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Hypernatremia in hyperosmolar hyperglycemic syndrome

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Resumen

Mujer de 82 años con estado hiperosmolar hiperglucémico asociado con hipernatremia, manejada con solución hipotónica de cloruro de sodio a 0.2 %, intravenosa, con mejoría en su estado neurológico en un periodo corto, en el que disminuyó la glucemia, el sodio plasmático y, consecuentemente, la osmolaridad sérica. Señalamos la ventaja y utilidad de la solución de cloruro de sodio a 0.2 % en este síndrome y hacemos énfasis en su importancia como parte del tratamiento. Conforme nuestro conocimiento es el primer informe en la literatura médica donde se describe esta solución como terapia sin que exista daño neurológico.

Palabras clave

hipernatremia
estado hiperglucémico hiperosmolar
cloruro de sodio

Summary

We report the case of a 82 year-old woman with hyperosmolar hyperglycemic state (HHS) associated with hypernatremia, who was treated using 0.2 % sodium chloride in 5 % dextrose in water which resulted in the amelioration of the neurological symptoms in a short period of time, followed the decrease in serum glucose and the serum sodium, consequently the serum osmolality. We discuss the advantages and usefulness of 0.2 % sodium chloride in 5 % dextrose in water in this syndrome and emphasize its importance in the treatment. We reviewed the literature on HHS and we assume that this case reported was treated with hypotonic solution without subsequent neurological damage.

Key words

hypernatremia
hyperosmolar hyperglycemic state
sodium chloride

Introduction

Hyperosmolar hyperglycemia (HH) is an endocrine emergency associated with a mortality ranging between 10 to 50 %. It has been observed in all age groups,¹ though predominantly in older patients suffering from type 2 diabetes,^{2,3} and is diagnosed when serum glucose is over 600 mg/dL and serum osmolarity over 320 mosm/L. Common presenting manifestations include fatigue, polydipsia, polyuria, nausea and alteration of consciousness.⁴ The mainstay of therapy is vigorous intravenous fluid replacement with close monitoring of blood glucose.

While hypernatremia in diabetic HH can be treated with infusions of 0.45 % or 0.9 % normal saline,⁵ in which case alternate measures, such as intravenous glucose or sterile water,^{6,7} or delivery of water through a nasogastric tube are avail-

able.⁸ In elderly patient, there is a risk of cerebral edema with the administration of hypotonic solution but it is low, whereas under-treatment may be complicated by vascular thrombosis and is associated with a high mortality.⁹ We report the case of an adult patient who presented with HH and severe hypernatremia, and who was treated with infusions of 0.2 % sodium chloride in 5 % dextrose in water without suffering neurological consequence.

Case report

An 82-year-old woman, who had a 10-year history of type 2 diabetes, was admitted to the hospital for management of severe hyperglycemia. According to her family, she had omitted to take her treatment with glibenclamide and metformin

for approximately two weeks and, in the meantime, she had lost six kg of body weight. On the day before her hospital admission, the patient developed fever and consciousness alteration. Her medical history was otherwise negative. Upon initial presentation, the patient was unresponsive her respiration was shallow, and she exhibited signs of volume depletion, including diminished skin turgor and dry mucous membranes. Her blood pressure was 90/60 mmHg in the supine position, heart rate 100 bpm, and axillary temperature 37 °C. Examination of the heart and lungs, abdomen, and extremities was normal, and she was responsiveness to deep pain stimulus.

The screening blood tests performed upon her admission to the hospital were consistent with severe HH (table I). Other laboratory tests, including electrocardiogram, chest roentgen rays and computerized axial tomography scan of the brain contributed no further pertinent information.

The patient was admitted to the medical intensive care unit, where she received of isotonic saline infused during an hour, along with a 10 U loading dose of regular insulin, followed by a continuous insulin infusion, beginning at 0.1 U/kg/h, and subsequently adjusted according to the results of regular measurements of the serum glucose concentration. The water deficit was estimated in 4.5 L, and the calculated serum osmolality was 386 mosm/L.

