

Case Report

Hyperglycaemic Hyperosmolar Non Ketotic Coma

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Abstract

A case report discussing the management of a patient with hyperosmolar hyperglycaemic state. This is intended to raise awareness about this complication of Type 2 diabetes and the factors that lead to it. HSS is mostly caused by severe dehydration and sepsis. Elderly patients often do not drink enough and infection is commonly seen in patients living in a hospitalised environment

Key Words

Dehydration, Sepsis, Hyperglycaemia, Hyperosmolar, Altered mental state, Ketones

Overview

This case discusses a 75-year-old diabetic patient who suffered hyperosmolar hyperglycaemic state (HSS) previously known as hyperglycaemic hyperosmolar non-ketotic coma (HONK).

Introduction

HSS is a syndrome characterised by hyperosmolarity, hyperglycaemia and dehydration in the absence of ketoacidosis. Most cases are seen in elderly with type 2 diabetes. It is estimated to account for less than 1% of hospital admissions in patient with diabetes, (*Francisco J. Pasquel and Guillermo E. Umpierrez 2014*).

Treatment of HSS include treating the underlying cause, replacing fluid loss and normalising blood glucose. It also aims to prevent complications such as arterial/ venous thrombosis and foot ulcers.

Case Presentation

History: Mrs D.V. is a 75 year old female, residing in a long term care facility. A proper history could not be obtained as she was a known case of dementia. Apart from dementia the patient also suffered from chronic heart failure, cerebral-vascular disease, type 2 diabetes mellitus, chronic kidney disease and hypertension. Nurses and health care workers noted her to be more lethargic and less responsive than usual.

Examination: No abnormalities were detected apart from an altered mental state. The patient was not in respiratory distress, parameters were stable and there was no obvious source of infection.

Investigations: Initial bloods showed that the patient had an acute kidney injury with rising urea and creatinine (see table) in addition to a septic picture with high white cell count and raised C reactive protein (see table). Mrs. D.V. was also noted to have increased capillary blood glucose.

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Table 1: Blood Results and Calculated Osmolality

	Normal Values	Day 1	Day 2	Day 3	Day 3	Day 4	Day 5	Day 6	Day 7	Day 8
WCC	4.3-11.4	17.99	14.56	17.29		12.55	9.96	8.83	8.31	7.59
CRP	0-5	49.7	102.5	253.5		250.7	236.5	225.8	122	46.9
UREA	1.7-8.3	42.6	41.2	43.6	37.3	32.1	24	21.5	11.4	3.3
CREAT	44-80	283	283	282	258	239	200	179	130	91
Na	135-145	171	172	162	153	154	146	148	138	140
K	3.5-5.1		4.54		3.41	3.79			3.98	4.36
Calc. Serum Osmolality	275-295	380								292

Management: She was started on intravenous (IV) fluids; 5% dextrose as the patient had hypernatraemia. (see table). Mrs. D.V was started on broad spectrum antibiotics (co-amoxiclav) as the source of infection was unknown. In view of the acute kidney injury, a catheter was also inserted to monitor urinary output. A urine sample was sent for culture but no organisms were cultivated. Ketones were not present in the urine.

The patient remained in a poor general condition and her inflammatory markers continued to rise in spite of the antibiotics. A Chest X ray was performed and showed congested lung fields but no consolidation. Therefore the source of infection was not yet found.

The addition of insulin to the dextrose infusion was not enough to control capillary blood glucose and so the patient was started on an intravenous insulin pump.

In spite all of this, Mrs D.V. continued to deteriorate. Antibiotics were switched from Co-amoxiclav to Tazobactam/Piperacillin. An ECG was performed which showed a 1st degree heart block. Blood tests showed further deterioration in renal function and increase in inflammatory markers. Blood cultures remained negative.

A differential diagnosis of HHS was made and patient was started on IV fluids twelve-hourly, as per NHS guidelines. Despite treatment with fluids, patient deteriorated. She was responsive only to pain the following day with a blood pressure of

90/70 mmHg and a pulse of 140 b/m. A bolus of 500 ml of intravenous fluids was given with minimal improvement. Relatives were contacted and informed about the poor prognosis. She was deemed not fit for CPR and an AOS (administration of sacraments) was done. Patient was started on oxygen and kept comfortable. IV fluids were increased to eight- hourly.

A microbiologist was contacted and informed about the case. He advised that the patient should be started on Meropenem. Repeated renal function tests showed low Potassium (due to the continuous infusion of Insulin), hence the patient was started on intravenous potassium supplement.

After increasing the fluids and using Meropenem the patient started to improve slowly. Parameters remained stable and blood results slowly improved back to normal. Capillary blood glucose was controlled and the patient could be weaned off the insulin pump. She was reviewed by the speech and language pathologist who started her on oral trials. When the patient started eating and drinking, IV fluids were stopped and catheter was removed.

Discussion

Hyperglycaemic Hyperosmolar State is a complication brought on by glucose levels more than 33mmol/L in patients with type 2 diabetes. Most commonly it occurs due to reduced fluid intake and infection is the most common preceding

illness.

According to NHS guidelines, diagnostic features include:

- high osmolality, often 320 mosmol/kg or more
- high blood glucose, usually 30 mmol/L or more
- severely dehydrated and unwell.

In HHS there is usually no significant ketosis/ketonaemia (less than 3 mmol/L), though a mild acidosis (pH greater than 7.3, bicarbonate greater than 15 mmol/L) may accompany the pre-renal failure.

Immediate management includes fluids usually 0.9% sodium chloride and finding the underlying cause for HSS. Underlying causes could be sepsis, or vascular events. Investigations such as blood glucose, osmolality, full blood count, chest X ray and ECG should be done. Control of blood glucose with insulin is of outmost importance however one should also avoid hypoglycaemia. Therefore, when blood glucose falls below 14 mmol/L 5% or 10% glucose should be commenced with the sodium chloride solution. Assessment for complications should be done throughout the management.

Mrs DV was severely dehydrated. One factor that may have led to this is the reduction in ability to drink water. As confirmed by the raised inflammatory markers there was also a source of infection. These 2 lead to the hyperosmolar hyperglycaemic state of the patient. Continuous follow up and appropriate management with fluids and antibiotics were critical for the wellbeing of the patient. Although the source of infection could not be elicited, the use of empirical antibiotics followed by the microbiology guided use of specific ones showed immediate results and was lifesaving to such a patient with frailty.

References

1. Hyperosmolar Hyperglycemic State: A Historic Review of the Clinical Presentation, Diagnosis, and Treatment; Francisco J. Pasquel and Guillermo E. Umpierrez; 2014
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