East of England Paediatric Critical Care Operational Delivery Network

Collaborative working to deliver high quality care to our children and their families

Management of Children & Young People with Diabetic Ketoacidosis (DKA)

Resource Book

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Endorsed by
East of England
Children and Young People
Diabetes network

Use in conjunction with British Society for Paediatric Endocrine and Diabetes. Guideline for the Management of Children and Young People under the age of 18 years with Diabetic Ketoacidosis

Further copies of all documents are available on our website:

http://www.eoeneonatalpccsicnetwork.nhs.uk/

What is Diabetic Ketoacidosis?

DKA is a serious and potentially fatal complication of type 1 diabetes mellitus, which is a disease of impaired glucose utilisation caused by a lack of insulin.

Incidence

There are over 3.9 million people in the UK with a diagnosis of diabetes; it is estimated that in the USA and Europe up to 70% of cases are at diagnosis of type 1 diabetes. Patients under the age of 5yrs are most at risk of developing DKA at diagnosis. [3,4]

Pathophysiology

DKA is a life threatening metabolic decompensation which should be treated as a medical emergency, caused by a combination of:

- Insulin deficiency
- The action of counter regulatory hormones [2]

DKA starts happening when there is a lack of insulin to enable the use of glucose as an energy source. Glucose is unable to enter the cells to act as an energy provider for cell metabolism, fats are used as an alternative source of energy production.

DKA is characterised by the biochemical triad of:

1. Hyperglycaemia *Diabetic*
2. Ketonaemia *Keto*
3. Metabolic acidosis *Acidosis*

Hypergylcaemia

Glucose that is consumed cannot enter the cells and accumulates in the blood, causing hyperglycaemia. Normally, insulin production suppresses the production of glucose and lipolysis (fat breakdown) in the liver.

Counter-regulatory hormones (responsible for glucose raising) are glucagon, growth hormone and cortisol (among others).
A lack of insulin results in increasing levels of glucagon and triggers over production of glucose in the liver (hepatic).
The elevation of any of these hormones leads to an increase in hepatic glucose production. Two processes contribute to this, gluconeogenesis and glycogenolysis.

Gluconeogenesis (production of new glucose)

Gluco	Neo	Genesis
glucose	new	production

The metabolic process by which new glucose is formed through the breakdown of non carbohydrate sources. This takes place mainly in the liver through the conversion of certain amino acids and lactate (lactic acid).

Glycogenolysis
The breakdown of complex glycogen into simple glucose - glycogen is stored in the liver and muscle, the glucose generated enters into the blood stream (to make it available to the cells who are demanding energy for metabolism) however, still lacking the insulin to enter the cells it merely contributes to an increasing blood glucose level.
Glycogenolysis is also involved with the flight and fight hormonal response, the production of the hormone adrenaline (epinephrine) through stress further stimulates the process of glycogenolysis (because the body perceives a need for fuel for the fight or to flee).

The body is stressed due to its unwell state and it is thought this stimulates the release of adrenaline in this context.

Both these processes serve to increase the blood glucose, further contributing to and driving the hyperglycaemia.

Ketonaemia

Insulin deficiency causes lipolysis of triglycerides - the mobilisation of fat as an alternative energy source produces free fatty acids and glycerol. The high glucose levels and low or non existent insulin levels allows lipolysis to occur unopposed, these result in ketone body production. In 'normal starvation' states these would be used in the Krebbs cycle for energy production but in DKA they quickly over-whelm the capacity of the Krebs cycle and they generate ketone bodies - acetone, acetoacetate and beta-hydroxybutyrate (usually the main ketone that is elevated) which spill over into the blood stream and urine.

Ketones can be measured in both the blood and urine via near patient testing, blood ketone measuring is the gold standard and should be used routinely in suspected cases of DKA.[1]

Acidosis

Metabolic acidosis occurs when there is too much acid in the bodily fluids; in DKA that occurs when the levels of ketone bodies build up. The ketone bodies need to be buffered if they are to remain harmless. In DKA the buffering system is overwhelmed and so the build up of ketone bodies leads to metabolic acidosis.

