

Letters to the Editor

Vigilant Monitoring Is Essential in Unwell Diabetic Patients After the Initiation of Treatment

In the April 2014 edition, Young et al reported a case of Hyperosmolar Hyperglycaemia Syndrome (HHS) in a 13-year-old obese boy with life-threatening complications.¹ There are further learning points from this case in addition to the recognition and management of this rare diagnosis discussed by the authors.

The patient was initially treated as a case of diabetic ketoacidosis (DKA) and after the initiation of intravenous (i.v.) fluid and insulin therapy, his blood glucose levels dropped rapidly from 105.6 to 64.7 mmol/L within the first 6 hours. A number of national / international DKA protocols have emphasised the importance of ongoing monitoring, such as capillary blood glucose levels hourly, and repeat laboratory glucose, electrolytes and blood gas every 2 hours after the start of resuscitation alongside strict fluid management.^{2,3} It was unclear to readers whether these investigations were performed and, if the management plan was re-evaluated accordingly during this period. Early and gradual actions taken in response to biochemical changes and the patient's fluid status may have prevented some of the morbidities, resulting from large osmotic shifts, due to the large reduction in plasma glucose levels and aggressive fluid resuscitation later in the patient.

The overlapping features between DKA and HHS can lead to great diagnostic challenges. Patients with HHS, who are severely dehydrated, may have mild or moderate acidosis. On the contrary, patients with type 1 diabetes may also show features of HHS if high carbohydrate containing drinks have been consumed to quench thirst and replace urinary losses prior to the diagnosis.³ The only early indication of possible HHS instead of DKA in this patient was the presence of relatively mild ketosis with only 1+ ketones in the patient's urine at the time he was catheterised. Further ketone testing throughout the course of his treatment was not stated in the report. Urine ketones lag behind the clinical status and persistent low level of ketosis relative to the degree of acidosis may have allowed an earlier diagnosis of HHS. The authors may like to consider the use of

capillary ketones monitoring, if not already implemented in their unit, for the management of future unwell diabetic patients. This device provides realtime and hence more accurate information at the bedside to facilitate timely decision making in the patients' management.⁴

HHS cannot be assumed in all obese patients presenting with a DKA-like picture as it is not uncommon to encounter type 1 diabetic patients with co-existing obesity. Although the authors stated it has been suggested that insulin may be harmful to patients with HHS,⁵ when faced with diagnostic uncertainty, it is acceptable to initiate insulin therapy. However, this must be followed by vigilant glucose and biochemical monitoring with appropriate dosage adjustments.

In summary, this case report has not only raised the awareness of HHS in the paediatric population, but also highlighted the importance of careful ongoing biochemical and fluid monitoring, as well as treatment re-evaluation in diabetic patients who are started on i.v. fluids and insulin for presumed DKA.

References

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