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Diabetic emergencies in adults

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Aim and intended learning outcomes

The aim of this article is to improve knowledge and understanding of the three main diabetic emergencies that nurses encounter in clinical practice. Through relevant identification, quick and appropriate intervention should follow. After reading this article you should be able to:

- Understand the control of blood glucose.
- Identify the causes of diabetic ketoacidosis (DKA), hyperosmolar non-ketotic syndrome and hypoglycaemia.
- Prioritise the nursing assessment and intervention for each diabetic emergency.
- Understand the fundamental principles of fluid resuscitation and management in DKA and hyperosmolar non-ketotic syndrome.
- Discuss the management of a patient who has hypoglycaemia.
- Consider health promotion strategies to help minimise and prevent such emergencies occurring.

Introduction

Some diabetic emergencies are quick in onset while others are more insidious, but all could have fatal consequences if they are not identified promptly and treated appropriately. Anecdotal evidence suggests that many of these emergencies are preventable, mainly through patient education. Thus the role of the nurse as a health educator and promoter is essential. However, to date there have been no major studies or implications for nurse education in the literature relating to health promotion and prevention of diabetes (Hjelm *et al* 2003).

Control of blood glucose

Through a process of metabolism, carbohydrates and fats are used and stored for cellular energy. Glucose is essential to maintain brain, renal medulla and erythrocyte energy sources. Digestive enzymes break carbohydrates and fats down into smaller molecules. They are then absorbed into the bloodstream via the lymphatic system (causing a rise in blood concentration of glucose). Amino acids (a product of protein metabolism), fats and excess carbohydrates are stored as fat (fat tissue) or glycogen (in the liver) (Marieb 2004).

The pancreas is the organ involved in regulation of blood glucose. Insulin and glucagon are two hormones that are central in the metabolism of carbohydrate and in the control of blood glucose. These hormones, including pancreatic polypeptide, are secreted by the islets of Langerhans in the pancreas. The islets of Langerhans are clusters of endocrine cells that secrete glucagon via the alpha cells, insulin via the beta cells and pancreatic peptide via the pancreatic cells (Waugh and Grant 2001). Their harmonious functioning determines hyperglycaemia, hypoglycaemia or normal glucose levels (3.6-5.8mmol/l) (Kruszynska 2003).

After a meal as much as two-thirds of the glucose from the intestine is stored in the liver (Guyton and Hall 2000) in the form of glycogen. The blood glucose level is kept constant by breaking down the glycogen stores in the liver and by the synthesis of new glucose. Insulin is required for these adjustments to be made. Insulin promotes glucose uptake and synthesis of proteins, carbohydrates and lipids. It is released by the rise or surge of serum glucose during a meal. Glucagon, growth hormone and catecholamines are released when the blood glucose is low. Insulin binds to the plasma membrane of the target cell, increasing the permeability of the cell to glucose and resulting in an increased uptake of glucose. Excessive glucose is stored as glycogen or converted to fat in the adipose tissue cell (Ganong 2003). Figure 1 provides a summary of blood glucose regulation.

In brief

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Summary
In this article the author discusses three diabetic emergencies: diabetic ketoacidosis, hyperosmolar non-ketotic syndrome and hypoglycaemia, which all require prompt recognition and appropriate intervention.

Key words
- Diabetes
- Diabetes: health promotion
- Emergencies

These key words are based on subject headings from the British Nursing Index. This article has been subject to double-blind review.

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TIME OUT 1

1. Draw a flowchart indicating the metabolism of glucose and the control of blood glucose.

2. Using the information that you have read and your clinical experience, what symptoms do you think may occur in patients who have a high or a low blood glucose level?

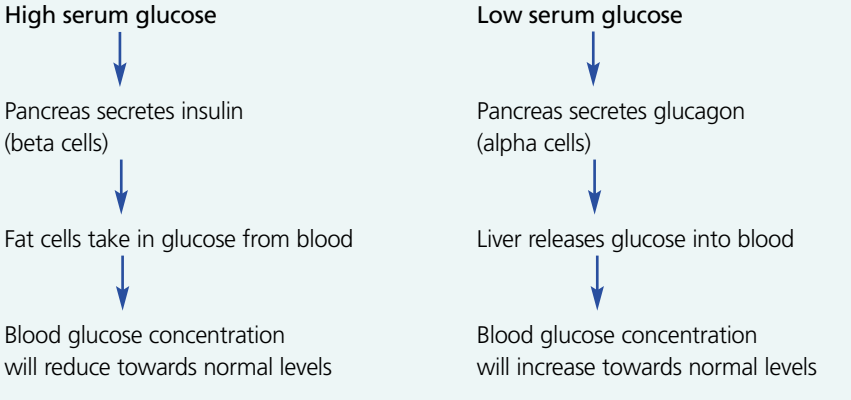
Diabetes mellitus

Diabetes mellitus is a growing, global, public health problem. The World Health Organization (1997) estimates that the problem could escalate to an estimated 300 million people by 2025. This is a burden for the affected individuals and for the societies in which they live (Zimmet *et al* 2001).

