

# Joint British Diabetes Societies Inpatient Care Group

## The Management of Diabetic Ketoacidosis in Adults

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## Writing Group

Mark W Savage (Chair of Sub Group)

Maggie Sinclair-Hammersley (Chair of JBDS IP Care Group)

Gerry Rayman

Hamish Courtney

Ketan Dhatariya

Philip Dyer

Julie Edge

Philip Evans

Michelle Greenwood

Girly Hallahan

Louise Hilton

Anne Kilvert

Alan Rees

and many others

**Groups represented:** Association of British Clinical Diabetologists; British Society of Paediatric 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; Welsh Endocrine and Diabetes Society; Scottish Diabetes Group.

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British Society of Paediatric Endocrinology and Diabetes (BSPED) guidelines for management of DKA in young people under the age of 18 years can be found at:  
<http://www.bsped.org.uk/professional/guidelines/docs/DKAGuideline.pdf>

# Contents

	<b>Page</b>
<b>Foreword</b>	<b>4</b>
<b>Introduction</b>	<b>5</b>
<b>Rationale for Best Practice</b>	<b>6</b>
<b>Controversial Areas</b>	<b>8</b>
<b>Serious Complications of DKA or its treatment</b>	<b>10</b>
<b>DKA Pathway of Care</b>	<b>11</b>
<b>Implementation and Audit</b>	<b>16</b>
<b>References</b>	<b>17</b>
<b>Appendices</b>	<b>20</b>
1. Conversion to subcutaneous insulin	
2. Joint British Societies Audit Standards	
Appendix to follow	
Integrated Care Pathway	

# Foreword

Diabetic ketoacidosis (DKA) though preventable remains a frequent and life threatening complication of type 1 diabetes. Unfortunately, errors in its management are not uncommon and importantly are associated with significant morbidity and mortality. Most acute hospitals have guidelines for the management of DKA but it is not unusual to find these out of date and at variance to those of other hospitals. Even when specific hospital guidelines are available audits have shown that adherence to and indeed the use of these is variable amongst the admitting teams. These teams infrequently refer early to the diabetes specialist team and it is not uncommon for the most junior member of the admitting team, who is least likely to be aware of the hospital guidance, to be given responsibility for the initial management of this complex and challenging condition.

To address these issues the Joint British Diabetes Societies, supported by NHS Diabetes has produced up-to-date guidance developed by a multidisciplinary group of practicing specialists, with considerable experience in this area. Where possible the guidance is evidenced based but also draws from accumulated professional experience. A number of new recommendations have been introduced including the use of bedside ketone meters (though management based on bicarbonate and glucose are retained for those yet to introduce ketone meters), the use of fixed rate intravenous insulin infusion, and mandatory and prompt referral to the diabetes specialist team in all cases. The management is clearly presented and divided into a number of key steps in the care pathway; the first hour, the next six hours, next twelve hours etc. Importantly, conversion to subcutaneous insulin and preparing for discharge home are included. Audit is encouraged against defined standards.

The guideline is clearly written and accompanied by a practical and easy to follow flow chart to be used in admitting departments and wards managing DKA.

The authors should be congratulated on their achievement. These guidelines are recommended to all diabetes hospital teams for rapid introduction and for acceptance as the national guideline for managing DKA. Their widespread introduction should significantly improve the care of people admitted with DKA.

**Dr G Rayman**  
**NHS Diabetes Clinical Lead for Inpatient Diabetes Care**

# Introduction

There are several currently available national and international guidelines for the management of Diabetic Ketoacidosis (DKA) in both adults and children. (ISPAD 2009, McGeoch 2007, Savage 2006, BSPED 2004, Kitabchi 2009). In the last decade, however, there has been a change in the way patients with DKA present clinically and in addition there has been rapid development of near-patient testing technology. Until recently there was no easily available assay for ketone bodies hence capillary glucose, venous pH and bicarbonate were used to diagnose and monitor response to treatment in DKA. Near patient testing for 3-beta-hydroxybutyrate is now readily available for the monitoring of the abnormal metabolite allowing for a shift away from using glucose levels to drive treatment decisions in the management of DKA. These guidelines have been developed to reflect the development in technology and reflect new practice in the UK. They are evidence based where possible but are also drawn from accumulated professional knowledge and consensus agreement. They are intended for use by any health care professional that manages DKA in adults.

## Pathophysiology

Diabetic ketoacidosis (DKA) is a complex disordered metabolic state characterised by hyperglycaemia, acidosis, and ketonaemia. DKA usually occurs as a consequence of absolute or relative insulin deficiency that is accompanied by an increase in counter-regulatory hormones (ie, glucagon, cortisol, growth hormone, epinephrine). This type of hormonal imbalance enhances hepatic gluconeogenesis and glycogenolysis resulting in severe hyperglycaemia. Enhanced lipolysis increases serum free fatty acids that are then metabolised as an alternative energy source in the process of ketogenesis. This results in accumulation of large quantities of ketone bodies and subsequent metabolic acidosis. Ketones include acetone, 3-beta-hydroxybutyrate, and acetoacetate. The predominant ketone in DKA is 3-beta-hydroxybutyrate.

There are several mechanisms responsible for fluid depletion in DKA. These include osmotic diuresis due to hyperglycaemia, vomiting commonly associated with DKA, and eventually, inability to take in fluid due to a diminished level of consciousness. Electrolyte shifts and depletion are in part related to the osmotic diuresis. Hyper and hypokalaemia need particular attention.

## Definition and Diagnosis

DKA consists of the biochemical triad of ketonaemia, hyperglycaemia, and acidaemia.

## Epidemiology

The true incidence is difficult to establish. Population-based studies range from 4.6 to 8 episodes per 1,000 patients with diabetes (Johnson 1980, Faich 1983). DKA remains a significant clinical problem in spite of improvements in diabetes care (Fishbein 1995, Umpierrez 19997).

## Mortality and Morbidity

Improved understanding of the pathophysiology of DKA with close monitoring and correction of electrolytes has resulted in a significant reduction in the overall mortality rate from this life-threatening condition. Mortality rates have fallen significantly in the last 20 years from 7.96% to 0.67% (Lin 2005). The mortality rate is still high in developing countries and among non hospitalised patients (Otieno 2005). This high mortality rate illustrates the necessity of early diagnosis and the implementation of effective prevention programmes.

Cerebral oedema remains the most common cause of mortality, particularly in young children and adolescents. The main causes of mortality in the adult population include severe hypokalaemia, adult respiratory distress syndrome, and co-morbid states such as pneumonia, acute myocardial infarction and sepsis (Hamblin 1989).

