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Managing diabetic ketoacidosis

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Summary

Diabetic ketoacidosis is a common life-threatening complication, which usually occurs in patients with type 1 diabetes, but it may also occur in those with type 2 diabetes during severe concurrent illness, such as sepsis, myocardial infarction or corticosteroid treatment. This article provides an overview of the diagnosis, pathophysiology and management of diabetic ketoacidosis. The differential diagnosis and treatment of hyperosmolar non-ketotic coma is also discussed, as is the nurse's role in promoting and providing health education to patients with diabetic ketoacidosis.

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Blood glucose, diabetes, diabetic ketoacidosis, insulin

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Aims and intended learning outcomes

This article aims to update nurses on the latest guidelines on treating diabetic ketoacidosis. It outlines the pathophysiology of the condition, the aims of treatment, and the differential diagnosis of hyperosmolar non-ketotic coma and diabetic ketoacidosis. After reading this article and completing the time out activities you should be able to:

- Recognise the signs and symptoms of diabetic ketoacidosis.
- Understand the need for maintaining strict fluid balance and accurate blood glucose estimation, with clear documentation of all observations.
- Document and report any deterioration in the patient's condition.
- Administer fluids and insulin as prescribed, accurately recording insulin dose per hour.
- Educate patients with type 1 diabetes on how to prevent diabetic ketoacidosis during concurrent illness or infection.

Time out 1

List the signs and symptoms of hyperglycaemia. What signs and symptoms indicate progression to diabetic ketoacidosis?

Diagnosis

Alberti (1974) defined diabetic ketoacidosis as 'severe uncontrolled diabetes requiring emergency treatment with insulin and emergency fluids with a blood ketone body (acetoacetate and 3-hydroxybutyrate) concentration of greater than 5 mmol/L'. Diagnosis is usually straightforward and symptoms include polyuria and polydipsia, as a result of hyperglycaemia, vomiting, abdominal pain and shortness of breath (Savage and Kilvert 2006). Signs are non-specific and relate to dehydration and acidosis, including tachycardia, hypotension, hyperventilation (Kussmaul breathing), the smell of ketones on the breath and drowsiness or coma. Not everyone is able to smell ketones, so this should not be relied solely on during diagnosis. Criteria for the diagnosis of diabetic ketoacidosis include (Kitabchi *et al* 2006, Savage and Kilvert 2006):

- Raised blood glucose (>11mmol/L) – levels may be normal if large doses of insulin have been given before admission, or the patient has

not eaten adequate amounts of food containing carbohydrate for a long time.

- At least moderate ketonuria or blood ketone levels above 3mmol/L. Note that reagent strips measure beta-hydroxybutyrate, not total ketones.
- Significant acidosis, for example a plasma bicarbonate concentration (capillary or arterial) ≤15mmol/L or an arterial pH <7.3.

Signs and symptoms of severe diabetic ketoacidosis include (Kitabchi *et al* 2006, Savage and Kilvert 2006):

- Reduced consciousness.
- Hyperventilation.
- Hypotension (systolic ≤90mmHg).
- Venous bicarbonate concentration <10mmol/L, arterial pH <7 .

Precipitating factors These include situations that increase the amount of stress hormone production, such as infection, myocardial infarction or surgery. New cases of diabetes and management errors such as inappropriate changes in insulin dosage are also precipitating factors (Savage and Kilvert 2006). The deliberate omission of insulin by the patient, for example because of the fear of weight gain, is a common cause (Kitabchi *et al* 2006). Error in dosage or manipulation of insulin was identified in 42% of young adults and abnormal insulin treatment behaviour, such as discontinuation of insulin, erratic compliance with insulin and eating difficulties, was highlighted as a major cause of diabetic ketoacidosis in young adults with diabetes.

