A case of inaugural hyperosmolar diabetic coma reported from Côte d'Ivoire

DERE Kwadjo A. L.1,2, Kouame Andre B.2, KOFFI Konan G.3, MANHAN Kahissié2, TIAHOU G. George5

1,2M.D, Biologist, 3-4M.D, Interne in Medicine, Unit of Training and Research, Medical School Bouaké, Côte d'Ivoire

*Corresponding Author:
Email: dere.kwadjoanictluc@gmail.com

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Abstract
A forty eight years old man was brought into the hospital in a deeply comatose and dehydrated state. In Côte d’Ivoire, few works related to hyperosmolar hyperglycemic syndrome state have been carried out from different teams but all of them have been made in Abidjan. This case we reported, diagnosed in the centre of the Côte d’Ivoire. Patient referred from a private hospital in a fever context with fasting glycemia at 101.95 mmol/l. The onset of symptomatology began one week before his admission by general pains and headache. One day before admission, occurred a weak of conscious such as coma that motivated his admission in hospital. In order to get a good therapy, he has been evacuated in the medical school of Bouaké. At admission, we notified severe hyperglycemia 101.95 mmol/l associated with hyperkalemia at 2.75g/l, hypercreatininemia at 125.5mg/l, massive glycosuria and only just a weak presence of ketonuria. Related to electrolytes, exams showed: hypokalemia (3.30mEq/l) and normal value of natremia (143mEq/l). These values permitted us to calculate osmolality which was estimated to 440.41mosmol/l. On the basis of the laboratory results and clinical examination, the diagnosis of hyperglycemic and hyperosmolar syndrome was confirmed. Moreover, fever at 39°C could be the primus movens of this complication and as well as a sign of infectious complications. Probably originate from lung and inherent to this disease. Rapidly fatal evolution of this case don’t allow us to lead other investigations precisely X-ray; although infectious syndrome seemed to be the case of the triggering of the hyperosmolar hyperglycemic state (HHS).

Keywords: Hyperosmolar Hyperglycemic state, Inaugural, Coma, Diabetes.

Introduction
According to the recent previsions of World health Organization (WHO), the rate of this disease increased importantly during these latest years and outlooks for the forthcoming years are not better. This rate is more important in the North of America and Europe. In Africa, particularly in West Africa this disease is more and more confirmed. In our practice, if the diagnosis is easy it isn’t the case for the treatment and for the monitoring because of the complications those appeared certainly after many years of evolution such as diabetic ketoacidosis (DKA) or hyperosmolar hyperglycemic state (HHS). If the treatment of DKA is known and the issue is favorable commonly, it’s not the case of HHS. This complication is less frequent and occurs in particular circumstances.1

The hyperosmolar hyperglycemic state (HHS) is a syndrome characterized by severe hyperglycemia, hyperosmolality, and dehydration in the absence of ketoacidosis. The exact incidence of HHS is not known, but it is estimated to account for 1% of hospital admissions in patients with diabetes.2 Most cases of HHS are seen in elderly patients with type 2 diabetes; however, it has also been reported in children and young adults.3 The global mortality rate is estimated to be as high as 20%, which is about 10 times higher than the mortality in patients with diabetic ketoacidosis (DKA).4-6 The prognosis is determined by the severity of dehydration, presence of comorbidities, and advanced age.5,7,8

It is not easy to distinguish DKA and HHS from a patient’s history because of their similar characteristic features. HHS presents as severe hyperglycemia, hyperosmolality, with no evidence of severe ketosis or acidosis. Although HHS is a rare presentation of childhood diabetes, the importance of proper management cannot be emphasized enough due to the high mortality rate.9,10

In Côte d’Ivoire, few works related to hyperosmolar hyperglycemic syndrome state have been carried out from different teams but all of them have been made in Abidjan. Lokrou et al.11 have conducted many works concerning diabetes and its complications in Abidjan and their results concentrated in one town; don’t argue the trend in the country.