Ketonaemia 3 mmol/L and over **or** significant ketonuria (more than 2+ on standard urine sticks)  
Blood glucose over 11 mmol/L or known diabetes mellitus  
Bicarbonate ( $\text{HCO}_3^-$ ) below 15 mmol/L **and/or** venous pH less than 7.3

# Rationale for Best Practice - The New Paradigm

## Ketones and Acidosis

Until recently, management of DKA has focussed on lowering the elevated blood glucose with fluids and insulin, using arterial pH and serum bicarbonate to assess metabolic improvement. This is based on the assumption that this would efficiently suppress ketogenesis and reverse acidosis. This strategy recognised that blood glucose is only a surrogate for the underlying metabolic abnormality. Recent developments now allow us to focus on the underlying metabolic abnormality (ketonaemia) which simplifies treatment of those who present with modest elevation of blood glucose but with acidosis secondary to ketonaemia 'euglycaemic diabetic ketoacidosis' (Munro 1973, Johnson 1980, Jenkins 1993). This clinical presentation is being encountered more frequently. Improved patient education with increased blood glucose and ketone monitoring has led to partial treatment of DKA prior to admission with consequent lower blood glucose levels at presentation.

## Bedside Monitoring

These guidelines recommend that management is based on bedside monitoring of patients with DKA. Blood glucose is routinely checked at the bedside, but portable ketone meters now also allow bedside measurement of blood ketones (3-beta-hydroxybutyrate). This is an important advance in the management of DKA (Sheikh-Ali 2008, Bektas 2004, Khan 2004, Wallace 2004, Vaneli 2003, Naunheim 2006). The resolution of DKA depends upon the suppression of ketonaemia, therefore measurement of blood ketones now represents best practice in monitoring the response to treatment (Wiggam 1997).

Access to blood gas and blood electrolyte measurement is now relatively easy and available within a few minutes of blood being taken. Therefore glucose, ketones and electrolytes, including bicarbonate and venous pH should be assessed at or near the bedside.

This recommendation raises important issues:

- Staff must be trained in the use of blood glucose and ketone meters
- The meters should be subject to rigorous quality assurance
- Laboratory measurement will be required in certain circumstances, such as when blood glucose or ketone meters are 'out of range'.

It is recognised that not all units have access to ketone meters. Thus, guidance is also given on monitoring treatment using of the rate of rise of bicarbonate and fall in blood glucose as alternative measures.

## The Involvement of Diabetes Specialist Teams (DST)

The diabetes specialist team must always be involved in the care of those admitted to hospital with DKA. Their involvement shortens patient stay and improves safety (Levetan 1995, Cavan 2001, Davies 2001, Sampson 2006). This should occur as soon as possible during the acute phase but will depend on local circumstances. Specialists must also be involved in the assessment of the precipitating cause of DKA, management, discharge, and follow up. This will include assessment of the patient's understanding of diabetes plus their attitudes and beliefs. Specialist involvement is essential to ensure regular audit and continuous quality improvement in the implementation of DKA guidelines. The practice of admitting, treating and discharging patients with DKA without the involvement of the diabetes specialist team is unsafe and likely to compromise safe patient care. This is a governance issue (Clement 2004).

## Recommended changes in management

- Measurement of blood ketones, venous (not arterial) pH and bicarbonate and their use as treatment markers
- Monitoring of ketones and glucose using bedside meters when available and operating within their quality assurance range
- Replacing 'sliding scale' insulin with weight-based fixed rate intravenous insulin infusion (IVI)

- Use of venous blood rather than arterial blood in blood gas analysers
- Monitoring of electrolytes on the blood gas analyser with intermittent laboratory confirmation
- Continuation of long acting insulin analogues (Lantus® or Levemir®) as normal
- Involvement diabetes specialist team as soon as possible

## General Management Issues

### Fluid administration and deficits

There is universal agreement that the most important initial therapeutic intervention in DKA is appropriate fluid replacement followed by insulin administration. The main aims for fluid replacement are:

- Restoration of circulatory volume
- Clearance of ketones
- Correction of electrolyte imbalance

The typical fluid and electrolyte deficits are shown in the table below. For example, an adult weighing 70kg presenting with DKA may be up to 7 litres in deficit. This should be replaced as crystalloid. In patients with kidney failure or heart failure, as well as the elderly and adolescents, the rate and volume of fluid replacement may need to be modified. The aim of the first few litres of fluid is to correct any hypotension, replenish the intravascular deficit, and counteract the effects of the osmotic diuresis with correction of electrolyte disturbance.

### Typical Deficits in DKA:

Water (ml/kg)	100
Sodium (mmol/kg)	7-10
Chloride (mmol/kg)	3-5
Potassium (mmol/kg))	3-5

The type of fluid to be used is discussed in detail in *Controversial Areas*.

### Insulin therapy

A fixed rate IVII calculated on 0.1 units/ per kilogram infusion is recommended. It may be necessary to estimate the weight of the patient. See *Controversial Areas*. Insulin has the following effects:

- Suppression of ketogenesis
- Reduction of blood glucose
- Correction of electrolyte imbalance

### Metabolic treatment targets

The recommended targets are

- Reduction of the blood ketone concentration by 0.5 mmol/L/hour

- Increase the venous bicarbonate by 3 mmol/L/hour
- Reduce capillary blood glucose by 3 mmol/L/hour
- Potassium should be maintained between 4.0 and 5.0 mmol/L

If these rates are not achieved then the fixed rate IVII rate should be increased (see *Management of DKA* Section B, Action 2).

### Intravenous glucose concentration

The management should be focussed on clearing ketones as well as normalising blood glucose. It is often necessary to administer an intravenous infusion of 10% glucose in order to avoid hypoglycaemia and permit the continuation of a fixed rate IVII to suppress ketogenesis. Introduction of 10% glucose is recommended when the blood glucose falls below 14 mmol/L. It is important to continue 0.9% sodium chloride solution to correct circulatory volume. It may be necessary to infuse these solutions concurrently (Section B, Action 2). Glucose should not be discontinued until the patient is eating and drinking normally.

### Special patient groups

The following groups of patients need specialist input as soon as possible and special attention needs to be paid to fluid balance.

- Elderly
- Pregnant
- Young people 18 to 25 years of age (see cerebral oedema)
- Heart or kidney failure
- Other serious co-morbidities

### Patient considerations

Patients with diabetes who are admitted with DKA should be counselled about the precipitating cause and early warning symptoms of DKA. Failure to do so is a missed educational opportunity. Things to consider are:

- Identification of precipitating factor(s) e.g. infection or omission of insulin injections
- Prevention of recurrence e.g. provision of written sick day rules
- Insulin ineffective e.g. the patient's own insulin may be expired or denatured. This should be checked prior to reuse
- Provision of handheld ketone meters and education on management of ketonaemia

# Controversial Areas

The clinical assessment and aims of treatment in the management of DKA are not controversial. However, there is still disagreement about the optimum treatment regimen and where the evidence base is not strong, recommendations are based on consensus and experience. Some of the more controversial points will now be considered and good practice recommendations are made. The recommendations are given first followed by the rationale.

