Bittersweet
Lessons in the Management of Severe DKA in Children

Dr E Auer, Dr K Berry, Dr V Stanley, Dr K Sykes, Dr L Van Boxel

University Hospital Southampton NHS
NHS Foundation Trust

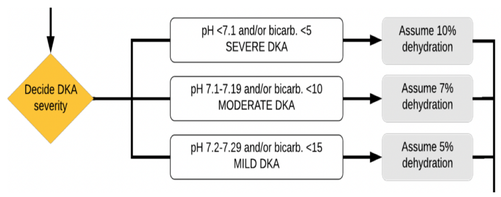
Introduction

Diabetic Ketoacidosis (DKA) is a life-threatening condition characterised by hyperglycaemia, ketoacidosis and dehydration. Symptoms of Type 1 diabetes are often missed in young children, thus DKA is the presenting feature in up to 25% of under 5s (1). Management of DKA in children is different to that in adults and can be confounded by difficult IV access and unfamiliarity with paediatric guidelines and drug dosing. Complications such as cerebral oedema and electrolyte disturbance are more common in smaller children (2) and make ongoing management more complex. We discuss the case of a 5-year-old with DKA to illustrate these issues.

The Case

A 5 year-old child presented with severe DKA as her first presentation of T1DM. She had experienced general malaise, abdominal pain and vomiting and was now unusually drowsy. On admission, a blood gas showed a pH of 6.8, glucose 19.1, ketones 5.7, HCO3- 6 and K+ 3.5mmol/L consistent with severe DKA. Her GCS was 11. She was managed using the 2020 British Society of Paediatric Endocrine and Diabetes (BSPED) DKA guideline (3).

Fig 1 BSPED 2020 DKA guideline



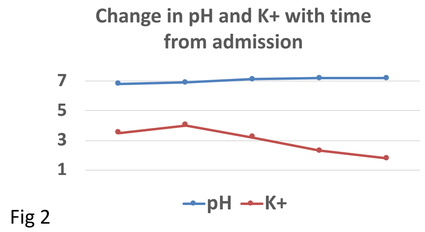


Fig 2

She developed signs of raised ICP and received hypertonic saline, following which her GCS improved. Her acidosis gradually improved, but her K+ fell (fig.2) despite supplementation. That evening she became bradycardic and was found to have a K+ of 1.8mmol/L. She developed irritable arrhythmias (fig.4) culminating in ventricular fibrillation (VF).

Fig 3: Hypokaleamic ECG. Life in The Fast Lane.

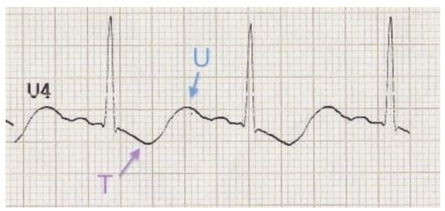
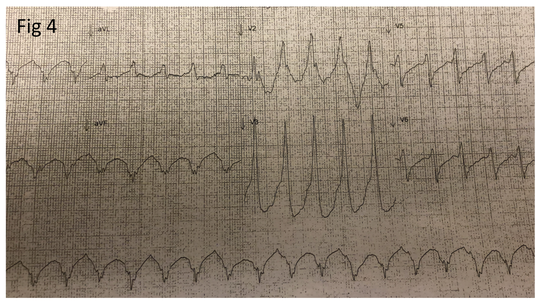


Fig 4



DC shock at 100J was administered following which she was asystolic. APLS guidelines were followed, and she received K+ 0.5mmol/kg peripherally. ROSC occurred 8-9 minutes after the initial VF. Central and arterial lines were sited, and central potassium replacement was given. The insulin infusion was paused. She was retrieved to the regional PICU where she made a full metabolic and neurological recovery. She was repatriated 3 days after PICU admission and is now coping well at home with her new diagnosis of diabetes.

References

1. Diabetes UK position statement 2015
2. Paediatric diabetic ketoacidosis. Steel s, Tibby s. Cont Edu Anaes Crit care & Pain. 2009;9 (6):194-199
3. BSPED guideline 2020 https://www.sort.nhs.uk/Media/Guidelines/BSPED-DKA-guideline-2020-update.pdf
4. Agarwal HS. Subclinical cerebral edema in diabetic ketoacidosis in children. *Clin Case Rep*. 2018;7(2):264-267.
5. Davis SM, Maddux AB, Alonso GT, Okada CR, Mourani PM, Maahs DM. Profound hypokalemia associated with severe diabetic ketoacidosis. *Pediatr Diabetes*. 2016;17(1):61-65. do

Childhood DKA	Adult DKA
Diagnosis of diabetes often delayed due to vague symptoms	Classical triad of polydipsia, polyuria and weight loss
DKA likely to be more severe and more likely to be 1st presentation of T1DM	Symptoms of DKA usually recognized earlier especially if known diabetic
Electrolyte imbalance less likely to be prevented: difficult phlebotomy reduces monitoring, lower body stores and more difficult to calculate replacement	Emphasis on risk of hypokalemia, monitoring and aggressive K+ replacement
Cerebral oedema commonest cause of death	Cerebral oedema rare, other comorbidities e.g. sepsis more likely
Dehydration corrected over 48-72 hours, unless shocked, % dehydration assumed based on initial pH	Generous fluid approach

Discussion

This child was admitted with severe DKA and cerebral oedema and developed severe hypokalaemia, despite moderate K+ replacement, suffering a hypokaleamic cardiac arrest. On discussion with her parents, she had a long history of fatigue and is a 'different more energetic person' after treatment. It is likely that she developed metabolic disturbance and DKA over a long period, predisposing her to the complications she suffered and demonstrating the difficulty in diagnosis of T1DM in small children. Subclinical cerebral oedema is a relatively common and easily missed complication of paediatric DKA (4). Clinically apparent cerebral oedema affects 1% of admissions but is the most common cause of morbidity and mortality in childhood DKA. Hypokalaemia develops during resuscitation, in all age groups, but is rarely as pronounced, and not as common a focus in paediatric guidelines as it is in adults. Aggressive K+ replacement in children may be unfamiliar to clinicians outside PICU and it is often inadequately replaced. This can result in severe hypokalaemia and the need for central access, anaesthesia and PICU transfer, which is traumatic for the family and costly to the NHS. The 2020 BSPED guidelines provide clear guidance and a calculator to assist in management of DKA but perhaps do not go far enough with regards to hypokalaemia. All children with DKA require paediatric involvement and HDU care, but the need for PICU and interhospital transfer might be avoided by seeking early advice from practitioners familiar with safe methods of aggressive electrolyte replacement.

Tips	
General	• Isotonic fluids e.g. plasmalyte reduce risk of hyperchloremic acidosis • Start insulin at least 1 hour after rehydration begun: 0.05-0.1u/kg/hr • Do not bolus insulin: increases risk of hypoglycaemia/kalaemia (5) • Avoid bicarbonate: increases risk of cerebral oedema and hypokalaemia (5) • Ensure paeds nurses check and prepare drugs: familiarity prevents drug errors
Hypokalaemia	• Risk factors: lower age and weight, low K+ at presentation, longer duration of acidosis • 40mmol/L K+ in all fluids from outset unless anuric/hyperkalaemic • NGT and early replacement of enteral K+ • If K+ low (<2.6) delay/pause insulin and consider need for central access • Central access is possible awake e.g. Leadercath in femoral, basilic or ext.jugular vein. • K+ 0.5mmol/kg can be diluted in 20mls saline and given via a large peripheral cannula if K <2mmol/L and the patient is dysrhythmic • Ensure continuous ECG monitoring