This case we reported, diagnosed in the centre of the Côte d’Ivoire, is an unexpected discovery of an important hyperglycemia surroundings 101.95 mmol/l occurred suddenly by a patient aged 48 years old without any past record known precisely type 2 diabetes. This uncommonly circumstance of HHS discovering, has aroused our interest and justified this work.

Case Description
Patient referred from a private hospital in a fever coma state (39°C) with fasting glycemia at 101.95 mmol/l. The onset of symptomatology began one week before his admission by general pains and headache growing into a fever context. One day before admission, occurred a weak of conscious such as coma.
that motivated his admission in hospital. In order to get a good therapy, he has been evacuated in the medical school of Bouaké.

A forty eight years old, man, without particular report knew admitted to medical emergency for monitoring and treatment of fever coma evolves for 24 hours. No personal history or family history of endocrine disease including mellitus diabetes was reported. At admission, the exam showed: temperature 40°c, respiratory rate: 28 beats/min, blood pressure: 90/60 mmHg. He looked cachectic and lethargic, his mental status was alert, sensation was intact, and motor strength was weak revealing a deficit difficult to appreciate because of the coma. We noted located convulsions on the face together with a weak reaction of pupils and signs of global dehydratation.

Focused on this presentation of fever coma out of trauma, biological investigations were done, within different parameters prescribed figured: glycemia, urea, creatininemia, electrolytes (Na+, K+, Cl-); blood numeration, quantitative buffy coat (QBC) and glycosuria and ketonuria in urine. At admission, we notified severe hyperglycemia 101.95 mmol/l associated with hyperuremia at 45.83mmol/l, hypercreatininemia at 1114.44µmol/l, massive glycosuria and only just a weak presence of ketonuria (Table 1). Related to electrolytes, exams showed: hypokaliemia (3.30mEq/l) and normal value of natremia (143mEq/l). These values permitted us to calculate osmolality which was estimated to 440.41mosm/l. On the basis of the laboratory results and clinical examination, the diagnosis of hyperglycemic and hyperosmolar syndrome was confirmed. Intensive intravenous fluid and regular insulin were administrated and laboratory explorations were performed every two hours in order to oversee the evolution by checking and modulating parameters changes (table 2). Thus, glycemia, natremia, kaliema, uremia, creatininemia, glycosuria and ketonuria have been regularly measured. Under insulinothrapy and hydratation, glycemia decreased from 101.95mmol/l to 83.25mmol/l two hours after onset, then 66.6mmol/l and lastly 54.39mmol/l at the sixth hour. Contrary to this trend, uremia and creatininemia increased gradually before stabilization. We noted reduction of kaliemia while natremia remained steady. Overall, we observed a HHS with decreasing value of osmolality value during the monitoring. Awaiting results to follow and perform the therapy, death unfortunately occurs in the same day surroundings 22H30min.

Table 1: Laboratory results of the case at 2 pm

| Parameters       | Results       | References |
|------------------|---------------|------------|
| **Blood**        |               |            |
| Glycemia         | 101.95mmol/l  | 3.60 – 6.10 mmol/l |
| HbA1C            | 15.5%         | < 5.6%     |
| Urea             | 45.83mmol/l   | 2.77 – 8.33 µmol/l |
| Creatininemia    | 1114.44 µmol/l| 53.28 – 106.56 µmol/l |
| Na+              | 143mEq/l      | 135 – 155 mEq/l |
| K+               | 3.30mEq/l     | 3.6 – 5.5 mEq/l |
| QBC              | négative      | -          |
| WBC              | 11.18, 10³/ul | 4.5 – 10.5, 10³/ul |
| Osmolality       | 440.41mOsm/l  | 292 – 308 mOsm/l |
| **Urine**        |               |            |
| Glycosuria       | ++++          | 0          |
| Urine ketones    | ±             | 0          |