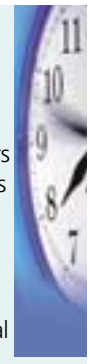
Diabetes mellitus is categorised into two types: type 1 and type 2. Type 1 usually occurs in childhood and adolescence. It results from a cell-mediated autoimmune destruction of the beta cells of the pancreas (Expert Committee 1997). Diabetes refers to the absence or inadequate production of insulin. Insulin is required to enable the uptake of glucose in the cells. Type 2 diabetes accounts for more than 80 per cent of cases with more than one million people diagnosed in the UK (Audit Commission 2000). It usually occurs in older, often overweight, individuals (Katsilambros and Tentolouris 2003). This type of diabetes is now becoming increasingly more common among adolescents and children (Rosenbloom *et al* 1999), particularly in people of African, American and Indian descent. Some of the risk factors include obesity, reduced physical activity, family history and genetic factors that influence insulin resistance. The management of type 2 diabetes includes modification of lifestyle (Katsilambros and Tentolouris 2003), for example, reducing fat intake, particularly for the obese with a higher intake of complex carbohydrates and increased physical activity. It can also include oral hypoglycaemic medication and possibly insulin.

Gestational diabetes mellitus occurs when there is a degree of glucose intolerance related to pregnancy. If left untreated, fetal macrosomia, hypoglycaemia, hypocalcaemia and hyperbilirubinaemia may occur. In addition, mothers may develop hypertension (James *et al* 1999).

Figure 1. Summary of blood glucose regulation



TIME OUT 2



Before reading the next section, consider the following scenario (also reflecting on your own experiences), then answer the question below. Veronica, who is 18 years old, has type 1 diabetes mellitus. She was diagnosed three years ago. She has had several admissions to hospital with diabetic ketoacidosis. Recently she has been feeling unwell and has a two-day history of fever and sore throat. Her visual appearance indicates dehydration, her skin is thin and dry, and her tongue is furred.

On admission she is semiconscious. The following observations were recorded:

- Glasgow Coma Scale: 12
- Pulse: 140 beats/minute
- Blood pressure: 90/55mmHg
- Respirations: 30/minute (Kussmaul breathing)
- Oxygen saturation: 85 per cent (on air)
- Blood values (normal ranges in brackets) pH 7.12 (7.35-7.45)
- PaO_2 18.4 (11.5-13.5kPa)
- HCO_3 18mmol/l (22-26mmol/l)
- $PaCO_2$ 2.1 (4.5-6.0kPa)
- Base excess 24.6

Urinalysis indicates ketones ++++ and glucose ++

Her clinical symptoms and laboratory results confirm a diagnosis of diabetic ketoacidosis. From the assessment data relate Veronica's signs and symptoms to the underlying physiological changes and identify her main problems.

Diabetic ketoacidosis

DKA is a common complication of diabetes mellitus (mainly type 1); approximately one-quarter of DKA cases occur in people with newly presenting type 1 diabetes and for many it is avoidable (NHS 2001). DKA may also occur in type 2 diabetes. DKA results from severe insulin deficiency leading to the disordered metabolism of proteins, carbohydrates and fats.

The outcomes of such pathological actions are hyperglycaemia, hyperosmolality, ketoacidosis and volume depletion (Lewis 2000). DKA is the largest single cause of death among those with diabetes aged less than 20 years (Marinac and Mesa 2000). DKA constitutes an emergency, life-threatening condition in which the onset usually occurs in hours or sometimes days. Patients suspected of DKA should be appropriately and quickly assessed by observing for symptoms, for example, glycosuria, ketonuria and reduction in blood pressure, to confirm diagnosis so that treatment can be started as soon as

possible. The ability of healthcare professionals (including nurses) to recognise the clinical features of DKA profoundly affects outcome and survival rates (Gillespie and Campbell 2002).

