



Diabetes and cancer

Diabetes and cancer are common long-term conditions which are highly prevalent in the general population. The co-existence of diabetes and cancer in a person is, therefore, common. In people living with diabetes, the risk of mortality from cancer and morbidity from treatment of cancer is significantly increased. Some cancer therapies cause hyperglycaemia. Many primary care clinicians will deal with a person living with diabetes and cancer. This factsheet summarises the potential links between diabetes and cancer, and provides some practical recommendations on management of hyperglycaemia in patients undergoing cancer therapy.

Links between diabetes and cancer

Diabetes and cancer appear to be linked, independent of obesity and other risk factors common to the two conditions.¹ Meta-analyses show that the relative risks of cancer in people with diabetes are greatest for the following:^{2,3}

- About two-fold or higher:
 - Pancreas.
 - Liver.
 - Endometrium.
- Moderate (about 1.2–1.5-fold):
 - Colon and rectum.
 - Breast.
 - Bladder.
- Conversely, diabetes appears to be protective against cancer of the prostate.⁴

Pre-existing diabetes increases risk of mortality from cancer. Meta-analyses suggests that diabetes is associated with an approximate 40% increased risk of mortality in patients with cancer compared with normoglycaemic individuals.⁵

Possible pathogenic mechanisms

Hyperglycaemia: Rapidly proliferating tumour cells consume glucose at a high rate compared with normal cells, and hyperglycaemia may contribute to cancer by way of providing energy at a cellular level.⁶

Hyperinsulinaemia is associated with type 2 diabetes, and leads to reduced hepatic synthesis of sex hormone-binding globulin (SHBG), leading to increased free levels of oestrogen in both men and women, and increased levels of free testosterone in women. The association of hyperinsulinaemia with cancer appears highest with **breast** and **endometrial** cancers.⁷

Insulin-like growth factor-1 (IGF-1): Insulin also appears to increase hepatic production of IGF-1, which promotes cellular growth. The insulin receptor is structurally related to the IGF-1 receptor and, at high concentrations, insulin may also activate the IGF-1 receptor. IGF-1 activates multiple signalling pathways known to be implicated in cancer pathogenesis. In large epidemiological surveys, elevated circulating insulin and/or IGF-1 levels were associated with **colorectal** cancer.⁸

Diabetes therapies and cancer risk

Metformin may provide some **protection** from cancer in epidemiological studies, possibly through modulation of insulin resistance and hence reduced circulating insulin levels.⁹

Thiazolidinediones (glitazones) have been associated with **urothelial cancers** in animal studies, and there may be a slight signal of increased risk of **bladder cancer** in people who take glitazones for more than two years.¹⁰

There has been some concern about **GLP-1 receptor agonists** and the risk of **pancreatic cancer**. A systematic review and meta-analysis of a number of studies, however, suggests GLP-1 analogues do not increase the risk of pancreatic cancer when compared to other treatments.¹¹ **Medullary thyroid cancer** has also been seen in animal studies, but no increased risk in humans has been reported as yet.¹²

Cancer therapies and diabetes/hyperglycaemia¹³

Corticosteroids are commonly used in patients with cancer to manage nausea or relieve fatigue and pain. They often cause **steroid-induced hyperglycaemia** or exacerbate pre-existing diabetes.

Androgen deprivation therapy (ADT) used to treat prostate cancer is associated with risk of **metabolic syndrome**.

Other cancer chemotherapeutics, such as **5-fluorouracil** and **carboplatin/paclitaxel**, have also been shown to cause **hyperglycaemia**.

Immune checkpoint inhibitors may lead to the development of **new-onset autoimmune type 1 diabetes**, as well as **other endocrinopathies**.

Managing hyperglycaemia in people with diabetes and cancer

Managing hyperglycaemia in people with cancer can be challenging.

- Cancer therapies may lead to hyperglycaemia, which can be exacerbated by nutritional supplements and enteral feeding.
- Nausea may lead to variable oral intake, increasing risk of hypoglycaemia.
- There is no good evidence that tight glucose control improves outcomes. Management of hyperglycaemia should, therefore, be pragmatic and aimed at avoiding symptoms of hypo- or hyperglycaemia.
- Joint British Diabetes Societies for Inpatient Care (JBDS-IP) guidelines suggest that all people undergoing systemic anti-cancer therapy should ideally have their HbA_{1c} and random plasma glucose checked prior to therapy.¹⁴
- A summary of some common clinical pathways for managing hyperglycaemia in patients with cancer is shown in [Table 1](#) (overleaf).