Dehydration also occurs; an excessive amount of glucose in the blood stream enters the renal tubules, it exceeds the threshold of the renal absorption of glucose at which point it spills over into the urine causing glycosuria. The glucose molecules provides an osmotic effect and draws large amounts of water across the semi-permeable membrane leading to the production of large volumes of urine. This is known as osmotic diuresis.

Ketones also collect in the urine, these are buffered by sodium and where sodium goes water follows, further adding to the large water movement into the renal tubules and contributing to the increased urine output.

The loss of large volumes of fluid contributes to the acidosis and causes further electrolyte disturbance.

Lactic acidosis from any sepsis will also contribute to the acidosis.

Any vomiting may contribute to dehydration and worsen the acidosis.

Acidosis occurs when there is either a reduction in the amount of bicarbonate (which acts as a buffer) resulting in a reduction in the body's ability to 'neutralise' the acid; or an increase in the amount of circulating hydrogen ions resulting in a change in the pH. In this case when the ketone bodies, which are strong acids, dissociate (breakdown) in water they release one or more hydrogen ions, contributing to the acidosis.

Compensation

Is the physical response to an acid –base imbalance that attempts to normalize the blood pH. In response to metabolic acidosis in DKA:

Respiratory

Respiratory rate, depth and alveolar ventilation will increase, bicarbonate as a buffer joins with hydrogen to form carbonic acid, this is converted to water and carbon dioxide and breathed out. This change in the respiratory pattern increases the amount of acid that is expelled through exhalation. In DKA this is often quickly over-whelmed and the child presents with Kussmaul respirations, a heavy sighing breathing pattern.

Renal

In a state of acidosis, the renal tubules will increase production of bicarbonate in order to buffer the increased hydrogen ions, however it is a slow process that happens over hours to days and cannot keep pace with the abnormal increase in hydrogen ions and is soon over-whelmed, The hydrogen ion concentration increases and the pH becomes acidotic.

Symptoms

Patients with DKA are often undiagnosed diabetics and present for the first time in DKA. Classic symptoms of a newly diagnosed diabetes include:

- Excessive thirst
- Excessive urination
- Hunger
- Weight loss
- Fatigue
-

In DKA the child may additionally present with:

- Clinical dehydration

Due to the osmostic diuresis

- Sighing respirations (Kussmaul breathing)

Due to the respiratory compensatory mechanism.

- Ketotic breath (likened to alcohol or pear drop sweets)

Due to the acetone production as a result of ketogenesis

- Nausea and / or vomiting

Due to the electrolyte disturbance

- Abdominal pain

possibly due to decreased gut motility, mesenteric ischaemia, or rapid expansion of the hepatic capsule.

- Reduction in level of consciousness.

Because the glucose circulating in the blood stream does not require insulin to cross the blood brain barrier, the brain is adequately supplied with the glucose it requires. Therefore children and young people with hyperglycaemia do not usually experience the sudden onset of neurological signs and symptoms, such as sudden collapse or seizure that hypoglycaemic patients do. Hyperglycaemia can lead to lethargy with a decreased responsiveness however, children in DKA are at risk of cerebral oedema, a potentially fatal consequence.

Diagnosis

DKA should be suspected in patients presenting with the classic symptoms, however diagnosis can only be confirmed with a blood glucose measurement and a blood gas.

Diagnosis of DKA is:

Blood glucose > 11 mmol/L (although it is often much higher)

And

pH below 7.3 **or** a plasma bicarbonate below 15 mmol/L

And

Blood ketones greater than 3 mmol/L

The management of DKA depends on the severity of the condition on presentation, but the goals are the same;

- Switch off ketosis and correct acidosis.
- Rehydration and correction of electrolyte disturbance
- Prevention of complications

Management

Children with DKA require critical care and often 1:1 nursing by a senior nurse, they are at risk of potentially fatal complications. Their fluid balance should be recorded accurately throughout and any concerns about their condition should be escalated to the most senior paediatrician without delay.

The management of patients in DKA will depend on the severity of the presentation, DKA is split into 3 categories, mild, moderate and severe, however a child who's illness is classified as mild still requires continual monitoring and strict management in order to prevent deterioration. The care of all children in DKA should be led by the most senior paediatrician available.

