check

Independent learning program for GPs

Unit 510 October 2014

Diabetes and obesity

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The five domains of general practice

- Communication skills and the patient-doctor relationship
- Applied professional knowledge and skills
- Population health and the context of general practice
- Professional and ethical role
- Organisational and legal dimensions

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ABOUT THIS ACTIVITY

The incidence of diabetes has doubled in the last two decades and in 2006–2007 diabetes accounted for 2.5% of all GP consultations in Australia.[1] Diabetes is predicted to be the seventh leading cause of death by 2030.[2] The global prevalence of obesity has also almost doubled since 1980.[1] Excess body weight is a major risk factor for non-communicable diseases,[3] including diabetes mellitus, cardiovascular disease, musculoskeletal disorders and certain cancers (eg endometrial, breast and colon).[3] This edition of *check* will consider the management of diabetes and obesity in general practice.

LEARNING OUTCOMES

At the end of this activity, participants will be able to:

- describe the diagnosis and management of type 1 diabetes
- outline the management of type 2 diabetes
- describe the management of vision loss in people with type 2 diabetes
- discuss palliative care for patients with diabetes
- summarise the potential benefits of weight loss and exercise for people who are obese and/or have diabetes
- discuss the risks and benefits of laparoscopic adjustable gastric banding surgery.

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GUIDE TO ABBREVIATIONS AND ACRONYMS IN THIS UNIT OF *CHECK*

ACEI	angiotensin converting enzyme inhibitor
ADA	American Diabetes Association
BMI	body mass index
COPD	chronic obstructive pulmonary disease
CORE	Centre for Obesity Research and Education
CrCl	creatinine clearance
DCCT	Diabetes Control and Complications Trial
DMO	diabetic macular oedema
DPP4	dipeptidyl peptidase-4
DPT	Diabetes Prevention Trial
EDIC	Epidemiology of Diabetes Interventions and Complications
ENDIT	European Nicotinamide Diabetes Intervention Trial
GAD	glutamate decarboxylase
GLP-1	glucagon-like peptide-1
HbA1c	glycated haemoglobin
HLA	human leukocyte antibody
IA2	tyrosine phosphatase
LAGB	laparoscopic adjustable gastric banding
LFT	liver function test
NHMRC	National Health and Medical Research Council
OCT	optical coherence tomography
OGTT	oral glucose tolerance test
QoL	quality of life
PBS	Pharmaceutical Benefits Scheme
SGLT2	sodium-glucose transporter 2
T1DM	type 1 diabetes mellitus
T2DM	type 2 diabetes mellitus
TFT	thyroid function test
TGA	Therapeutics Goods Administration
VEGF	vascular endothelial growth factor

CASE 1

ANNIE HAS SIGNS OF DIABETES

Annie is 15 years of age. She presents with a 3-week history of polyuria and polydipsia. Annie is obese and you are concerned that she has developed diabetes. You tell Annie that you suspect a diagnosis of diabetes mellitus. Her mother, who has accompanied Annie, asks if you think Annie has type 1 (T1DM) or type 2 diabetes mellitus (T2DM).

QUESTION 1

What would you tell her?

QUESTION 2

What features of Annie's history are important?

FURTHER INFORMATION

On further questioning, Annie reveals that she is pleased she lost about 5 kg recently 'without even trying'.

Annie's parents' families were originally from the Indian subcontinent and there is a strong family history of diabetes on both sides. Her mother was diagnosed with gestational diabetes in her first pregnancy, with Annie's older brother. Her father and paternal grandmother were diagnosed with T2DM at the age of 45 years. Several other family members on Annie's father's side have T2DM, but Annie and her mother are unsure of the details. All family members are managed with either oral hypoglycaemic agents or diet, except for Annie's paternal grandmother who was commenced on subcutaneous insulin at the age of 70 years.

Annie has been overweight or obese since late childhood. This has been attributed to a combination of inappropriate food choices in the family home and lack of physical activity. This became more of an issue after the time of menarche, which occurred when Annie was 12 years of age. Annie's periods have been regular since that time. Annie's body mass index (BMI) is now greater than the 97th centile for her age, again raising your suspicions of T2DM, given Annie's family history. Neither acanthosis nigricans nor hirsutism was present.

QUESTION 3

Which test is the most appropriate first-line investigation?

FURTHER INFORMATION

Annie's random blood glucose level is 15 mmol/L and her blood ketones are 0.2 mmol/L.

QUESTION 4

What is the appropriate next step?

FURTHER INFORMATION

Annie was appropriately referred to the local hospital endocrinology service for assessment. On presentation to the emergency department, Annie's venous pH was normal at 7.34 (7.35–7.45) and her ketones were borderline positive at 0.6 mmol/L (normal <0.6 mmol/L). She was therefore not in diabetic ketoacidosis and was only mildly dehydrated. She was commenced on subcutaneous basal bolus insulin (1 unit/kg/day) with oral rehydration. Blood samples were sent for autoantibody testing, thyroid function tests (TFTs), screening for coeliac disease, liver function tests (LFTs) and cholesterol. All tests were normal except for those shown in *Table 1*. Measurement of C-peptide was not included as it is not part of the diagnostic workup for diabetes in childhood.[1]

Table 1. Abnormal blood test results

Test	Result
GAD antibodies	20 U/mL (normal <5 U/mL)
HbA1c	10.5% (91.3 mmol/mol)

GAD, glutamate decarboxylase; HBA1c, glycated haemoglobin

Annie and her parents were counselled that this was most probably T1DM and should be managed as such. Annie and her family received comprehensive education on diabetes aetiology and pathogenesis, insulin treatment, practical aspects of self-management and dietary advice. She was discharged 2 days later, when she was comfortable with self-administration of insulin, and was given a plan for regular monitoring of diabetes control at medical specialist appointments every 3–4 months.[2]

After commencing basal bolus insulin, Annie returns to you for review 3 weeks later. She is adjusting to the diagnosis of diabetes but is concerned that she is the only one in her immediate family who has to use insulin. She asks if she can take tablets instead of injections.

QUESTION 5

Do oral hypoglycaemic agents have a role in the management of Annie's diabetes?

QUESTION 6

What advice/information would you provide to help Annie adjust to her diagnosis?

CASE 1 ANSWERS

ANSWER 1

Statistically, people of Annie's age are more likely to develop T1DM than T2DM regardless of body size/weight and family history. The incidence of T1DM is increasing[3] and now affects 500,000 children worldwide.[4] T1DM accounts for >90% cases of childhood and adolescent diabetes in most western countries.[5] In Australia, 14–23 per 100,000 children (0–14 years) are diagnosed with T1DM annually.[6,7] Despite the increasing prevalence of overweight and obesity,[8,9] T2DM is still uncommon in the Australian paediatric group.[10]

There is no feature that can definitively discriminate between the two types of diabetes at diagnosis and, therefore, Annie must be assumed to have T1DM until proven otherwise, as misdiagnosis of T1DM may result in clinical deterioration within hours, which is potentially life-threatening.[11,12] Missed cases and delayed referral for insulin commencement are the most common reasons for diabetic ketoacidosis in patients newly diagnosed with T1DM,[13,14] which is a major cause for morbidity and mortality in this population.[11,12] The osmotic symptoms that Annie describes should increase your suspicion of T1DM.[1]

ANSWER 2

The following points are important:

- **Presenting features:** the classical osmotic symptoms of hyperglycaemia (polyuria, polydipsia, weight loss and fatigue) strongly favour a diagnosis of T1DM.[1]
- **Family history:** family history is not usually positive in a child with T1DM but a personal or family history of autoimmune conditions (thyroid disease, coeliac disease, vitiligo, pernicious anaemia, inflammatory bowel disease, rheumatological conditions) may increase the suspicion for a primary autoimmune disorder.[15] Family

history is often contributory in T2DM[15] and although it is important to ask about it, family history does not help to delineate between T1DM and T2DM diabetes in the first instance.

- **Features of the metabolic syndrome:** the presence of acanthosis nigricans in the neck creases, axillae, antecubital fossae, groin and inframammary areas is suggestive of insulin resistance but does not definitively confer a diagnosis of T2DM.[15,16] Other features, such as the presence of non-alcoholic fatty liver disease, hypertension or manifestations of polycystic ovarian syndrome, may also increase your suspicion of T2DM, but may also co-exist in an individual with T1DM.
- **Ethnicity:** ethnicity is important but does not supersede the clinical presentation. Aboriginal and Torres Strait Islander peoples, people from the Pacific Islands, Indian subcontinent and China are at higher risk of T2DM, compared with other groups[15] but even in these populations T1DM should still be the first consideration in childhood.

ANSWER 3

In the primary care setting, if there is a suspicion of diabetes in a child, the first-line investigation should be a random blood glucose level on fingerprick point-of-care testing. As per the American Diabetes Association (ADA) position statement, a diagnosis of diabetes is made where there is a random blood glucose level of ≥11.1 mmol/L or a fasting plasma glucose of ≥7.0 mmol/L, with typical symptoms of hyperglycaemia,[17] which should then be confirmed by a laboratory sample. Note that the ADA diagnostic criteria are recognised worldwide. A fingerprick assessment of ketones using a point-of-care meter should also be done where available. Frequently, children present to the emergency department with typical symptoms of diabetes and are referred for outpatient phlebotomy, with no immediate follow-up of abnormal results. This is concerning as clinical deterioration of these children can lead to diabetic ketoacidosis, a potentially life-threatening situation. Indeed, up to one-third of children who present in diabetic ketoacidosis have had at least one medical contact in the week before presentation.[18]

The use of an oral glucose tolerance test (OGTT) or fasting blood glucose levels are generally not recommended as part of the diagnostic workup for suspected T1DM in childhood.[1] As second-line investigations, these should be reserved for a child with no osmotic symptoms and a normal blood sugar on random testing, when there is a high suspicion of T2DM. When an OGTT or fasting plasma glucose is requested, there should be immediate notification of abnormal results to the requesting physician, with same-day specialist referral for any positive results.

If the above results are equivocal, there should be discussion about further options with a paediatric endocrinologist, who is likely to advise referral for inpatient blood glucose monitoring given the nature of this presentation.

ANSWER 4

If a random blood glucose level is diagnostic or strongly indicative of diabetes, same-day referral for specialist assessment is warranted, as any delay in confirmation of the diagnosis may increase the risk of diabetic ketoacidosis and associated complications.[13] Additional investigations are unnecessary at this time, except to confirm the findings in a laboratory sample.

High levels of blood ketones are indicative of severe insulin deficiency.[19] Recall that ketones are a toxic by-product arising from the degradation of fat stores for energy in the absence of insulin[20] which can lead to the development of diabetic ketoacidosis, a potentially fatal medical emergency. At this time Annie's blood ketone level is within range.

ANSWER 5

Annie's clinical history and laboratory results are suggestive of T1DM and therefore subcutaneous insulin is the appropriate first-line treatment for her.[1] Emphasis should be placed on the underlying diagnosis with its associated insulin deficiency, and insulin therapy should be optimised in the first instance. Insulin resistance in T1DM increases during adolescence[21] and may be a contributing factor to the suboptimal glycaemic control frequently seen in this population. If Annie's insulin requirement approaches 2 units/kg/day, this should be discussed with her endocrinologist as an apparently large insulin requirement may in fact reflect insulin omission (intentionally omitting or reducing the dose of insulin).[22]

In families where there is a strong history of T2DM, managed with either diet alone or oral hypoglycaemic agents, there may be some resistance to insulin therapy. Annie may feel unsupported in her home environment and appropriate education of her family members may be required. Tools are available to assess diabetes-related distress and depression.[15]

ANSWER 6

It is important to emphasise to Annie that T1DM requires lifelong medical management with insulin therapy. Education and psychological support will be essential in helping Annie to adjust to the diagnosis. These should be provided in a culturally, developmentally and age-appropriate manner.[19] You should advise Annie to maintain regular contact with her diabetes healthcare team and ensure that she receives information about:[19]

- preventive interventions at key developmental stages: these interventions emphasise appropriate family involvement and support for Annie in managing her diabetes
- access to mental health professionals for psychological support
- flexible insulin therapy programs.