Resource: JBDS-IP (2023) [Management of glycaemic control in people with cancer](#).

Enteral feeding

Due to the high carbohydrate content of enteral feeds, hyperglycaemia is common and achieving good glucose control is challenging.

JBDS-IP guidance recommends glucose targets of 6–10 mmol/L, in an attempt to limit the risk of hypoglycaemia.¹⁶ CBG should be checked pre-feed, every 4–6 hours during feed, and 2 hours post-feed.

Metformin powder/liquid may be considered for mild hyperglycaemia, but frequently insulin is required, and the type and dosing depends on the duration and composition of the feed. An example would be a pre-mixed (30/70) human insulin at the start and a second dose at the midpoint of the feed. It is also important to titrate insulin dose when the feed composition is changed.

End-of-life care

End-of-life care should facilitate a painless, symptom-free death, with minimal medical intervention. In discussion with the person and their family, it may be appropriate to consider cessation of non-essential medications (e.g. statins, cardiovascular risk protection drugs) to reduce tablet burden.

Glycaemic management should aim to avoid metabolic decompensation and symptomatic hyperglycaemia, but also to avoid hypoglycaemia. It may be appropriate to continue insulin once daily.¹⁷

Resource: Trend Diabetes (2024) [End of life guidance for diabetes care](#).

Management of hyperglycaemia associated with glucocorticoid therapy

Glucocorticoids induce hyperglycaemia via effects on insulin receptors in liver, muscle and adipose tissue, causing insulin resistance, increased hepatic gluconeogenesis and decreased insulin secretion.

Typically, they are administered in the morning to avoid causing any sleep disturbance. The pattern of hyperglycaemia tends to be a predominantly post-prandial hyperglycaemia. In order to anticipate this, capillary blood glucose (CBG) levels should be checked regularly in people on glucocorticoids, especially in the afternoon/evening. JBDS-IP guidelines on the management of hyperglycaemia with glucocorticoids offer practical recommendations to manage this problem.¹⁵

For people at risk of steroid-induced diabetes, HbA_{1c} should be measured prior to commencing glucocorticoids. At commencement of glucocorticoid therapy, CBG testing should be done once a day, before either lunch or the evening meal.

Treatment options include metformin, sulfonylureas or morning NPH insulin (e.g. Insulatard® or Humulin I®). A dose of 10 units should be commenced and rapidly titrated according to response. Further addition of prandial insulin may be required.

If glucocorticoids are weaned or stopped, insulin or sulfonylurea therapy should be proactively reduced to reduce risk of hypoglycaemia.

Learning points

- The co-diagnosis of diabetes and cancer is common.
- Diabetes increases the risk of poorer outcomes in people with cancer.
- There is no good data to suggest that tight glycaemic control is beneficial.
- Screen all patients undergoing systemic anti-cancer therapy for diabetes with HbA_{1c} and random plasma glucose tests.
- Avoid extremes of hypo- and hyperglycaemia.
- Metformin is useful and may improve outcomes.
- Monitor and treat post-prandial glucose levels in patients on glucocorticoid therapy.
- Monitor for new-onset type 1 diabetes or diabetic ketoacidosis in people on immune checkpoint inhibitors.
- Discuss withdrawal of non-essential treatment in patients with limited life expectancy.

References are available in the [online version of this article](#).

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Table 1. Summary of relevant guidelines for management of hyperglycaemia in people with cancer.