Precipitating factors Accidental or deliberate omission of insulin, for example, patients undergoing surgery, can precipitate DKA. Some patients may omit insulin during acute illness such as severe infection (Kitabchi *et al* 2001). Sometimes patients who are ill are treated with inappropriate or slow-acting insulin or may receive incorrect advice from healthcare professionals about stopping insulin. Patients may have a history of uncontrolled insulin-dependent type 1 diabetes. Serious medical illness and surgery can produce a state of increased insulin resistance and relative insulin deficiency (Votey and Peters 2004), resulting in hyperglycaemia. Other causative factors include anything that would initiate a physiological or psychological stress response, for example, sepsis.

The stress response relates to a cascade of metabolic and neurohormonal changes that create a defensive mechanism for coping to take place. Stress-induced hyperglycaemia is attributed to hypersecretion of the counter-regulatory hormones: catecholamines (adrenaline [epinephrine] and noradrenaline [norepinephrine]), glucagon, cortisol and growth hormones that antagonise the effect of insulin by increasing glucose production (Ganong 2003). The effect is a surge in circulating glucose derived from glycogenolysis (conversion of glycogen into glucose) and impaired use of glucose (hyperglycaemia impairs glucose use and residual insulin secretion).

Patients who are insulinopenic have reduced levels of insulin secretion and are unable to increase insulin production (occurs more commonly in type 1 but can occur in type 2). Secretion is inadequate to counteract these intense catabolic effects. Autonomic physiological responses also occur, that is, sympathetic nervous activity is increased affecting the cardiovascular system. This results in increased heart rate, respiratory rate and blood pressure. The patient's physiological response to stress can cause considerable added strain on already failing organs.

Clinical features These can be categorised as general (for example, hyperglycaemia, polyuria, nocturia) and severe (for example, signs of metabolic acidosis, large amounts of ketones in the urine). Classical symptoms of DKA are shown in Box 1. In the older patient, symptoms can be less overt. The main reason for this is that some symptoms may be equated to disorders of ageing and other diseases may mask symptoms, for example, urinary frequency may be associated with prostatism in men or infection in women. Kussmaul breathing may be mistaken for respiratory problems associated with severe heart failure or chest infection.

Clinical features of DKA include: metabolic acidosis, hyperglycaemia, glycosuria, ketones in the blood and urine, Kussmaul respirations, dehydration and electrolyte imbalances.

Metabolic acidosis The severity of the condition is determined by the pH of the blood. Young and Oh (2003) provide a widely accepted criterion for severe DKA: pH less than 7.30, bicarbonate (HCO_3) less than 15mmol/l, blood glucose greater than 14mmol/l and ketonuria (Charalambos *et al* 1999).

The normal range for pH is 7.35-7.45. This is maintained by a buffer system, namely, bicarbonate. The buffer system neutralises acids by combining with excess hydrogen ions. Bases are neutralised by releasing hydrogen. The weak acid (carbonic acid) pairs with bicarbonate and then combines with a strong acid, such as hydrochloric acid, to weaken them (Schell and Puntillo 2001). If the concentration of hydrogen ions continues to rise, levels of bicarbonate (acting as a buffer) become insufficient to maintain normal pH levels, causing metabolic acidosis (Abelow 1998).

Metabolic acidosis is a clinical disturbance characterised by an increase in total body acid. It causes a disruption of the body's acid base balance and, as a result, creates an imbalance in the pH level. Ketone bodies accumulate in the blood, lowering the blood pH level and leading to metabolic acidosis (Waugh and Grant 2001).

Clinical features of metabolic acidosis include headache and lethargy (early symptoms). Severe acidosis can cause deep, rapid respirations – the respiratory system is stimulated to increase the excretion of carbon dioxide in an attempt to restore acid base balance – dysrhythmias and coma (Huether 1998).

Hyperglycaemia Osmotic diuresis causes large volumes of water and electrolytes to be lost. Dehydration depletes the intracellular compartment (this is the largest of the body's fluid spaces) and an increased serum osmolality causes movement of water out of the cells. Hyperosmolality causes a reduction in the level of the patient's consciousness, possibly leading to coma. Counter-regulatory hormones are released and their catabolic action further exacerbates the hyperglycaemia (Ganong 2003). Box 2 summarises problems associated with hyperglycaemia.

Glycosuria This occurs when the renal threshold for glucose of 10mmol/l is exceeded, and up to 200g of glucose is lost in the urine per day (Young and Oh 2003). Glycosuria and hyperglycaemia create a favourable medium for the growth of yeast organisms, consequently the patient may complain of pruritus (itching), particularly around the genitalia.

