Anaesthetic management of diabetes

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Abstract

Diabetes is a complex, chronic metabolic disorder affecting approximately 8.5% of the adult population with the number of people living with diabetes worldwide having almost quadrupled since 1980. This increase has largely been attributed to global urbanization and lifestyle changes. Diabetes affects 10–15% of the surgical population. These patients are now frequently elderly, have complex medical comorbidities and present for both high-risk elective and emergency surgery. This multisystem disease poses a significant challenge to both anaesthesia and surgery with diabetic patients demonstrating higher morbidity and mortality rates compared to their non-diabetic counterparts. As the management of diabetes becomes more complex, it is vital that the anaesthetist, as a member of the multidisciplinary team, remains up-to-date and plays a key role in patient optimization and perioperative glycaemic control. It is crucial that good glycaemic control is maintained throughout the perioperative period as this has been shown to correlate with positive patient outcomes. Patients themselves are well experienced in managing their own diabetes and should be involved in doing so whenever possible.

Keywords Diabetes; fasting blood sugar; glycosylated haemoglobin; insulin; oral hypoglycaemics; variable rate intravenous insulin infusion

Royal College of Anaesthetists CPD Matrix: 1A01, 1A02, 2A03, 3I00

Learning objectives

After reading this article you should be able to:

- describe the pathophysiological basis of type 1 and type 2 diabetes
- discuss the various management options available
- discuss the important diabetic complications and how they influence anaesthetic practice
- outline the safe perioperative management of patients with diabetes

Introduction

Diabetes is a group of metabolic disorders where defective insulin secretion, peripheral action or both result in disordered carbohydrate, lipid and protein metabolism. Uncontrolled disease typically manifests as hyperglycaemia. Over time this leads to macro- and microvascular disease with subsequent end-organ dysfunction and failure. Organs typically involved include the eyes, kidneys, nerves and heart.[1]

Pathophysiology

Diabetes is a heterogeneous disease with complex pathophysiological mechanisms resulting in deficient pancreatic B cell secretion of insulin. This can range from an absolute insulin deficiency secondary to autoimmune destruction of the pancreatic islets of Langerhans, to a relative deficiency, setting the scene for insulin resistance and subsequent B cell dysfunction. All mechanisms ultimately result in hyperglycaemia. The mechanism by which poor glycaemic control leads to accelerated multisystem disease is not fully understood. In chronic hyperglycaemia, glucose non-enzymatically combines with free amino acids on circulating tissue proteins that over time result in advanced glycation end products. Net accumulation overtime may contribute to diabetic microvascular complications. Hyperglycaemia may also promote an inflammatory state with cytokine release and expression of vascular growth factors that overtime lead to cardiovascular complications.

Classification

Box 1 demonstrates the aetiological classification of diabetes. The three most common clinical presentations are type 1, type 2 and gestational diabetes.

Type 1 diabetes

This is characterized by autoimmune destruction of β cells in the pancreas resulting in an absolute insulin deficiency. Patients can be identified by genetic markers or serological evidence of autoantibodies. It has an incidence of 5–10% with multiple genetic predispositions. Environmental factors also play a role.

Type 2 diabetes

The process of β cell dysfunction and death is less well defined with defective and insufficient insulin secretion being seen in the setting of insulin resistance. It accounts for 90–95% of all diabetes and appears to have a stronger genetic predisposition. Autoimmune pancreatic destruction does not occur. Instead insulin secretory defects appear to be related to metabolic stress in conjunction with other factors.

Gestational diabetes

Placental hormones are thought to play a key role in inducing maternal insulin resistance being most marked during the third trimester. Gestational diabetes is first diagnosed in the second or third trimester that is not clearly pre-existing type 1 or 2 diabetes. It has an incidence of between 5% and 7%, which is increasing in parallel with type 2 diabetes.

Clinical presentation and diagnosis

The presentation and disease progression of both type 1 and 2 diabetes varies considerably. Both diseases can occur in adults and children with the traditional paradigm that type 2 diabetes only occurs in adults and type 1 in children being inaccurate.