	Screening/monitoring	Management	Ref
Prior to starting systemic anti-cancer therapies	<ul style="list-style-type: none"> HbA_{1c} and random plasma glucose (RPG) 	<ul style="list-style-type: none"> If HbA_{1c} >47 mmol/mol, refer to GP If RPG ≥20.1 mmol/L, check for hyperglycaemic emergency; once excluded, refer urgently to the diabetes team for treatment If RPG 12–20 mmol/L, check for ketones and commence gliclazide therapy If RPG <12 mmol/L, continue screening at each visit 	14
Glucocorticoid-induced hyperglycaemia in a person with known T2DM on oral agents	<ul style="list-style-type: none"> HbA_{1c} prior to commencement Test capillary blood glucose (CBG) four times daily 	<ul style="list-style-type: none"> Acceptable CBG range 4–12 mmol/L Add or titrate gliclazide 40 mg daily, titrate to maximum 240 mg in morning and 80 mg in evening Add NPH insulin 10 units daily and titrate by 10–20% daily to achieve target Upon cessation of steroids, reduce glucose-lowering treatment to pre-steroid levels Proactive monitoring and treatment at next cycle of treatment 	15
Glucocorticoid-induced hyperglycaemia in a person with known T1DM or T2DM on insulin	<ul style="list-style-type: none"> HbA_{1c} prior to commencement Test CBG four times daily 	<ul style="list-style-type: none"> Acceptable CBG range 4–12 mmol/L, but might be individualised to patients' needs Involve the specialist diabetes team if needed If on once-daily night-time insulin: Transfer night-time insulin injection to the morning; titrate by 10–20% daily; if targets not achieved, consider twice-daily insulin If on twice-daily insulin: Increase morning dose by 10–20% according to pre-evening-meal glucose readings If on basal-bolus insulin: Consider transferring basal insulin dose to morning instead of evening; increase short/fast-acting insulin dose by 10–20% daily until target reached Upon cessation of steroids, reduce treatment to pre-steroid levels Proactive monitoring and treatment at next cycle of treatment 	15
Glucocorticoid-induced hyperglycaemia in a person not known to have diabetes	<ul style="list-style-type: none"> HbA_{1c} prior to commencement At least once-daily monitoring of CBG, prior to lunch or evening meal If CBG >12 mmol/L consistently, enter treatment algorithm 	<ul style="list-style-type: none"> Acceptable CBG range 4–12 mmol/L Gliclazide 40 mg with breakfast; titrate to maximum 240 mg in morning and 80 mg in evening Add NPH insulin 10 units daily and titrate by 10–20% daily to achieve target Upon cessation of steroids, reduce treatment to pre-steroid levels Proactive monitoring and treatment at next cycle of treatment 	15
Enteral feeding in a person with T1DM	<ul style="list-style-type: none"> HbA_{1c} prior to commencement CBG four times daily 	<ul style="list-style-type: none"> Involve the diabetes specialist team Avoid VRIII if possible; if on insulin pump, stop and convert to multiple-dose insulin regimen Continue basal insulin and consider bolus doses of short/rapid-acting insulin at start of feed, and at 6 and 12 hours into feed 	16
Enteral feeding in a person with well-controlled T2DM	<ul style="list-style-type: none"> HbA_{1c} prior to commencement CBG four times daily 	<ul style="list-style-type: none"> Acceptable CBG range 4–12 mmol/L Consider metformin powder via nasogastric tube if CBG is >12 mmol/L consistently 	16
Enteral feeding in a person with poorly controlled T2DM	<ul style="list-style-type: none"> HbA_{1c} prior to commencement CBG four times daily 	<ul style="list-style-type: none"> Acceptable CBG range 4–12 mmol/L Continue metformin as powder via nasogastric tube If CBG is >12 mmol/L consistently, consider: <ul style="list-style-type: none"> Pre-mixed (30/70) insulin at start and middle of feed NPH insulin at start and middle of feed Continue basal insulin and consider bolus doses of short/rapid-acting insulin at start of feed, and at 6 and 12 hours into feed 	16
End-of-life care	<ul style="list-style-type: none"> Consider reduction in monitoring 	<ul style="list-style-type: none"> Careful discussion of diabetes management with patient and carers Offer reduction in monitoring and prescribing in patients with short life expectancy T2DM on diet or metformin alone: Stop monitoring CBG T2DM on tablets, GLP-1 RA or insulin: Consider: <ul style="list-style-type: none"> Stopping tablets and GLP-1 RA injections Stopping insulin depending on dose If insulin to continue, consider once-daily regimen T1DM on insulin: Continue once-daily basal insulin in the morning 	17

CBG=capillary blood glucose; NPH=neutral protamine Hagedorn insulin (e.g. Insuman Basal, Humulin I, Insulatard); RPG=random plasma glucose; T1DM=type 1 diabetes; T2DM=type 2 diabetes; VRIII=variable-rate intravenous infusion of insulin.