Education should also include nutritional/dietary and lifestyle advice, encouraging healthy lifelong eating habits, which includes advice about:[19]

- carbohydrate quantification and insulin-to-carbohydrate ratios
- the benefits of low-glycaemic index food choices
- avoiding high protein, low carbohydrate diets
- reducing intake of saturated fats and substitution of saturated fats with monounsturated and polyunsaturated fats.

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RESOURCES FOR PATIENTS

- The Better Health Channel provides a fact sheet on type 1 diabetes, www.betterhealth.vic.gov.au/bhcv2/bhcarticles.nsf/pages/Diabetes_Type_1_or_juvenile_diabetes [Accessed 27 August 2014].
- NPS Medicinewise provides information on diabetes for patients, www.nps.org.au/

RESOURCES FOR DOCTORS

- Craig ME, Twigg, Donaghue KC, et al. for the Australian Type 1 Diabetes Guidelines Expert Advisory Group. National evidenced-based clinical care guidelines for type 1 diabetes in children, adolescents and adults. Canberra: Australian Government Department of Health and Ageing, 2011, www.nhmrc.gov.au/_files_nhmrc/publications/attachments/ext004_type1_diabetes_children_adolescents_adults.pdf [Accessed 25 July 2014].
- NPS Medicinewise provides information on diabetes for health professionals, www.nps.org.au

CASE 2

JACK HAS BEEN WETTING HIS BED

Jack is 7 years of age and his mother, Sarah, has brought him to see you as he has recently started wetting his bed at night. She thinks the bedwetting is associated with Jack being thirsty all the time for the past few weeks and needing to drink lots of water. She is also worried because Jack has lost weight.

QUESTION 1

What differential diagnoses would you consider?

QUESTION 2

Sarah asks if Jack's diabetes could have been prevented. How would you respond?

QUESTION 3

What is the likelihood of Jack's siblings developing diabetes?

FURTHER INFORMATION

After confirming the diagnosis of type 1 diabetes mellitus (T1DM) and starting insulin therapy, Sarah calls you to say 'Jack's diabetes has gone away'.

QUESTION 4

What would you say to her?

QUESTION 5

What are the long-term consequences of T1DM? What routine screening for complications is recommended in T1DM?

QUESTION 6

Jack's mother asks you if he will lead a 'normal' life.

CASE 2 ANSWERS

ANSWER 1

Jack is a child who presents with a classical history of increasing polyuria, polydipsia and weight loss over a few weeks. This presentation should not usually pose a diagnostic difficulty for T1DM.[1] As in Case 1, if there is a suspicion of diabetes in a child in the primary care setting, the first-line investigation should be a random blood glucose level on fingerprick point-of-care testing and, where available, a fingerprick assessment of ketones. If a random blood glucose level is diagnostic or strongly indicative of diabetes, same-day referral for specialist assessment, management and initiation of therapy is warranted, as any delay in confirmation of the diagnosis may increase the risk of diabetic ketoacidosis and associated complications.[2]

It is important to be aware of the various non-emergency presentations of diabetes. As in Jack's case, a recent onset of secondary enuresis and polyuria in a previously toilet-trained child should raise suspicions of T1DM. However, it could be misdiagnosed as a urinary tract infection[1] or the result of excessive fluid ingestion. Note that it is possible to have a urinary tract infection in addition to T1DM. Similarly, polydipsia may be thought to be psychogenic.[1] Vomiting, if present, may be misdiagnosed as gastroenteritis or sepsis.[1] Chronic weight loss or failure to gain weight in a growing child is worrying, as are recurrent skin infections or vaginal candidiasis, especially in prepubertal girls. It is always important to enquire about irritability and decreasing performance at school.

Consider differential diagnoses carefully as some situations may result in a late diagnosis of diabetic ketoacidosis. For example, the hyperventilation of ketoacidosis (Kausmaul breathing) may be misdiagnosed as pneumonia or asthma, although cough and breathlessness distinguish these conditions from diabetic ketoacidosis. In addition, the abdominal pain associated with ketoacidosis may simulate an acute abdomen and lead to referral to a surgeon.[1] An Australian study found that a population awareness campaign was effective in reducing the number of children who presented in diabetic ketoacidosis by 64%. In the intervention group, all childcare centres, schools and doctors' offices in the region received an educational poster illustrating four common signs of diabetes, including weight loss, increased thirst, increasing urination and fatigue. The early recognition of symptoms, prompted diagnosis and treatment helped avoid diabetic ketoacidosis in the majority of children.[3]

We know that T1DM is a chronic autoimmune disease in the majority of patients and accounts for over 90% of childhood and adolescent diabetes in Australia.[4] T-cell-mediated destruction of the pancreatic beta cells leads to insulin deficiency.[1,5] T1DM includes those cases attributable to an idiopathic cause for which neither an aetiology nor a pathogenesis is known. Regardless of its aetiology, the clinical staging of T1DM reflects the new concept that diabetes progresses through several clinical stages during its natural history. These stages are characterised by preclinical, clinical, partial remission and chronic phases;[1,6] that is, normoglycaemia, preclinical diabetes (impaired glucose regulation, which includes impaired fasting glycaemia or impaired glucose tolerance) and then overt presentation of T1DM.[1,7] The important point to note is that individuals may move from stage to stage in either direction, and understanding this time continuum of presentation can aid diagnosis. In patients with classical T1DM, such as Jack, reversion to more normal glucose levels is not possible without insulin therapy.

ANSWER 2

Current guidelines do not recommend any interventions for use in clinical practice to delay or prevent T1DM as, to date, all clinical trials attempting to prevent or delay the onset of T1DM in those at high risk have been unsuccessful.[1] The most important of these intervention studies were the European Nicotinamide Diabetes Intervention Trial (ENDIT), which showed that nicotinamide did not delay or prevent the onset of T1DM in high-risk first-degree relatives,[8] and the Diabetes Prevention Trial (DPT), in which low-dose subcutaneous insulin therapy did not delay or prevent the onset of clinical diabetes in first-degree relatives.[9] Screening of any population or intervention in the preclinical phase should not occur outside the context of defined clinical studies and research settings.[10] Individuals who test positive for genetic or immunological markers of T1DM should have access to appropriate counselling and to centres participating in intervention and other defined studies.

In T1DM, progressive destruction of beta cells occurs at a variable rate and diabetes becomes clinically symptomatic when approximately 90% of the pancreatic beta cells are destroyed.[11] Insulin deficiency then manifests clinically as blood glucose levels rise to pathological levels. It is important to explain to the patient and the family our current understanding about the multifactorial pathogenesis of diabetes whereby T1DM results from an interplay between genetic predisposition and environmental factors.[1] Many parents experience feelings of guilt, assuming that T1DM occurred as a result of allowing their child to have a high-sugar diet. These misconceptions need to be addressed early on.

ANSWER 3

Families need to be aware that there is no recognisable pattern of T1DM inheritance. Susceptibility to autoimmune T1DM is determined by the interaction between multiple genes, with HLA genes having the strongest known association. These genetic markers can confer either an increased or decreased risk.[1] However, several studies have provided valuable insight in further characterising aspects of the disease. The overall risk for first-degree relatives in singleton families is 4–6%[12] and rises to 15% if two first-degree relatives are affected. Alternatively, concordance for T1DM between monozygotic twins is around 36%.[13] Hence, the genetic load on risk is relatively low. Interestingly, T1DM is transmitted more frequently to the offspring of diabetic men than those of diabetic women (6.1%, compared with 1.3 % of offspring).[14] In summary, Jack's siblings have a 95%

chance of not developing T1DM. However, as with everyone else in the population, their chances of developing type 2 diabetes mellitus (T2DM) diabetes at some time in their lives is greater than 1 in 3.[15]

Although we do not fully understand the multifactorial pathological processes leading to T1DM, we understand that these processes can start several months to years before clinical symptoms are manifested.[1] The most important information to convey about sibling risk is that although parents would not be able to alter the course of disease progression, if they notice any early symptoms, Jack's siblings should be investigated early and referred to a specialist diabetes centre for management, as discussed earlier. Prospective follow-up of high-risk individuals shows that diagnosis of T1DM can be made in asymptomatic individuals in the majority of cases, as shown in the DPT: when high-risk individuals were followed up, 73% of participants who were diagnosed with diabetes were asymptomatic.[9]

The onset of the disease is predictable, especially in the relatives of affected individuals, using a combination of auto-antibody measurements, glucose tolerance testing and genotyping.[16] The parameters currently helping to define the preclinical phase include the islet cell autoantibodies – glutamic acid decarboxylase (GAD) autoantibodies, IA2 (tyrosine phosphatase) autoantibodies, insulin autoantibodies – and human leukocyte antibody (HLA) typing.[1] Relatives with at least two of the autoantibodies were found to have a risk of 39% (95% CI, 27–52) and 68% (95% CI, 52–84) of developing diabetes within 3 and 5 years, respectively. For those with all three autoantibodies the risk of developing diabetes within 5 years was estimated to be 100%.[17] These findings suggest that the presence of two or more of the autoantibodies (IAA, GAAs and ICA512bdcAAs) is predictive of T1DM developing in relatives of affected individual.[17]

We also know that environmental triggers (chemical and/or viral) that start the process of autoimmune destruction of the pancreatic beta cells are yet to be fully described.[1] Congenital rubella[18] and other potential environmental triggers like enteroviral infections[19] (particularly coxsackie and ECHO viruses[20,21]), casein/cow's milk proteins[22] and gluten[23] have all been implicated. There is an international race on with trials that are investigating potential triggers, describing seasonal variation and researching protective factors in an attempt to understand the multifactorial nature of T1DM.

ANSWER 4

In approximately 80% of children and adolescents, insulin requirements decrease transiently following initiation of treatment.[24] This is known as the partial remission or honeymoon phase.[1,24] Most studies define a partial remission phase when the patient requires less than 0.5 units/kg/day of insulin and has an HbA1c <7%.[24] The honeymoon phase can commence within days or weeks of starting insulin therapy and may last for weeks to months.[10] During this phase, blood glucose levels are frequently stable within the normal range, despite fluctuations in diet and exercise, and insulin therapy may even be ceased.

Patients and families should be advised of the transient nature of the honeymoon period, to avoid the false hope that the diabetes has spontaneously gone into remission.[10] There is no evidence that any intervention has any effect on the length of the honeymoon period.[25,26] As the chronic phase of the lifelong dependence on insulin gradually occurs, it is important to support the patient and family as the only treatment for managing T1DM is insulin. There is no alternative and insulin is essential for survival.

ANSWER 5

Death of people with T1DM is usually due to diabetes-related complications.[1] The main long-term microvascular complications of diabetes can cause blindness due to diabetic retinopathy, renal failure due to diabetic nephropathy, and diabetic neuropathy which can be both peripheral and autonomic. Hypertension, dyslipidaemia and smoking influence the development of both microvascular and macrovascular complications.

Current Australian guidelines for routine screening or monitoring for complications in patients with T1DM are shown in *Table 1*.[1]

Table 1. NHMRC guidelines for screening and monitoring for complications in T1DM[1]

Complication	Commence screening	Monitoring frequency
Retinopathy	After 2 years duration in adolescents and adults, and after 5 years duration or from age 9 in children	Every second year or annually for select groups (eg high-risk groups)
Nephropathy	After 2 years duration in adolescents and adults, and after 5 years duration or from age 9 in children	Annually
Neuropathy	Annually	Annually
Lipids	At diagnosis if there is a family history or from 12 years	Every 5 years until puberty and then annually
Blood pressure	At diagnosis	At least annually
Macrovascular disease	In adulthood	At least annually

ANSWER 6

Some of the fundamentals of successful diabetes management, which can help patients lead a normal, healthy life, are discussed below.

