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"The nursing management of hyperglycaemia"

Abstract

Diabetes Mellitus is a metabolic disorder characterised by the presence of hyperglycaemia due to the bodies inability to produce or utilise insulin. Patients diagnosed with diabetes are at risk of short term potentially life-threatening conditions such as diabetic ketoacidosis (DKA) and hyperosmolar hyperglycaemic states (HHS) as well as long term complications. Nurses frequently care for patients diagnosed with diabetes in various clinical arenas; therefore, an awareness of the nursing management of hyperglycaemia is essential. This article details the underlying causes of hyperglycaemia, and how nurses should care for patients using current guidance to inform nursing management.

Key Words

diabetes, hyperglycaemia, diabetic ketoacidosis, hyperosmolar hyperglycaemic state, prevention

Key points

- The pathophysiology of type 1 and type 2 diabetes mellitus are different, it is important for nurses to understand and be able to distinguish between the two.
- Diabetes is characterised by the inability to either produce insulin or to utilise insulin, subsequently this leads to hyperglycaemia.
- Hyperglycaemia is defined as blood glucose levels persistently over 10mmol/l, however frequently symptoms will not be apparent until these levels are over 15mmol/l (UKPDS 2018).
- Hyperglycaemia is present in both type 1 and 2 diabetes. If left unmanaged it can be potentially life threatening. Historically type 2 diabetes mellitus has been dismissed as less serious, this is not the case.

- Causes of acute episodes of hyperglycaemia may be attributed to a variety of causes, some examples are; imbalances between medication dose and carbohydrate consumption, variation in normal activity, stress and illness.
- The current management of hyperglycaemia in primary and secondary care is systematically led by guidelines, whether this be national clinical guidelines from the Joint British Diabetes Societies 2012 and 2013, National Institute for Health and Care Excellence 2015 or via local policy.

Aims and intended learning outcomes

This article will facilitate nurses in understanding the causes of hyperglycaemia and support them to develop knowledge about the effects of it. In addition, it will encourage nurses to expand their understanding of how to recognise and care for patients who present with hyperglycaemia. The NMC Code of Conduct (2015) stipulates that nurses should be able to practice effectively and preserve safety. In terms of the nursing management of hyperglycaemia, this is particularly pertinent due to the life threatening nature of diabetic ketoacidosis (DKA) and hyperosmolar hyperglycaemic states (HHS) and the frequently devastating effects of long-term hyperglycaemia.

Four intended learning outcomes are proposed:

1. Understand the causes of hyperglycaemia.
2. Recognise and act upon the signs and symptoms of hyperglycaemia.
3. Appreciate the difference between DKA and HHS.
4. Understand how to support patients in preventing re-occurrence of hyperglycaemia.

Current context

Worldwide 347 million individuals are diagnosed with diabetes (World Health Organisation 2014). This is reflected in the United Kingdom where presently 3.7 million people live with diabetes at a cost of £10 billion pounds per year to the public purse (Diabetes UK 2019a). There are potentially an

additional 630 000 people who live with undiagnosed diabetes; both types of diabetes are increasing and a significant increase in type 2 diabetes mellitus (T2DM) within the developed world is observed (World Health Organisation 2018). For nurses this means that we meet patients diagnosed with diabetes in all areas of clinical practice, hence the need for knowledge of the condition to ensure confident and competent clinical practice.

Pathophysiology and clinical presentations

Diabetes is characterised by the inability to either produce insulin or to utilise insulin. Subsequently this leads to a chronic hyperglycaemic state often referred to as high blood glucose levels. The pathophysiology of type 1 and 2 are quite different and it is important to firstly understand how to distinguish the pathophysiology of the two different types as a foundation for building diabetes knowledge (Dunning 2014).