Ketones in the blood and urine Insulin deficiency prevents the normal use of serum glucose, leading to cellular starvation. The unmet energy requirements of the cells stimulate gluconeogenesis (increased hepatic glucose synthesis from amino acids) and glycogen conversion in the liver through the release of counter-regulatory hormones. The body is forced to break down fat and protein stores to meet energy requirements, exceeding the required amount (Guyton and Hall 2000).

Kussmaul respirations This type of breathing results

Box 1. Classical symptoms of diabetic ketoacidosis

- Abdominal pain, usually generalised or epigastric. Some symptoms may imitate gastrointestinal problems, making diagnosis more difficult
- Rigid abdomen and irregular bowel sounds
- Polyuria
- Polydipsia
- Nocturia
- Blurring of vision
- Leg cramps
- Nausea and vomiting
- Infection

(Young and Oh 2003)

from a low pH, which stimulates the respiratory centre, producing an increased rate and depth of breathing (Ganong 2003). Hyperventilation is the body's attempt to excrete carbon dioxide to compensate for acidosis. The odour of the breath smells fruity because small quantities of acetoacetic acid in the blood (which increases in severe diabetes) are converted to acetone. This is volatile and vaporised into expired air, hence being smelt on the breath of the patient (Guyton and Hall 2000).

Dehydration Glucose exerts a large amount of osmotic pressure in the extracellular fluid and if the glucose concentration rises excessively this can cause considerable dehydration (Guyton and Hall 2000). Between five to six litres of free water can be lost (Simpson 2001). Fluid is also lost via hyperventilation, nausea, vomiting, increased perspiration and decreased intake, leading to dehydration, hypotension and hypovolaemic shock. Compensatory mechanisms stimulated by hypovolaemia aid the maintenance of hydration in the short term. However, if the patient is vomiting and therefore unable to replace lost fluids orally, his or her condition will quickly worsen, and if not treated promptly, he or she may develop acute renal failure (Moore 2004).

Electrolyte imbalances Low serum concentrations of potassium, phosphate and magnesium can cause cardiac dysrhythmias, including asystole. Hypokalaemia can result from haemodilution following fluid resuscitation and inadequate potassium replacement. Initially, plasma potassium is decreased. However, as the vascular volumes fall, renal function will be affected, and as a result the excretion of potassium will also be affected (excretion of potassium relies on exchange of sodium), causing the level of potassium to rise (Hudak *et al* 1998). Vomiting will also worsen electrolyte loss and imbalances, especially potassium, precipitating hypokalaemia and possible cardiac complications. Sodium plasma concentrations are usually normal or slightly low (130-140mmol/l). Hyponatraemia is caused by the movement of water from the intracellular compartment to the extracellular compartment, via the osmotic gradient. Hypophosphataemia (low serum phosphate), which is also present in DKA, can cause muscle weakness, malaise, confusion, respiratory failure and decreased oxygen delivery (Clark 2000). Diagnostic findings of DKA can be seen in Box 3.

Box 2. Problems associated with hyperglycaemia

It is important that glucose concentrations are well controlled and are not permitted to become too raised for the following reasons:

- Cellular dehydration can be caused by glucose exerting a large amount of osmotic pressure in the extracellular space
- Glycosuria is caused by an excessively high glucose concentration
- Glycosuria causes increased fluid loss by the kidneys which can lead to fluid and electrolyte depletion

(Guyton and Hall 2000)

TIME OUT 3

- After reading the above text identify the nursing management for Veronica.
- Briefly outline a care plan, listing the complications that could occur and strategies to help prevent these occurring.
- Compare your notes with the following text.

Management Patients who present with severe DKA are usually semiconscious with marked hypotension, severe acidosis or electrolyte disturbance. The first 24 hours are critical and require close monitoring by nurses and medical staff. Managing the patient with severe DKA may present a challenging situation.

The *National Service Framework for Diabetes* (DoH 2001) outlines a proactive approach and advocates health education before the development of DKA, thus trying to avoid the situation. It is, however, unhelpful in its guidance when such an emergency occurs (Moore 2004). Local protocols exist in trusts and nurses are encouraged to become familiar with the guidance offered.

Generally, the aims of treatment are to:

- Decrease serum glucose.
- Correct dehydration.
- Correct metabolic acidosis.
- Identify precipitating causes.