Mild DKA

pH 7.20 – 7.29 **and / or** plasma bicarbonate under 15 mmol/L

Moderate DKA

pH 7.10 – 7.19 **and / or** plasma bicarbonate under 10mmol/L

Severe DKA

pH less than 7.10 **and / or** plasma bicarbonate under 5 mmol/L

Sepsis

Children who are in DKA may also have sepsis, fever is not a feature of DKA and so if a child has fever they should also be treated for sepsis, however IV fluid resuscitation should follow the DKA guideline and there should be early use of inotropes to support the blood pressure.

Suspect sepsis? Think sepsis six:

- Give high flow oxygen
- Obtain IV/IO access take bloods/ do LP if clinically indicated
- Give IV / IO antibiotics
- Consider IV/IO fluids
- Ensure senior paediatrician in attendance
- Consider inotropic support early

Shock

Children who present in DKA may be also be shocked due either to the hypovolaemia or any concurrent sepsis. This is assessed according to the APLS guidelines so, if children have tachycardia, prolonged capillary refill time, poor peripheral pulses and hypotension (although this is a late sign) they should be treated as shocked, NICE emphasised weak thread pulses and hypotension as an appropriate clinical indicator of shock. The UK Resuscitation Council suggests isotonic crystalloids for the initial treatment of shock (eg Plasmalyte 148 or Ringers lactate) and 0.9% Saline if these are not available.

Shocked

Give 10 ml/kg bolus of 0.9% saline over 15 minutes.

Following the initial 10 ml/kg bolus patient should be reassessed further boluses of 10 ml/kg may be given if required to restore adequate circulation **up to a total of 40 ml/kg** at which stage inotropes should be considered.

Non shock

All other children should follow the non shocked pathway and be given a 10mls/kg fluid bolus over 30 mins.

Not Shocked

All children and young people with mild, moderate or severe DKA
who are not shocked and will require IV fluids/insulin
should receive a **10 ml/kg** 0.9% Nacl bolus over 60 mins.

Fluids

Fluid calculations and fluid balance charting is an essential part of the management of DKA, large volumes of fluid have been displaced and their replacement needs careful management. It is sensible if both the medical and nursing staff calculate the fluid requirement independently of each other, this acts a double check.
The fluid calculation is split into two elements, which when combined give an hourly rate of infusion for the first 48hours of the management.

Deficit

First, the **fluid deficit** is calculated, this is the amount of fluid that it is estimated the patient has lost prior to medical care. In order to calculate the deficit, there needs to be a decision made about the degree of the deficit or how much fluid the patient has lost, usually this is based on clinical findings, however with DKA this is based on their blood gas analysis.

Deficit fluids

calculations are based on the following results:

Mild DKA
pH 7.20 – 7.29 and / or plasma bicarbonate under 15 mmol/L
Assume a 5% deficit

Moderate DKA
pH 7.10 – 7.19 and / or plasma bicarbonate under 10mmol/L
Assume a 5% deficit

Severe DKA
pH less than 7.10 and / or plasma bicarbonate under 5 mmol/L
Assume a 10% deficit

The formula for calculating the deficit is:

Deficit = Weight x % deficit x 10

It is important to know whether the patient was treated for shock as this affects their fluid calculations. A non shocked patient, given 10mls/kg must have that volume deducted from their deficit calculations. However a shocked patient does not have any of their bolus fluids deducted from their deficit calculation. The deficit volume is then divided by 48 so that it replaced slowly over 48 hours.

Examples:

A) 24kg child - 5% deficit **shocked** - received 30mls / kg fluid bolus.

Deficit = 24 x 5 x 10 = 1200 mls

Divided by 48 hours = 25 mls/hr

B) 24kg child—5% deficit **non shocked** - received 10mls/kg only

Deficit = 24 x 5 x 10 = 1200 deduct 240mls = 960 mls

Divided by 48 hours = 20 mls/hr.

Maintenance fluids

Secondly the maintenance fluids need to be calculated, this is done using the Holliday-Segar formula, which you're probably familiar with.

100mls/kg/day for each kg up to 10kg of body weight
50mls/kg/day for each kg up to the next 20 kgs (or part therof)
20mls/kg/day for each additional kilogram above 20kgs

Example: patient weighs 24kgs.