## Recommendations

1. Measure venous rather than arterial bicarbonate and pH
2. Blood ketone meters should be used for near patient testing
3. Crystalloid rather than colloid solutions are recommended for fluid resuscitation
4. Cautious fluid replacement in young adults
5. 0.9% sodium chloride solution is the recommended fluid of choice
6. Subcutaneous long-acting analogue insulin should be continued
7. Insulin should be administered at a fixed rate IVII calculated on body weight
8. Do not use a priming dose (bolus) of insulin
9. Bicarbonate administration is not recommended routinely
10. Phosphate should not be supplemented routinely

### 1. Arterial or venous measurements?

Recent evidence shows that the difference between venous and arterial pH is 0.02-0.15 pH units and the difference between arterial and venous bicarbonate is 1.88 mmol/L (Kelly 2006, Gokel 2000). This will change neither diagnosis nor management of DKA and it is not necessary to use arterial blood to measure acid base status (Ma 2003). Venous blood can be used in portable and fixed blood gas analysers and therefore venous measurements (bicarbonate, pH and potassium) are easily obtained in most admitting units. Arterial line insertion should only be performed if its use will influence management i.e.

for frequent arterial oxygen level measurements or monitoring blood pressure in the critically unwell patient.

### 2. Blood ketone measurement?

Ketonaemia is the hallmark of DKA. Frequent repeated measurement of blood 3-beta-hydroxybutyrate has only recently become a practical option due to the availability of meters which can measure blood ketone levels. Compelling evidence supports the use of this technology for diagnosis and management of DKA (Sheikh-Ali 2008, Bektas 2004, Vaneli 2003, Naunheim 2006). The resolution of DKA depends upon the suppression of ketonaemia and measurement of blood ketones now represents best practice in monitoring the response to treatment.

### 3. Colloid versus crystalloid?

Many guidelines suggest that in hypotensive patients initial fluid resuscitation should be with colloid. However, the hypotension results from a loss of electrolyte solution and it is more physiological to replace with crystalloid. Moreover, a recent Cochrane review did not support the use of colloid in preference to crystalloid fluid (Perel 2007).

### 4. Rate of fluid replacement?

There is concern that rapid fluid replacement may lead to cerebral oedema in children and young adults. National and international paediatric guidelines recommend cautious fluid replacement over 48 hours. Existing adult guidelines (ADA, ABCD, SIGN) all recommend rapid initial fluid replacement in the first few hours. No randomised controlled trials exist to guide decision making in this area. We therefore recommend cautious fluid replacement in small young adults who are not shocked at presentation.

### 5. 0.9% sodium chloride solution or Hartmann's solution for resuscitation?

There has been much debate recently about the relative merits of these two solutions (Dhatariya 2007 and NPSA Alerts 2002, 2007).

Infusion solution	Advantages	Disadvantages
<b>0.9% sodium chloride</b>	<ul style="list-style-type: none"> <li>• Decades of clinical experience</li> <li>• Readily available in clinical areas</li> <li>• Commercially available ready mixed with potassium at required concentrations, 20mmol/L (0.15%) or 40mmol/L (0.3%)</li> <li>• Supports safe practice with injectable potassium (NPSA compliant (NPSA alert 2002))</li> </ul>	<ul style="list-style-type: none"> <li>• Hyperchloraemic metabolic acidosis which may cause renal arteriolar vasoconstriction leading to oliguria and a slowing of resolution of acidosis</li> </ul>
<b>Compound sodium</b>	<ul style="list-style-type: none"> <li>• Balanced crystalloid with minimal tendency to hyperchloraemic metabolic acidosis</li> </ul>	<ul style="list-style-type: none"> <li>• Insufficient potassium if used alone</li> <li>• Not commercially available with adequate pre-mixed potassium. Potassium addition in general clinical areas is unsafe. (NPSA alert 2002)</li> <li>• Unfamiliar and not routinely kept on medical wards</li> </ul>

It is recommended that 0.9% sodium chloride solution should be the fluid of choice for resuscitation in all clinical areas as it supports safe practice and is available ready to use with adequate ready-mixed potassium. In theory replacement with glucose and compound sodium lactate (Hartmann's solution) with potassium, would prevent hyperchloraemic metabolic acidosis, as well as allow appropriate potassium replacement. However, at present this is not readily available as a licensed infusion fluid.

### 6. Continuation of long-acting insulin analogues?

In the last few years the use of long acting basal insulin analogues (Levemir®, Lantus®) has become widespread. Continuation of subcutaneous analogues during the initial management of DKA provides background insulin when the IV insulin is discontinued. This avoids rebound hyperglycaemia when IV insulin is stopped and should avoid excess length of stay. This only applies to long acting analogues and does not obviate the need to give short acting insulin before discontinuing the intravenous insulin infusion.

### 7. Fixed-rate intravenous insulin infusion (fixed rate IVII) versus variable rate?

Patient demographics are changing and patients with DKA are now more likely to be obese or suffering with other insulin-resistant states including pregnancy. Evidence has led to the re-emergence of fixed rate IVII in adults in the USA and international paediatric practice (Kitabchi 2009, BSPED 2009, ISPAD 2009). Fixed dose(s) per kilogram body weight enable rapid blood ketone clearance, which is readily monitored using bed-side ketone measurement. The fixed rate may need to be adjusted in insulin resistant states if

the ketone concentration is not falling fast enough, and/or the bicarbonate level is not rising fast enough.

### 8. Initiating treatment with a priming dose (bolus) of insulin?

A priming dose of insulin in the treatment in DKA is not necessary provided that the insulin infusion is started promptly at a dose of at least 0.1 unit/kg/hour (Kitabchi 2008).

### 9. Intravenous bicarbonate?

Adequate fluid and insulin therapy will resolve the acidosis in DKA and the use of bicarbonate is not indicated (Morris 1986, Hale 1984). The acidosis may be an adaptive response as it improves oxygen delivery to the tissues by causing a right shift of the oxygen dissociation curve. Excessive bicarbonate may cause a rise in the CO<sub>2</sub> partial pressure in the cerebrospinal fluid (CSF) and may lead to a paradoxical increase in CSF acidosis (Ohman 1971). In addition, the use of bicarbonate in DKA may delay the fall in blood lactate: pyruvate ratio and ketones when compared to intravenous 0.9% sodium chloride infusion (Hale 1984). There is some evidence to suggest that bicarbonate treatment may be implicated in the development of cerebral oedema in children and young adults (Glaser 2001).

### 10. Use of intravenous phosphate?

Whole-body phosphate deficits in DKA are substantial, averaging 1 mmol/kg of body weight. There is no evidence of benefit of phosphate replacement (Wilson 1982) thus we do not recommend the routine measurement or replacement of phosphate. However, in the presence of respiratory and skeletal muscle weakness, phosphate measurement and replacement should be considered (Liu 2004).