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Aetiological classification of diabetes

- Type 1 diabetes
  - autoimmune β-cell destruction
- Type 2 diabetes
  - progressive loss of β-cell insulin secretion on a background of insulin resistance
- Gestational diabetes mellitus
  - diagnosed in the second or third trimester of pregnancy that was not clearly overt diabetes prior to gestation
- Specific types of diabetes due to other causes
  - Monogenic diabetes syndromes (e.g. maturity onset diabetes of the young)
  - Diseases of exocrine pancreas (e.g. cystic fibrosis)
  - Drug- or chemical-induced diabetes (e.g. glucocorticoid use, HIV treatment or post-organ transplantation)

Taken from Diabetes Care 2017; 40 Supplement 1.

Box 1

Hyperglycaemia is classically associated with polyuria, polydipsia and weight loss however its presentation in adults can be variable. Approximately one-third of type 1 diabetics present as an emergency with diabetic ketoacidosis (DKA), a triad of ketonaemia, hyperglycaemia and acidosis. The onset of type 2 diabetes is more insidious with hyperglycaemia frequently going undiagnosed for years. Patients are often obese which itself increases insulin resistance. Occasionally type 2 diabetics can present with diabetic ketoacidosis however they are more likely to suffer from a hyperglycaemic hyperosmolar non-ketotic state. Both conditions can result in coma and are life threatening.

The diagnostic criteria for diabetes are outlined in Box 2.

Those whose glucose levels do not meet the diagnostic criteria for diabetes however are too high to be considered normal are classified as pre-diabetic. These patients are at high risk of developing type 2 diabetes in the future along with obesity, hyperlipidaemia and hypertension.[1] Glycosylated haemoglobin (HbA_{1c}) is a marker of glycaemic control over the preceding 3-month period. It not only can be used to identify patients with diabetes (HbA_{1c} >48 mmol/mol (6.5%), but also those who have a very high risk of developing diabetes in the future (HbA_{1c} 5.7–6.4%).

Criteria for the diagnosis of diabetes

Fasting plasma glucose > 7.0 mmol/litre
(Fasting defined as no caloric intake for at least 8 hours)

OR

Two-hour plasma glucose > 11.1 mmol/litre during an oral glucose tolerance test
(The test should be performed using a glucose load containing the equivalent of 75 g anhydrous glucose dissolved in water)
OR

HbA_{1C} > 6.5% *(48 mmol/mol)*

Taken from Diabetes Care 2017; 40 Supplement 1.

Box 2

Management

Hyperglycaemia defines diabetes therefore glycaemic control is fundamental to its management. This can be achieved be different means depending on disease aetiology. Patient self-management, education and support is also critical in preventing acute and long-term complications.

Lifestyle management

- There is strong, consistent evidence that obesity management can both delay the progression to type 2 diabetes and improve glycaemic control in those with established disease.[2]

Antidiabetic agents

- Oral and injectable hypoglycaemic agents are commonly used to manage type 2 diabetes in conjunction with lifestyle management. HbA_{1c} is used to monitor glycaemic control having a strong predictive valve for diabetic complications. An acceptable target is an HbA_{1c} of less than 48–53 mmol/mol (6.5–7%).[1]
- Metformin is typically used as the first-line monotherapy as it has good efficacy, is inexpensive and has few side-effects. Depending on glycaemic control, dual, triple or combination injectable therapies, including insulin, may be required.
- The management of diabetic patients is becoming increasingly complex with patients frequently being on multiple agents with which the anaesthetist may not be familiar. Commonly used antidiabetic agents are outlined in Table 1.

Insulin

- Insulin analogues are the mainstay of management for type 1 diabetes and are increasing used to manage type 2 and other forms of diabetes. The aim is to mimic the normal physiological pattern of insulin release, covering both basal requirements and the postprandial response.
- Insulins are available as fast, intermediate and long-acting analogues. Their onset of action is dependent on the rate of absorption from the injection site which is in turn related to the products solubility.
- Fast-acting analogues (e.g. Novorapid®) have a rapid onset of action ranging from 5 to 15 minutes, reach peak activity 2 hours after injection and a duration of action up to 6 hours. Long-acting insulins (e.g. Levemir®) are released slowly and can have peak concentrations anywhere between 6 and 10 hours after injection with a duration of action between 18 and 26 hours. Premixed biphasic insulins are also available (e.g. Novomix 30®) and consist of a mixture of fast and intermediate acting insulins.
- Commonly used insulins and regimes are outlined in Table 2.

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