In contrast, 75% of people over 25 years had identifiable precipitants to diabetic ketoacidosis, such as infection or myocardial infarction (Thompson et al 1995). Recreational drugs such as ecstasy, ketamine, methadone and cocaine have also been shown to precipitate diabetic ketoacidosis in people with type 1 diabetes (Lee et al 2009), as do alcohol binges. Historically high rates of diabetic ketoacidosis in people using continuous subcutaneous insulin pumps, when compared with conventional injection therapy, have been reported (Peden et al 1984). With more modern pumps and structured education programmes, most studies show that the frequency of diabetic ketoacidosis is similar for patients using continuous subcutaneous insulin pumps and people on injection therapy. However, diabetic ketoacidosis can develop much more quickly in patients using continuous subcutaneous insulin if the infusion is interrupted because of the much smaller depot of subcutaneous insulin (Pickup and Keen 2002).

Incidence and prognosis

The incidence of diabetic ketoacidosis in Europe and the United States (US) is estimated to be one to five episodes per 100 patients with type 1 diabetes per year. Diabetic ketoacidosis is twice as common in females (Krentz and Natress 2003). Before the introduction of insulin treatment in 1923, diabetic ketoacidosis was fatal. Mortality rates today in Europe and the US vary. Mortality is higher in patients treated by a non-specialist (Levetan *et al* 1999), particularly in the older population (Gale *et al* 1981). Krentz and Natress (2003) estimated the average mortality to be 5-10% in western countries. A consensus statement from the American Diabetes Association suggested a 5% mortality rate (Kitabchi *et al* 2006). Diabetic ketoacidosis was found to be the greatest single cause of death in patients with diabetes under the age of 20 in England and Wales, accounting for 16% of deaths (Tunbridge1981).

Pathophysiology

Diabetic ketoacidosis occurs as a consequence of absolute or relative insulin deficiency. This lack of insulin results in the failure of glucose to enter insulin-sensitive tissues, such as muscle, liver and adipose tissue, while gluconeogenesis (glucose production from non-carbohydrate sources) is suppressed in the liver. As the insulin-sensitive tissues are starved of glucose, the body reacts by increasing production of counter-regulatory hormones, for example glucagon, catecholamines, cortisol and growth hormone, which increase glucose levels in the blood. As there is not enough insulin available to use the glucose, ketones are used to provide energy. Ketones are normal in starvation and can be used as a source of energy by the brain and kidney. In ketoacidosis, ketones are produced faster than they can be used or excreted. Hepatic gluconeogenesis and glycogenolysis (glucose production from glycogen) increase and lipolysis increases the production of free fatty acids that are later metabolised to ketones. These changes occur due to insulin deficiency and increases in counter-regulatory hormones. Lipolysis occurs because the level of insulin is too low to inhibit hormone-sensitive lipase, the key regulator of the breakdown of fat stores. The liver uses free fatty acids as an alternative energy source (ketogenesis) and this results in the accumulation of end metabolities (ketones). Ketones include acetone, beta-hydroxybutyrate and acetoacetate. If these are not reduced, accumulation can lead to metabolic acidosis with a drop in pH and bicarbonate levels.

In an attempt to correct this metabolic acidosis, breathing increases (Kussmaul breathing) to excrete ketone bodies. The presence of ketones induces nausea and vomiting, which further increases fluid and electrolyte loss. Hyperglycaemia results in glycosuria, which raises water and sodium loss, leading to dehydration, thirst and abdominal pain. Potassium moves from the intracellular to the extracellular space because of increased entry of hydrogen ions into cells, which displace potassium ions. Potassium is subsequently lost through vomit and urine. Hence, although there is total body depletion of potassium, plasma potassium levels at presentation are usually normal or high. These levels can fluctuate dramatically during the treatment of diabetic ketoacidosis because insulin promotes uptake into cells. Hypokalaemia increases the risk of cardiac arrhythmias and, on average, body water is depleted by approximately five litres in diabetic ketoacidosis (Krentz and Natress 2003). Contraction of extracellular fluid volume causes a reduction in renal blood flow, which impairs the kidney's ability to clear glucose and ketone bodies. Correction of dehydration is thus as vital as insulin replacement in the acute management of patients with diabetic ketoacidosis.

Acute management

Treatment of patients with diabetic ketoacidosis should commence in hospital, in intensive or high-dependency units if the condition is severe. It involves rehydration with intravenous (IV) fluids, administration of insulin and replacement of electrolytes. The following recommendations are based on a combination of guidelines (Krentz and Natress 2003, Savage and Kilvert 2006, Jerreat *et al* 2009).