# Serious complications of DKA and its treatment

## Hypokalaemia and hyperkalaemia

Hypokalaemia and hyperkalaemia are potentially life-threatening conditions during the management of DKA. There is a risk of acute pre-renal failure associated with severe dehydration and it is therefore recommended that no potassium be prescribed with the initial fluid resuscitation or if the serum potassium level remains above 5.5 mmol/L. However, potassium will almost always fall as the DKA is treated with insulin, thus it is recommended that 0.9% sodium chloride solution with potassium 40 mmol/L (ready-mixed) is prescribed as long as the serum potassium level is below 5.5 mmol/L and the patient is passing urine. If the serum potassium level falls below 3.5 mmol/L the potassium regimen needs review. Where fluid balance permits, an increase in rate of 0.9% sodium chloride solution with potassium 40 mmol/L infusion is possible. Otherwise, a more concentrated potassium infusion will be needed and to ensure safe practice, all aspects of its use must comply with local and national guidance (NPSA 2002, 2009). Trusts need to ensure that they have local protocols in place which allow for the safe administration of concentrated potassium solutions. This may require transfer to a higher care environment. Electrolyte measurements can be obtained from most modern blood gas analysers and should be used to monitor sodium, potassium and bicarbonate levels.

## Hypoglycaemia

The blood glucose may fall very rapidly as ketoacidosis is corrected and a common mistake is to allow the blood glucose to drop to hypoglycaemic levels. This may result in a rebound ketosis driven by counter-regulatory hormones. Rebound ketosis lengthens duration of treatment. Severe hypoglycaemia is also associated with cardiac arrhythmias, acute brain injury and death. Once the blood glucose falls to 14 mmol/L intravenous glucose 10% needs to be commenced to prevent hypoglycaemia.

## Cerebral oedema

Cerebral oedema causing symptoms is relatively uncommon in adults during DKA although asymptomatic cerebral oedema may be a common occurrence (Rosenbloom 1990). The observation that cerebral oedema usually occurs within a few hours of initiation of treatment has led to the speculation that it is iatrogenic (Hillman 1987). However, this is disputed since subclinical cerebral oedema may be present before treatment is started (Hoffmann 1988). The exact cause of this phenomenon is unknown; recent studies suggest that cerebral hypoperfusion with subsequent reperfusion may be the mechanism operating (Glaser 2001, Glaser 2008, Yuen 2008).

Cerebral oedema associated with DKA is more common in children than in adults. In the UK around 70 to 80% of diabetes-related deaths in children under 12 years of age are caused as a result of cerebral oedema (Edge 1999). The UK case control study of cerebral oedema complicating DKA showed that children who developed cerebral oedema were more acidotic and, after severity of acidosis was corrected for, insulin administration in the first hour and volume of fluid administered over the first 4 hours were associated with increased risk (Edge 2006).

## Pulmonary oedema

Pulmonary oedema has only been rarely reported in DKA. As with cerebral oedema, the observation that pulmonary oedema usually occurs within a few hours of initiation of treatment has led to the speculation that the complication is iatrogenic and that rapid infusion of crystalloids over a short period of time increases the likelihood of this complication (Dixon 2006). Elderly patients and those with impaired cardiac function are at particular risk and monitoring of central venous pressure should be considered.

# DKA Care Pathway

Diabetic Ketoacidosis is a medical emergency with a significant morbidity and mortality. It should be diagnosed promptly and managed intensively. The specialist diabetes team should always be involved as soon as possible and ideally within 24 hours because this has been demonstrated to be associated with a better patient experience and reduced length of stay.

**For young people under the age of 18 years, contact your paediatric diabetes service and use the BSPED DKA guidelines which can be found at <http://www.bsped.org.uk/professional/guidelines/docs/DKAGuideline.pdf>**

## Assessment of severity

The presence of one or more of the following may indicate severe DKA and admission to a Level 2/HDU (High Dependency Unit) environment, insertion of a central line and immediate senior review should be considered:

- Blood ketones over 6 mmol/L
- Bicarbonate level below 5 mmol/L
- Venous/arterial pH below 7.1
- Hypokalaemia on admission (under 3.5 mmol/L)
- GCS less than 12 or abnormal AVPU scale
- Oxygen saturation below 92% on air (assuming normal baseline respiratory function)
- Systolic BP below 90 mmHg
- Pulse over 100 or below 60 bpm
- Anion gap above 16 [Anion Gap =  $(\text{Na}^+ + \text{K}^+) - (\text{Cl}^- + \text{HCO}_3^-)$  ]

## Provision of care

Local care pathways should identify the units that are to care for DKA patients. Nursing staff appropriately trained in Level 2/HDU should take the lead in hands-on patient care.

## New principles

The insulin infusion rate is calculated by weight, which may need to be estimated. Administration by weight allows insulin resistant states to be accommodated. Reliance on standard variable rate

IVII regimens will fail to accommodate for the very obese or the pregnant patient and risks premature reduction of insulin dosage. Where blood ketone measurements are available the adequacy of the insulin regimen is determined by the rate of fall of the ketones and will need revision if this is inadequate. If bedside ketone measurement is not available the bicarbonate level can be used to assess response during the first 6 hours, but may be less reliable thereafter. This is particularly important when glucose levels are relatively normal. Supplementary glucose solution may need to be infused at some stage in treatment to provide substrate. This will permit the fixed rate IVII to be maintained, avoid hypoglycaemia and allow the full suppression of ketone production.

## A. Hour 1: Immediate management upon diagnosis: 0 to 60 minutes.

**T=0 at time intravenous fluids are commenced If there is a problem with intravenous access critical care support should be requested immediately**

### Aims

- Commence IV 0.9% sodium chloride solution
- Commence a fixed rate IVII but only after fluid therapy has been commenced
- Establish monitoring regime appropriate to patient; generally hourly blood glucose (BG) and hourly ketone measurement, with at least 2 hourly serum potassium for the first six hours
- Clinical and biochemical assessment of the patient
- Involvement of the diabetes specialist diabetes team at the earliest possible stage

## Action 1 - Intravenous access and initial investigations

- Rapid ABC (Airway, Breathing, Circulation)
- Large bore iv cannulae and commence iv fluid replacement (See action 2)
- Clinical assessment
  - o Respiratory rate; temperature; blood pressure; pulse; oxygen saturation

- o Glasgow Coma Scale. NB: a drowsy patient in the context of DKA is serious and the patient requires critical care input. Consider NG tube with airway protection to prevent aspiration
- o Full clinical examination
- Initial investigations should include:
  - o Blood ketones
  - o Capillary blood glucose
  - o Venous plasma glucose
  - o Urea and electrolytes
  - o Venous blood gases
  - o Full blood count
  - o Blood cultures
  - o ECG
  - o Chest radiograph
  - o Urinalysis and culture
- Continuous cardiac monitoring
- Continuous pulse oximetry
- Consider precipitating causes and treat appropriately
- Establish usual medication for diabetes

## Action 2 – Restoration of circulating volume

Assess the severity of dehydration using pulse and blood pressure. As a guide 90mmHg may be used as a measure of hydration but take age, gender and concomitant medication into account.

