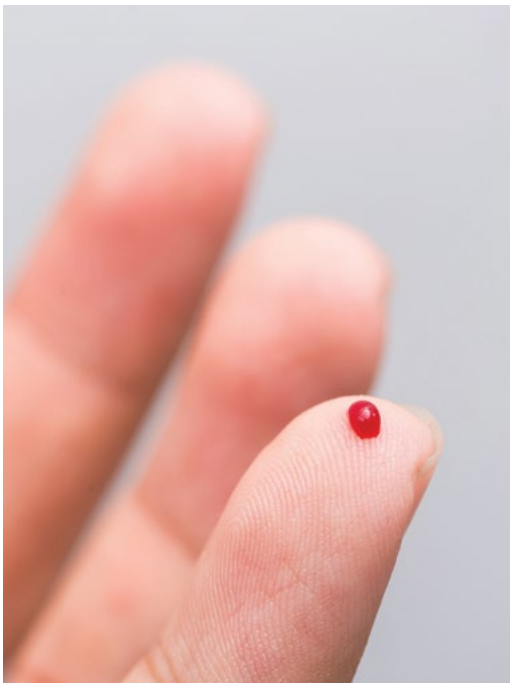
Palliative management of type 2 diabetes mellitus in patients with advanced cancer



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Background
Patients with advanced cancer often have type 2 diabetes mellitus (T2DM) and are on multiple medications that affect glycaemic control. Patients can have pre-existing diabetes or treatment- and glucocorticoid-induced hyperglycaemia. Optimal management of glycaemic control differs at varying stages of life and depends on overall goals of care.

Objective
This article summarises the evidence-based management of T2DM in patients with advanced cancer, including management of glucocorticoid-induced hyperglycaemia, and appropriate targets for glycated haemoglobin (HbA1c).

Discussion
T2DM often co-exists in patients with advanced cancer. Management is aimed at reducing the complications of hyperglycaemia and hypoglycaemia, and maximising quality of life. Interventions and treatments need to be balanced against quality of life and prognosis.

THE PREVALENCE of diabetes mellitus has increased, with an estimated 1.3 million people (5.1%) living with the condition in Australia.[1] Approximately 8–18% of patients with cancer have diabetes, and glycaemic control often worsens after a cancer diagnosis.[2,3] Managing diabetes mellitus in advanced cancer is complex, as malignancy and its treatments interfere with metabolism and glycaemic control.

There is no consistent evidence base to guide the management of type 2 diabetes mellitus (T2DM) in advanced cancer or end-of-life care.[4] Although routine management of T2DM aims to prevent long-term complications, this approach might not be appropriate as the goals of care shift. This article reviews available evidence and expert opinion on the management of T2DM in patients with cancer who are receiving palliative care.

Effect of hyperglycaemia and hypoglycaemia

CASE STUDY

Mrs PH, aged 76 years, with metastatic pancreatic cancer was admitted to an inpatient palliative care unit with functional decline and inability to manage at home. She had a background of multiple comorbidities including T2DM, which was well controlled on non-insulin glucose-lowering agents. She was slowly deteriorating from her disease over a period of weeks. During her admission, multiple drugs were deprescribed, including her diabetes medications. On a routine ward round review, Mrs PH reported that she was sleeping poorly because she had multiple episodes of nocturia. Her blood glucose levels (BGLs) were closely monitored for the next few days, and she was found to have BGLs up to 23 mmol/L. After discussion with Mrs PH, she elected to have a dose of long-acting insulin (insulin glargine) together with thrice daily short-acting insulin (insulin aspart) on a sliding scale. The upper level of her BGLs was maintained between 10 and 15 mmol/L, and her nocturia improved.

Patients with cancer have an increased risk of hypoglycaemia due to weight loss, reduced oral intake and depleted hepatic glycogen stores.[5] Hypoglycaemia can cause sweating, palpitations, dizziness, difficulty concentrating, confusion, lethargy, seizures and coma.