T1DM is a chronic disease among children and adolescents and its diagnosis and management usually involves a significant burden on the patient and their families, as they must change various aspects of their lifestyle to fulfil the demands of treatment.

Clinicians need to be aware that psychological disorders commonly co-exist in those with T1DM and that validated screening tools for psychological disorders in T1DM are available.[1] They should be able to provide appropriate support to the patient and family for a healthy

adjustment to the diagnosis of T1DM or referral to other providers as necessary.[27] Psychological interventions have been shown to improve psychosocial outcomes and have an effect on HbA1c reduction, although modest. A pooled analysis of 10 studies found a 0.5% reduction in HbA1c following psychological interventions.[1] Although there was significant variability in these reports on the benefits of psychological interventions on metabolic control, distress was significantly reduced. Results of a 2012 systematic review reported that generic quality of life (QoL) of children and adolescents with T1DM was not impaired when compared with healthy peers, despite having to live with a demanding treatment regime. Nevertheless, disease-specific QoL problems such as a negative impact of diabetes on daily functioning and diabetes-related worries were present.[27]

Diabetes education and access to care by a multidisciplinary team trained in childhood diabetes is essential.[7] Education provision should be adapted to each individual's age, maturity, stage of diabetes, lifestyle and culture. Any insulin regimen should be considered in the wider context of a total diabetes management package, which must include dietary management, exercise and physical activity, blood glucose monitoring, initial and ongoing education, regular medical follow-up and psychological care.[7]

Intensive management (including multiple daily injections or pump therapy, education, intensive monitoring and psychosocial support) of T1DM in adolescents improves metabolic control and reduces the risk of microvascular complications.[28] However, the long-term benefits of intensifying management need to be weighed against the risks of frequency and severity of hypoglycaemia. This is because severe hypoglycaemia has been associated with lower verbal and full-scale intelligence quotient scores; attention, processing speed and executive skills were mostly affected in children with younger-onset of diabetes.[29] To this end, current guidelines recommend making every effort to achieve glycaemic targets to help minimise the potential impact of diabetes on cognitive function. The guidelines also recommend minimising acute episodes of hypoglycaemia and hyperglycaemia to help maintain optimal cognitive performance.[1]

The Diabetes Control and Complications Trial (DCCT) confirmed the benefit of maintaining near normal glycaemia in reducing the development and progression of diabetic microvascular complications in adolescents and adults.[30,31] The Epidemiology of Diabetes Interventions and Complications (EDIC) study reported that people with T1DM (aged 13–39 years at baseline) receiving intensive diabetes treatment had decreased progression of intima–media thickness, a marker for coronary and cerebrovascular disease, compared with patients receiving conventional therapy, over the mean follow-up period of 6.5 years.[32] In older children and adolescents, the target HbA1c should be <7.5%.[33]

In summary, T1DM in childhood imposes a number of psychological stresses on both the child and the family. However, open and frequent communication will help everyone to continue to work towards the primary goals of treatment, which include maintaining near-normoglycaemia through intensive insulin therapy, avoiding acute and long-term complications through regular screening, while balancing these needs to maintain as close to a normal, fulfilled life as possible for the person with diabetes. General practice management plans and team care arrangements could also be considered to help improve clinical outcomes for patients.

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RESOURCES FOR PATIENTS

- The Better Health Channel provides a fact sheet on type 1 diabetes, www.betterhealth.vic.gov.au/bhcv2/bhcarticles.nsf/pages/Diabetes_Type_1_or_juvenile_diabetes [Accessed 27 August 2014].
- NPS Medicinewise provides information on diabetes for patients, www.nps.org.au

RESOURCES FOR DOCTORS

- Craig ME, Twigg, Donaghue KC, et al. for the Australian Type 1 Diabetes Guidelines Expert Advisory Group. National evidenced-based clinical care guidelines for type 1 diabetes in children, adolescents and adults. Canberra: Australian Government Department of Health and Ageing, 2011, www.nhmrc.gov.au/_files_nhmrc/publications/attachments/ext004_type1_diabetes_children_adolescents_adults.pdf [Accessed 25 July 2014].
- NPS Medicinewise provides information on diabetes for health professionals, www.nps.org.au

CASE 3

ERICA IS STRUGGLING WITH BLOOD SUGAR CONTROL AND HER WEIGHT

Erica, a retired schoolteacher aged 62 years, was diagnosed with type 2 diabetes (T2DM) 3 years ago. She has been struggling with glycaemic control and her weight. At her last visit she stated that her 'sugars' must be bad as she is up all night urinating. She is on metformin slow release 2 g/day and gliclazide slow release 120 mg at night. Her last HbA1c was 7.6% (60 mmol/mol) 1 month ago. She has come to see you today for her diabetes review and states that her main concerns are her weight and frequent urination.

QUESTION 1

What clinical possibilities need to be considered at this time?

QUESTION 2

Does ethnicity affect the classification of her obesity?

FURTHER INFORMATION

Erica is often hungry in the mornings and therefore eats a large breakfast. She has gained weight since she started gliclazide. You advise Erica that she may have some hypoglycaemia leading to compensatory 'defensive snacking', which could be contributing to her weight gain. Erica does not self-monitor her glucose levels.

QUESTION 3

What are the current recommendations for self-monitoring of blood glucose for patients using oral medications?

QUESTION 4

Which glucose-lowering agents commonly cause hypoglycaemia? Which agents have low rates of hypoglycaemia?

FURTHER INFORMATION

Erica would like to lose some weight and would like to know your thoughts on this. Her body mass index (BMI) is 34 kg/m^2 and her waist circumference is 90 cm.

QUESTION 5

What are the key elements of a weight management plan? How would you incorporate physical activity and weight loss advice?

FURTHER INFORMATION

You referred Erica to a dietitian and they have started working on a weight-loss plan. Erica has also been regularly attending aerobic classes and yoga.

QUESTION 6

Which diabetes medications have been shown to increase weight? Which medication(s) assist weight loss? Which medications may assist Erica? Why?

CONCLUSION

Erica lost 7 kg and has maintained this weight loss. She continues to exercise regularly and has added 30–60-minute walks several times a week to her exercise program. No changes to the doses of her diabetes medications were required as her glycaemic levels were at target following her weight loss.

CASE 3 ANSWERS

ANSWER 1

An HbA1c of 7.6% is unlikely to cause polyuria. Either blood glucose levels have risen since this HbA1c measurement or the HbA1c is not accurately reflecting Erica's level of glycaemia or there is another cause. T2DM can be associated with increased risks of fungal or bacterial infection[1] such as urogenital infections. It may be relevant to assess Erica for the presence of other potential causes of her frequent urination, such as bladder prolapse, or excessive caffeine or alcohol intake, or metabolic causes such as hypercalcaemia or renal disease.

It is important to review Erica's history and seek possibilities for alternative causes of weight gain, such as poor diet, depression, hypothyroidism or less common conditions such as Cushing syndrome, as well as cardiovascular diseases such cardiac failure. Obesity and T2DM combined may present with other comorbidities that aggravate glycaemia and also further increase obesity risks. Examples include obstructive sleep apnoea, non-alcoholic liver steatosis, gastro-oesophageal reflux and arthritis. Obesity may also aggravate urinary incontinence, which has relevance to her presenting symptoms.[1]

ANSWER 2

The standard classification for overweight or obesity is a BMI of 25.0–29.9 kg/m^2 and >30.0 kg/m^2, respectively. People from South Asian, Chinese and Japanese population groups may have more body fat at lower weights and may be at greater risk of ill health than people from other population groups, so guidelines suggest considering a lower BMI threshold (ie >23 kg/m^2) for these populations.[2] By contrast, people from the Pacific Islands (including Torres Strait Islander peoples and Maoris) tend to have a higher proportion of lean body mass and guidelines suggest considering a higher BMI threshold.[2] Aboriginal peoples have a high limb-to-trunk ratio, compared with other groups, so guidelines suggest considering a lower BMI threshold.[2]

ANSWER 3

Current guidelines on self-monitoring of blood glucose are outlined in section 8.1 of *General practice management of type 2 diabetes 2014–15*.[1]

Self-monitoring is recommended for people with diabetes using insulin, those with hyperglycaemia arising from illness or with haemoglobinopathies, for pregnant women or for people with any other condition where data on glycaemic patterns are required. Self-monitoring is also recommended in patients who are on medications that may cause hypoglycaemia, such as sulphonylureas, which Erica is currently taking. It would be useful to educate Erica on the practicalities and benefits of self-monitoring her blood glucose levels. Note that with the exception of patients using sulphonylureas, routine self-monitoring is not recommended in low-risk patients using oral glucose-lowering drugs.[1]

ANSWER 4

Insulin and sulphonylureas can cause hypoglycaemia. Of the sulphonylureas, gliclazide and glipizide are least likely to cause hypyglycaemia.[1] Other agents including metformin, acarbose, glitazones, glucagon-like peptide-1 (GLP-1)-mimetics and dipeptidyl peptidase-4 (DPP4) inhibitors will not cause hypoglycaemia when used as monotherapy.[1]

The risks of hypoglycemia may escalate when dual or triple combination glucose-lowering therapy is used.

ANSWER 5

A weight management plan should incorporate general advice about weight loss and physical activity and provide dietary advice. Weight management has been shown to be more difficult in people with T2DM.[4–6]

Weight loss

Weight loss in people with T2DM often results in improved glycaemic control, blood pressure and lipid profiles. A sustained weight reduction of about 5 kg has been associated with a reduction in HbA1c of 0.5–1%. In adults with a BMI <35 kg/m^2 and dysglycaemic states or hypertension, weight loss of at least 2–3 kg achieved with lifestyle interventions may result in clinically meaningful systolic blood pressure reductions (an average of 4.5 mmHg systolic and 3–3.5 mmHg diastolic).[2] Any level of weight loss should be encouraged and even losses of 5–10% will improve glycaemic control.[1,7,8]

Physical activity

The greatest health benefits attributable to physical activity are seen in those who change from doing no physical activity, or very little, to doing more.[9] *Table 1* shows the different intensities of physical activity, which is useful information that could be discussed with patients.

Table 1. Levels of intensity of physical activity[2]

Intensity	Description	Examples
Sedentary	Activities that involve sitting or lying down, with little energy expenditure	Occupational (eg sitting at work) Leisure (eg watching TV, reading, sewing, computer use for games, social networking) Transport (eg sitting in a car, train, bus or tram)
Light	Activities that require standing up and moving around in the home, workplace or community	Housework (eg hanging out washing, ironing, dusting) Working at a standing workstation
Moderate	Activities are at an intensity that requires some effort, but allow a conversation to be held	Brisk walking Gentle swimming Social tennis
Vigorous	Activities that lead to harder breathing or puffing and panting (depending on fitness)	Aerobics Jogging Some competitive sports

Any commencement of increased activity should be supported and reinforced by clinicians. Health benefits are achieved with 150–300 minutes of moderate-intensity activity or 75–150 minutes of vigorous activity (or a combination of moderate-intensity and vigorous activity), accumulated over a week.[9] Current evidence suggests that physical activity has little effect on weight loss unless it is combined with a reduced energy intake.[10–12] Even so, increased physical activity has been associated with a range of health benefits, including improved cardiovascular disease risk factors, even in the absence of weight is loss.[11,12] Note that initial weight gain, due to an increase in muscle mass, has been associated with the commencement of muscle strengthening exercises.[2]

Dietary advice

Aiming for a reduction of 2000–2500 kilojoules (478–598 calories or 10%) in total daily energy intake per day should result in a weight loss of about 0.5 kg per week.[1]

Practical advice in constructing a weight management plan for adults who are obese can be found in the National Health and Medical Research Council (NHMRC) *Summary guide for the management of overweight and obesity in primary care 2013*,[13] which is based on the 5As approach (ask and assess, advise, assist, arrange).