First 10 kg = 10 x 100 = 1000 mls

Next 10 kgs = 10 x 50 = 500 mls

Next 4 kgs = 4x 20 = 80 mls

Total maintenance is 1580 mls per 24hours

Maintenance per hour is 66mls/hr

Look out for max volumes in patients > 75kgs

So now we have calculated both elements of the fluids , deficit and maintenance we can calculate the hourly fluid infusion rate. In DKA this is done for a 48 hour period using the following formula:

$$\frac{\text{Fluid deficit (- 10mls/kg for non shocked patients)}}{48} + \text{Maintenance fluids hourly rate}$$

Example: 24kg not shocked patient with 5% deficit

$$\frac{\text{Deficit = 1200 mls — 10mls/kg given (240mls)}}{48} + 66\text{mls/hr}$$

= 86 mls/hr for 48hrs.

Fluid balance
It is crucially important that all fluids given are accurately recorded and urine output is measured to allow an accurate fluid balance to be calculated.
Urine output is calculated in mls/kg/hour
When the patient passes urine, measure the volume and divide this by their weight and the number of hours since they last passed urine to get mls/kg/hour.

Fluid type
It is important to give the correct fluid to assist with switching off the ketotic process and correcting the electrolytes. The glucose concentration of the IV fluids will depend on the blood sugar of the patient.

Any fluids given for bolus' should be normal saline 0.9%
(some centres use PlasmaLyte 148 due to its lower chloride content, however additional potassium will need to be added to prevent hypokalaemia)

Any fluids given for IV maintenance should contain potassium, for every 500mls fluid there should be 20mmols potassium. This is the maximum concentration of potassium that is suitable for infusion via a peripheral IV line.

Potassium
Normally, the majority of potassium is in the cells (intracellular), the presence of insulin shifts it from the blood to the cells, however the lack of insulin that precipitates DKA means that large amounts of potassium will remain in the blood stream (extracellular). The high glucose in the blood spills into the urine, taking water with it, (osmotic diuresis) the excess water also carries sodium and potassium into the urine. Children presenting in DKA may have been losing potassium in their urine for some time.

Depending at what stage in the process the child presents they may have low, normal or high serum potassium levels, however they may have a very low intracellular potassium. Patients therefore who present with hypokalaemia are described as having a severe total body potassium depletion.

Renal failure
Severe dehydration can lead to renal failure; where the potassium is at the high end of normal range or raised on admission and there is concern about renal function it may be prudent to avoid potassium in fluids until the child has passed urine. (to confirm they are not becoming anuric).

As fluids and insulin are given, potassium will move back in to the cells from the intravascular space, and rapidly reduce the serum potassium, it is therefore important to ensure hypokalaemia does not occur through giving potassium in the IV fluids.

Giving IV fluids will:

- replace the extracellular volume (intravascular volume)
- decrease the counter-regulatory hormones (reducing their potency)
- decrease the blood sugar.

It is important that the blood glucose level does not fall too rapidly, this could cause a rapid fluid shift and can lead to cerebral oedema. In order to control this, it is important to give the least amount of insulin required whilst also giving glucose to enable normal cell respiration to occur once again; this will switch off the hepatic energy production, which in turn reduces the production of ketone bodies and reduces the acidosis, allowing the pH and bicarbonate to slowly correct.

Initial fluids

The fluids should be started as soon as the patient is stabilised, it is good practice for nursing and medical staff to calculate the fluid requirement independently to reduce the risk of error.

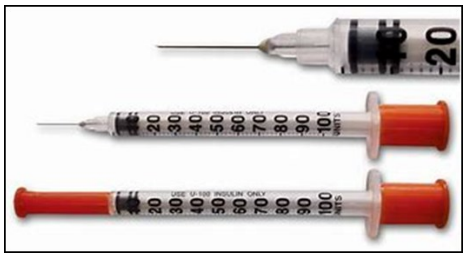
Insulin

Before starting insulin the fluids will have already started to lower the blood glucose level, it is important therefore to wait 1-2 hours after the maintenance fluids have been started before starting the insulin infusion so that the blood glucose doesn't drop too rapidly. Rapid changes in the blood glucose level will cause rapid fluid shifts and may lead to cerebral oedema.
Insulin is required to switch off the ketosis (remember the glucose generating processes gluconeogenesis and glycogenolysis discussed earlier that are normally suppressed by the presence of insulin.)