These patients are also at increased risk of hyperglycaemia because of systemic therapy, glucocorticoids, intercurrent

illness and challenges prioritising self-care.[6] Hyperglycaemia causes polyuria (which can have distressing consequences, as outlined in the case study above), thirst, thrush, lethargy, pain exacerbation, irritability, delirium and non-ketotic hyperosmolar states. BGLs >20 mmol/L typically cause symptoms, and it can be hard to differentiate these symptoms from those caused directly by the cancer.

Cancer and treatment effects on BGLs

Glycaemic control affects outcomes for patients with cancer. It can have an effect on the risk of infections and hospitalisation.[7]

Pharmacologic and dietary management of diabetes is often a secondary priority during chemotherapy.[8] Cancer itself alters metabolic balance and glycaemic control. Some systemic therapies raise blood glucose in the short term, but this can persist and lead to a new diagnosis of diabetes. Chemotherapy agents like 5-fluorouracil (5-FU) and platinum-based drugs contribute to hyperglycaemia and can worsen diabetic neuropathy. Hormone therapies, immunotherapies (eg pembrolizumab, nivolumab) and some targeted therapies (eg everolimus, nilotinib) can also cause hyperglycaemia. It is important to remember that certain drugs, such as immune checkpoint inhibitors (eg pembrolizumab, nivolumab, atezolizumab, avelumab), can cause new-onset type 1 diabetes mellitus and worsen glycaemic control in people with T2DM, with rapid progression to insulin deficiency. These patients will need insulin and, if not treated quickly, can progress to diabetic ketoacidosis.

Conversely, some haematologic treatments induce hypoglycaemia. Immune checkpoint inhibitors can cause hypopituitarism leading to adrenal insufficiency and hypoglycaemia. Radiation therapy, surgery and metastases affecting the adrenal and pituitary glands can also affect glycaemic control.[9]

General principles of diabetes management in patients with advanced cancer

The approach to management of T2DM in this population depends on disease trajectory and expected prognosis (Tables 1 and 2).

Most of the guidance is based on low-level evidence and expert consensus. For patients with good functional status and a prognosis of weeks to months, closer monitoring of BGLs and management with non-insulin glucose-lowering agents or insulin would be reasonable. However, for patients with a shorter prognosis and poor performance status, it becomes more appropriate to relax glycaemic targets, as the burden of treatment might outweigh the potential benefits. In these cases, minimising monitoring and interventions is the preferred approach. The primary focus shifts to maintaining a safe blood glucose range, avoiding significant variability and emphasising symptom management over strict glycaemic control. Preventing long-term complications secondary to diabetes is deprioritised, as the remaining quality of life is unlikely to allow for meaningful benefits from such interventions. Oral intake is encouraged as tolerated, and dietary restrictions can be loosened.[10,11]

A blood glucose range of 6–15 mmol/L is generally acceptable.[12,13] Higher levels are accepted if patients are asymptomatic and have significant cancer progression. Glucose-lowering medications might need to be reduced or ceased, aiming for the simplest regimen. If using insulin, cautious use of long-acting insulin or a basal bolus regimen is reasonable.[14] Short-acting insulin alone might suffice when intake is unpredictable, but this is likely to cause greater variability in glycaemia.[13,15] It is important to bear in mind that people with long-standing T2DM might have beta-cell burnout and be unable to produce endogenous insulin. This group will require ongoing insulin to prevent complications such as diabetic ketoacidosis. This is a similar consideration for people who develop type 3c diabetes after partial or total pancreatectomies as part of their treatment for cancer. They would also require insulin to prevent complications, until the last few hours to short days of their lives. We discuss our approach to deprescribing in Tables 1 and 2.