Ask and assess

- Measure waist circumference and calculate BMI.
- Discuss readiness to change lifestyle behaviours as well as other psychological factors, such as comorbid depression, using primary practice tools such as *The Patient Health Questionnaire-2* (PHQ2) and *Problem Areas in Diabetes Questionnaire* (PAID).

Advise

- Convey the message that even small amounts of weight loss improve health and wellbeing.

Assist

- Use multicomponent approaches as these work better than single interventions.
- Refer appropriately to assist people to make lifestyle changes or for further intervention.

Arrange

- Support a self-management approach and provide ongoing monitoring.
- Use a multidisciplinary approach to support.

ANSWER 6

In a meta-analysis of 27 randomised controlled trials of about 11,200 participants, the addition of sulphonylureas and thiazolidinediones to maximal doses of metformin was associated with weight gain of 2.06 kg and 2.08 kg respectively. No weight change was reported with DPP4 inhibitors, whereas GLP1 agonists were associated with weight loss of 0.74 kg. All of the drugs had a similar effect on glycaemic control.[14] In a systematic review of sodium-glucose transporter 2 (SGLT2) receptor inhibitors available on the Pharmaceutical Benefits Scheme (PBS), dapagliflozin was associated

with weight loss of 1.81 kg and canagliflozin with a loss of 2.3 kg compared to placebo.[3]

There is no correct answer regarding the use of diabetes medications for Erica. If she can use lifestyle interventions to achieve weight loss, this may lead to improvements in her glycaemic control and she may not need to increase the doses of any of her medications or to use alternative or additional medications. Additionally, depending on her weight loss, her medication needs may decrease.

Diabetes often requires combination therapy to allow patients to remain at their glycaemia targets as the disease progresses. Metformin remains the drug of first choice for initial drug therapy in T2DM,[1,15] but decisions regarding which drug to add next and combination therapy are hampered by a lack of long-term, high-quality outcome data, particularly for cardiovascular endpoints. To date the most studied agents include metformin and sulphonylureas.[1]

If Erica's blood glucose levels remain uncontrolled and above target (>7% or 53 mmol/mol) it may be worth considering a combination of metformin and a GLP1 agonist or metformin and an SGLT2 receptor inhibitor; triple combination of metformin, sulphonylurea and GLP1 agonist (or alternatively an SGLT2 receptor agonist) may also be an option. These combinations may not all be supported by reimbursement under the PBS, so prescribers would need to check current PBS indications for available prescribing choices.

It is also important to consider the potential side effects of any additional agents that may be prescribed. For example, there is a risk of nausea or vomiting with use of GLP1 agonists, which occurs in up to 50% of patients but, in most cases, disappears after 1–2 weeks of continued treatment.[16] There is an increased risk of urogenital infections and possible urinary tract infections for the SGLT2 receptor inhibitors.[1] Newer agents such as these do not as yet have high-quality, long-term prospective cardiovascular safety trials. This should be discussed with patients when considering their clinical use.

Renal dysfunction may require a change in medication and/or dosage:

- Metformin: reduce maximum dose when creatinine clearance (CrCl) is <90 mL/minute[17]
  - 2 g recommend daily for when CrCl is 60–90 mL/minute
  - 1 g daily when CrCl is 30–60mL/minute
  - contraindicated when <30 mL/minute.
- SGLT 2 receptor inhibitors:[18,19]
  - dapagliflozin is contraindicated if CrCl <60 mL/minute
  - canagliflozin is contraindicated if CrCl <45 mL/minute)
  - care in combination with loop diuretics.

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RESOURCES FOR PATIENTS

- Diabetes Australia provides resources including guidance on diet and physical activity, www.diabetesaustralia.com.au/Living-with-Diabetes/Eating-Well

RESOURCES FOR DOCTORS

- www.healthyactive.gov.au
- www.health.gov.au/internet/main/publishing.nsf/Content/Nutrition+and+Physical+Activity-1

- General Practice management of type 2 diabetes 2014-15, www.racgp.org.au/your-practice/guidelines/diabetes
- NHMRC summary guide for the management of overweight and obesity in primary care 2013, www.nhmrc.gov.au/_files_nhmrc/publications/attachments/n57b_obesity_guidelines_summary_guide_131219.pdf
- NHMRC clinical practice guidelines for the management of overweight and obesity in adults, adolescents and children in Australia, www.nhmrc.gov.au/_files_nhmrc/publications/attachments/n57_obesity_guidelines_131204_0.pdf P25
- Guidelines for preventive activities in general practice 8th edition Section 7.2, overweight, www.racgp.org.au/your-practice/guidelines/redbook/prevention-of-chronic-disease/overweight/
- Diabetes Australia Position paper on bariatric surgery. Diabetes Australia 2011; www.diabetesaustralia.com.au/PageFiles/3/BariatricSurgery_PositionStatement_211211.pdf
- Indigenous focussed healthy lifestyle practice resources, www.aihw.gov.au/uploadedFiles/ClosingTheGap/Content/Publications/2012/ctgc-rs09.pdf

CASE 4

FRANK IS SHORT OF BREATH

Frank is 86 years of age and is one of your long-term nursing home patients. He has been deteriorating slowly over the past 12 months and is known to the local palliative care team. He has become increasingly frail, has been less mobile and has a decreased appetite. Recently he has had difficulty swallowing some of his medication. He has a past history of end-stage chronic obstructive pulmonary disease (COPD), type 2 diabetes mellitus (T2DM), several strokes and dementia. His medications include metformin, glicazide, tiotropium bromide, salbutamol, aspirin, a statin and an angiotensin converting enzyme inhibitor (ACEI).

Today when you visit he is very quiet and withdrawn. Increasingly, he experiences shortness of breath but his temperature is normal and his heart rate and blood pressure are stable. He has decreased air entry at the base of both lungs but normal percussion. His abdomen is normal and a urine dipstick test shows 2+ glucose, but is otherwise negative. A chest X-ray shows COPD but no evidence of consolidation. His blood sugar level is 19 mmol/L.

QUESTION 1

Why is diabetes mellitus important when considering the needs of a patient requiring palliative care?

QUESTION 2

What are appropriate blood glucose readings at the end of life?

QUESTION 3

What factors affect glycaemic control in patients with diabetes at the end of life?

QUESTION 4

How can poor glycaemic control negatively impact on a patient's quality of life at end of life?

QUESTION 5

How would you manage Frank's diabetes now?

CASE 4 ANSWERS

ANSWER 1

Diabetes mellitus is a common condition and its prevalence is increasing. Globally it has been estimated that from 2010 to 2030 there will be a 69% increase in adults aged 20–79 years with diabetes in developing countries, and a 20% increase in developed countries.[1] At the current rate of growth, it is estimated that the number of Australians with T2DM will increase from about 950,000 to over 2.5 million in 20 years.[2] As the population ages, there will be more patients with diabetes who will require palliative care. Diabetes is also one of the most common comorbidities in cancer patients. It is associated with increased risk of death from several forms of cancer including liver, pancreas, endometrial, colon and breast cancer in women, and breast, liver, oral cavity/pharynx, pancreas, bladder and colon cancer in men.[3]

The management of diabetes in palliative care settings aims to give the best quality of life to the patient, while aiming to achieve glucose readings in a range that do not cause symptoms. In particular, the avoidance of metabolic decompensation and diabetes-related emergencies (eg frequent and unnecessary hypoglycaemia, persistent symptomatic hyperglycaemia, diabetic ketoacidosis) are key principles of care. In most cases, tight glycaemic control is no longer appropriate in patients nearing the end of life.[4]

ANSWER 2

The consensus of several guidelines suggest a range of 6–15 mmol/L is an appropriate blood glucose target for most patients in palliative care.[4–6] Readings at this level are optimal for maintaining patient wellbeing and cognitive function.[7,8] As patients will be aware of the blood glucose targets usually set in diabetes and those set for them previously, explanation and reassurance may be required.[4] If patients are stable and are not in the terminal phase of care, maintaining HbA1c at no lower than 59 mmol/mol (7.5%) when taking oral hypoglycaemic medication will avoid hypoglycaemia.[6] Hypoglycaemia is always more of a risk if the patient is taking insulin or sulphonylureas and these medications should be reviewed and the dose changed or stopped if appropriate.[4] Frank's blood sugar level is currently 19 mmol/L, despite being on metformin and gliclazide treatment.

There is minimal evidence for managing diabetes at the end of life, including information about an optimal blood glucose range, as patients at the end of life are a vulnerable and heterogenic group, and recruitment to studies is difficult.[4,5] Moreover, tight glycaemic control is unlikely to be of benefit in people with major life-limiting comorbidities, given that diabetic complications take years to develop.[9]

ANSWER 3

Factors that can affect blood glucose levels at the end of life include the stress response to sustained illness, organ failure leading to renal or liver dysfunction, the malignancy, chemotherapy, use of steroids for symptom control, frequent infections, anorexia and cachexia, nausea and vomiting, dehydration, swallowing problems and weight loss.[6]

ANSWER 4

Hyperglycaemia may worsen confusion, thirst and incontinence, and impair cognitive function.[7,8] Blood glucose levels above 15 mmol/L may cause polyuria and an increased risk of infections. Diabetic ketoacidosis can mimic terminal illness and if not recognised and treated it can be fatal.[6]

Hypoglycaemia can cause discomfort, confusion and impaired cognitive function.[6]

ANSWER 5

Frank's blood glucose, which is 19 mmol/L, is above the recommended optimal level, despite his being on treatment. This may be attributable to his decreased mobility, decrease in appetite or deterioration of his clinical condition.[6] However, hyperglycaemia can negatively impact on Frank's cognition and comfort. Depending on Frank's prognosis, additional treatment of his diabetes may be warranted. If Frank has weeks or months to live then his hyperglycaemia could be treated while rationalising his medication (*Figure 1*). If, however, Frank is in his final days of life, then the focus of his diabetes care should be ensuring his comfort (*Figure 2*).

When rationalising medication, several key points should be considered. Changes in appetite or difficulty with swallowing require a review of tablet dose and frequency. Metformin in particular can cause gastrointestinal symptoms and worsen appetite, so review of its use may be warranted.[4] Glucagon-like peptide 1 (GLP-1) agonists can cause nausea, vomiting, weight loss and reduced appetite.[4,5] Avoidance of dietary sugars may no longer be appropriate as food choices become limited,[4] and so involving a dietitian may be useful in addition to adjusting therapy. Avoiding long-acting sulfonylurea preparations can decrease the risk of hypoglycaemia in patients with poor dietary intake, and renal or hepatic failure.[4,6] Low-dose insulin may be the only option for patients whose glucose levels are high and are symptomatic despite significantly reduced dietary intake.[4] Patients who were on insulin for type 1 (T1DM) or T2DM previously will need lower doses if dietary intake is poor and a daily long-acting insulin may be a more appropriate regime.[4,6] In complex cases, liaison with the local palliative care team or diabetes team is recommended.

It is important to discuss any changes to a patient's diabetic management with the patient where possible, as well as family members or carers. Managing diabetes during the end of life can be challenging for the practitioner, patient and family.[10] For some people the change or even withdrawal of medication, especially insulin, can be distressing as some may view it as life-sustaining,[10] whereas other people may feel that managing diabetes in addition to their terminal illness is 'pointless'.[6] Palliative Care Australia has information sheets that can be accessed via their website that can help with patient or carer education.