The composition of fluids administered was based on the corrected serum sodium, by the following formula, which was result 173.5 mEq/l:

$$\text{Measured Na}^+ (\text{mEq/l}) + \frac{1.65 (\text{Glucose (mg/dL)} - 100)}{100} = 173.5 \text{ mEq/l (corrected Na}^+)$$

We treated the hyponatremia with an infusion of 0.2 % sodium chloride in 5 % dextrose in water (750 mL of 5 % glucose + 250 mL of 0.9 Na+Cl in water) + 20 mEq KCl, at a rate of up to 250 mL/h for the next 24 h. Within 12 h, the serum sodium was lowered since 162 to 147 mEq/L (table I), and the patient began to move spontaneously and responded to verbal stimuli. On the second hospital day, the serum sodium had returned to normal, the patient's neurological status continued to improve, and she began to eat. Treatment with glargine insulin was initiated, fluid administration remained unchanged, and 50 % of the calculated water deficit was replaced over the next 24 h. The patient was discharged from the hospital without further complication five days later.

Discussion

Despite published expert recommendations on the management of hyponatremia in HH syndrome, we found no report of intravenous hypotonic fluids administration, specifically of 0.2 % sodium chloride in 5 % dextrose in water (1/4 isotonic saline). While adverse effects have been observed during rapid correction of acute hyponatremia, particularly in children, the risks in adults presenting in a hyperglycemic state associated with changes in mental status are not well known.¹⁰ We used the formula described by Adroque et al. to calculate the volume of infusion and estimate the rate required to correct the severe hyponatremia presented by our patient.^{11,12} While the optimal correction rate in adults is not known, we judged that a 1 mmol/l/h decrease in Na+ would be appropriate, although it was a chronic,

Table I | Selected laboratory tests since the admission after to 24 hours

| | Admission | Time in hours after admission | | |
|------------------------------------|-----------|-------------------------------|--------|--------|
| | 0 | 8 | 12 | 24 |
| Hemoglobin (g/dL) | 14.00 | – | – | – |
| Hematocrit (%) | 43.00 | – | – | – |
| White blood cells (count/ μ L) | 6,800.00 | – | – | – |
| Serum glucose (mg/dL) | 600.00 | 410.00 | 280.00 | 130.00 |
| Blood urea nitrogen (mg/dL) | 61.20 | 55.10 | 50.00 | 28.00 |
| Serum creatinine (mg/dL) | 1.90 | 1.40 | 1.36 | 1.31 |
| Serum sodium (mmol/L) | 165.00 | 147.00 | 140.00 | 140.00 |
| Corrected serum sodium | 173.50 | 152.00 | 143.00 | 140.00 |
| Serum potassium (mmol/L) | 3.40 | 3.30 | 3.10 | 3.20 |
| Plasma osmolality (mosmol/L) | 386.00 | 336.00 | 313.00 | 297.00 |
| Plasma osmolality (mosmol/kg) | 364.00 | 317.00 | 295.00 | 287.00 |
| Blood gases | – | – | – | – |
| pH | 7.40 | – | – | – |
| PaO ₂ (mm Hg) | 93.00 | – | – | – |
| PaCO ₂ (mm Hg) | 45.00 | – | – | – |
| Bicarbonate (mEq/L) | 28.00 | – | – | – |

rather than an acute hyponatremia. Close monitoring of the patient's clinical status and of the laboratory tests results the key in guiding the administration of fluids, particularly with regard to the "paradoxical" infusion of glucose in presence of pre-existing hyperglycemia. As we were monitoring and adjusting the infusion of insulin according to the blood glucose measurements, the patient regained consciousness within 12 h. It seems, therefore, highly unlikely that the rapid decrease in blood glucose and sodium concentration had caused no brain edema. While, in adults, the risk of cerebral edema is known to be low, the consequences of under-treatment, on the other hand are serious, and include vascular thrombosis and increased risk of death.

Another advantage of our treatment strategy was the absence of need to vary the administration of fluids, as recommended by most guidelines. Instead, when the serum glucose falls below 300 mg/dL, 5 % dextrose with 0.45 % sodium chloride infusion can be substituted.¹³ when our patient was able to eat, she was placed on her previous treatment regimen and, the rate of fluid infusion was regulated according to her hydration status.

Conclusions

An expeditious fluid replacement, perhaps including isotonic fluids or plasma expanders, is the first and most important step to take in the management of HH syndrome. In presence of mild hypotension and high serum sodium, an infusion of 0.2 % sodium chloride solution in 5 % dextrose water should be administered, at a rate adjusted according to the degree of dehydration. The proper rate of correction of hyponatremia must be calculated when the serum sodium has been corrected for the blood glucose concentration.¹⁴ The rate of sodium correction should not exceed 0.7 mmol/L/h, or approximately 10 % of the corrected per day.¹⁵

This case report indicated that it is possible to treat hyponatremia in HH syndrome with intravenous hypotonic solutions infused at a rate of correction faster than usually practiced, without the development of neurologic complications.

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