Monitoring BGLs can be a useful adjunct to addressing symptoms such as nausea, headache and polyuria.[16] It can be challenging to determine if symptoms are secondary to glycaemic control or if there are other factors contributing. There is no guideline for testing frequency. Symptomatic patients should be tested regularly until stable, and then the frequency of monitoring can be reduced as appropriate. Reduction in frequency of monitoring from thrice-daily to once-daily is reasonable if stable.[17] Aim to cease monitoring quickly in patients who are stable. Starting corticosteroids introduces a complicating factor – it is advisable to

Table 1. General principles of managing patients on insulin receiving palliative care

Principles of managing patients on insulin receiving palliative care		
Care for all: • Symptom evaluation and management • Collaborative care: palliative/endocrinology/GP/patient • Avoid tight glycaemic control • Family and patient education regarding goals		
What is the prognosis?		
Hours	**Days**	**Weeks–Months**
• Stop BGL monitoring • Stop insulin	• Stop or reduce frequency of BGL monitoring • Reasonable to give insulin with liberal targets of BGLs (under 20 mmol/L) – change to LA or SA insulin with meals	• Continue monitoring BGLs • Continue usual treatment – consider ceasing insulin if T2DM (exercising caution – refer to 'Deprescribing' section) • Consider reducing insulin dose if hypoglycaemic (or) LA insulin (or) SA insulin if eating meals

BGL, blood glucose level; GP, general practitioner; LA, long acting; SA, short acting; T2DM, type 2 diabetes mellitus.

Table 2. General principles of managing patients on oral hypoglycaemic agents receiving palliative care

Principles of managing patients on non-insulin glucose-lowering drugs receiving palliative care	
Care for all:	
• Symptom evaluation and management	
• Collaborative care: palliative/endocrinology/GP/patient	
• Consider deprescribing SU/GLP-1RA/SGLT2i	
• Consider DPP-4i ± metformin	
• Avoid tight glycaemic control	
• Family and patient education regarding goals	
What is the prognosis?	
Hours–Days	**Weeks–Months**
• Stop BGL monitoring • Stop glucose-lowering drugs	• Monitor BGLs – tailor frequency on the basis of glycaemic variability • Deprescribe as appropriate • If increased risk of hypoglycaemia – reduce dose of glucose-lowering drugs, cease LA SU • Consider ceasing glucose-lowering drugs depending on BGLs if prognosis is weeks

BGL, blood glucose level; DPP-4i, dipeptidyl peptidase-4 inhibitor; GLP-1RA, glucagon-like peptide-1 receptor agonist; GP, general practitioner; LA, long acting; SGLT2i, sodium glucose cotransporter 2 inhibitor; SU, sulfonylurea.

monitor glycaemic control for a few days after commencement to enable detection of hyperglycaemia.

Although there are a lack of studies investigating glycaemic control at the end of life, expert advice recommends avoiding hypoglycaemia.[18] Some guidelines suggest blood glucose targets can be relaxed to 10–20 mmol/L in the last days of life, though our palliative physician practice is to stop checking BGLs at this stage. Reduce or cease insulin and non-insulin glucose-lowering drugs, especially as the swallow becomes weaker. Glucose monitoring is not recommended in this situation.

Glucocorticoid-induced hyperglycaemia

Glucocorticoids cause hyperglycaemia in 10–30% of patients receiving chemotherapy.[9] They are commonly used in advanced cancer for symptom management and oncological emergencies.

Table 3 shows the pharmacokinetic and hyperglycaemic effects of different glucocorticoids, which should guide insulin choice. Intermediate-acting glucocorticoids typically increase BGLs 4–6 hours after administration, peaking between lunch and dinner when given in the morning. Levels often normalise overnight, so it is important to tailor treatment to avoid nocturnal and early-morning hypoglycaemia.[19]

Predicting hyperglycaemia risk from glucocorticoids is difficult. Risk factors include dose, potency and duration. It is worthwhile noting that the pharmacological studies looking at relative potency of glucocorticoids have been based on factors that might not be relevant to our clinical situation.[20]

Most consensus guidelines for the management of glucocorticoid-induced hyperglycaemia target inpatients. For outpatients, it is recommended to test patients who have risk factors and pre-existing diabetes twice weekly at a minimum, up to four times daily. If BGLs exceed 11.1 mmol/L, increase the frequency of monitoring and start pharmacotherapy.[21]

It has been suggested that short-acting sulfonylureas can be used in the palliative setting; however, caution should be exercised when using these hypoglycaemic agents, and the same principles should apply as discussed above.[22]

If hyperglycaemia persists despite the use of non-insulin glucose-lowering medications, it would be reasonable to consider insulin.[21,23] Using an intermediate insulin is a good choice to cover the glucocorticoid affected period, and there are reference tables available to estimate the daily insulin dose required.[24] If the patient is already on insulin, it is considered safe to increase the dose by 10–20% to achieve glycaemic targets; however, caution is needed, especially when using long-acting insulin in patients receiving prednisolone, as this might increase the risk of nocturnal hypoglycaemia due to the lack of a glucose-stimulating effect overnight.[19] A tailored approach based on the specific insulin regimen and glucocorticoid type is recommended.