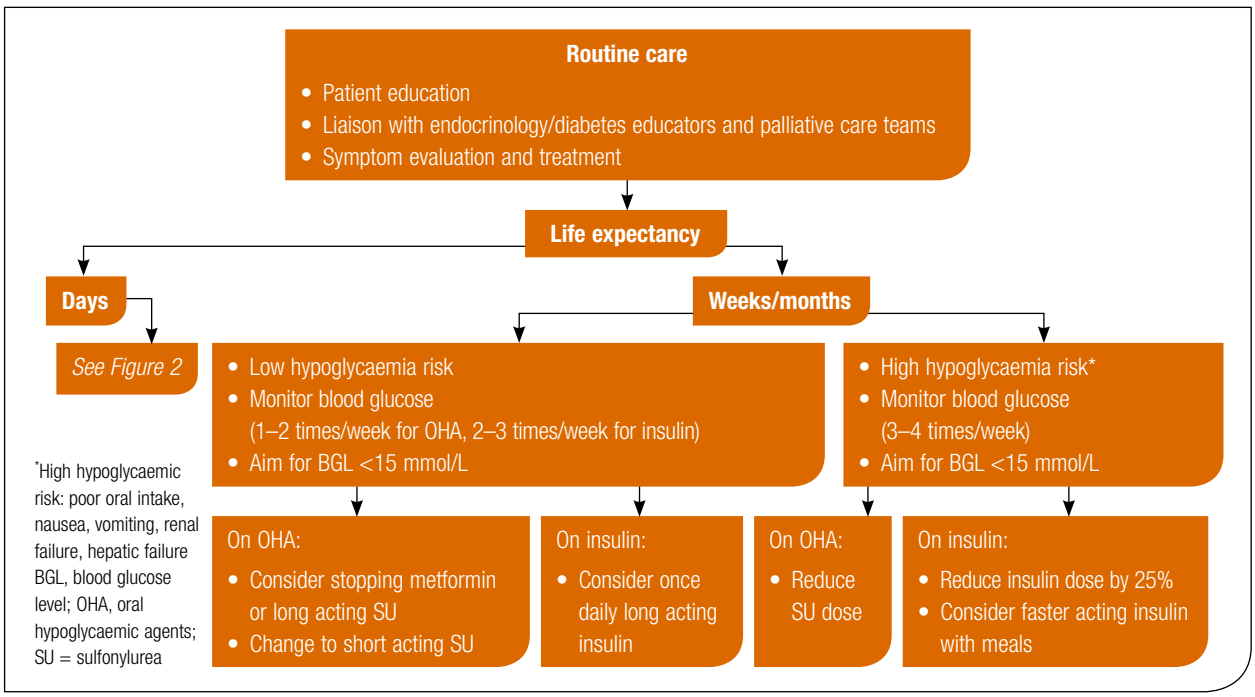

Figure 1. Algorithm for management of patients with diabetes with weeks or months to live[11]

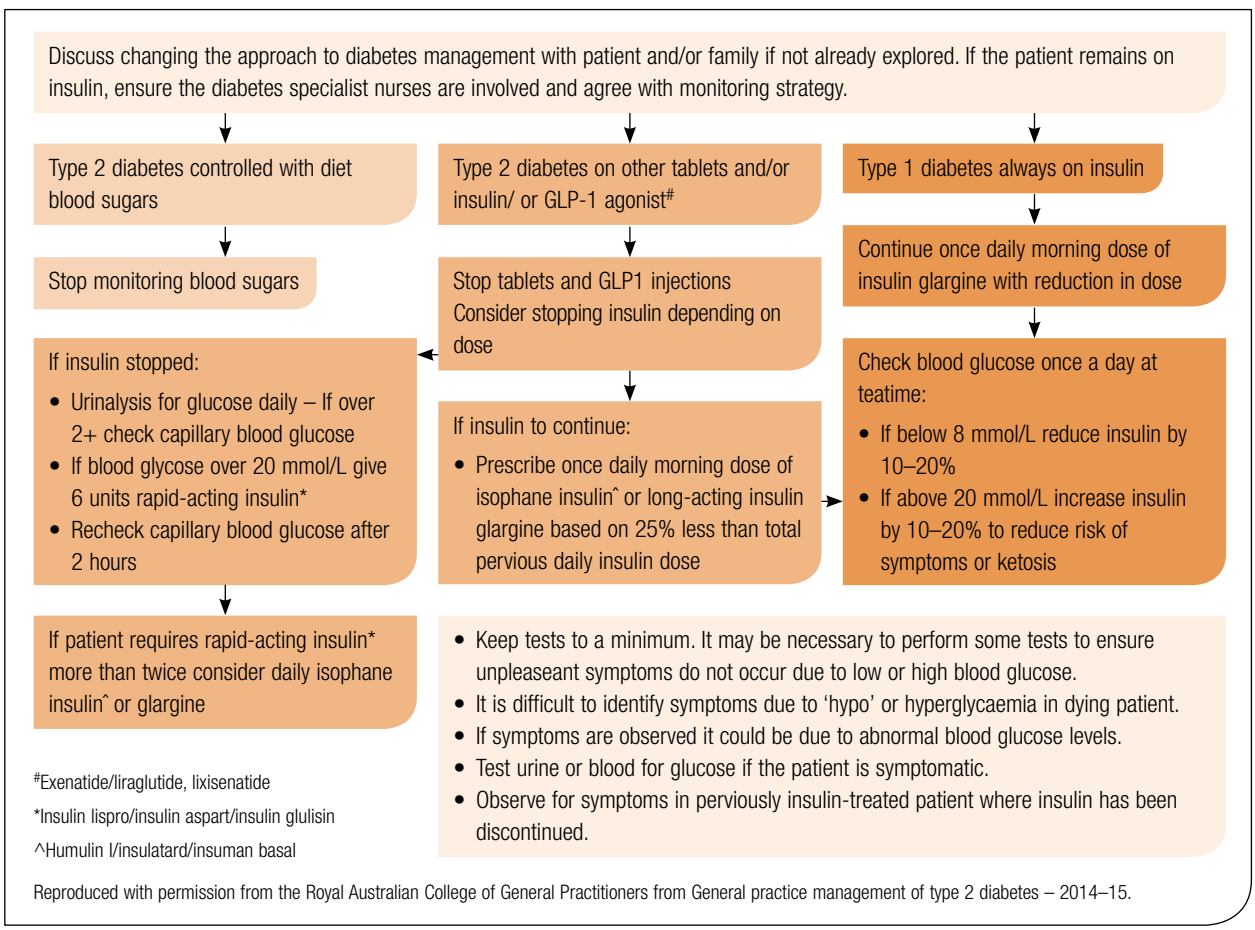

Figure 2. Algorithm for an end-of-life diabetes care management strategy for a patient with days to live[7]

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RESOURCES FOR PATIENTS AND CARERS

- Diabetes and Palliative Care. Information for people with diabetes and family members of people with diabetes, www.palliativecare.org.au/Resources/Consumerresources.aspx

RESOURCES FOR DOCTORS

- The Royal College of General Practitioners and Diabetes Australia. General practice management of type 2 diabetes 2014–2015. Melbourne: RACGP and Diabetes Australia, www.racgp.org.au/your-practice/guidelines/diabetes and www.diabetesaustralia.com.au/PageFiles/763/RACP%20Guidelines%20PDF.pdf
- Diabetes UK. End-of-life diabetes care: clinical care recommendations, www.trend-uk.org/documents/End_of_Life%20clinical%20 recommendations.pdf

CASE 5

FRED IS WORRIED ABOUT HIS WEIGHT

Fred is a busy professional aged 48 years. Recently he was diagnosed with type 2 diabetes (T2DM), which is being controlled through dietary intervention. He currently weighs 118 kg and has a body mass index (BMI) of 38 kg/m^2. His weight has steadily increased over the past 20 years. He has tried various diets, including commercial weight loss programs, slimming programs from the chemist, duromine and medication bought over the internet. None of these approaches had led to a sustainable and substantial weight loss, despite some initial successes. His wife has been urging him to do something about his weight for several years. He wants to do something definitive and with his busy work and family schedule does not feel another diet will be of any value. He would like information about adjustable gastric band surgery and requests a referral to see a surgeon.

QUESTION 1

How would you respond, before discussing surgery with Fred?

QUESTION 2

What would you tell Fred about weight loss surgery in general and laparoscopic adjustable gastric banding (LAGB) surgery more specifically?

QUESTION 3

What are the patient selection criteria for bariatric surgery in Australia?

QUESTION 4

What is the mechanism of action of LAGB surgery?

FURTHER INFORMATION

Several months later, Fred has lost 4 kg, but regained 2 kg. Having thought objectively about his options, he has decided that LAGB is his only alternative. He elects to undergo surgery. Placement of a LAGB is performed uneventfully as a day procedure and he is back working 4 days later. As he is well educated about the process and has realistic expectations, Fred adapts well. He achieves a loss of 18 kg in the first 12 months. His diabetes remits. He feels much better and feels energetic enough to go to the gym three times per week and now regularly plays 18 holes of golf. He attends for routine review.

QUESTION 5

What is the role of the GP in managing a patient following LAGB surgery?

FURTHER INFORMATION

Fred returns 4 years later. After losing a peak of 26 kg, he has regained 4 kg. He is worried that he has developed a complication. He has found that he is eating only softer (high caloric) foods and is suffering from heartburn. He is struggling to swallow most solid foods and is regurgitating every second day. He had some saline added to the LAGB eight months ago, but has not returned to the bariatric surgical follow-up team since then.

QUESTION 6

What complications can arise after LAGB?

CASE 5 ANSWERS

ANSWER 1

Although it is likely that bariatric surgery will be beneficial to Fred, several things can be considered prior to surgery to help address his weight problem. For example:

- Encourage Fred to pursue substantial weight loss.
- Gently discuss weight-loss approaches (eg reduced energy diets, increased physical activity) recommended by the National Health and Medical Research Council (NHMRC) guidelines.[1] These approaches include dietary intervention (reduced energy intake), increased physical activity and lifestyle. Specifically, mention that even a small amount of weight loss will have substantial benefits to his overall health.
- Develop a weight management plan for Fred, which could include referral to other health professionals (eg dietitian).
- Advise Fred that even if he undergoes surgery, he will still need to continue to make substantial efforts to control his weight and improve his overall health for the remainder of his life.
- Use this visit as an opportunity to discuss and screen for other medical problems such as complications of his diabetes, obstructive sleep apnoea, hypercholesterolaemia, hypertension, non-alcoholic fatty liver disease and related conditions, as well as psychosocial and psychiatric issues; where appropriate referrals and review should be arranged.
- Emphasise the three key benefits of weight loss, which include improved physical quality of life, improvements in medical problems and long-term reduction of cardiovascular and mortality risks.
- Ensure that Fred has adequate information to make a decision about surgery. Suggest that he look at reputable websites such as Obesity Surgeons Society of Australia and New Zealand (refer to *Resources for doctors*) and obtain further information, for instance by reading the *LAP-BAND Solution* book.[2]
- Schedule a follow-up appointment to review Fred's progress and discuss surgery at that time if appropriate.