Treatment with IV saline and insulin should be started immediately as delay could be fatal. Initial procedures should include:

- Obtaining good IV access.
- Inserting a nasogastric tube if the patient is unconscious.
- Inserting a urinary catheter if the patient is unconscious, incontinent or anuric after two hours.
- Consideration of thromboprophylaxis in older adults or high-risk patients, unless it is contraindicated.

Essential investigations need to be carried out and the nurse should check the following (Krentz and Natress 2003, Savage and Kilvert 2006, Jerreat *et al* 2009):

- Capillary and laboratory blood glucose levels (re-check laboratory glucose after two hours).
- The presence of ketones in blood and urine.
- Urea and electrolytes (re-check after two hours and then four-hourly until stable).
- Venous bicarbonate concentration and blood gases. Arterial gases need to be checked if the patient displays reduced consciousness, is in respiratory distress or is hypotensive (re-check venous bicarbonate as for electrolytes).
- Full blood count. A raised white cell count is common in patients with diabetic ketoacidosis and does not necessarily indicate infection.
- Other investigations such as troponin T, amylase (may be elevated and does not necessarily indicate pancreatitis), blood cultures, culture of urine, throat swab, chest X-ray, electrocardiogram, lumbar puncture and a computed tomography (CT) brain scan may be required.

The nurse should ensure strict fluid balance with hourly monitoring. Capillary blood glucose estimation (until on subcutaneous insulin and then pre-meal and pre-bed) and capillary blood ketone estimation (as per instruction for capillary blood glucose estimation) should be carried out hourly.

Rehydration of patients with IV fluids is essential in the management of diabetic ketoacidosis and should be as follows (Krentz and Natress 2003, Savage and Kilvert 2006, Jerreat *et al* 2009):

- Administration of one litre of 0.9% sodium chloride solution over the first hour.
- Rate of fluids thereafter depends on the age and fitness of the patient, typically:
  – One litre over the following one to two hours.
  – Two litres over the following four hours
  – One litre every four to six hours.
  The rate should be reduced in older adults, in cardiac or renal disease, or in mild diabetic ketoacidosis (plasma bicarbonate concentration >10mmol/L). It is important to note that more rapid infusion increases the risk of respiratory distress syndrome and possibly cerebral oedema.
- Switch to 5-10% glucose over the following eight hours once blood glucose is ≤15mmol/L.
- Continue 0.9% sodium chloride solution in addition to glucose if the patient remains volume depleted. It is important not to switch back to 0.9% sodium chloride if blood glucose increases. Increase the insulin infusion rate. It is lack of insulin, rather than too much glucose, that will be increasing blood glucose levels.

Box 1 provides information on managing potassium levels. Opinion is divided on whether

insulin should be administered via a syringe driver, thus separating the insulin from the glucose and potassium, or whether it is best to use the glucose, potassium and insulin in one bag. There is no evidence to suggest that one method is superior to the other. For the purpose of this article the separate syringe driver method is described.

Time out 2

After two hours on an IV sliding scale regimen of six units of insulin per hour, the patient's laboratory blood glucose has risen. It was 16mmol/L on admission and is now 24mmol/L. How would you manage this situation and what treatment would you recommend?

When administering insulin the following procedure is recommended (Krentz and Natress 2003, Savage and Kilvert 2006, Jerreat *et al* 2009):

- Dilute 50 units of soluble insulin, for example Actrapid, Humulin S, to 50ml of 0.9% sodium chloride in a syringe (as prescribed). Label and ensure the insulin is witnessed and signed for.
- Infuse intravenously using a syringe driver, starting at six units per hour.
- Ten units of soluble insulin should be given intramuscularly if venous access is proving difficult or a delay, for example regarding venous access or setting up the insulin infusion, is anticipated.
- Laboratory venous blood glucose should be checked at two hours if blood glucose has not fallen. Pump and IV connections should be checked and, if satisfactory, the rate of insulin should be increased to ten units per hour.
- When blood glucose is ≤15mmol/L, the infusion rate and fluids should be changed to a variable rate/sliding scale regimen (Box 2).