The pancreatic islets are responsible for the secretion of insulin and glucagon in response to changes in blood glucose level. Following a meal, the levels of glucose within the blood begins to rise. In response to this the pancreatic beta cells release insulin (Skyler 2016). Insulin acts on body cells to support the diffusion of this glucose into the body cells. It also increases the conversion of glucose into glycogen (glycogenesis) and increases the uptake of amino acids and promotes protein synthesis (Dunning 2014). This subsequently decreases blood glucose levels and homeostasis is restored. In contrast, when blood glucose levels begin to fall, perhaps after exercise or if a meal is due, the pancreatic alpha cells begin to release glucagon. Glucagon acts upon hepatocytes within the liver which causes them to release glucose which restores blood glucose levels back to normal levels and homeostasis is maintained (Dunning 2014).

Time Out; Make notes on the pathophysiological differences of T1DM and T2DM.

Type 1 diabetes mellitus (T1DM) is defined by absolute deficiency of insulin. In the majority of cases this is caused by an autoimmune response where the body begins to attack its own pancreatic beta

cells thus destroying them and rendering them incapable of insulin production (Nair and Peate 2013). Onset of T1DM is characterised by polyuria, polydipsia, lethargy, weight loss, hyperglycaemia, glycosuria and presence of blood and urinary ketones. Onset is normally rapid and generally occurs in younger patients. Patients diagnosed with T1DM typically require lifelong insulin delivered via injection in order to maintain normal homeostasis (Porth 2015).

T2DM is defined by inability to utilise circulating blood glucose coupled with decreasing insulin production (Nair and Peate 2013). Often patients diagnosed with T2DM have begun to develop long term complications by the time they are diagnosed. Onset is generally slower and affects older people. However, the age of onset for newly diagnosed T2DM is decreasing. Often, within T2DM, symptoms occur so gradually that they may go unnoticed or be attributed to other illness or life style factors. Historically T2DM was managed by a combination of lifestyle modification and oral hypoglycaemic agents. However, increasingly those diagnosed with T2DM who are unable to maintain glycaemic control through use of oral medications alone are now prescribed insulin (NICE 2015).

Defined as excess concentrations of glucose in circulating blood plasma, hyperglycaemia is noted to be blood glucose level of > 7mmols/L when fasting and >11mmols post prandial (Diabetes UK 2019b).

Hyperglycaemic crisis

Incidence of hyperglycaemic crisis is increasing commensurately with the increase worldwide in prevalence of diabetes (Hackel 2014). DKA, a life threatening condition, occurs when the body is unable to use blood glucose due to lack of insulin (Porth 2015). Consequently, the body begins to use fat as an alternative energy source, and a build-up of ketones within the body occurs. DKA is the most frequently regarded hyperglycaemic crisis with incidence of HHS occurring less frequently. Commonly there is lack of recognition of HHS within clinical practice and less understanding of its causes and presentations. HHS is a different condition in which the body becomes dangerously dehydrated due to very high blood glucose levels, however normally there remains enough insulin production within

the body to prevent ketosis. Alarming, HHS confers much higher mortality rates than DKA (McNaughton et al 2011). Observations from the clinical arena indicate that nurses frequently believe that only those with T1DM are at risk of hyperglycaemic crisis. Undeniably those with T1DM are more commonly at risk but the risk to those with T2DM should not be dismissed.

Precipitating factors for hyperglycaemic crisis often include infection, surgery or periods of acute illness which causes production of stress hormones and a consequent rise in blood glucose levels (Hackel 2014). Stress-related hyperglycaemia continues to be a common occurrence in acute settings with patients being admitted to ICU in many cases. Stress-related hyperglycaemia has direct correlations with high mortality and morbidity, which has been evidenced from many research studies performed in acute care settings (Olariu et al 2018). During episodes of critical illness, hyperglycaemia is seen in response to stress, however, there is yet to be a universal definition in relation to blood glucose levels. This lends itself to varying approximations of its occurrence (from 19.9% in cases with blood glucose levels > 8.5 mmol/L to 75% in cases where blood glucose levels > 6.1 mmol/L (Giles 2018).

Time out; consider the body's normal stress response.