Table 2: Evolution of value every 2 hours

| Parameters      | Value at 4pm  | Value at 6 pm  | Value at 8pm  |
|-----------------|---------------|----------------|---------------|
| Glycemia        | 83.62 mmol/l  | 66.6 mmol/l    | 54.39 mmol/l  |
| Uremia          | 46.66 mmol/l  | 43.33 mmol/l   | 44.16 mmol/l  |
| Creatininemia   | 1154.4 µmol/l | 1136.64 µmol/l | 1243 µmol/l   |
| Kaliemia        | 3.50 mEq/l    | 3.45 mEq/l     | 3.35 mEq/l    |
| Natremia        | 142 mEq/l     | 140 mEq/l      | 145 mEq/l     |
| Glycosuria      | ++++          | ++++           | ++++          |
| Urine ketones   | ±             | ±              | ±             |
| Osmolality      | 420.65 mOsm/l | 396.82 mOsm/l  | 395.24 mOsm/l |

Discussion

This case highlighted the lack of sanitary oversee in our practice. Several people don’t receive medical assistance and much of them get many financial problems that represent a burden for the population in our undeveloped countries. This unhappy situation is frequent but mostly evolves without to be diagnosed. This case, reported is a typical presentation of HHS as
describe in literature including serum osmolality above 400mosmol/l, serious neurological injuries and severity of dehydration. In addition to these perturbations, we perceived renal dysfunction markedly by renal failure probably inorganic might the consequence of dehydration state. The dissociation of urea and creatininemia justifies the mechanism of incoming renal failure. Indeed, urea increasingly is more important than creatininemia. Serum urea enhance quickly by the kidney injury have been reported to be associated with higher risks of case-fatality in previous studies, but most of them failed to differentiate specific types of patients’ cardiovascular diseases and infections in more detail. In addition, prognosis for the patients with HHS also tended to be worse for individuals with advanced age or severe comorbidity. Moreover, compared to that of DKA, the case-fatality rate of HHS was much higher varying from 12 to 46%.22

The treatment based on rehydration, insulinotherapy and intensive monitoring has been done but in spite of this therapy the outcome has been fatal to the patient the same day, six hours after his admission. HHS usually causes greater morbidity and mortality than DKA, depending on the severity of dehydration, hyper-osmolality, and patient age.23-25 Pinies et al26 reported 132 cases of HHS, according to their report, the mortality rate was 16.9%, and the incidence rate of complications associated with HHS were as follows: septic shock, 5.4%; cardiac failure, 2.3%; digestive bleeding, 1.5%; stroke, 1.5%; pulmonary embolism, 0.77%; and hypoglycemia due to insulin therapy, 0.77%. A few cases of HHS associated with rhabdomyolysis and acute kidney injury have been reported,27 but their frequency is unclear. In a study by Singhal et al, the incidence of acute renal failure in diabetic emergencies (diabetic ketoacidosis and hyperosmolar non-ketotic diabetic coma) was 25%.18 As dehydration is more severe in HHS than in diabetic ketoacidosis, the incidence of acute renal failure in HHS may be higher. HHS is reported to be a rare cause of rhabdomyolysis the electrogenic sodium pump is inhibited by hyperosmolar states, acidosis, hypernatremia, and potassium deficiency, as well as by decreases in intramuscular energy supply due to insulin deficiency.29

Conclusion
Making the diagnosis of mellitus diabetes during the state of complication is not so uncommon in our daily practice, however the conditions of a good therapy are not always guaranteed which lead in the most cases to fatal issue. In order to prevent and reduce death related to chronic diseases and particularly mellitus diabetes many actions have to be conduct so as to screen early this disease in our population and ensure a good treatment. Since the prognosis of hyperosmolar hyperglycemic syndrome is more serious than the other manifestations, we greatly recommend information and sensibilization attending to the population about this disease in order to make early diagnosis and enhance the treatment monitoring.
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