In the unit where the author works, it has been found that basing the rate of insulin that is infused on the patient's usual insulin requirements ensures that particularly insulin-resistant patients, such as pregnant women or obese patients, are not given too little. For example, if a patient usually requires 100 units of insulin a day or four units an hour it seems unlikely that two units an hour will control his or her blood glucose adequately.

Bicarbonate replacement is controversial in the treatment of diabetic ketoacidosis and it is generally not used. Small doses may, however, be beneficial if pH is less than 7.0 and cardiorespiratory collapse appears imminent (Krentz and Natress 2003). Savage and Kilvert (2006) recommended that it should only be used in intensive care units or in a high dependency setting.

Time out 3

When should sliding scale insulin be stopped? Identify how you would implement this.

Discontinuing intravenous insulin

IV insulin should be discontinued once the patient is able to eat and drink normally, and ketones are negative or trace (≤0.6mmol/L on the optimum blood ketone meter). In the unit where the author works, it is recommended that if the patient has pre-existing type 1 diabetes, the basal insulin should be continued subcutaneously while the patient is receiving IV insulin. This ensures that there is basal or background insulin available when the IV insulin is stopped, making ketones less likely to reappear. IV insulin should not be stopped until 30 minutes after subcutaneous insulin has been given with a meal. Patients with established diabetes should return to their previous regimen, which the diabetes specialist nurse or diabetes medical team should be asked to review. They should also try to identify the cause of diabetic ketoacidosis and provide educate the patient on how to deal with concurrent illness and recreational drugs and alcohol use. This may prevent further episodes occurring.

If the patient with diabetic ketoacidosis was previously undiagnosed with diabetes then a subcutaneous dose of insulin should be calculated using the total IV insulin requirements infused over the last 24 hours. This highlights the importance of good

BOX 1

Potassium replacement

- Do not add to the first litre of fluid infused.
- Establish potassium level (exclude hyperkalaemia).
- Use pre-mixed potassium solutions.
- Monitor potassium every two hours until stable and then monitor daily.

Serum potassium (mmol/L)	Potassium chloride to be added to each litre of fluid
>5.5	Nil, recheck potassium in two hours
3.5-5.5	20mmol/L
3.0-3.4	40mmol/L
<3.0	Higher rates of potassium should be administered in the intensive care unit.

(Jerreat *et al* 2009)

documentation of insulin rates. If the total daily dose in the previous 24 hours was 48 units, a regimen of 50% background or basal insulin and 50% quick acting insulin could be given. For example, Humalog eight units three times a day and glargine 24 units or NovoRapid eight units three times a day and Levemir 12 units twice a day.

Complications

Treatment of diabetic ketoacidosis is usually uncomplicated and successful. However, some complications may occur (Krentz and Natress 2003, Savage and Kilvert 2006, Jerreat *et al* 2009):

- Hypoglycaemia and hypo/hyperkalemia – blood glucose levels and potassium levels should be checked as suggested previously, and insulin, carbohydrate intake and potassium should be adjusted as required.
- Renal failure – strict fluid balance should be maintained.
- Aspiration – a nasogastric tube should be passed in to prevent aspiration of stomach content by an unconscious patient.
- Cerebral oedema – this is rare in adults and results from the excessive entry of water into central nervous system cells, causing swelling to the brain. Signs and symptoms include headache, bradycardia, rising blood pressure, decreased consciousness, restlessness, irritability and convulsions. Management should exclude hypoglycaemia. The consultant should be informed and the patient should be transferred to the intensive care unit. Mannitol 20% 5ml per kg of body weight should be administered intravenously over 20 minutes, and a CT scan of the head should be carried out to exclude other causes.
- Thromboembolic complications such as venous thrombosis, stroke and rarely disseminated intravascular coagulation – prophylactic anticoagulation is recommended in some areas (enoxaparin sodium or heparin is recommended in the trust policy where the author works), but not by all. It is not recommended routinely in the management of diabetic ketoacidosis by Krentz and Natress (2003) and Savage and Kilvert (2006).
- Adult respiratory distress syndrome – this may be caused by excessive fluid replacement and rapid correction of osmolarity, leading to shifts in intracellular fluid. Signs and symptoms include dyspnoea, tachypnoea, central cyanosis and arterial hypoxia. Respiratory support with intermittent positive pressure ventilation may be required.