ANSWER 2

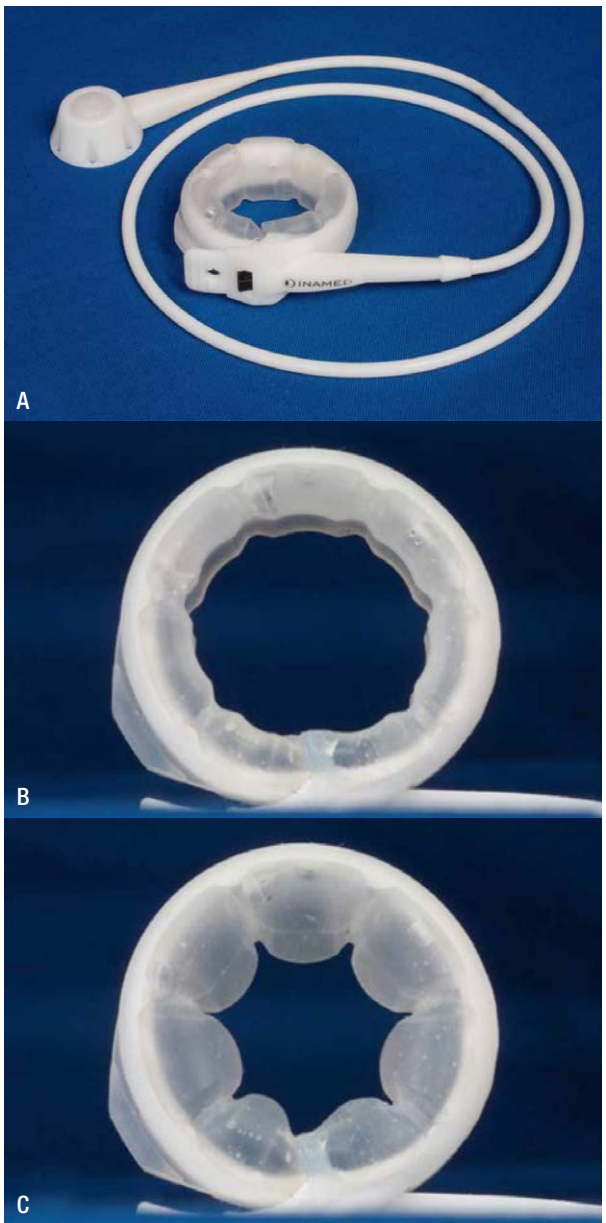
The term bariatric surgery covers a range of surgical procedures that aim to reduce weight and to maintain weight loss. Techniques include LAGB. Another procedure is laparoscopic sleeve or tube gastrectomy, where most of the fundus and gastric body are removed, leaving an approximately 200-ml capacity residual gastric sleeve or tube. Gastric bypass (Roux-en-Y or RYGB) involves dividing the upper stomach, creating a 30-mL proximal gastric pouch to which the jejunum is anastomosed, thereby bypassing the remainder of the stomach. All bariatric procedures induce weight loss by either reducing caloric intake or limiting caloric absorption. Depending on the surgical technique used, weight loss may also be achieved via additional mechanisms (eg delayed digestion, changes in the levels of hormones that control hunger).[3]

Different procedures carry differing risk profiles and follow-up requirements. All procedures have been shown to be highly effective at inducing substantial weight loss, inducing remission of comorbidities, improving quality of life and reducing long-term mortality.[4]

The risk and benefits of weight loss surgery should be explained to Fred. LAGB is a very effective weight loss procedure. It has the advantages of safety, efficacy, adjustability and reversibility. It is often performed as a day-case procedure.[5] *Figure 1* shows the LAGB device and port with the balloon inflated and deflated. *Figure 2* shows the band correctly positioned at the very top of the stomach around the cardia. There are several different devices in use, which are generally similar in design.

LAGB requires lifelong follow-up. This usually involves at least 6–8 visits in the first year, 4–6 visits in the second year and then review every 3–6 months in the long term. Follow-up involves discussion about food intake, nutrition, physical activity, understanding the effects of the band, ensuring the band is adjusted appropriately, and monitoring for complications.[6]

LAGB requires major behavioural change, an alteration in the approach to eating and continued efforts to lose and control weight, paying attention to food selection, quantity and portion control, as well as ensuring that regular exercise (20–30 minutes) is incorporated into the daily schedule.[2]



A. A solid outer silicone shell attaches to an inflatable balloon, which can be filled with varying volumes of saline to specifically tailor effects. The balloon is attached via tubing to an access port, usually sited on the rectus muscle in the left upper quadrant. Saline is added or removed by accessing the port with a non-coring needle as a 30 second office based procedure. B and C show the band and port with the balloon inflated (B) and deflated (C).

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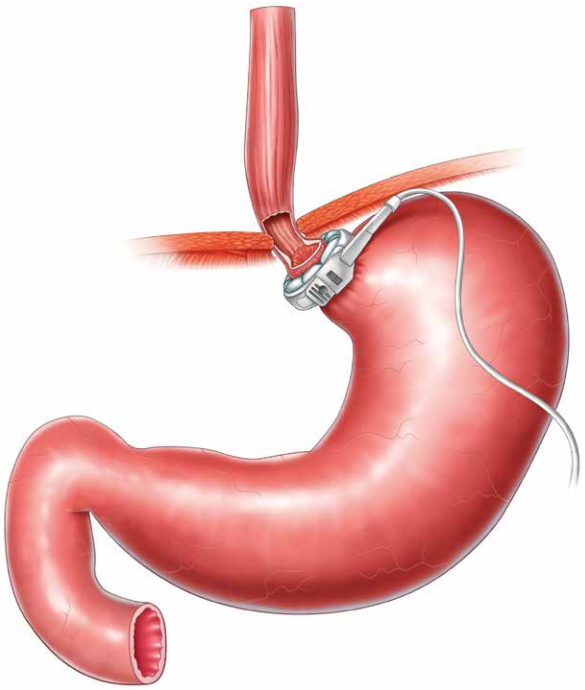
Figure 1. Laparoscopic adjustable gastric band



The LAGB is always placed around the apex of the stomach, leaving only a 20–30-mL pouch of stomach above the band. This is critical to achieving optimal early and prolonged satiety and substantial weight loss. A lower positioning of the band will induce reflux and regurgitation, as well as facilitating a larger volume food intake. A band that is too high results in dysphagia.

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Figure 2. Laparoscopic adjustable gastric band correctly positioned

Weight loss of 1 kg/week is possible in the first year following bariatric surgery but a more realistic weight loss is around 0.5 kg/week. Depending on the procedure undertaken, a steady weight loss is achieved within 12–24 months post surgery.[3] There is a high chance of achieving substantial weight loss (about 50% of excess weight) with LAGB, which has been shown to be maintained for at least 15 years.[7] Improvements in diabetes outcomes, including remission of diabetes, have been reported following LAGB.[8–10]

ANSWER 3

NHMRC guidelines suggest that bariatric surgery may be considered for adults with BMI >40 kg/m^2, or BMI >35 kg/m^2 and an obesity-related comorbidity. LAGB surgery by a specialist bariatric paediatric team may be considered in postpubertal adolescents with BMI >40 kg/m^2, or BMI >35 kg/m^2 with obesity-related complications, where other interventions have been unsuccessful.[1] These criteria are similar to other international guidelines. Medicare defines the indication for bariatric surgery as clinically severe obesity.[11] Other factors that are often used to select suitable patients include:[3]

- age 18–65 years
- fitness to undergo surgery
- ability to give informed consent
- willing to participate in long-term follow-up
- selected patients, with BMI >30 kg/m^2 and an obesity-related comorbidity.

A recent randomised trial in 51 patients with BMI 25–30 kg/m^2 and T2DM evaluated the addition of LAGB to multidisciplinary diabetes care. The addition of LAGB improved glycaemic control with an acceptable adverse event profile. Diabetes remission was reported at follow-up 2 years later in 12 (52%) participants in the multidisciplinary care plus gastric band group and two (8%) participants in the multidisciplinary care only group.[10]

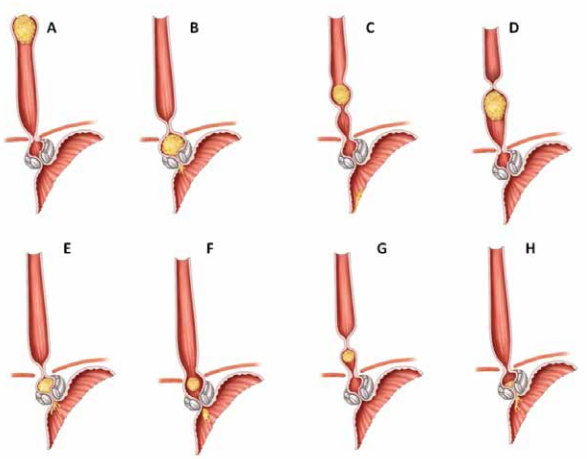
Contraindications to surgery include irreversible end-organ dysfunction, cirrhosis with portal hypertension, medical problems precluding general anaesthesia and centrally mediated obesity syndromes such as Prader-Willi syndrome or craniopharingioma.

ANSWER 4

LAGB induces early and prolonged satiety,[12] which results in decreased overall energy intake and weight loss. Daily energy intake is reduced from approximately 10,545 kJ (2497 cal) to 4271 kJ (1020 cal) in the 12 months following LAGB.[13]

Compression of nerve endings in the proximal stomach results in suppression of hunger between meals and particularly in the mornings.[14]

An appropriately adjusted LAGB results in significant slowing of food transit into the stomach below the band.[15] Each bite must be appropriately sized, chewed well and a period of 45–60 seconds allowed for several oesophageal peristaltic contractions to transit the swallowed bolus in portions, through the band. *Figure 3* illustrates the resultant effect, which leads to a feeling of fullness and meal termination after consumption of a small volume of food.



A. An appropriately selected and chewed food bolus is swallowed. B. Peristalsis drives the bolus to the level just above the LAGB and generates flow across the LAGB. C. The residual bolus refluxes back into the oesophagus. D. Another peristaltic contraction drives the bolus back to the LAGB, where flow commences again. E–G. This process is repeated, with a progressively smaller bolus. H. Complete transit is achieved. The overall effect is to delay transit and induce satiety by stimulation of the cardia above the band.

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Figure 3. Effects of LAGBs on swallowing

During follow-up, adjustments can be made to the band, with saline added and removed. Patients are presented with the schema shown in *Figure 4*.[2] This divides a patient's eating and sensations into three zones:

- Yellow: indicates inadequate saline and too much hunger.
- Green (optimal zone): patients consume small meals and are satisfied, and weight loss is good.
- Red: there is too much saline and adverse symptoms, such as reflux and regurgitation, are experienced.

ANSWER 5

The GP is a key part of the bariatric follow-up team, while needing to continue to monitor other aspects of the patient's care.[16] For example the GP could:

- Remain central to overseeing management of all the patient's medical problem
- Adjust/review medical care and medication(s) as requirements change during weight loss, particularly if diabetic medications or anti-hypertensive agents are being used
- Monitor patients for adverse symptoms, complications and nutritional status relating to bariatric surgery
- Encourage patients to continue attendance at bariatric follow-up visits
- Reinforce healthy eating and exercise messages.

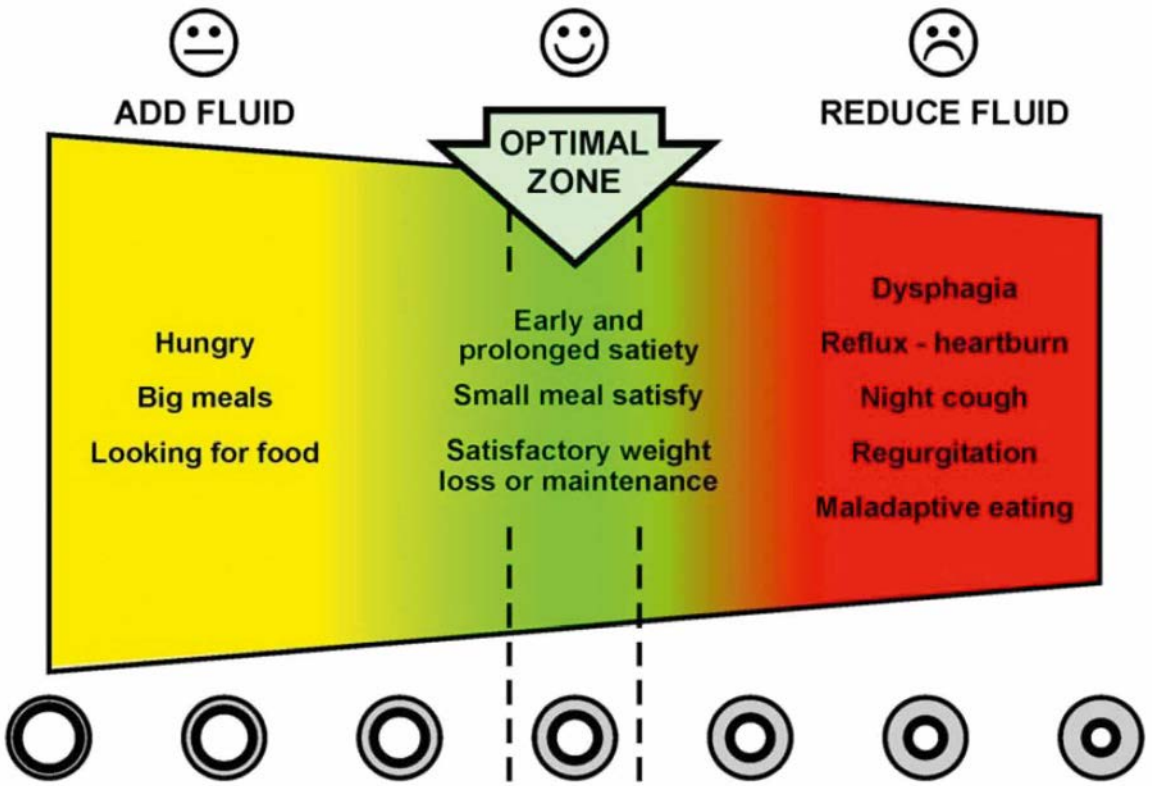
The GP should be aware of what is 'normal', following LAGB surgery. When the amount of fluid in the band is optimal the patient feels satisfied with 2–3 small meals per day of solid food with weight loss of about 0.5–1 kg per week[3] (or weight stability after substantial weight loss) and does not experience adverse symptoms.