### **Systolic BP (SBP) on admission below 90mmHg**

Hypotension is likely to be due to low circulating volume, but consider other causes such as heart failure, sepsis, etc.

- Give 500 ml of 0.9% sodium chloride solution over 10-15 minutes. If SBP remains below 90mmHg this may be repeated **whilst awaiting senior input**. In practice most patients require between 500 to 1000 ml given rapidly.
- If there has been no clinical improvement re-consider other causes of hypotension and seek **immediate senior assessment**. Consider involving the ITU/critical care team.
- Once SBP above 90mmHg follow fluid replacement as below

### **Systolic BP on admission 90 mmHg and over**

Below is a table outlining a typical fluid replacement regimen for a previously well 70kg adult. This is an illustrative guide only. A slower infusion rate should be considered in young adults (see Controversial Areas).

Fluid	Volume
0.9% sodium chloride 1L *	1000ml over 1st hour
0.9% sodium chloride 1L with potassium chloride	1000ml over next 2 hours
0.9% sodium chloride 1L with potassium chloride	1000ml over next 2 hours
0.9% sodium chloride 1L with potassium chloride	1000ml over next 4 hours
0.9% sodium chloride 1L with potassium chloride	1000ml over next 4 hours
0.9% sodium chloride 1L with potassium chloride	1000ml over next 6 hours
<b>Re-assessment of cardiovascular status at 12 hours is mandatory, further fluid may be required</b>	

\*Potassium chloride may be required if more than 1 litre of sodium chloride has been given already to resuscitate hypotensive patients

### **Exercise caution in the following patients**

- Young people aged 18-25 years
- Elderly
- Pregnant
- Heart or kidney failure
- Other serious co-morbidities

In these situations admission to a Level 2/HDU facility should be considered. Fluids should be replaced cautiously, and if appropriate, guided by the central venous pressure measurements.

### Action 3 - Potassium replacement

Hypokalaemia and hyperkalaemia are life threatening conditions and are common in DKA. Serum potassium is often high on admission (although total body potassium is low) but falls precipitously upon treatment with insulin. Regular monitoring is mandatory.

Potassium level in first 24 hours (mmol/L)	Potassium replacement in mmol /L of infusion solution
Over 5.5	Nil
3.5-5.5	40
Below 3.5	Senior review as additional potassium needs to be given (see serious complications section)

### Action 4 - Commence a fixed rate intravenous insulin infusion (IVII)

- If weight not available from patient, estimate patient weight (in kg)
- If pregnant use present weight and consider calling for senior obstetric help as well.
- Start continuous fixed rate IVII via an infusion pump. 50units human soluble insulin (Actrapid®, Humulin S®) made up to 50ml with 0.9% sodium chloride solution. Ideally this should be provided as a ready-made infusion
- Infuse at a fixed rate of 0.1unit/kg/hr (i.e. 7ml/hr if weight is 70kg)
- Only give a stat dose of intramuscular insulin (0.1 unit/kg) if there is a delay in setting up a fixed rate IVII.
- If the patient normally takes insulin Lantus® or Levemir® subcutaneously continue this at the usual dose and usual time
- Insulin may be infused in the same line as the intravenous replacement fluid provided that a Y connector with a one way, anti-syphon valve is used and a large-bore cannula has been placed

## B. 60 minutes to 6 hours

### Aims:

- Clear the blood of ketones and suppress ketogenesis
- Achieve a rate of fall of ketones of at least 0.5 mmol/L/hr
- In the absence of ketone measurement, bicarbonate should rise by 3 mmol/L/hr and blood glucose should fall by 3 mmol/L/hr

- Maintain serum potassium in normal range
- Avoid hypoglycaemia

### Action 1 – Re-assess patient, monitor vital signs

- Consider urinary catheterisation if incontinent or anuric (i.e. not passed urine by 60 minutes)
- Consider naso-gastric tube if patient obtunded or if persistently vomiting
- If oxygen saturation falling perform arterial blood gases and request repeat chest radiograph
- Regular observations and Early Warning Score (EWS) charting as appropriate
- Accurate fluid balance chart, minimum urine output 0.5ml/kg/hr
- Continuous cardiac monitoring in those with severe DKA
- Give low molecular weight heparin as per NICE guidance (CG 92 Jan 2010)

### Action 2 – Review metabolic parameters

- Measure blood ketones and capillary glucose hourly (note: if meter reads "blood glucose over 20 mmol/L" or "HI" venous blood should be sent to the laboratory hourly or measured using venous blood in a blood gas analyser until the bedside meter is within its QA range)
- Review patient's response to fixed rate IVII hourly by calculating rate of change of ketone level fall (or rise in bicarbonate or fall in glucose).
- Assess resolution of ketoacidosis

- o If blood ketone measurement available and blood ketones not falling by at least 0.5 mmol/L/hr call a prescribing clinician to increase insulin infusion rate by 1 unit/hr increments hourly until ketones falling at target rates (also check infusion\*\*)
- o If blood ketone measurement not available use venous bicarbonate. If the bicarbonate is not rising by at least 3 mmol/L/hr call a prescribing clinician to increase insulin infusion rate by 1 unit/hr increments hourly until bicarbonate is rising at this rate\*\*
- o Alternatively use plasma glucose. If glucose is not falling by at least 3 mmol/L/hr call a prescribing clinician to increase insulin infusion rate by 1 unit/hr increments hourly until glucose falls at this rate. Glucose level is not an accurate indicator of resolution of acidosis in euglycaemic ketoacidosis, so the acidosis resolution should be verified by venous gas analysis\*\*

**\*\* If ketones and glucose are not falling as expected always check the insulin infusion pump is working and connected and that the correct insulin residual volume is present (to check for pump malfunction)**

- Measure venous blood gas for pH, bicarbonate and potassium at 60 minutes and 2 hours and 2 hourly thereafter.
- If the potassium is outside the reference range, assess the appropriateness of potassium replacement and check it hourly. If it is abnormal next hour seek immediate senior medical advice. (See Action 3 p14).
- Continue fixed rate IVII until ketones less than 0.3 mmol/L, venous pH over 7.3 and/or venous bicarbonate over 18 mmol/L. (See section C)
- Do not rely on urinary ketone clearance to indicate resolution of DKA, because these will still be present when DKA has resolved.
- If glucose falls below 14 mmol/L commence 10% glucose given at 125mls/hour alongside the 0.9% sodium chloride solution.
- Monitor and replace potassium as it may fall rapidly.

### Action 3 – Identify and treat precipitating factors

#### C. 6 to 12 hours.

**Aim:** The aim within this time period is to:

- Ensure that clinical and biochemical parameters are improving
- Continue IV fluid replacement
- Continue insulin administration
- Assess for complications of treatment e.g. fluid overload, cerebral oedema
- Continue to treat precipitating factors as necessary
- Avoid hypoglycaemia

#### Action 1 – Re-assess patient, monitor vital signs

- If patient not improving seek senior advice
- Ensure referral has been made to diabetes team

#### Action 2 – Review biochemical and metabolic parameters

- At 6 hours check venous pH, bicarbonate, potassium, as well as blood ketones and glucose
- Resolution is defined as ketones less than 0.3mmol/L, venous pH over 7.3 (do not use bicarbonate as a surrogate at this stage – see box Section D).