If the patient is unconscious, airway assessment and management are vital. If the patient is vomiting, intubation may be required to protect the airway. Vital signs such as blood pressure, pulse pressures, mean arterial pressures and central venous pressure (CVP) should be closely monitored. This may be performed initially every 15-30 minutes depending on the severity of the patient's condition. The patient should also be monitored for cardiac dysrhythmias associated with initial hyperkalaemia (peaked T waves; widening QRS complex; prolonged PR interval; and flattened or absent P waves). Hypokalaemia may show depressed ST segments; flat or inverted T waves; or increased ventricular dysrhythmias. Continuous observation can be achieved using a cardiac monitor. Potassium analysis should be conducted at regular intervals for hyperkalaemia and hypokalaemia, as these conditions can have potentially fatal consequences (life-threatening dysrhythmias) (Urden and Stacey 2000).

Resuscitation involves fluid and electrolyte replacement in addition to the administration of insulin. For treatment to prove successful, the underlying cause should wherever possible be identified and treated.

Fluid replacement By rehydrating the patient, hyperglycaemia should be reduced as a result of improvement in glomerular filtration. Replacement of fluid should take account of the patient's age, degree of dehydration and history of cardiac disease (Hand 2000).

In severe dehydration, fluid is lost initially from the largest spaces (intracellular). Rehydration using a similar sodium concentration may be used, for example hypotonic saline 0.45% (Harrison and Daly 2001). Some patients may develop hypovolaemic shock. Fluid replacement should be governed by CVP readings. In clinical practice there appears to be a consistent approach to fluid replacement. The choice of fluid is isotonic saline (0.9% saline) via rapid infusion to restore renal blood flow. During the first one to two hours, 1-2 litres is given (Charalambos *et al* 1999) and a total of 6-8 litres is administered in the first six to eight hours.

Hypotonic saline may be used if sodium levels are greater than 160mmol/l and then only 1 litre is infused over eight hours. Plasma expanders may be indicated in some cases if diastolic pressures are less than 100mmHg. In the case of hypernatraemia, half-strength normal saline is used – 0.45%. Fluid can be replaced using colloids. Colloids aid fluid retention in the intravascular space (only one-quarter to one-third of crystalloid remains in the intravascular space, the remainder enters the interstitial space), depending on the fluid as well as capillary permeability. However, resuscitating the severely hypovolaemic patient with crystalloid fluids only may cause a massive expansion in the intracellular spaces and can result in peripheral, pulmonary or cerebral oedema. When blood glucose levels are less than 15mmol/l, infusion fluid should be changed to 5% dextrose (Fleckman 1993).

Attention should be paid to fluid balance as impaired renal function, particularly in older patients, could lead to circulatory overload or prolonged hypotension. Nurses should monitor the patient's fluid balance by completing a fluid balance chart hourly. Clinical signs of dehydration should also be noted, for example, dry skin, furred tongue, reduced blood pressure and reduced CVP readings (Moore 2004).

Serum potassium levels can fall further with rehydration, thus patients are at risk of cardiac dysrhythmias and cardiac arrest. Regular laboratory monitoring of potassium should be performed to check the levels and potassium replacement therapy should be started when indicated (Page and Hall 1999).

Insulin replacement Initially the patient should be treated with an intravenous (IV) infusion of insulin. This is administered using 50 units of soluble insulin in 50ml normal saline (via a sliding scale) (Goldberg *et al* 2004). Before administering the infusion, 50-100ml of this solution should be run through the tubing because insulin is absorbed by polyvinyl chloride in both the IV tubing and bag (Fain 1986). An IV infusion using a sliding-scale insulin regimen helps to lower serum glucose, inhibits ketogenesis (production of ketones) and also begins the reversal process for metabolic acidosis. Capillary blood is tested at appropriate intervals and the IV infusion of insulin adjusted according to the patient's blood glucose levels. Serum glucose should be analysed (initially one to two hourly) in the laboratory rather than using a portable gluocometer to ensure the accuracy of results.

Insulin resistance may occur because of the effects of counter-regulatory hormones and acidosis. This may be significant to nurses when weaning the patient from IV insulin to the subcutaneous route. As a result, patients may require a 10 per cent increase in their usual insulin dose for several days after the onset of DKA (Page and Hall 1999). IV insulin infusions should not be stopped abruptly as the patient can become insulin deficient within ten minutes (Gillespie and Campbell 2002). Instead, the infusion dosage should be reduced on an hourly basis. Despite normal blood glucose levels, the infusion should not be stopped until the urine is free of ketones (Charalambos *et al* 1999). Once the urine is clear of ketones and blood glucose is maintained within normal limits and the patient is eating, subcutaneous insulin can be commenced. IV infusion regimens should not be discontinued at the same time as the first dose of subcutaneous insulin is given. The infusion should be discontinued at least half an hour later.