A significant number of GP's have been trained to conduct LAGB follow up and perform adjustments. This can occur at bariatric surgical centres or within the general practice setting. Many regional centres have GPs able to perform LAGB follow-up. A small amount of training/mentoring is required to gain those skills.

ANSWER 6

A number of complications can arise following laparoscopic gastric band surgery, which may explain Fred's situation. The most likely problem is that Fred is in the red zone (*Figure 4*) and this can be remitted with removal of some saline and more frequent follow up. It is unlikely further investigations will be required. Weight loss can be worsened if the band is too tight as patients migrate to eating softer, high caloric foods. The other possibility is that Fred has developed a complication of pouch dilatation or prolapse (*Table 1*). This may be suspected if simple measures of optimally adjusting the band and better patient education do not improve symptoms. Fred should be advised to return to his bariatric surgeon for review. Common symptoms and complications following laparoscopic gastric band surgery are listed in *Table 1*.

LAGB is very low risk, however serious peri-operative events such as perforation/infection can occur. Surgical mortality is estimated at 1–5/10,000.[7,17]



This is a conceptual way of identifying the optimal level of band adjustment on the basis of sensations and symptoms and communicating these zones simply to patients. When patients are in the green zone or optimal zone, they do not feel hungry, are satisfied with a small amount of food, and are achieving weight loss or maintaining a satisfactory weight. The yellow zone indicates that the band is too loose. Patients in this zone will feel hungry and eat too easily. Weight loss will be inadequate or may not occur. The red zone indicates that the band is too tight. Patients in this zone may experience reflux, heartburn and vomiting. The range of food the patient in the red zone can eat will be limited as dysphagia or regurgitation will occur with solid foods. A patient may favour softer, smoother high-energy foods such as ice cream and chocolate. While in the red zone, patients will not lose weight as effectively and they may even gain weight due to consumption of poor-quality foods.

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Figure 4. Clinical zones in patients with LAGBs[2]

A 15-year review of over 3000 patients identified a revisional surgery rate of 30%.[7] This included surgery on the port and tubing because of device wear and tear, and operations for symptomatic gastric enlargements above the band. A randomised controlled trial followed up to 10 years showed maintenance of weight loss (14 kg) in the LAGB group. The non-surgical group lost minimal weight (0.4 kg). Revisional surgery occurred in 30% of the LAGB patients and during the follow-up decade 12% had the LAGB removed.

Other series have shown very high failure and LAGB explant rates.[18] One possible explanation for the differences in reported outcomes is ensuring patients are diligently followed up in the long term.[19] Without that follow-up, LAGB cannot be expected to work. If LAGB patients elect to have the LAGB removed or have complications, then conversion to an alternative procedure (such as gastric bypass or sleeve gastrectomy) is generally safe and successful, although risks may be higher than with primary surgery.[20,21]

Longer-term prospective data or comparative randomised controlled trials are rare in bariatric surgery. The largest randomised controlled trial (n = 250) comparing laparoscopic gastric bypass to LAGB found higher percentage excess weight loss at 4 years (68 ± 19% versus 45 ± 28%) in the bypass group but also a higher complication rate and one mortality, compared with none. The authors concluded that both procedures were effective: gastric bypass resulted in better weight loss but was associated with more perioperative and late complications and a higher 30-day readmission rate.[22]

One issue with LAGB is the incidence of dysphagia and patients cite the inability to consume certain types of foods and the necessity to substantially alter eating intake as the biggest problem following LAGB.[15] This is a reality of the procedure and a dysphagia score of around 20 out of 45 (with 0 being no dysphagia and 45 being the inability to consume liquid) being observed in patients with good weight loss and in a regular follow-up program.[15]

Nutritional deficiencies are rare with the LAGB and weight loss has been shown to be primarily of the fat mass.[23,24] Patients who have undergone procedures that permanently alter gastrointestinal anatomy or absorptive capacity, such as gastric bypass or sleeve gastrectomy are at higher risk of nutritional deficiencies.[25] These can include fat soluble vitamins such as vitamin A or D (including calcium and parathyroid hormone), folate and B_{12}, iron and micronutrients such as zinc.[25,26]

Table 1. Common problems and complications after laparoscopic adjustable gastric banding

Complication/ problem	Features	Investigations/ management
Too much saline in the band	Red zone: Heartburn, dysphagia, regurgitation	Usually easily resolved with removal of saline and patient education
Too little saline in the band	Yellow zone: Weight gain, eating freely, inadequate satiety	More frequent follow up, addition of saline to the LAGB, patient education
Pouch dilatation – also known as slip or prolapse of the band	Regurgitation and reflux, or heartburn, particularly recumbent or nocturnal	Barium swallow
Port problem – leakage of saline	Weight gain, loss of satiety, usually associated with needlestick injury to tubing or hub of port	Check volume of saline in system, if a significant discrepancy then may need a port revision
Erosion of band into stomach	Loss of satiety, increased appetite, ability to eat freely Possible spontaneous port infection	Gastroscopy

The obesity surgeons society of Australia and New Zealand (www.ossanz.com.au) strongly advocates life long follow-up for all patients who have undergone bariatric surgery to ensure a good outcome is maintained, healthy lifestyle measures continue, complications are identified early and nutritional status is monitored (refer to *Resources for doctors*).

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RESOURCES FOR DOCTORS

- Obesity Surgeons Society of Australia and New Zealand, www.ossanz.com.au
- National Health and Medical Research Council clinical practice guidelines for the management of overweight and obesity in adults, adolescents and children in Australia, 2013, www.nhmrc.gov.au/guidelines/publications/n57
- Monash University Centre for Obesity Research and Education (CORE), www.core.monash.org
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CASE 6

ROBERT IS HAVING BLURRED VISION

Robert, aged 65 years, has had type 2 diabetes mellitus (T2DM) for 15 years and is on insulin treatment. He visits you to discuss his eyes. For the past 4–6 months he has had increased blurring of his right eye and his glasses do not seem to help anymore. He is finding it harder to read the newspaper and driving has become more difficult. These are tasks he found easy to perform 6 months ago. He also has a history of hypertension and raised cholesterol for which he takes medications.

QUESTION 1

What are the possible causes of Robert's blurred vision?

QUESTION 2

What would be your initial management plan?

FURTHER INFORMATION

Robert saw an optometrist 18 months ago and at the time his distance vision was corrected to 6/6 with glasses in both eyes. He went back to the optometrist after seeing you. The optometrist was unable to refract his right eye beyond 6/18, although his left eye was still 6/6. No significant lens opacity was noted in either eye and no drusen or significant retinal pigment change was noted at either macula to suggest age-related macular degeneration. A copy of Robert's fundus photos (*Figure 1*) and optical coherence tomography (OCT) scan (*Figure 2*) for the right eye were sent to you with the report and a recommendation for referral.

Figure 1. A colour fundus photograph of Robert's right eye

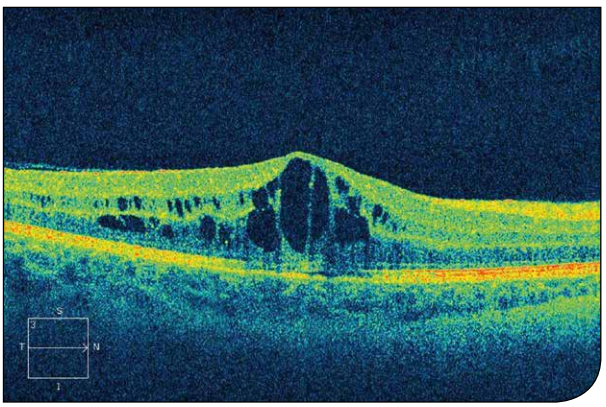

Figure 2. An OCT scan of Robert's right central macula (scanned through the fovea)

QUESTION 3

What do the images show?

QUESTION 4

What would you do next?

FURTHER INFORMATION

Robert returns 6 months after treatment for his right diabetic macular oedema. He has had a number of injections of an agent that inhibits vascular endothelial growth factor (anti-VEGF) for his right eye. He now finds it easier to read the newspaper and feels that his vision has improved overall.

QUESTION 5

What is your long-term management of Robert's eyes?

CASE 6 ANSWERS

ANSWER 1

Common causes of vision loss for Robert would be refractive error, cataract, macular degeneration and diabetic retinopathy (a microvascular complication of diabetes). In Western countries, macular degeneration is currently the most important cause of blindness; however, uncorrected refractive error continues to be the leading cause of moderate and severe vision impairment.[1] Refractive error does not usually change significantly over 6 months, and age-related cataracts also progress slowly, so these are less likely to be responsible for Robert's vision changes. His blurred vision may be due to progression of the atrophic, or 'dry' form of age-related macular degeneration (choroidal neovascularisation or 'wet' age-related macular degeneration tends to cause sudden loss of vision and distortion). In any patient with diabetes, diabetic maculopathy is also a consideration.

ANSWER 2

In the first instance it would useful to check Robert's vision. If his vision improves through a pinhole, this suggests his visual blur is at least partly due to refractive error.[2] Robert needs to have his refractive error checked and have a dilated ocular examination to determine the cause of his blurred vision. You may wish to perform the dilated examination in your rooms, or refer Robert to an optometrist or ophthalmologist for this initial review. It is recommended that this review occur within 1–2 weeks, as long-term visual outcomes for the treatment of wet age-related macular degeneration and diabetic retinopathy are correlated to the time from first symptoms to the time of initial treatment,[3,4] whereas long-term visual outcomes for the treatment of diabetic retinopathy are correlated to less severe diabetic retinopathy at presentation.[5] Current treatment options for both conditions aim to prevent further vision loss, but do not always restore vision already lost.

Checking Robert's blood pressure and blood glucose control would also be useful. Depending on the outcomes of these checks, it may be necessary to adjust his current medications to ensure that his blood pressure and blood glucose are within appropriate targets for him.

ANSWER 3

The first image is a fundus photograph of the right macula. It shows severe diabetic maculopathy with retinal haemorrhages and hard exudates, particularly in the superior macula, and these extend into the fovea. Severe diabetic maculopathy is defined as fovea-involving diabetic change.

The second image is an optical coherence tomography (OCT) scan. OCT images are increasingly being used to assess macular pathology and monitor progress with any treatment.[6] Robert's OCT scan shows cystoid macular oedema with multiple dark cystic spaces of fluid involving the fovea. Vision is usually significantly affected once the fovea is involved.

ANSWER 4

Robert has visually significant diabetic macular oedema (DMO) in his right eye. At this point he requires referral to an ophthalmologist for treatment. A 2012 systematic review using data from approximately 23,000 patients with diabetes estimated the following prevalence of diabetic eye problems:[7]

- any diabetic retinopathy: 34.6%
- diabetic macular oedema: 6.81%
- proliferative diabetic retinopathy: 6.96%
- any vision-threatening diabetic retinopathy: 10.2%.