**If DKA resolved go to section E.**

**If DKA not resolved refer to Action 2 in Section B.**

#### D. 12 to 24 HOURS

**Expectation:** By 24 hours the ketonaemia and acidosis should have resolved

**Aim:**

- Ensure that clinical and biochemical parameters are improving or have normalised
- Continue IV fluids if not eating and drinking.
- If patient is not eating and drinking and there is no ketonaemia move to a variable rate IVII as per local guidelines
- Re-assess for complications of treatment e.g. fluid overload, cerebral oedema

- Continue to treat precipitating factors as necessary
- Transfer to subcutaneous insulin if patient is eating and drinking normally. Ensure subcutaneous insulin is started before IV insulin is discontinued. Ideally give subcutaneous fast acting insulin and a meal and discontinue IV insulin one hour later.

### Action 1 – Re-assess patient, monitor vital signs

### Action 2 – Review biochemical and metabolic parameters

- At 12 hours check venous pH, bicarbonate, potassium, as well as blood ketones and glucose
- Resolution is defined as ketones <0.3mmol/L, venous pH>7.3

**If DKA resolved go to section E.**

**If DKA not resolved refer to Action 2 in Section B and seek senior specialist advice as a matter of urgency.**

NB: Do not rely on bicarbonate to assess resolution of DKA at this point due to possible hyperchloraemia secondary to high volumes of 0.9% sodium chloride solution. The hyperchloraemic acidosis may cause renal vasoconstriction and be a cause of oliguria. However, there is no evidence that the hyperchloraemic acidosis causes significant morbidity or prolongs length of stay.

**Expectation:** Patients should be eating and drinking and back on normal insulin.

If expectation is not met within this time period it is important to identify and treat the reasons for the failure to respond to treatment. **This situation is unusual** and requires senior and specialist input.

### E. Conversion to subcutaneous insulin.

- Convert back to an appropriate subcutaneous regime when biochemically stable (blood ketones less than 0.3, pH over 7.3) and the patient is ready and able to eat.

Conversion to subcutaneous insulin is ideally managed by the Specialist Diabetes Team. If the team is not available see Appendix 1. If the patient is newly diagnosed it is essential they are seen by a member of the specialist team prior to discharge.

### Specialist diabetes team input

If not already involved the local diabetes team should be informed and the patient reviewed within 24 hours of admission. Diabetes team input is important to allow re-education, to reduce the chance of recurrence and to facilitate follow up.

# Implementation of the guidelines

Repeated audits by many diabetes units in all constituent UK countries have consistently demonstrated poor adherence to local (or national) guidelines in the management of DKA. There are two main problems to be addressed:

1. the guidelines must be implemented
2. the guidelines must be audited

The guidelines must be reviewed regularly: next planned review is 2013.

## Commissioning of Care

Diabetic Ketoacidosis is a recognised common medical emergency and must be treated appropriately. For this to occur the Health Economies within the United Kingdom must address management of DKA in the context of provision of expert medical and nursing input within secondary care. Commissioners, Primary Care Providers, Local Diabetes Networks and Diabetes Directorates within the Acute Trusts, should co-operate and ensure the Quality Indicators and Audit Standards set out below are met.

## Audit

### Quality Indicators

Every Acute Trust should have a local management plan in place based upon these, or other authoritative guidelines. Guidelines must be current and valid and should not be used if the review date has expired. If there is no review date, they should not be used.

Every Acute Trust should have nominated care areas for patients with diabetic ketoacidosis

Every Acute Trust should have trained Health Care Workers available to measure blood ketone levels 24 hours per day.

Every Acute Trust should have a Quality Assurance Scheme in place to ensure accuracy of blood glucose and ketone meters.

We recommend that every Acute Trust use performance indicators to assess the quality of care given (examples given in Appendix 2). A Treatment Pathway document may be beneficial, as adherence to guidelines for this condition is very poor and integrated pathway documents would improve compliance.

# References

- Bektas F, Eray O, Sari R, Akbas H. Point of care testing of diabetic patients in the emergency department. *Endocr Res* 2004(30):395-402
- British Society for Paediatric Endocrinology and Diabetes (BSPED) guidelines for the management of DKA. <http://www.bsped.org.uk/professional/guidelines/docs/DKAGuideline.pdf> accessed 14th Dec 2009
- Cavan DA, Hamilton P, Everett J, Kerr D. Reducing hospital inpatient length of stay for patients with diabetes. *Diabet Med* 2001; **18**:162-164
- Clement S, Braithwaite SS, Magee MF, Ahmann A, Smith EP, Schafer RG, Hirsch IB. Management of Diabetes and Hyperglycemia in Hospitals. *Diabetes Care* 2004; **27**, 553-591.
- Davies M, Dixon S, Currie CJ, Davis RE, Peters JR. Evaluation of a hospital diabetes specialist nursing service: a randomized controlled trial. *Diabet Med* 2001; **18**: 301–307.
- Dhatariya K. Editorial. Diabetic Ketoacidosis. *Brit Med J* 2007 (**334**):1284-1285
- Dixon AN, Jude EB, Banerjee AK, Bain SC. Simultaneous pulmonary and cerebral oedema and multiple CNS infarctions as complications of diabetic ketoacidosis: a case report. *Diabetic Medicine* 2006; **23**:571-3.
- Edge JA, Jakes RW, Roy Y, Hawkins M, Winter D et al. The UK case-control study of cerebral oedema complicating diabetic ketoacidosis in children. *Diabetologia* 2006; **49**:2002–2009
- Edge JA, Ford-Adams ME, Dunger DB. Causes of death in children with insulin dependent diabetes 1990-1996. *Arch Dis Child* 1999; **81**:318-323  
doi:10.1136/adc.81.4.318
- Faich GA, Fishbein HA, Ellis SE: The epidemiology of diabetic acidosis: a population-based study *Am J Epidemiol* **117** : 551-558,1983
- Fishbein HA, Palumbo PJ: Acute metabolic complications in diabetes. In *Diabetes in America*. National Diabetes Data Group, National Institutes of Health, 1995, p.283 -291 (NIH publ. no.: 95-1468)
- Glaser N, Barnett P, McCaslin I, et al. Pediatric Emergency Medicine Collaborative Research Committee of the American Academy of Pediatrics. Risk factors for cerebral edema in children with diabetic ketoacidosis. The Pediatric Emergency Medicine Collaborative Research Committee of the American Academy of Pediatrics. *N Engl J Med* 2001; **344**(4):264-9.
- Glaser NS, Marcin JP, Wootton-Gorges SL, et al. Correlation of clinical and biochemical findings with diabetic ketoacidosis-related cerebral edema in children using magnetic resonance diffusion-weighted imaging. *J Pediatr*. 2008 Oct; **153**(4):541-6.
- Gokel Y, Paydas S, Koseoglu Z et al. Comparison of blood gas and acid-base measurements on arterial and venous blood samples in patients with uremic acidosis and diabetic ketoacidosis in the emergency room. *Am J Nephrol* 2000(**4**): 319-323
- Hale PJ, Crase J, Natrass M. Metabolic effects of bicarbonate in the treatment of diabetic ketoacidosis. *BMJ (Clin Res Ed)* 1984; **289**(6451): 1035–1038
- Hamblin PS, Topliss DJ, Chosich N, et al. Deaths associated with diabetic ketoacidosis and hyperosmolar coma, 1973-1988. *Medical Journal of Australia* 1989; **151**: 439-444
- Hillman K. Fluid resuscitation in diabetic emergencies – a reappraisal. *Intensive Care Med* 1987; **13**:4-8.
- Hoffman WH, Steinhart CM, el Gammal T, Steele S, Cuadrado AR, Morse PK. Cranial CT in children and adolescents with diabetic ketoacidosis *Am J of Neuroradiol*, 1988; **9**, 733-739,
- International Society for Pediatric and Adolescent Diabetes. 2009.  
<http://www.ispad.org/FileCenter.html?CategoryID=5> accessed 14th December 2009
- Jenkins D, Close CE, Krentz AJ, Natrass M, and Wright AD. Euglycaemic diabetic ketoacidosis: does it exist? *Acta Diabetol* 1993; **30**:251-253.