During episodes of stress the body overcompensates to try to reduce the stress it is under. One action in this stress response is to release hormones including adrenaline and epinephrine which collectively enable the body's fight or flight response. Clinical manifestations of these actions are rises in blood pressure and heart rate and alterations in metabolic state in order that blood glucose levels are maximised. In addition, stress fuels the release of the hormone adrenocorticotrophic, made in the cells of the pituitary gland. This action stimulates production of cortisol from the cortex of the adrenal glands, which subsequently modifies the body's metabolism, resulting in rises in plasma glucose. Whilst this is central in acute episodes of stress, in chronic stress this could result in persistently raised levels of circulating blood glucose (Giles 2018).

Patients living with undiagnosed diabetes, those who have recently been diagnosed and those who have had recent medication titration may be particularly at risk of developing DKA and HHS. Specifically, omissions or inadequate insulin dose are frequent precipitating factors for DKA. Stroke, silent myocardial infarction, pancreatitis and certain medications such as steroids are also associated with incidence of HHS (Chiasson et al 2003). Diagnoses of hyperglycaemic crisis, more specifically DKA or HHS are usually confirmed by a combination of clinical presentation, history and biomedical markers. In both DKA and HHS a raised blood glucose level will be present.

Diabetic ketoacidosis

DKA is largely associated with T1DM; those with T2DM rarely develop DKA. DKA in type 2 diabetes is more commonly referred to as Ketosis prone type 2 diabetes or Flatbush diabetes (Misra et al 2013) and commonly occurs in non-white ethnic groups. Normally in T2DM there remains sufficient beta cell function and subsequent insulin production to prevent lipolysis and ketoacidosis (Kitabchi et al 2008). However, the possibility of DKA in T2DM should not be excluded. In the absence of insulin, glucose is unable to enter the body's cells; consequently, high blood glucose levels develop. A lack of the hormone insulin leads to release of catecholamine's, lipolysis and free fatty acid mobilisation. This causes the formation of B-hydroxybutyrate, acetone and acetoacetate; consequently, metabolic acidosis develops. Simultaneously, protein catabolism occurs and forms a substrate for glycogenesis, further contributing to a rise in blood glucose levels. To further exacerbate this hyperglycaemic crisis, tissue glucose utilisation is impaired (Wolfsdorof et al 2014). Diagnosis of DKA is normally made if clinical investigations meet the following criteria: ketonaemia $> 3.0\text{mmol/L}$ or significant ketonuria (more than 2+ on standard urine sticks) Blood glucose $> 11.0\text{mmol/L}$ or known diabetes mellitus, Bicarbonate (HCO_3^-) $< 15.0\text{mmol/L}$ and/or venous pH < 7.3 (Joint British Diabetes Societies Inpatient Care Group 2013).

Pathophysiological manifestations include osmotic diuresis, ketonuria and glycosuria as the filtered load within the kidney exceeds re-absorption capacity leading to polyuria. Cellular dehydration causes polydipsia and, despite patients reporting drinking in response to excessive thirst, the thirst is unquenchable as the body continues to dehydrate. As the body becomes increasingly acidotic in an attempt to correct acidosis this manifests clinically as breathing difficulties: specifically, tachypnea and Kussmaul respiration (Garcia-Pascual and Kidby, 2012). Kussmaul's respiration is a compensatory mechanism, as the body attempts to overcome acidosis. This manifests as deep, laboured breathing and gasping as the body struggles to restore homeostasis. Patients frequently report abdominal pain, nausea and vomiting all of which are caused by ketones. Consequently, vomiting causes loss of sodium, potassium and other vital electrolytes, further exacerbating the acidosis (Kitabchi et al 2006).

In severe prolonged DKA the patient will present as hypothermic but due to peripheral vasodilatation may feel warm to touch. As renal function is impaired, dangerously ill patients may become oliguric as acute kidney injury occurs. Kussmaul's respiration will cease as bradypnea occurs. The patient's vital signs will indicate hypotension and bradycardia. If the patient does not receive treatment to correct the DKA they will progress to coma and death (Nair and Peate, 2013).

Text box; Criteria for diagnosing DKA – text box –Ed to advise

Diagnosis should be confirmed by using the JBDS (2013) guideline.