For many years diabetic macular oedema was treated with argon laser photocoagulation in accordance with findings from the Early Treatment Diabetic Retinopathy Study.[8] This study showed that macular laser treatment for clinically significant diabetic macular oedema reduced the risk of losing three lines on a vision chart by 50% at 2 years, compared with no treatment.[3]

More recently intravitreal steroid and intravitreal anti-VEGF agents have been used. The duration of the effects of intravitreal triamcinolone acetonide (a steroid) is 4–6 months[9] and that of intravitreal anti-VEGF injection is approximately 1 month.[10]

In patients with DMO that has persisted or recurred after laser treatment, intravitreal triamcinolone acetonide has been shown to improve vision and reduce macular thickness. With repeated treatment, this benefit was seen for up to 2 years.[4] Adverse events associated with this treatment, however, include acceleration of cataract and raised intraocular pressure, which may lead to glaucoma[11] and, uncommonly, endophthalmitis.[12]

The Diabetic Retinopathy Clinical Research Network reported on a randomised trial evaluating intravitreal ranibizumab (an anti-VEGF agent) or 4 mg triamcinolone acetonide combined with macular laser, compared with macular laser alone, for treatment of DMO. The 2-year results for all patients showed an improvement in the mean change from baseline in the visual acuity letter score in the combined ranibizumab and laser groups, but was marginally worse in the combined triamcinolone and laser group, compared with laser treatment alone.[13] It was believed that part of the vision loss in the intravitreal steroid group was related to cataract formation. For patients who had already undergone cataract surgery, however, the combination of ranibizumab and laser had equivalent outcomes for vision when compared with steroid and laser. This finding has led many ophthalmologists to use anti-VEGF therapy as a first-line treatment for patients with DMO.[14] The anti-VEGF therapies have been associated with a doubling of the likelihood of vision gain and have one-third fewer cases of severe vision loss compared with previous laser treatment.[13]

ANSWER 5

The ophthalmic treatment of Robert's diabetic macular oedema addressed the diabetic eye changes he had at the time.

Continuing to monitor his diabetes and hypertension is important to prevent the development of further diabetic retinopathy, which may be associated with future vision loss. Optimising blood glucose levels and blood pressure, and possibly blood lipid levels, is very important for reducing the risk of development or progression of diabetic retinopathy.[15]

The FIELD study, a randomised controlled study assessing the effects of fenofibrate, showed that patients with T2DM on fenofibrate 200 mg daily had a significantly lower requirement for first laser treatment, compared with placebo (3.4% fenofibrate group versus 4.9% placebo group).[16] This outcome seems to be independent of its effect on plasma lipid concentrations. Fenofibrate may be considered for any patient with diabetic retinopathy, and does not need to be limited to those cases with diabetic retinopathy that affects visual acuity. Note, fenofibrate is indicated for use in dyslipidaemia associated with T2DM, severe hypertriglyceridaemia and hypercholesterolaemia (second-line treatment for those unable to tolerate other lipid-lowering agents).[17] The Therapeutic Goods Administration (TGA) recently approved the use of fenofibrate in patients with diabetic retinopathy. The 200 mg dose used in the FIELD study is bioequivalent to three tablets of 48 mg or one tablet of 145 mg, which are available in Australia.[18]

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RESOURCES FOR PATIENTS AND DOCTORS

- National Health and Medical Research Council. Guidelines for the management of diabetic retinopathy, www.nhmrc.gov.au/_files_nhmrc/publications/attachments/di15.pdf
- Mohamed Q, Gillies MC, Wong TY. Management of diabetic retinopathy: a systematic review, http://jama.jamanetwork.com/article.aspx?articleid=208502
- The Royal College of Ophthalmologists (UK). Diabetic retinopathy guidelines, www.icoph.org/dynamic/attachments/taskforce_documents/2012-sci-267_diabetic_retinopathy_guidelines_december_2012.pdf

Diabetes and obesity (Activity ID: 9894)

In order to qualify for 6 Category 2 points for the QI&CPD activity associated with this unit:

- read and complete the unit of *check* in hard copy or online at the *gplearning* website at http://gplearning.racgp.org.au
- log into the *gplearning* website at http://gplearning.racgp.org.au and answer the following 10 multiple choice questions (MCQs) online
- complete the online evaluation.

If you are not an RACGP member, please contact the *gplearning* helpdesk on 1800 284 789 to register in the first instance. You will be provided with a username and password that will enable you access to the test.

The expected time to complete this activity is 3 hours.

Do not send answers to the MCQs into the *check* office. This activity can only be completed online at http://gplearning.racgp.org.au

If you have any queries or technical issues accessing the test online, please contact the *gplearning* helpdesk on 1800 284 789.

FOR A FULL LIST OF ABBREVIATIONS AND ACRONYMS USED IN THESE QUESTIONS PLEASE GO TO PAGE 3.
FOR EACH QUESTION BELOW SELECT ONE OPTION ONLY.

CASE 1 – MABEL

Mabel, aged 72 years, has had type 2 diabetes mellitus (T2DM) for the past 18 years, hypertension for 16 years and dyslipidaemia for 12 years. Vision in both of her eyes has deteriorated over the past 6 months, making reading and driving difficult.

QUESTION 1

Which of the following is the best way to manage this presentation?

A. Reassure Mabel that the deterioration in her vision is normal for her age and advise her to see you again in 6 months.

B. Refer Mabel to an ophthalmologist for assessment of her vision problems.

C. Increase the dose of Mabel's antihypertensive medication.

D. Increase the dose of Mabel's glucose-lowering medication.

E. Advise Mabel to get stronger reading glasses.

QUESTION 2

A report from her optometrist says Mabel has central (fovea-involving) diabetic macular oedema (DMO). Which statement is correct regarding treatment for her DMO?

A. The benefits of intravitreal triamcinolone acetonide injection last for approximately 1 month.

B. Mabel should be referred for argon laser photocoagulation as first-line therapy.

C. Optimising blood glucose levels, blood pressure and possibly blood lipid levels, is important in reducing the risk of development and/or progression of diabetic retinopathy.

D. Optimising blood glucose levels, blood pressure and possibly blood lipid levels, is not important for reducing the risk of development and/or progression of diabetic retinopathy.

E. Intravitreal anti-vascular endothelial growth factor (anti-VEGF) agents are last-line treatments for diabetic macular oedema.

CASE 2 – CONNIE

Connie is 79 years of age and is receiving end-of-life care. Her life expectancy is weeks to 1 month. Her condition has been deteriorating rapidly and she needs assistance with activities of daily living. She has difficulty eating, and nausea, significant gastrointestinal upset and problems swallowing. She has T2DM and moderate vision loss. She has also had several minor transient ischaemic attacks/strokes and endometrial cancer 10 years ago. Her medications include metformin, insulin, a statin, an angiotension converting enzyme inhibitor (ACEI) and aspirin.

QUESTION 3

Which one of the following statements is correct regarding Connie's blood glucose levels?

A. The optimal blood glucose level for Connie is 6–15 mmol/L.

B. The optimal blood glucose level for Connie is 6–20 mmol/L.

C. The optimal blood glucose level for Connie is the same as that for all people with diabetes.

D. Tight glycaemic control is beneficial in people with limited life expectancy.

E. Poor glycaemic control does not have a negative impact on a patient's quality of life.

CASE 3 – JOSIE

Josie is 5 years of age and attends your clinic with her mother. Josie's mother tells you that she is very concerned about Josie's weight loss, occasional vomiting, increased thirst and polyuria over the last few weeks. When examining Josie you note abdominal pain.

QUESTION 4

Which of the following statements is the most correct?

A. Her polydipsia is psychogenic.

B. Her polyuria is due to a urinary tract infection.

C. Her abdominal pain and occasional vomiting coupled with weight is due to gastroenteritis.

D. The presenting symptoms are suggestive of type 1 diabetes mellitus (T1DM).

E. The presenting symptoms are not suggestive of T1DM.

QUESTION 5

Using point-of-care testing at your clinic, you find that Josie's random blood glucose is 16 mmol/L and her blood ketones are 0.6 mmol/L. Which of the following statements is the most correct regarding the next steps you should take?

A. A blood sample should be taken to confirm a diagnosis of T1DM.

B. Josie should be sent for an oral glucose tolerance test (OGGT).

C. Blood samples should be taken to confirm the random blood glucose and ketones test results.

D. Josie should be referred for a specialist assessment over the next few weeks/month.

E. Josie should be referred immediately (same day referral) for specialist assessment.

QUESTION 6

Josie was referred to the local emergency department. She was assessed and managed for ketoacidosis. Her T1DM was confirmed, insulin treatment initiated and diabetes education and support offered. Which of the statements below is the most correct regarding Josie's ongoing management?

A. Josie and her family will require ongoing support and advice and about nutrition and healthy lifestyle practices.

B. When Josie is older her insulin therapy could be changed to an oral anti-hypoglycaemic agent.

C. Routine annual screening for retinopathy complications should commence immediately for Josie.

D. Routine annual screening for nephropathy complications should commence immediately for Josie.

E. Josie should have her lipid levels checked annually until puberty and from then every 5 years.

CASE 4 – SIEW

Siew is a Chinese woman aged 59 years and is a new to your practice. She tells you that she was diagnosed with T2DM 12 months ago and that she is due for her annual diabetes review. She has lost 5 kg since her diagnosis and would like to lose more weight. Her medications include metformin 2 g daily and ramipril 10 mg daily.

QUESTION 7

You determine that her body mass index (BMI) is 30.1 kg/m^2. Which one of the following statements is correct?

A. Siew is a good candidate for bariatric surgery.

B. Use of a lower BMI threshold is recommended for Siew.

C. Weight loss of 10 kg or more is required before any reduction in HbA1c levels may be observed.

D. Siew should be able to lose weight with just a vigorous exercise program.

E. Weight loss may improve her glycaemic control but will not improve her blood pressure.

QUESTION 8

A blood sample taken at the annual review showed an HBA1c of 7.7% (61 mmol/mol) and a CrCL of 73 mL/minute. What would you do now?

A. You would do nothing.

B. Increase Siew's dose of metformin.

C. Increase Siew's metformin to total daily doses and if this does not improve her glycaemic control introduce a short-acting sulfonylurea.

D. Switch Siew to another oral hypoglycaemic agent.

E. In the short term, encourage further weight reduction and review Siew's HBA1c levels, with the view to adding a second oral hypoglycaemic agent if her HBA1c continues to be above target.

CASE 5 – GINNY

Ginny is 36 years of age and wants to discuss laparoscopic adjustable gastric band (LAGB) surgery after watching a documentary. Her BMI is 37.6 kg/m^2 and her weight is the highest she has experienced. Recently she was started on metformin for her T2DM, a statin for her lipids and a diuretic to control her blood pressure.

QUESTION 9

Which statement is the correct regarding LAGB surgery for Ginny?

A. Ginny does not qualify for LAGB surgery.

B. To qualify for LAGB surgery Ginny's BMI needs to be >40 kg/m^2.

C. Ginny's diabetes might improve or remit if she had LAGB surgery.

D. There would be no change in Ginny's diabetes status as LAGB surgery does not improve diabetes outcomes.

E. If Ginny were 17 years of age LAGB surgery would be contraindicated for her.

QUESTION 10

Ginny comes back for a routine Pap smear. She is scheduled for LAGB surgery and is keen to discuss the risk and benefits of surgery with you again. Which statement is correct?

A. Weight loss of 2–3 kg per week is normal following LAGB surgery.

B. Surgical mortality is estimated at 10–20/10,000.

C. The risk of long-term complications is 3% over 15 years.

D. Weight loss following LAGB surgery has been shown to be maintained for up to 15 years.

E. There are no long-term complications following LAGB surgery.

check

Independent learning program for GPs