Bolus doses of IV insulin should never be given.

If a patient uses an insulin pump and it is still attached when they present in DKA this should be stopped (the failure of the pump may have contributed to the DKA and this may need to be investigated by the diabetes specialist team)

Where available pre filled insulin infusions should be used, otherwise they will need to be made up, insulin is measured in units (not mls)



An insulin syringe must be used

Insulin syringes comes in different unit measures (50units/ 100unitsetc) - always check the syringe and ensure you draw up 50 units of soluble insulin eg. actrapid.

Add the insulin to 49.5mls of normal saline 0.9% in a 50ml syringe. The resulting solution is:

50units insulin in 50mls normal saline 0.9%

Ensure the syringe and giving set at the patient end are clearly labelled to say they contain insulin.

Never flush the cannula /extension set that contains insulin.

Rates of insulin infusion

Insulin infusions are described in terms of units / kg / hour

The recommended infusion rates vary depending on the severity of the DKA, but a dose of 0.05 units /kg/hour is recommended except for cases of severe DKA or for DKA in adolescents where 0.1 units/kg/ hour may be indicated.

It is good practice and often local policy that any changes to insulin infusion rates are independently calculated and checked by two registered nurses.

Example:
6yr old in moderate DKA weighs 24 kgs
24kgs x 0.05units = 1.2 units
50units in 50 mls means the infusion will run at 1.2 mls/hr to deliver a dose of 0.05units/kg/hour.

14 yr old in DKA weighs 42kgs
42kgs x 0.1 units = 4.2 units
50units in 50mls means the infusion will run at 4.2mls/hr to deliver a dose of 0.1units/kg/hour.

The consultant paediatrician may wish to start some sub cutaneous long acting insulin alongside the IV infusion as there is a school of thought that this shortens the recovery and aids transition back to a maintenance sub cut insulin regime, however there is no strong evidence either way.

The insulin infusion must not be stopped whilst the IV fluids are infusing or this may lead to increasing blood glucose levels.

The following regime should be followed for IV fluids and insulin depending on the blood glucose levels:

Blood glucose > 14mmols/L

0.9% Sodium Chloride containing 20mmols per 500ml

Blood glucose < 14mmols/L

Change the fluid to contain 5% glucose - 500 ml bags of 0.9% sodium chloride with 5% glucose and 20 mmol potassium chloride.

and

Reduce insulin infusion rate to 0.05 units/kg/hr from 0.1 Units/kg/hour (or maintain at that rate if patient initiated on 0.05 units/kg/hr)

Blood glucose < 6 mmols/L

increase the glucose concentration of the intravenous fluid infusion – 500mls bag of sodium chloride 0.9% with 10% glucose and 20mmol potassium chloride.

and

if there is persisting ketosis, continue to give insulin at a dosage of least 0.05 units/kg/hour

Blood glucose < 4 mmols/L

give a bolus of 2 ml/kg of 10% glucose

and

increase the glucose concentration of the infusion. Insulin can temporarily be reduced for 1 hour.

DO NOT stop the insulin infusion while glucose is being infused, insulin is needed to switch off ketosis

Do not allow the patient to have oral fluids until the 48hr rehydration period is completed.

Nursing considerations

All children in DKA should be nursed in a critical care bed, by a nurse with experience in DKA management. Children in DKA often require 1:1 nurse: patient ratio until their condition has stabilised.