Correcting metabolic acidosis The role of bicarbonate in treating DKA remains controversial. Sodium bicarbonate (8.4%) can cause alkalosis, hypokalaemia and hypocalcaemia, leading to pulmonary oedema, thrombosis of the peripheral vein and tissue necrosis, resulting in extravasation (Simpson 2001). Wherever possible other treatments should be considered, bicarbonate may be used as a last resort with very close monitoring of the patient, particularly his or her cardiovascular status. Swearingen and Keen (2000) suggest that acidosis is best corrected by insulin therapy. Arterial blood gas monitoring is also required to establish the degree of metabolic acidosis and to evaluate acid base balance.

It is suggested that bicarbonate administration is unnecessary via the blood if pH is more than 7.1 (Okuda *et al* 1996). For a blood pH of 6.9-7.0 it is recommended that 50mmol/l (one ampoule) of sodium bicarbonate is diluted in normal saline. If the pH is less than 6.9, double the dosage is given (two ampoules) diluted in normal saline (Miller 1999).

Effective treatment of DKA should alleviate abdominal pain within 6-12 hours. If pain persists, another cause is likely. Infection is treated with IV antibiotics. A nasogastric tube should be inserted in patients who are drowsy or vomiting. Patients are at risk of developing deep vein thrombosis, therefore low doses of heparin should be administered (Jerreat 2003).

Complications Gastric stasis can lead to acute abdominal distension with copious vomiting and a high risk of aspiration pneumonia. A nasogastric tube should be inserted to aspirate stomach contents. Salt and water depletion can cause shock and renal insufficiency. Over-rapid correction of biochemical abnormalities can cause hypoglycaemia, hypocalcaemia

Box 3. Diagnostic test findings for diabetic ketoacidosis

- Elevated serum glucose level (greater than 14mmol/l)
- Positive serum ketone level
- Positive urine ketone level
- Decreased serum sodium
- Increased serum potassium level initially then decreased because of increased diuresis and reversal of acidosis
- Metabolic acidosis evident on arterial blood gas analysis
- Elevated haemoglobin and haematosis levels because of diuresis and dehydration
- Lowered haemodynamic pressures – below the patient's normal
- Dysrhythmias on electrocardiogram associated with potassium imbalance

(Krentz and Nattrass 2003)

and hyperkalaemia. Respiratory complications including failure can manifest with rapidly progressive shortness of breath and hypoxaemia. Bilateral alveolar pulmonary shadowing can also be seen on X-ray. The pathophysiology is unclear but possible links have been made to a decrease in osmotic pressures and an increase in left atrial pressure from excessively rapid fluid replacement (Page and Hall 1999). Mediastinal surgical emphysema, which usually affects the chest and neck, can occur in patients with severe acidosis. Symptoms include prolonged hyperventilation and vomiting. Increased alveolar pressure can damage the alveolar walls and enable air to escape into the interstitial lung tissue (Chang and Barton 2004).

Hyperosmolar non-ketotic syndrome

This condition is less common than DKA and relates predominantly to patients aged more than 60 years, presenting with type 2 diabetes (indicating some insulin production) (Krentz and Nattrass 2003). The mortality rate remains high (Clark 2000, NHS 2001).

This condition is characterised by insulin deficiency (a raised plasma glucose, usually more than 30mmol/l), dehydration, plasma hyperosmolality and renal impairment. This condition is the second major clinical presentation of uncontrolled diabetes mellitus. In comparison to DKA, the onset is more insidious with clinical features developing gradually, sometimes taking up to two weeks (Clark 2000).

Precipitating factors These include drugs that reduce insulin secretion such as beta-blockers, thiazides and loop diuretics, and those that increase insulin resistance such as corticosteroids. Other precipitating factors include increase in the osmotic load caused by enteral and parenteral feeding and excessive IV glucose administration. Finally, a preceding illness resulting in several days of increasing dehydration may precipitate this condition, for example excessive vomiting (Krentz and Nattrass 2003).

Clinical features Diagnosis is made on a number of criteria, which are similar to DKA but with marked dehydration and the absence of ketones in the urine. A summary is shown in Box 4. Frequently, older patients do not manifest acute clinical symptoms of illness and can become critically ill before symptoms are recognised. Patients may present with non-specific symptoms, for example, anorexia, malaise and weakness. Hyperglycaemia is more severe than in DKA, resulting in a significant serum hyperosmolality and pronounced osmotic diuresis (Box 4). Severe dehydration can occur; patients can lose up to 25 per cent of their body weight, resulting in intracellular dehydration. The blood becomes more viscous and flow is impeded, increasing the risk of thromboemboli. Increased cardiac workload and decreased renal and cerebral blood flow may result in myocardial infarction, renal failure and stroke (Ganong 2003).