Johnson DD, Palumbo PJ, Chu C-P: Diabetic ketoacidosis in a community-based population. *Mayo Clin Proc* 1980; 55: 83-88.

Kelly AM. The case for venous rather than arterial blood gases in diabetic Ketoacidosis. *Emerg Med Australas* 2006(**18**): 64-67

Khan ASA, Talbot JA, Tiezen KL, Gardener EA, Gibson JM and New JP. Evaluation of a bedside blood ketone sensor: the effects of acidosis, hyperglycaemia and acetoacetate on sensor performance. *Diab Med* 2004;**21**: 782-785

Kitabchi, AE, Umpierrez, GE, Miles, JM, Fisher, JN. Hyperglycemic crises in adult patients with diabetes: a consensus statement from the American Diabetes Association. *Diabetes Care* 2009; 32:1335.

Levetan CS, Salas JR, Wilets IF, Zumoff B. Impact of endocrine and diabetes team consultation on hospital length of stay for patients with diabetes. *Am J Med* 1995; **99**: 22-28.

Lin SF, Lin JD, Huang YY. Diabetic ketoacidosis: comparisons of patient characteristics, clinical presentations and outcomes today and 20 years ago. *Chang Gung Med Journal*. 2005;**28**:24-30

Liu P, Jeng C.. Case Report - Severe hypophosphatemia in a patient with diabetic ketoacidosis and acute respiratory failure. *J Chin Med Assoc* 2004;**76**:355-359

Ma OJ, Rush MD, Godfrey MM, Gaddis G. Arterial blood gas results rarely influence emergency physician management of patients with suspected diabetic ketoacidosis. *Acad Emerg Med* 2004(**8**): 836-841

McGeoch SC, Hutcheon SD, Vaughan SM, et al. Development of a national Scottish diabetic ketoacidosis protocol *Pract Diabetes Int* 2007; **24**: 257-261

Morris LR, Murphy MB, Kitabchi AE. Bicarbonate therapy in severe diabetic ketoacidosis. *Ann Intern Med* 1986; **105**:836-40

Munro JF, Campbell IW, Mccuish AC, Duncan LJP. Euglycaemic Diabetic Ketoacidosis. *British Medical Journal*, 1973; **2**: 578-580.

Naunheim R, Jang TJ, Banet G, Richmond A, McGill J. Point of care testing identifies diabetic Ketoacidosis at triage. *Acad Emerg Med*. 2006;**6**:683-5.

NPSA. National Reporting & Learning System (NRLS). Never Events Framework 2009/10. London 2009

NPSA. Patient Safety Alert 22. Reducing the risk of hyponatraemia when administering intravenous infusions to children. London 2007

NPSA. Patient Safety Alert. Potassium solutions: risks to patients from errors occurring during intravenous administration. London 2002

Ohman JL Jr, Marliss EB, Aoki TT, Munichoodappa CS, Khanna VV, Kozak GP. The cerebrospinal fluid in diabetic ketoacidosis. *N Engl J Med* 1971;**284**:283-290

Otieno CF, Kayima JK, Omonge EO, Oyoo GO. Diabetic ketoacidosis: Risk factors, mechanisms and management strategies in sub-Saharan Africa: A review. *East African Medical Journal* 2005; 82 (12 Suppl):S197-203.

Perel P, Roberts I. Colloids versus crystalloids for fluid resuscitation in critically ill patients. *Cochrane Database of Systematic Reviews* 2007, Issue 3. Art. No.: CD000567. DOI: 10.1002/14651858.CD000567.pub3

Rapid Responses :  
[bmj.com/cgi/eletters/334/7607/1284](http://bmj.com/cgi/eletters/334/7607/1284) (accessed 14th December 2009)

Rosenbloom AL. Intracerebral crises during treatment of diabetic ketoacidosis. *Diabetes Care* 1990; **13**:22-33

Savage M, Kilvert A; on behalf of ABCD. ABCD guidelines for the management of hyperglycaemic emergencies in adults. *Pract Diabetes Int* 2006; **23**: 227-231

Sheikj-Ali M, Karon BS, Basu A et al. Can serum beta-hydroxybutyrate be used to diagnose diabetic ketoacidosis? *Diabetes Care* 2008(**4**):643-647

Umpierrez GE, Kelly JP, Navarrete JE, Casals MMC, Kitabchi AE: Hyperglycemic crises in urban blacks. *Arch Intern Med* 1997;**157**: 669-675

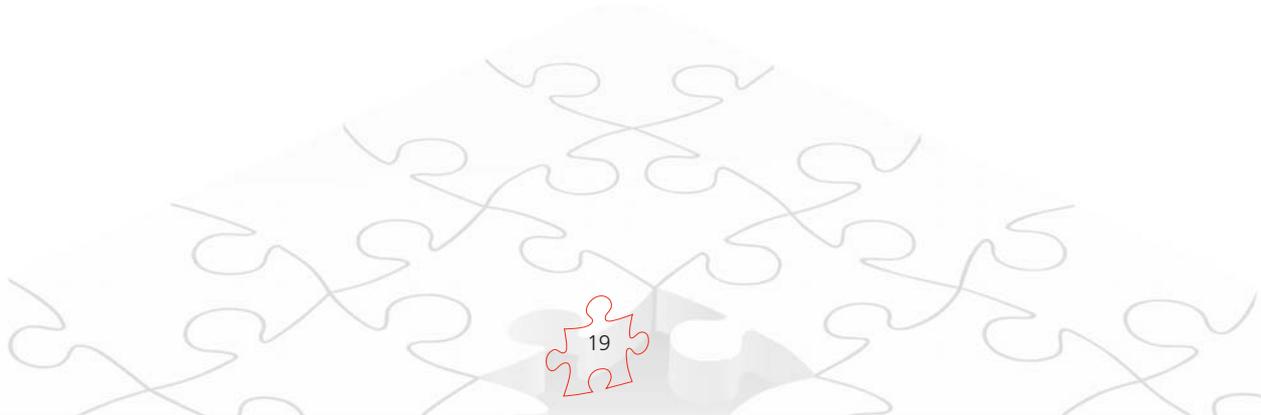
Vanelli M, Chiari G, Capuano C et al. The direct measurement of 3-beta-hydroxy butyrate enhances the management of diabetic ketoacidosis in children and reduces time and costs of treatment. *Diabetes Nutr Metab* 2003;**16**): 312-316