Children in severe DKA (pH , 7.10) and those under the age of 2 years are at increased risk of cerebral oedema and must have:

- **half-hourly** neurological observations, (including blood pressure and heart rate) and assessment of level of consciousness (using the modified Glasgow coma score as necessary)

All children in DKA must have:

- strict fluid balance including oral fluids and urine output, using fluid balance charts (urinary catheterisation may be required in young/sick children but should not be done routinely)
- hourly **capillary blood glucose** measurements (these may be inaccurate with severe dehydration/acidosis but are useful in documenting the trends. Do not rely on any sudden changes but re check and send a venous laboratory glucose measurement)
- **capillary blood ketone** levels every 1-2 hours
- hourly BP and basic observations
- hourly level of consciousness initially, using the modified Glasgow coma score
- reporting **immediately** to the medical staff, even at night, symptoms of **headache**, or slowing of pulse rate, or any change in either conscious level or behaviour
- reporting any changes in the ECG trace, especially signs of hypokalaemia, including ST-segment depression and prominent U-waves
- twice daily weight; can be helpful in assessing fluid balance

Cerebral oedema

Children in DKA are at risk of cerebral oedema, a potentially fatal consequence of the fluid shifts seen in DKA. Those who present in severe DKA, and those under 2 are thought to be at greater risk of developing cerebral oedema.

Cerebral oedema is swelling in the brain.

In DKA there are several factors that may contribute to cerebral swelling:

High blood glucose levels have caused a shift in water from the intracellular space to the extracellular space (and then contributed to the increased diuresis) and possibly caused the cell to contract in size. Correction through the administration of IV fluids and insulin is directed at slowly reversing the fluid shift through a gradual change in the osmolality (concentration) of the extracellular fluid, in particular the sodium level. If there are rapid changes in the sodium, water will follow rapidly too, causing the cells to be 'flooded' with water and expand in size rapidly causing the brain to swell.

The correction of an acidotic pH also has an impact; in the brain carbon dioxide is known to cause vasodilation, so in DKA where metabolic acidosis has led to respiratory compensation, and a dramatically reduced PCO2, (sometimes as low as around 1.0) a rapid correction of the pH and associated rapid rise in PCO2 will lead to vasodilation causing constriction and swelling in the brain.

The purpose of very frequent neuro observations is to look for signs and symptoms of cerebral oedema, which leads to raised intracranial pressure. Diagnosis is based on clinical findings, and if suspected should be treated immediately.

Symptoms of cerebral oedema

- Confusion / Reduced conscious level / obtunded
- Severe headache
- Vomiting
- Hypertension
- Bradycardia
- Apnoea or altered respiratory pattern

Treatment for oedema should not be delayed to obtain imaging, however imaging is necessary to rule out other causes of the clinical findings that suggest cerebral oedema or raised intracranial pressure.

If cerebral oedema is suspected, treat immediately with the most readily available of either -

hypertonic saline (2.7% or 3%) 2.5-5 ml/kg over 10-15 minutes
or
mannitol 20% 0.5-1 g/kg over 10-15 minutes

Untreated cerebral oedema will lead to catastrophic raised intracranial pressure.

If a child or young person develops any of these signs:

- deterioration in level of consciousness
- abnormalities of breathing pattern, for example respiratory pauses &/or drop in SaO2.
- oculomotor palsies
- abnormal posturing
- pupillary inequality or dilatation.

Treat immediately for raised intracranial pressure with the most readily available of either -

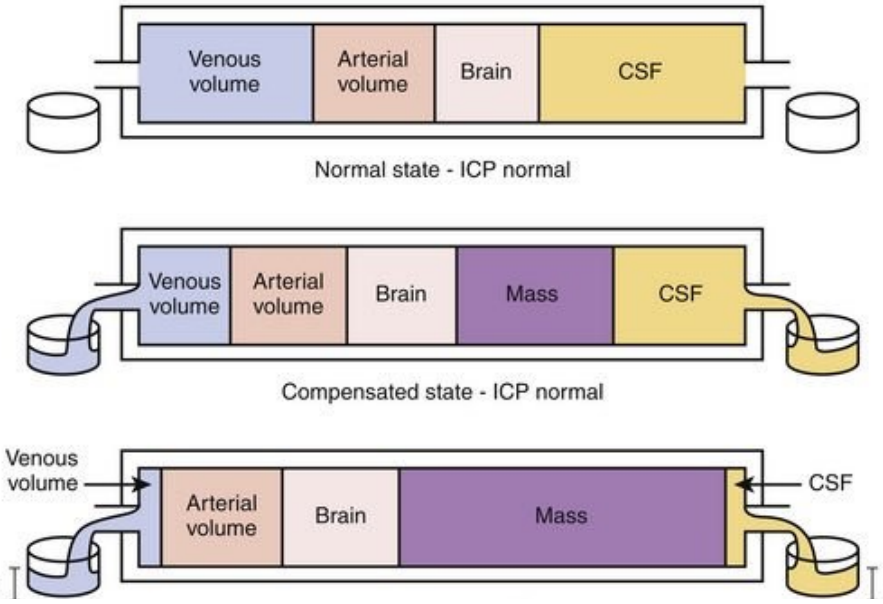
hypertonic saline (2.7% or 3%) 2.5-5 ml/kg over 10-15 minutes
or
mannitol 20% 0.5-1 g/kg over 10-15 minutes