Other features include polydipsia and polyuria, tachycardia, hypotension, tachypnoea with shallow respirations, profound weakness, focal seizures and hypokalaemia (Young and Oh 2003). Neurological deficits may be mistaken for senility in the older patient. Some patients may be unconscious or have impairment of conscious levels. This is proportional to the severity of hyperosmolality (Young and Oh 2003). Although the body's available insulin is insufficient to control blood glucose, it usually is adequate to prevent the formation of ketone bodies, thus avoiding metabolic acidosis.

Management Treatment is similar to DKA but patients have a much greater sensitivity to insulin. Insulin is usually administered in low doses via the IV route (because of poor tissue perfusion). This condition can sometimes be treated with fluid replacement alone (Swearingen and Keen 2000). Fluid and electrolyte replacement are achieved via a CVP line of 0.9% saline. Potassium, sodium, phosphate and magnesium supplements are given on the basis of laboratory values. Fluid replacement requires flexibility in relation to the patient's level of dehydration, electrolyte imbalances and blood glucose levels.

Initially, a large volume of fluid may be given rapidly, for example, 1-2 litres over the first two hours. The aim is to replenish intravascular volume and correct hyperosmolality. Many patients respond to fluid resuscitation alone, but IV insulin can correct hyperglycaemia (Sagarin and McAfee 2001). The aim is usually to replace the fluid deficit (average 8-10 litres) over approximately 48 hours.

Box 4. Diagnostic test findings for hyperosmolar non-ketotic syndrome

- Markedly elevated serum glucose level: greater than 14mmol/l
- Urine glucose positive
- Serum ketone usually absent
- Urine ketones negative
- Elevated serum sodium, normal or low (usually low due to the osmotic effect of glucose drawing H_20 into the vascular spaces)
- Normal or slightly lowered serum potassium level
- Decreased serum phosphate: less than 1.4mmol/l
- Decreased serum magnesium: less than 1.5 mmol/l
- Impaired phagocytosis with serum glucose levels equal to or greater than 200mg/dl. This can delay wound healing and predispose the patient to infection

(Huddleston and Ferguson 1997)

For patients who are in a coma, safety is the priority and airway management is required. Prophylactic anti-coagulation should be given to prevent venous thromboembolism (NHS 2001).

TIME OUT 4

Outline the major differences between diabetic ketoacidosis and hyperosmolar non-ketotic syndrome. Consider the aetiology, precipitating factors, clinical features and management.

Hypoglycaemia

Hypoglycaemia can be caused by the inappropriately raised insulin concentrations caused by inadequate insulin delivery following subcutaneous injection of agents that stimulate insulin release, such as sulphonylureas (Heller 2003).

Patients are vulnerable to hypoglycaemia from approximately two hours after a meal until they eat again. This is because a typical meal raises blood glucose concentrations within a few minutes, reaching maximum at about one hour. Peak insulin concentrations occur between one and two hours later (Heller 2003). Other predisposing factors include excessive dosage error by either the patient or healthcare professionals, accelerated absorption through exercise or injection into the abdomen, mismatch between insulin, carbohydrate intake and the use of carbohydrate, missed or delayed meals and excessive alcohol consumption.

During a non-acute hypoglycaemic episode the patient usually relies on early warning signs, for example, sweating, trembling and palpitations (Jerreat 2003). In this instance, the patient should ingest 20g of quick-acting carbohydrate, for example 100ml of a soft drink such as Lucozade™, or four teaspoons of sugar.

In severe hypoglycaemia serum glucose levels fall below 1mmol/l. Glucagon and catecholamines (especially adrenaline [epinephrine]) are the major hormonal defence against hypoglycaemia and glucagon plays the dominant role (Shoemaker *et al* 1995). Glucagon limits a blood glucose fall by reversing the suppression of hepatic glucose output induced by insulin (Heller 2003). Adrenaline (epinephrine) stimulates hepatic glucose output but also inhibits peripheral glucose uptake.

During prolonged hypoglycaemia growth hormones and cortisol are secreted, which reduce the rate of glucose used by most cells of the body, converting instead to greater amounts of fat use. This helps the blood to return the level of glucose towards normal.

TIME OUT 5

As a result of her diabetes Veronica may experience the effects of hypoglycaemia. Distinguish between mild hypoglycaemia and an emergency hypoglycaemic situation.

Clinical features Blood glucose levels can fluctuate greatly in healthy people, therefore symptoms of hypoglycaemia can be vague and non-specific, making its diagnosis difficult.

