Aneurin Bevan Health Board

Guidelines for Diabetic Ketoacidosis in Adult Patients (over 18 years)

There is a separate All Wales Pathway for paediatric and young adult patients

N.B. Staff should be discouraged from printing this document. This is to avoid the risk of out of date printed versions of the document. The Intranet should be referred to for the current version of the document.

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Contents:

1 Summary

The joint British Diabetes Societies In Patient Care Group represent the Association of British Clinical Diabetologists, British Society for Endocrinology and Diabetes and association of Children's Diabetes Clinicians; Diabetes Inpatient Specialist Nurse (DISN) Group, Diabetes UK; NHS Diabetes (England); Northern Irish Diabetologists; Society of Acute Medicine and the Welsh Endocrine and Diabetes Society. In March 2010 the joint British Societies Inpatient Care Group developed and approved a management guideline for diabetic ketoacidosis (DKA) in adult patients. The guidelines were based on the review of current evidence where possible and accumulated professional experience. The guidelines that follow are largely based on that policy document. The aim is to treat DKA in keeping with the pathophysiological process allowing more effective management and faster resolution of ketosis.

1.1 Scope of policy

All adult (> 18 years) admitted with diabetic ketoacidosis.

The JBS guidelines can be accessed on:
http://www.diabetes.nhs.uk/publications_and_resources/reports_and_guidance/

1.2 Essential Implementation Criteria

When this new guideline is implemented it will need to occur with education and training of the staff admitting and caring for patients with DKA. This will be clinical teams in A&E, EAU, ITU and admitting wards. A&E staff are already familiar with the all Wales paediatric guidelines which are similar in principle.

This policy has a bedside chart containing all essential information and monitoring of the treatment of patients with DKA. Remember these are guidelines and should not override the decisions of an experienced clinician.

2 Aims

To achieve safe, effective and consistent management of DKA.

3 Background

DKA consists of the triad of ketonaemia, hyperglycaemia and acidaemia. Now bedside testing for 3-beta hydroxybutyrate, the predominant ketone body in DKA is available in ABHB. This allows treatment decisions to be made on the principle of suppression of ketogenesis rather than the surrogate marker of blood glucose levels. DKA remains a significant problem and estimates of prevalence vary from 4.6 to 8 episodes per 1000 patients with diabetes. On average there are 2 admissions per month (NHH data). Mortality rates have

now fallen, the most recent data suggests 0.67%. improved patient education with increased blood glucose and ketone monitoring has lead to partial treatment of DKA prior to admission so blood glucose levels may be lower at presentation.

4 Definition of DKA

- **Ketonaemia > 3mmol/l or more (or significant ketonuria > 2+)**
- **Blood glucose > 11 mmol/l or known diabetes mellitus**
- **Bicarbonate , 15 mmol/l and/or venous pH less than 7.3**

5 Pathophysiology

Absolute or relative insulin deficiency accompanied by and increase in counter regulatory hormones (glucagon, cortisol, Growth Hormone, adrenaline) enhance hepatic gluconeogenesis and glycogenolysis causing hyperglycaemia. Enhanced liplolysis increase serum free fatty acids which are metabolised as an alternative energy substrate leading to ketogenesis. Ketone bodies are the 3-beta-hydroxy butyrate (detected in blood ketones not urine ketones) and acetoacetate.

6 Clinical presentation

- Thirst
- Dehydration
- Vomiting
- Unwell
- Raised blood glucose
- Abdominal pain
- Dyspnoea
- Decreased conscious level

7 Causes

- New diagnosis of diabetes
- Omission of insulin
- Relative insulin deficiency (self management)
- Any acute illness/infection
- Increased risk in pregnancy

8 Recommended changes in management compared to existing practice

Treatment markers:

- Measurement of blood ketones not urinary ketones

- Venous (not arterial pH)
- Bicarbonate

Monitoring:

- Blood ketone and glucose meters at the bedside
- Venous pH and electrolytes on blood gas analyser

Therapy:

- Replace " sliding scale" insulin with a weight based fixed dose insulin infusion (more physiological, patients more likely to be insulin resistant)
- Continuation of long acting insulin analogues (insulin glargine= lantus, insulin detemir= levemir) and basal human insulins (Insulatard, Humulin-I). Prescribe on the insulin chart. (avoids rebound hyperglycaemia when iv insulin stopped)
- Cautious fluid replacement in young (small) adults (cerebral oedema in children and young adults)
- Normal saline as the fluid of choice for rehydration (or Hartmann's)

9 Initial investigations & assessment of severity

1. Clinical assessment and history

2. Bedside capillary blood glucose and blood ketones.

3. Obtain venous access. Send venous blood in a heparinised ABG syringe for venous pH, bicarbonate and electrolytes mark as URGENT. Send additional laboratory samples for FBC, UECr, blood & urine cultures. Troponin only if indicated.