Raised Intracranial Pressure

The skull contains 3 things:

- brain matter
- cerebral spinal fluid (CSF)
- blood (in arteries and veins)

In adults and older children this is a rigid fixed box, the volume of these components must remain constant to avoid complications. If any of these components increases in size or there is an additional component (space occupying lesion, brain bleed, infection) the body will try to compensate by reducing one of the other components as shown in this graphic.



In infants in whom the sutures and fontanelle remain open the skull is less fixed in volume and allows for a small degree of swelling to be accommodated in addition to these mechanisms.

If this happens the infant will have a bulging fontanelle, this should be escalated to a senior doctor without delay.

If left untreated, raised intracranial pressure will result in herniation of the brain stem through the foramen magnum. This is irrevocable brain damage and will cause brain stem death.

The primary signs of critical raised intracranial pressure are known as '**cushings triad'**

- Hypertension
- Bradycardia
- Decreased respirations or apnoea

These are more easily remembered as the opposite to the symptoms seen in shock - hypotension, tachycardia, tachypnoea

Treatment with mannitol or hypertonic saline aims to rapidly reverse the serum osmolality - raising the sodium content of the fluid in the intravascular space will cause water to move from the cells into the circulating volume where it can be diuresed.

For the child in DKA with cerebral oedema or raised intracranial pressure, there should be discussion with a PICU about their management and potential retrieval into a PICU bed.

Other manoeuvres to assist with reducing intracranial pressure should be considered -

- Maintain the patient in a head up posture to promote venous drainage
- Keep the patients head mid line to promote venous drainage
- Maintain good systolic blood pressure using inotropes if necessary. In order to perfuse the brain with oxygen the arterial blood pressure needs to be higher than the intracranial pressure. Hypotension will exacerbate the risk of a brain injury.
- Restrict fluids (after seeking senior advice)

Response to treatment

With careful management a patient who presents in DKA should gradually improve. The blood chemistry (blood glucose and gases) should rectify towards more normal levels, and clinically they should become more responsive and return to their normal self.

If there are fluctuations in the blood glucose or blood gas results, the patient fails to respond to treatment, or responds and then deteriorates this should be escalated to the senior paediatrician.

Consider

- Whether there could be an additional diagnosis, particularly consider sepsis
- Inadequate fluid resuscitation or failure to recognise patient was shocked at presentation.
- Use of recreational drugs or overdose of prescription medications
- Re-doing all the fluid calculations to check they are correct
- Re making all the IV fluids, including insulin.

Discontinuing IV fluids and insulin regime

Once ketones are < 1.0 mmol/l, and oral fluids can be tolerated / nausea and vomiting has ceased, consider switching from intravenous to subcutaneous insulin.

This is usually guided by a senior paediatric doctor, or the diabetes specialist nurse, essentially the principle is that the child receives a dose of subcutaneous insulin appropriate to the food they will eat, then after 30 mins the IV insulin can be stopped.

Further care

Once children are diagnosed as diabetic, or a known diabetic child or young person has presented in DKA they will need close follow up and education with the diabetic specialist nursing team. This may focus around, for known diabetic patients, the factors that may have contributed to the DKA episode, and for newly diagnosed diabetics will include all the education and support that is required following such a diagnosis.

Please remember , be mindful in all your conversations around the bed space and with colleagues that you may be overheard by the patient and or family, consider your language and the way you interact with the family. Potentially they are just absorbing the news of this life changing diagnosis, and will be engaging with health care for the rest of their lives, they need to feel confident in our ability to manage their life long condition and ensure they have the best outcomes.

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Further reading

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