ALL 3 OF THE FOLLOWING MUST BE PRESENT TO CONFIRM DKA:

1. Capillary blood glucose (CBG) $\geq 11.0\text{mmol/L}$ or known diabetes
2. Capillary blood ketones $>3.0\text{mmol/L}$ or 2+ ketonuria
3. Venous $\text{pH} < 7.3$ and/or venous bicarbonate $< 15\text{mmol/L}$ HCO_3^- -mmol/L

Hyperosmolar Hyperglycaemic State

HHS occurs more frequently in but is not exclusive to those diagnosed with T2DM. HHS is characterised by significant hyperglycaemia (>30 mmols/l) and an increase in serum osmolality (>320 mosmol/kg) (Innes 2016). In contrast to DKA, where presence of ketones is a significant factor, patients with HHS do not normally present with significant ketosis (<3.0 mmol/L) (Joint British Diabetes Societies Inpatient Care Group 2012). Hypovolaemia coupled with profound dehydration are trade mark presentations of HHS; patients may present with a fluid deficit of up to 7 litres (Chaisson et al 2003).

Clinical manifestations of patients with HHS are visible signs such as sunken eyes and longitudinal furrows on the tongue. Patients will feel profoundly weak and initially may present with altered mental status, such as inability to concentrate or confusion. If left untreated the condition will progress to manifest focal neurological deficits such as slurred speech. HHS shares some clinical manifestations with DKA such as polydipsia and polyuria in the early stages progressing to oliguria, fitting, coma and death if untreated. Onset and progression of HHS is significantly slower than DKA and normally occurs in older patients (Wolfsdorf et al 2014). The symptoms of HHS can be confused with other medical conditions such as urinary tract infection, silent myocardial infarction, dementia or stroke. This is particularly dangerous given the age demographic for which HHS frequently occurs; as those over 65 years have often already suffered from cardiovascular or mental health conditions such as dementia; correctly identifying HHS may be difficult. Frequently these co-morbidities are causative or can occur at the same time as HHS, but may in fact also disguise it.

Treatment of hyperglycaemic crisis

The Joint British Diabetes Societies Inpatient Care Group have issued guidance and care pathways on both the clinical management of HHS (2012) and DKA (2013). This guidance is being adapted within the clinical arena with the DKA guidance being utilised more frequently within the clinical arena than the HHS pathway.

Time out; find the National Guidelines for Diabetic Ketoacidosis (DKA) and Hyperosmolar Hyperglycaemic State (HHS) make notes on: What are the main treatment goals for DKA? What are the main treatment goals for HHS?

The aims of medical management for patients with DKA are: to restore circulating fluid volume and correct urea and electrolyte imbalance, to clear blood ketones and suppress ketogenesis. Guiding principles of DKA management advise that bedside capillary ketones monitoring should be employed and fixed rate insulin infusion regimes (FRII) should be used (Joint British Diabetes Societies Inpatient Care Group 2013). Blood glucose monitoring is also necessary but it is the rate of ketone reduction that will determine the adequacy of the insulin regime in place and the patient's overall condition and intended recovery. The use of variable rate intravenous insulin infusion (VRIII) formerly seen as the gold standard in DKA management is no longer advocated. A move to FRII, which use patients weight as a guidance for hourly insulin dose rather than the capillary blood glucose level employed in VRIII's, is now viewed as the most effective treatment regime. This is rationalised as weight based administration accounts for insulin resistance (Joint British Diabetes Societies Inpatient Care Group 2013).

The aims of medical management for patients with HHS are: to normalise osmolality, replace fluid and electrolyte loss and normalise the blood glucose levels (Wolfsdorf et al 2014). This is achieved by intravenous (IV) fluid replacement using 0.9% sodium chloride; this must be done with particular caution as overly rapid rehydration may precipitate heart failure, particularly in older patients. An FRII should only be commenced if there is significant ketonamia or ketonuria or if the blood glucose levels are no longer falling with IV fluids only. As osmolality is normalised and fluids and electrolytes replaced, blood glucose level should return to normal range and unnecessary administration of insulin can put the patient at risk of hypoglycaemia (Joint British Diabetes Societies Inpatient Care Group 2012).