4. ECG (look for changes of hyperkalaemia or acute ischaemia). Request CXR.

5. Assess severity

One or more of the following may indicate severe DKA and admission to level 2/HDU environment should be considered:

- **Blood ketones > 6 mmol/l**
- **Bicarbonate < 5 mmol/l**
- **Venous/arterial pH < 7.1**
- **GCS < 12**
- **O2 saturations < 92%**
- **Systolic BP < 90 mmHg**
- **Anion gap > 16** [= (Na + K) - (Cl+HCO3)]

10 Hour 1: Immediate management on diagnosis

ABC

Commence iv 0.9% sodium chloride via large bore cannula (example below but tailor to patient's requirements)

Commence a fixed rate IVII (intravenous insulin infusion) after fluid started. This can be via same line as the insulin infusion but ONLY if a Y connector with a one way anti syphon valve is used & large bore cannula placed

Monitor hourly blood glucose and ketones, and at least 2 hourly serum potassium for the first 6 hours

Clinical and biochemical assessment of the patient. Involve critical care early if concerned.

Inform diabetes team

Use ABHB bedside chart for monitoring and management summary

11 Fluid management

Fluid	Volume
0.9% sodium chloride 1L	1000 ml over 1st hour
0.9% sodium chloride 1L with KCl	1000ml over next 2 hours
0.9% sodium chloride 1L with KCl	1000ml over next 2hours
0.9% sodium chloride 1L with KCl	1000ml over next 4 hours
0.9% sodium chloride 1L with KCl	1000ml over next 4 hours
0.9% sodium chloride 1L with KCl	1000ml over next 6 hours

Fluid replacement is a guide only. Exercise caution in young adults, elderly, heart or kidney failure and pregnancy. Consider HDU input in these situations. Reassess fluid status regularly.

11.1 Potassium replacement

Potassium level in first 24 hours (mmol/l)	Potassium replacement in mmol/L of infusion solution
>5.5	Nil
3.5-5.5	40 mmol/L
<3.5	Senior review additional potassium needed

12 Insulin infusion

Commence fixed rate insulin infusion (IVII)

Prescribe "*50 units of actrapid insulin made up to 50 mls in 0.9% sodium Chloride*"
(write as above on the once only prescription on front of the insulin chart)

Infuse at a fixed rate of 0.1 unit/Kg/Hr (e.g. 7 mls/hr = 7 units/Hr if weight is 70 Kg). Weigh/estimate weight of patient

If the patient normally takes insulin detemir (levemir), insulin glargine (lantus)' Insulatard or Humulin-I, **continue this** at the same time s.c.

13 60 minutes to 6 hours

Aim:

- Clear blood of ketones suppress ketogenesis
- Achieve a rate of fall of blood ketones of at least 0.5 mmol/l/Hr
- Rise in bicarbonate of 3 mmol/l/Hr and fall of blood glucose of 3 mmol/l/Hr
- Maintain serum potassium
- Avoid hypoglycaemia
- Maintain fluid balance

Reassess

- Consider urinary catheter if anuric/incontinent
- Consider NG tube if vomiting or obtunded
- If O2 sats fall do ABG and repeat clinical assessment and CXR
- MEWS
- Fluid balance/urine output minimum 0.5 ml/Kg/Hr
- Continuous cardiac monitoring (severe DKA)
- Prophylactic LMWH
- Foot/heel care. Check for active ulceration.
- **Measure venous blood gas for pH, bicarbonate and potassium at 60 minutes and 2 hours and 2 hourly thereafter**

If DKA not resolving check insulin infusion pump is connected/working/contains insulin and increase insulin infusion rate by 1 unit / hour.

If the blood glucose falls below 14 mmol/l commence 10% glucose given at 125 mls/hr alongside 0.9% sodium chloride.

14 6 to 12 hours

- Ensure clinical and biochemical improvement
- Continue iv fluid replacement and iv insulin
- Assess for treatment complications e.g. fluid overload, cerebral oedema, hypoglycaemia
- Avoid hypoglycaemia
- If not improving seek senior advice
- If DKA resolved and patient eating resume usual s.c. insulin as below.

By 12 to 24 hours the ketonaemia and acidosis should have resolved Resolution = ketones < 0.3 mmol/L, venous pH> 7.3 and/or venous bicarbonate > 18 mmol/l.

If this has occurred re start usual subcutaneous insulin before insulin infusion is discontinued. If not already done so inform diabetes team.

15 12 to 24 hours

- Continue iv fluids if not eating and drinking
- If not eating and drinking revert to variable rate insulin infusion as per ABHB guidelines
- If DKA not resolved seek urgent senior advice and repeat steps in section 13
- Do not rely on bicarbonate to assess resolution of DKA after 24 hours as there may be hyperchloraemia secondary to large volumes of sodium chloride.

16 Monitoring and evaluation

This policy and chart will be subject to regular audit undertaken by the diabetes team.

17 Appendices

The bedside chart contains all essential information and monitoring for the management of DKA. Forms can be printed using the following link: http://howis.wales.nhs.uk/sitesplus/documents/866/ABHB_Clinical_0337%20DKA%20Charts%20and%20Guidelines_Issue%201.pdf

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