Once glucocorticoids are ceased, it is recommended to monitor BGLs until euglycemia returns. Insulin and non-insulin glucose-lowering drugs can be reduced in tandem with the glucocorticoid taper. If hyperglycaemia persists, it is advised to continue glucose monitoring and formally test for diabetes at three months.[19,25]

Glycated haemoglobin: Role and pitfalls

The use of glycated haemoglobin (HbA1c) is limited in the palliative setting, and factors associated with cancer and its treatment can affect results. For example, recent blood transfusions, blood loss and liver disease can result in an inappropriately low HbA1c. Conversely, complications of cancer such as hyperbilirubinemia, uraemia and chronic renal failure can falsely elevate HbA1c. We (the authors) recommend against HbA1c measurement in patients with advanced cancer receiving palliative care because of inaccuracy of measurement as well as a shift in the goals of care.

Deprescribing

Polypharmacy is common in patients with T2DM and adds to the burden of disease. Deprescribing is warranted when the focus

Table 3. Variability in glucocorticoid pharmacokinetics and temporal association with hyperglycaemia

Glucocorticoid	Type	Time to peak plasma concentration (minutes)	Half-life (hours)	Duration of action (hours)	Impact on hyperglycaemia
Hydrocortisone	Short acting	10	2	8–12	1 hour (onset) 3 hours (peak) 6 hours (resolution)
Prednisolone	Intermediate acting	60–180	2.5	12–36	4 hours (onset) 8 hours (peak) 12–16 hours (resolution)
Methylprednisolone	Intermediate acting	60–180	2.5	12–36	4 hours (onset) 8 hours (peak) 12–16 hours (resolution)
Dexamethasone	Long acting	60–120	4	36–72	8 hours (onset) Variable (peak) 24–36 hours (resolution)

shifts from tight glycaemic targets to symptom management and harm minimisation (Table 4). Explanation and reassurance will be required as the focus of care changes.

Secretagogues and insulin pose a significant risk of hypoglycaemia.[26]

Metformin is generally safe but might worsen nausea. Caution also needs to be exercised when there is hepatic and renal impairment. Sulfonylureas increase the risk of hypoglycaemia, especially with reduced oral intake. Some dipeptidyl peptidase-4 (DPP-4) inhibitors need to be ceased or used cautiously in patients with renal failure. Glucagon-like peptide-1 (GLP-1) agonists might worsen anorexia and weight loss and are therefore less desirable in an oncology setting. Sodium-glucose cotransporter-2 (SGLT-2) inhibitors need to be ceased in cases of acute illness, reduced intake and dehydration because of the risk of ketoacidosis. They also increase the risk of urinary tract infections and urogenital candidiasis, especially in an immunocompromised population who are also often on glucocorticoids.[27]

Patients with T2DM might still produce endogenous insulin, which would make it safe to stop insulin use. This can be determined by measuring paired C-peptide and fasting BGLs. C-peptide is a byproduct of endogenous insulin production and is useful to assess the residual pancreatic function in patients with diabetes being treated with insulin. In the presence of a normal–high fasting BGL, C-peptide levels would be expected to be high in the presence of endogenous insulin. If the C-peptide levels were low, it can be inferred that there is minimal-to-no endogenous insulin production. If insulin is required, it is prudent to use the simplest regimen and train caregivers in administration.

From a practical experience point of view, continuing metformin and/or a DPP-4 inhibitor until the swallow has deteriorated or the medications become too burdensome is a reasonable solution.