Fundamental nursing considerations

Largely the nursing consideration for the care of patients with DKA and HHS are similar, with the exclusion of bedside ketones monitoring which is often futile in HHS management. Both groups of patients will require regular observations of vital signs, 24 hour cardiac monitoring and pulse oximetry as well as ongoing assessment employing the national early warning score system. The introduction of a urinary catheter and strict hourly fluid balance is vital to ensure hypovolaemia and dehydration is corrected and a minimum output of 0.5ml/kg/hr is advocated. An hourly blood glucose level test will evaluate if homeostasis is being restored with regard to circulating blood glucose levels (Joint British Diabetes Societies Inpatient Care Group 2013).

Patients diagnosed with HHS have been highlighted specifically as a high risk group for pressure damage to the feet and therefore it is good practice to assess foot risk score on admission (Joint British Diabetes Societies Inpatient Care Group 2012). Appropriate pressure relieving equipment should be procured for all patients and on-going checks and pressure relief should continue even as the patient begins to recover.

Frequency of blood glucose monitoring in non-crisis situations should be planned according to the individual patient and will depend on several factors such as: pharmacological treatments for diabetes that the patient is prescribed, for example insulin or oral hypoglycaemic agents (NICE 2015) lifestyle factors such as diet and exercise and special circumstances such as patients who are prescribed steroids or are on artificial feeding regimes require consideration. During periods of medication titration or episodes of common illness, for example diarrhoea and vomiting or the common cold, patients will be advised to monitor capillary blood glucose levels with increased frequency. In un-well patients diagnosed with T1DM incorporation of urine testing may be part of a 'sick day plan' agreed with the diabetologist or diabetes specialist nurse but for those with T2DM this is uncommon within the clinical arena (Lang, 2015).

Urinalysis should not be used as a regular method of blood glucose monitoring within UK practice. In patients presenting with HHS its uses are limited to detection of glycosuria and indicators of infection that may be a contributory factor to HHS. However, presence of an acutely unwell patient diagnosed with T2DM but absence of ketonuria in hyperglycaemic patients may be a red flag for HHS. Urinalysis is however critical in the management of DKA as presence of ketones in urine is an effective indicator that acidosis is present (Westerberg, 2013).

Education and follow up to prevent re occurrence is a vital nursing role and support from the diabetes specialist team should be sought in doing this (Runciman, 2013). Nurses are in an ideal position to develop therapeutic relationships with patients and offer education and support on how patients can self-manage and prevent reoccurrence of crisis in the future.

Prevention of re-occurrence:

Structured education alongside lifestyle management support to achieve normal glycaemic control is suggested in long term prevention of hyperglycaemia. Structured education programmes such as DESMOND for people diagnosed with T2DM and DAFNE for individuals diagnosed with T1DM can support patients in empowerment and improving quality of life through education (Chatterjee 2018). Clear and appropriate explanations of what can be complex self-management need to be presented by the health care professional to support empowerment of patients. Continuing education should be presented at every contact to maximise the opportunity of education relating to all elements of diabetes management using the Make Every Contact Count model (Hopkins 2012). The triad of diabetes management: Diet, Activity (or lifestyle intervention) and Medication is a simple formula to allow the health care professional to rapidly review and discuss the issues of hyperglycaemia in both type 1 and type 2 Diabetes.

DIAGRAM OF THE TRIAD IF POSSIBLE? Editor to advise.

DIET

Food is a fundamental element of diabetes management, frequently patients require guidance on which foods are most likely to result in hyperglycaemia. This is not a case of avoidance but making informed choices. Carbohydrate awareness is required in T2DM to empower patients to make educated choices (Dyson 2011) in T1DM support to undertake complex carbohydrate counting plans such as DAFNE may be appropriate.