Wallace TM, Matthews DR. Recent advances in the monitoring and management of diabetic ketoacidosis. *QJM*. 2004;**97**:773-80

Wiggam MI, O’Kane MJ, Harper R, et al. Treatment of diabetic ketoacidosis using normalization of blood 3-hydroxybutyrate concentration as the endpoint of emergency management. A randomized controlled study. *Diabetes Care* 1997; **20**: 1347–1352.

Wilson HK, Keuer SP, Lea AS, et al. Phosphate therapy in diabetic ketoacidosis. *Arch Intern Med* 1982;142(3):517-20

Yuen N, Anderson SE, Glaser N, Tancredi DJ, O’Donnell ME. Cerebral blood flow and cerebral edema in rats with diabetic ketoacidosis. *Diabetes*. 2008;57:2588-94



# Appendix 1

## Restarting subcutaneous insulin for patients already established on insulin

- Previous regimen should generally be re-started
- With all regimens the intravenous insulin infusion should not be discontinued for at least 30 to 60 minutes after the administration of the subcutaneous dose given in association with a meal.
- Patient on basal bolus insulin
  - There should be an overlap between the insulin infusion and first injection of fast acting insulin. The fast acting insulin should be injected with the meal and the intravenous insulin and fluids discontinued 30 minutes later.
  - If the patient was previously on a long acting insulin analogue such as Lantus® or Levemir®, this should have been continued and thus the only action should be to restart their normal short acting insulin at the next meal.
  - If basal insulin has been stopped in error, the insulin infusion should not be stopped until some form of background insulin has been given. If the basal analogue is normally taken once daily in the evening and the intention is to convert to subcutaneous insulin in the morning, give half the usual daily dose of basal insulin as isophane (Insulatard®, Humulin I®) in the morning. This will provide essential background insulin until the long acting analogue can be recommenced. Check blood ketone and glucose levels regularly

- Patient on twice daily fixed-mix insulin
  - Re-introduce before breakfast or evening meal. Do not change at any other time. Maintain insulin infusion until 30 minutes after subcutaneous insulin given
- Patient on CSII
  - Recommence at normal basal rate. Continue intravenous insulin infusion until meal bolus given. Do not recommence CSII at bedtime

## Calculating subcutaneous insulin dose in insulin-naïve patients

### Estimate Total Daily Dose (TDD) of insulin

This estimate is based on several factors, including the patient's sensitivity to insulin, degree of glycaemic control, insulin resistance, weight, and age. The TDD can be calculated by multiplying the patient's weight (in kg) by **0.5 to 0.75 units**. Use 0.75 units/kg for those thought to be more insulin resistant i.e. teens, obese.

**Example:** a 72 kg person would require approximately  $72 \times 0.5$  units or 36 units in 24 hours

### Calculating a Basal Bolus (QDS) Regimen:

Give 50% of total dose with the evening meal in the form of long acting insulin and divide remaining dose equally between pre-breakfast, pre-lunch and pre-evening meal.

	Pre-breakfast	Pre-lunch	Pre-evening meal	Bedtime
Rapid acting insulin, e.g. Apidra®/Humalog®/ NovoRapid®	6 units	6 units	6 units	
Long acting insulin, e.g. Lantus®/Levemir®			18 units	

Administer 1st dose of fast acting s/c insulin prior to breakfast or lunch preferably. Administer before evening meal if monitoring can be guaranteed. Do not convert to subcutaneous regimen at bed time.

In patients new to insulin therapy dose requirements may decrease within a few days as the insulin resistance associated with DKA resolves. Close supervision from the specialist diabetes team is required.

### Calculating a twice daily (BD) regimen:

If a twice daily pre-mixed insulin regimen is to be used, give two thirds of the total daily dose at breakfast, with the remaining third given with the evening meal.

# Appendix 2

## Standards of care

Purpose of standards

- Maximise patient safety and quality of care
- Support professional best practice
- Deliver enhanced patient satisfaction
- Reduce Trust operating costs (litigation, complaint procedures)
- Contribute to improved financial performance (reduced length of stay)

Diabetic Ketoacidosis	
Processes	
Protocol	Availability of diabetes management guidelines based on national examples of good practice
Implementation	Availability of hospital wide pathway agreed with diabetes speciality team and regular audit of key components. Evidence of rolling education program for all medical and nursing staff
Specialist review	People with diabetes who are admitted to hospital with diabetic ketoacidosis are reviewed by a specialist diabetes physician or nurse prior to discharge
Environment	HDU access or monitored bed for DKA
Outcome measures	
Incidence	Benchmark incidence of DKA against equivalent national and regional data for admissions using widely available local and national datasets
Income	Length of stay Time of conversion to subcutaneous regimen
Ketosis	Time to blood ketonaemia or acidosis resolution
Morbidity & mortality	Complication rate of DKA treatment (e.g.cerebral oedema) In hospital death rates In hospital complications cerebral oedema, pulmonary oedema, ARF, septicaemia Readmission rate for DKA over 12-month period

## Statement for Inpatient Guidelines

This guideline has been developed to advise the treatment and management of The Management of Diabetic Ketoacidosis in Adults.

The guideline recommendations have been developed by a multidisciplinary team led by the Joint British Diabetes Society (JBDS) and including representation from Diabetes UK. People with diabetes have been involved in the development of the guidelines via stakeholder events organised by Diabetes UK.

It is intended that the guideline will be useful to clinicians and service commissioners in planning, organising and delivering high quality diabetes inpatient care. There remains, however, an individual responsibility of healthcare professionals to make decisions appropriate to the circumstance of the individual patient, informed by the patient and/or their guardian or carer and taking full account of their medical condition and treatment.

When implementing this guideline full account should be taken of the local context and in line with statutory obligations required of the organisation and individual. No part of the guideline should be interpreted in a way that would knowingly put people, patient or clinician at risk.

We would like to thank the service user representatives whose input has informed the development of these guidelines.

[www.diabetes.nhs.uk](http://www.diabetes.nhs.uk)

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