Clinical manifestations are due to a combination of the adrenergic and neuroglucopenic responses to hypoglycaemia. The adrenergic response to hypoglycaemia is part of the complex counter-regulatory hormone response, which is implicated in correcting hypoglycaemia (Pinsky and Dhainaut 1993). Altered behaviours include inappropriate behaviour, withdrawal, irritability, difficulty in motor function (for example, walking) and slurred speech that gives the impression of drunkenness. Physiological symptoms include increased heart rate, sweating and tremor (due to the activation of the sympathetic nervous system). Sometimes epilepsy and in severe cases stupor, seizure and comas may be present (due to neuroglucopenia) (Williams and Pickup 1999).

Management If hypoglycaemia is not promptly corrected then irreversible brain damage and myocardial infarction can occur. Prompt reversal of hypoglycaemia occurs with the administration of concentrated glucose. There is the danger of precipitating hyperglycaemia with the administration of too much glucose.

If the patient is conscious, 20-30g oral glucose should be administered. Blood glucose should be checked after 15-20 minutes. If the patient is unconscious then IV glucose solution, for example 30-50ml of 50% should be given. Blood glucose should be checked 15-20 minutes after administration.

TIME OUT 6

Consider the type of health education Veronica will require in an attempt to reduce the likelihood of diabetic ketoacidosis recurring.

Patient education

Although small, death rates from DKA and hypoglycaemia in those under the age of 20 have not declined significantly (Laing *et al* 1999). This indicates the need for effective emergency services and ongoing support. Patients need help to prevent and manage acute episodes.

Diabetes is a chronic disease and because of the severity of the illness, patients may have neither the capacity nor the desire to be involved in health promotion.

However, once patients have passed the 'critical phase' of their illness and are assessed as 'competent' (ability to receive and understand information), health promotion should begin.

Health promotion is a group of activities that helps to prevent disease and improve health and well-being (Naidoo and Wills 1998). It is the process of enabling people to increase control over, and improve, their health (WHO 1986).

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The process of empowerment is a developmental one, giving power and control to patients by increasing their knowledge, skill and ability to participate. Key features of empowerment include:

- The need to clarify individuals' beliefs and values about themselves, health and health-influencing behaviour (Downie *et al* 1996). For example, a patient admitted with uncontrolled diabetes should be encouraged to articulate what diabetes means to them, what effect it has on them and how they can change their lifestyle and dietary habits.
- Fostering empowerment through raising patients' self-esteem, encouraging positive beliefs about self-efficacy and the acquisition of life skills (Tones 1991). For example, constant reassurance, allowing patients time to make decisions relating to how they will adapt and change their lifestyle. Nurses should not dictate to patients what they should be doing.
- A partnership model of communication, communicating with patients (Downie *et al* 1996).

The importance of preventing diseases as far as possible, for example, the common cold, should be emphasised. When patients are ill they may require more insulin than normal because blood glucose can rise even if they cannot eat a normal diet (Harrison and Daly 2001). Therefore it is important that patients continue their insulin therapy and medications even when feeling ill. Patients may need to eat an easily digestible liquid diet containing carbohydrates until they feel better, or less nauseous. If patients are not feeling well they should be informed of the importance of checking their urine for ketones. If their illness is persistent or they have ketonuria they should seek medical advice.

Patients should be seen and assessed by a dietician. Food nutrition management and the regular monitoring of capillary blood glucose in the regulation of diet should be explained.

The symptoms of hypoglycaemia should be revisited. In the event of hypoglycaemia if the patient is still conscious, oral glucose should be ingested.

Some patients find it difficult to implement the recommendations of healthcare professionals in relation to modifying diet and other lifestyle habits. They may also face difficulty in complying with self-monitoring, medication, home care instructions and return for follow up.

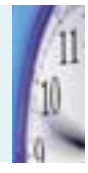
Nurses and other healthcare professionals should work with patients to identify and design solutions for remediable barriers and should use recommended techniques for patient education and counselling to give patients the factual information and motivational encouragement they need to initiate meaningful change.

Conclusion

Nurses need to understand aetiology, clinical features and management of the three main diabetic emergencies: diabetic ketoacidosis, hyperosmolar non-ketotic syndrome and hypoglycaemia. This article has provided information about the recognition, management and nursing intervention for these emergencies. Nurses should be in a position to quickly and appropriately identify these emergencies to ensure suitable management is provided

TIME OUT 7

Now that you have finished the article, you might like to write a practice profile. Guidelines to help you are on page 56.



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