Activity

In T2DM where insulin resistance is an issue weight loss approaches need to be addressed (Dyson 2011). The move to low calorie and low to no carbohydrate diets is becoming increasingly popular, however, the ability to maintain these strict and restrictive diets may not be achievable in the long term. Increasing activity can offer a way to address this in some patients, leading to positive outcomes (Polonsky 2016). However, this may not be a feasible option for some clients with other co-morbidities or accompanying disability (Sheri 2015). In such cases minimal increases can be significant but diet exploration maybe the safest approach in hyperglycaemic management. Improvement in hyperglycaemia with the introduction of controlled activity in people with T2DM is evocative in that people are more likely to increase activity in the early stages of diagnosis of T2DM. Engagement and encouragement needs to be continued to promote activity in the long term.

In patients with T1DM normal activity can be a different consideration, the indiscriminate occurrence of T1DM in the younger age range means consideration of involving the other members of the MDT is frequently necessary, specifically the diabetes dietitian. Understanding the bodies individual responses to activity is necessary as even moderate intensity aerobic activity can result in hypoglycaemia, with intensive and high intensity anaerobic activity potentially causing hyperglycaemia (Castonguay 2018).

TIME OUT; What are the guidelines for minimal type and time of activity? Think about the health promotion messages on TV that encourage activity, are these effective?

Medication

The final aspect of the triad management of hyperglycaemia is medication. In T1DM due to absolute insulin deficiency, the only option is insulin. When reviewing the glycaemic status blood glucose monitoring is a vital tool (UKPDS 2018) with no safe decision able to be made without this information. Setting targets for frequent and intense monitoring is necessary. Establishing regular blood glucose monitoring is a necessary undertaking in the management of hyperglycaemia in T1DM and T2DM in those prescribed insulin or sulphonyureas. Pre-meal blood glucose monitoring is preferable to allow the assessment of carbohydrate counting and management (Baghbanian 2012). Providing education about the actions of insulin is imperative. In cases where insulin doses require adjustment in line with blood glucose levels, long acting once daily analogues should only be increased to establish a safe morning fasting blood glucose level. It is essential that further titration only takes place following a time period of 2-4 days and following review of pre and post prandial blood glucose levels. Encouraging patients to keep a record of consumed meals, in particular the carbohydrate content combined with the blood glucose records encourages awareness not only of insulin requirements but aides in understanding the content of meals. Awareness of insulin dose adjustment is encouraged in T1DM following DAFNE education programmes. Insulin dose adjustment policy indicates a 10% increase is advocated in insulin dose in response to hyperglycaemia with a 20% reduction in response to hypoglycaemia.

Management of hyperglycaemia in T2DM currently has many options when considering oral medication (NICE 2015). Local guidelines and formularies need also to be considered due to the potential financial implication of certain medications, however Metformin continues to be first line consideration in hyperglycaemia management (Joint Formulary Committee 2018) with review of HbA1c at 3-month to evaluate effectiveness. Second line treatment options may include the

introduction of sulphonyureas, should a more rapid response to hyperglycaemia be required, specifically in patients who present with clinical manifestations due to other treatment options taking longer to be established. In significant hyperglycaemia with presenting symptoms in patients at high risk of developing HHS insulin may be considered as first line treatment. Insulin initiation should be closely monitored with patients required to monitor blood glucose and be provided with a clear plan of care. Transition from insulin to oral medication needs to be considered after the emergency situation is resolved.

TIME OUT; Talk to your patient who has diabetes about what their target blood glucose levels are? Do they have specific guideline regarding what to do in the case of a high blood glucose readings? What symptoms, if any do they have if their blood glucose levels are raised? What does the patient class as hyperglycaemia and does that differ from medical classifications?

Conclusion

Developing knowledge about the basic pathophysiological differences and treatment in terms of diabetes mellitus is useful for all practicing nurses. It is necessary for nurses to practice confidently and competently with regard to fundamental diabetes management as patients diagnosed with diabetes will almost certainly require care across almost all areas of clinical nursing practice. Possessing basic knowledge that allows nurses to recognise and respond to hyperglycaemic crisis is a useful skill in helping those who are vulnerable. For those working in more specialist areas where people with diabetes are reviewed more frequently current management of hyperglycaemia needs to be at the forefront of every contact between the practitioner and patient.

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