BOX 2

Calculations for variable rate/sliding scale insulin

- Infuse 5-10% glucose, ideally one litre of 10% glucose over eight hours.
- If the patient has been diagnosed with diabetes and receives insulin therapy, establish the patient's total daily dose (TDD) requirements of insulin (include all types used) and divide the total by 24 to give the basal rate. For example, eight units of Humalog or NovoRapid pre-meal equals 24 units of quick acting insulin plus 24 units of glargine pre-bed, giving a TDD of 48 units, making the basal rate two units per hour.
- If the patient has not been treated with insulin previously, assume the basal rate is two units.

Blood glucose (mmol/L)	Rate of insulin (units/hour)	Notes
3.9 or less	0.5	
4.0-6.9	2	Basal rate
7.0-9.9	3	Basal rate x 1.5
10.0-16.9	4	Basal rate x 2
≥17	6	Basal rate x 3 (and adjust scale)

(Jerreat *et al* 2009)

Time out 4

Sarah is 32 years old and has had type 1 diabetes for eight years. She is now an inpatient with diabetic ketoacidosis. It appears that a bout of influenza precipitated her admission. She had decreased her insulin dose as she was eating very little. How would you educate Sarah with regard to diabetes management during any future episodes of illness.

Prevention of diabetic ketoacidosis

All patients with type 1 diabetes should be given general advice on how to manage concurrent illness. Advice and sick day rules should include:

- Seek prompt medical treatment, for example antibiotics for infections and paracetamol or aspirin to relieve pyrexia, if necessary.
- Cough mixtures and cold remedies should be sugar-free.
- If unable to eat normally, carbohydrates should be replaced with cereals, soups or liquid carbohydrate. Withholding carbohydrates because of high blood glucose levels may make ketosis worse.
- Drink plenty of sugar-free liquids.
- If vomiting and unable to tolerate liquid carbohydrate, medical attention should be sought immediately.
- Insulin should be continued even if not eating usual amounts of food.

- It is likely that insulin doses will need to be increased during illness.
- Blood glucose, particularly if treated with insulin, should be tested more frequently, at least four times a day.

The aim of management and sick day rules is to prevent diabetic ketoacidosis, and it is likely that the insulin dose will need to be increased considerably. Historically, patients were told to test blood glucose levels before each meal and before bed, and to inject usual doses of insulin with additional doses of rapid-acting insulin at these times if blood glucose is greater than 13mmol/L. Urine or blood ketones are tested if blood glucose is 17mmol/L or above. Ideally, the doses of extra rapid-acting insulin were based on total daily insulin requirements, but usually ranged from six to ten units. Ten units are given if blood glucose is 20mmol/L or above, or if moderate or large ketones are present in the urine or blood (Jerreat 2003).

If the patient has completed the Dose Adjustment For Normal Eating (DAFNE) course, the advice differs and the amount of additional quick-acting insulin is dependent on the level of ketones, rather than blood glucose levels. If ketones are negative, trace, or less than 1.5mmol/L on an optimum meter, glucose and ketones are tested every four to six hours and corrective doses of insulin are used (one unit of insulin to reduce blood glucose by 2-3mmol/L, although it is acknowledged that slightly higher doses may be required). If ketones are higher and if ketones are moderate in the urine or 1.5mmol/L in the blood, and blood glucose is greater than 13mmol/L, 10% of the total daily dose of insulin is given every two hours in addition to the usual background dose and amounts needed to cover carbohydrate eaten in meals. If ketones are greater than 3mmol/L or large, 20% of the total daily dose should be given every two hours in addition to usual doses (DAFNE 2007).

