mush given via nasogastric tube thrice a day.

The same day the coma improved from grade 3 to grade 1 and plasmatic glucose lowered to 295 mg/dl. When it reached 139 mg/dl on day 2, the insulin therapy shifted to three doses of rapid insulin and 2 bolus of long-acting insulin. The blood glucose lowered to 93 mg/dl. C reactive protein raised up to 67 mg/dl on day 3. He also received intravenous artesunate at 2,4 mg/kg at hours zero, 12 and 24, then once daily. On day 4, blood glucose raised again (260 mg/dl) and the boy had abdominal distension. He died on day 5. Body temperature ranged from 37,1° to 39°C.

The second case was a girl of 6 years, with fever, polydipsia, headache and polyuria. She had 17 kg and a BMI of 12,7; 2.5th percentile of BMI for age and sex). There was a familial history of diabetes mellitus. The respiration was acidotic. She was agitated and comatose (Grade 1). The axillary temperature was 37°C and the random plasmatic glucose 500 mg/dl. Malarial exams were negative. The C reactive protein was 10, 76 and 36 mg/dl respectively on days 1, 3 and 6. Physicians diagnosed sepsis and DKA and prescribed 1,5 IU/kg/day of rapid insulin divided in equal doses given intravenously after every hour. She also received lactate ringer and saline infusions. Food of low glycemic index were given in mush via nasogastric tube. She received cefotaxime (Claforan®) and gentamycine. After the second CRP dosage, the antibiotherapy changed into Vancomycin (Sandoz) and

amikacin (Mylan). The axillary temperature lowered to 36°C on day 8 and plasmatic glucose lowered from 600 to 149 mg/dl. The insulin treatment was then revised: two thirds of the dose was rapid insulin given subcutaneously every 8 hours and the rest was long-acting insulin given at the same time as the first and third rapid insulin doses. Note that plasmatic glucose lowered difficultly and irregularly. The clinical state worsened with dyspnea, crackles and respiratory distress. HbAc level was 10,4%, blood pH 7. Urine dipstick revealed proteinuria, ketonuria, leucocyturia and hematuria and chest X-ray showed bilateral sparse opacities, leading to the diagnosis of pneumonia or pulmonary tuberculosis and pyelonephritis. The girl died two days later. The last dosage of blood glucose showed 122 mg/dl.

3. DISCUSSION

These two cases of diabetes mellitus DKA were diagnosed in underweight school-level children. Both had type 1 diabetes mellitus and cases definitions matched those of American Diabetes Association [8]. Both had serious sepsis co-morbidity that made control of blood glucose difficult. Despite glycemia improvement with insulin treatment, death occurred, probably due to cerebral edema for the first case and

pulmonary edema for the second. Ophthalmoscopy was not realized but clinical features looked like what many authors found. Cerebral edema is the most common cause of death in DKA. Cerebral edema may be exacerbated by factors related to both DKA presentation and therapy. Intravenous fluid boluses should be given cautiously [9,10]. The first case had sepsis and the girl pneumonia and urine tract infection.

Some studies found that cerebral edema, pulmonary edema and septic shock were predictors of mortality [10-12].

Patients came from high socio-economic level families. Richer patients and those living in urban area were reported by some studies [10,13]. One child had acute kidney injury (AKI), a risk factor increasing morbidity and mortality of children with type 1 DKA [14]. Death from diabetes in children and adolescents is potentially preventable through increased awareness of diabetes symptoms, genetically exposed children (with positive familial history), good nutrition practices (breastfeeding, avoidance of sugar beverages for example), earlier treatment and education related to diabetes, and adequate management of DKA [10,13,15,16,17].

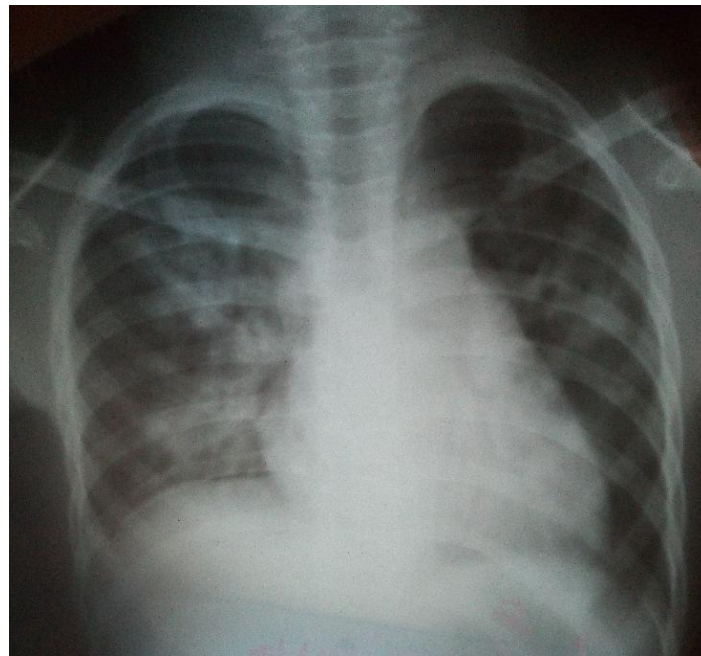


Image 1. Chest-X ray of the female case

Both children died despite availability of good quality insulin, antibiotics, fluids and nutritional assistance. This high mortality rate has been reported by many authors [3,7,9,10,11]. Despite appropriate use of insulin and fluids, and continuous clinical observation, the mortality rate has not improved, and has remained the same as that reported in the 1970s. Worldwide, infection is the most common precipitating cause for DKA, occurring in 30-50% of cases. Urinary tract infection and pneumonia account for the majority of infections [16]. Diabetes mellitus is also reported as a risk factor of tuberculosis [18,19].

This study had many limits. There was almost no biological data about electrolyte and acid-base balance at admission and during treatment because neither in The Nouveau Village de pédiatrie nor in local private laboratory such exams were available. So we could not precisely say the children died because of the sepsis, kidney injury, metabolic disorders due to DKA or insufficient hydro-electrolytic therapy. These limitations in the diagnostic work-up, treatment and outcome of the cases, compared to the international guidelines must help physicians, public and private laboratories, to address this issue. As one author said, in a lot of "countries in development", the burden of the infectious and parasitic pathology in pediatric environment made of the obesity and other non-infectious diseases a marginal preoccupation [20].

4. CONCLUSION

Diabetes mellitus type 1 in children, complicated with DKA and sepsis, have worse prognosis. More children death would be avoidable by correct *global* treatment including insulin and hydro-electrolytic balance. Underweight children and those with tuberculosis should realize routine screening for diabetes mellitus and inversely.

CONSENT

It is not applicable.

ETHICAL APPROVAL

This study had the agreement of research Authorities of the faculty of medicine and Pharmacy of the University of Kisangani.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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