Continuous glucose monitors and insulin pumps

Newer technologies like continuous glucose monitors (CGM) and insulin pumps allow closer monitoring of BGLs and facilitate closed-loop insulin delivery. These devices offer close glucose monitoring and accommodate variable insulin requirements;[28] however, these technologies are usually patient-controlled, which can lead to challenges and anxiety, particularly as many medical staff might be unfamiliar with these evolving technologies. Additionally, CGM measurements become less accurate at the end of life because of poor interstitial tissue perfusion. These 'abnormal' readings can be very distressing to carers, and focus needs to be shifted back to managing symptoms and comfort rather than biochemical markers.

Although there are no guidelines available around the use of CGMs in a palliative setting, the decision to continue or cease BGL monitoring (either by CGM or traditional finger-prick testing) should be individualised to take into account patient and carer preference, stage of illness and experience of the healthcare providers.[29] As patients deteriorate, the goals of care shift. In some cases, this might involve loosening glycaemic targets as previously described or ceasing insulin altogether. Pumps use short-acting insulin, so BGLs can rise quickly if it is ceased. Effective communication with both patients and families is at the heart of management, particularly as their expectations evolve. Like with the other aspects of management of diabetes in palliative care, the withdrawal of familiar treatments and devices can evoke a sense of abandonment, further exacerbating distress for patients and families grappling with the realities of end-of-life care. It is essential to convey clearly that the priority has shifted towards providing the best possible care to ensure a dignified death, rather than

Table 4. Medications used in the treatment of diabetes

Drug	Risk of hypoglycaemia	Dose consideration
Insulin	High	• Rapid-acting insulin might be beneficial if erratic appetite • Long-acting insulin might cause less hypoglycaemia because of smoother pharmacodynamics • Review dose if renal/liver dysfunction – risk of hypoglycaemia
Sulfonylurea (eg gliclazide/glipizide/glimepiride/tolbutamide)	High	• Review dose if renal/liver dysfunction, poor oral intake or significant weight loss
Biguanides (eg metformin)	None	• Caution if renal/liver dysfunction; cease if liver failure or eGFR <30 mL/min/1.73 m^2 – risk of lactic acidosis • Can cause distressing gastrointestinal symptoms (nausea, heartburn, diarrhoea, flatulence)
Thiazolidinediones (eg pioglitazone)	Very low risk	• Might cause fluid retention, oedema • Cease if liver failure, heart failure
GLP-1 receptor agonists (eg exenatide, liraglutide, semaglutide)	None	• Causes nausea – review if loss of appetite or significant weight loss • Cease if abdominal pain or pancreatitis • Review dose if renal failure
DPP-4 inhibitor (eg linagliptin, sitagliptin)	Low risk	• Review dose if renal failure – linagliptin safe to use if renal disease • Increased risk of hypoglycaemia when used with sulfonylureas
SGLT2 inhibitor (eg empagliflozin, dapagliflozin)	Low risk	• Cease if dehydrated, acutely unwell, poor oral intake – risk of euglycaemic ketoacidosis • Risk of urogenital infections, candidiasis

DPP-4, dipeptidyl peptidase-4; eGFR, estimated glomerular filtration rate; GLP-1, glucagon-like peptide-1; SGLT2, sodium-glucose cotransporter-2.

focusing on intensive glycaemic control. This understanding can help alleviate anxiety and foster a sense of comfort during a challenging time.

Conclusion

T2DM frequently co-exists in patients with advanced cancer receiving palliative care. It might be pre-existing or result from cancer and its treatments. Evidence is lacking in this area; however, the guiding principle of management is to balance interventions against quality of life, prognosis and symptoms. The focus shifts from often-burdensome treatments aimed at preventing long-term complications of diabetes to preventing symptoms related to extremes of glycaemia. Education of patients and carers about new targets and goals is vital, as many are accustomed to traditional approaches.

Key points

- Diabetes management in patients with advanced cancer focuses on symptom relief and quality of life.
- Extremes of glycaemia cause symptoms and require individualised treatment.
- HbA1c is unreliable and might not align with goals of care in a palliative population.
- Deprescribing and simplifying treatment of diabetes is key as goals of care shift.
- Clear, compassionate communication helps patients and families navigate this transition.

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