Follow up

It is important, where possible, to establish the underlying cause of diabetic ketoacidosis. If it was infection, how the patient managed it, and how that could be improved, should be discussed in an open and non-judgemental way. Those who have had diabetes for some time may feel embarrassed if they have not 'followed the rules'. Some may be newly diagnosed and take comfort from the fact that diabetic ketoacidosis can be prevented in the future. Others experience repeated admissions with the condition. In these cases, it is important to establish any of the patient's patterns or problems. Omitting insulin to become thin is common particularly in young women (Hurel et al 1997), and may affect compliance with an insulin regimen. Hurel et al (1997) claimed that at least one third of patients with diabetic ketoacidosis has problems with personality, for example eating disorders, or wishing to spend time in hospital, or compliance and social, domestic or psychiatric problems. Many diabetes services do not have access to psychologists and patients do not always obtain specialist support.

Ketosis-prone type 2 diabetes

Ketosis-prone type 2 diabetes has previously been termed 'flatbush diabetes', 'atypical diabetes' and 'type 1.5 diabetes' (Umpierrez *et al* 2006). Umpierrez *et al* (2006) described these patients as having varying degrees of insulin deficiency, no evidence of autoimmunity and their absolute requirement for insulin as variable 'coming and going'. Aggressive insulin treatment results in significant improvement in beta-cell function, and insulin can usually be discontinued within a few months. This period of remission can last for a few months to years and patients require education on ketone testing and sick day management. The condition is most common in African and Hispanic groups, although the reason for this is unknown (Umpierrez 2006).

Hyperosmolar non-ketotic coma

This condition occurs in patients with type 2 diabetes (usually in people over 60 years) and is characterised by hyperglycaemia and high plasma osmolality without significant ketones or acidosis. Blood glucose levels may be extremely high, usually more than 50mmol/L. The condition often occurs in previously undiagnosed patients . Diagnosis of hyperosmolar non-ketotic coma is confirmed by marked hyperglycaemia and with a calculated osmolarity (2{Na +K} + glucose) >350mosmole/L (Savage and Kilvert 2006). However, patients may be acidotic for other reasons such as sepsis, renal failure or cardiovascular collapse (Table 1). Management is the same as for diabetic ketoacidosis, with the following exceptions (Krentz and Natress 2003, Savage and Kilvert 2006, Jerreat *et al* 2009):

- If serum sodium is >155mmol/L, 0.45% sodium chloride rather than 0.9% should be considered initially.
- The risk of thromboembolic disease is high and patients should be given an anticoagulant.
- Older patients should have a central venous pressure catheter inserted.

- Lower rates of insulin infusion may be required as patients are often insulin sensitive.
- Although subcutaneous insulin may be required for some weeks, in the longer term patients will usually be managed with diet and oral agents.

Conclusion

The treatment of diabetic ketoacidosis needs to be rapid and is based on fluid, electrolyte and insulin replacement. It is vital to note any deterioration in the patient's condition. Strict fluid balance and observations of potassium, blood glucose, ketone levels, blood pressure, respirations, pulse, temperature and level of consciousness are vital. To prevent ketosis IV insulin administration should be continued for 30-60 minutes after subcutaneous insulin has been given. Where possible, reasons should be sought for diabetic ketoacidosis and patient education and support should be provided in a non-judgemental way **NS**

Time out 6

Now that you have completed the article you might like to write a practice profile. Guidelines to help you are on page 60.

TABLE 1

Differential diagnosis of hyperosmolar non-ketotic coma and diabetic ketoacidosis

Hyperosmolar non-ketotic coma	Diabetic ketoacidosis
Patients tend to have a high plasma sodium concentration.	Plasma sodium concentration usually normal or low.
Blood glucose >50mmol/L.	Blood glucose may be only slightly elevated and usually <40mmol/L.
Negative or small quantities of ketones in the urine and/or blood.	Moderate or large quantities of ketones in urine and/or blood.
Plasma bicarbonate usually ≥18mmol/L.	Low plasma bicarbonate usually <15mmol/L.
Slow onset of hyperglycaemic symptoms.	Rapid onset of hyperglycaemic symptoms.
Symptoms rarely include abdominal pain, vomiting or Kussmaul breathing (unless acidotic).	Symptoms include vomiting, abdominal pain and Kussmaul breathing.
If previously undiagnosed with diabetes, the patient is more likely to be over 60 years old.	A patient previously undiagnosed with diabetes is more likely to be, but not exclusively, under 30 years old and normal weight.

(Jerreat 2003)

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Diabetic ketoacidosis

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This self-assessment questionnaire (SAQ) will help you to test your knowledge. Each week you will find **ten multiple-choice questions that are broadly linked to the learning zone article.** Note: There is only one correct answer for each question.

Ways to use this assessment

- You could test your subject knowledge by attempting the questions before reading the article, and then go back over them to see if you would answer any differently.
- You might like to read the article to update yourself before attempting the questions.

Prize draw

Each week there is a draw for correct entries. Please send your answers on a postcard to: Zena Latcham, Nursing Standard, The Heights, 59-65 Lowlands Road, Harrow-on-the-Hill, Middlesex HA1 3AW, or send them by email to: zena.latcham@rcnpublishing.co.uk

Ensure you include your name and address and the SAQ number. This is SAQ no. 540. Entries must be received by 10am on Tuesday May 11 2010.

When you have completed your self-assessment, cut out this page and add it to your professional portfolio. You can record the amount of time it has taken. Space has been provided for comments.

You might like to consider writing a practice profile, see page 60.

1. Diabetic ketoacidosis is:

a) Controlled diabetes requiring daily treatment with insulin ❑
b) Uncontrolled diabetes requiring emergency treatment with insulin and rehydration ❑
c) More common in males ❑
d) More common in patients with type 2 than type 1 diabetes ❑

2. Signs of diabetic ketoacidosis include:

a) Hypotension ❑
b) Hyperventilation ❑
c) Tachycardia ❑
d) All of the above ❑

3. The average mortality rate of patients with diabetic ketoacidosis in western countries is:

a) 5-10% ❑
b) 10-20% ❑
c) 20-30% ❑
d) 30-40% ❑

4. Which of the following should not be relied on when diagnosing diabetic ketoacidosis

a) Polyuria ❑
b) Smell of ketones on the breath ❑
c) Polydipsia ❑
d) Drowsiness ❑

5. On average, how much body water is depleted in diabetic ketoacidosis?

a) 1 litre ❑
b) 3 litres ❑
c) 5 litres ❑
d) 7 litres ❑

6. When diabetes ketoacidosis is suspected the nurse should check:

a) Capillary and laboratory blood glucose ❑
b) Venous bicarbonate ❑
c) Urea and electrolytes ❑
d) All of the above ❑

7. Signs and symptoms of cerebral oedema include:

a) Rising blood pressure ❑
b) Decreasing blood pressure ❑
c) Reduced swelling in the brain ❑
d) Alertness ❑

8. How often should glucose and ketones be tested if ketones are less than 1.5mmol/L?

a) Every 1-2 hours ❑
b) Every 2-4 hours ❑
c) Every 4-6 hours ❑
d) Every 6-8 hours ❑

9. Which of the following is not a characteristic of hyperosmolar non-ketotic coma?

a) High plasma sodium concentration ❑
b) Rapid onset of hyperglycaemic symptoms ❑
c) Negative or low quantities of ketones in the urine and/or blood ❑
d) Plasma bicarbonate 18mmol/L or above ❑

10. Urine or blood ketones are tested if blood glucose is:

a) 5mmol/L ❑
b) 10mmol/L ❑
c) 15mmol/L ❑
d) 17mmol/L or above ❑

This self-assessment questionnaire was compiled by Tanya Fernandes

The answers to this questionnaire will be published on May 12

Report back

This activity has taken me ____ hours to complete.

Other comments:

_____

_____

_____

Now that I have read this article and completed this assessment, I think my knowledge is:

Excellent ❑
Good ❑
Satisfactory ❑
Unsatisfactory ❑
Poor ❑

As a result of this I intend to:

_____

_____

_____

Answers to audit questions

The answers to SAQ no. 538 on audit, which appeared in the April 14 issue, are:

1. d 2. c 3. a 4. c 5. b
6. b 7. d 8. b 9. a 10. a