**Keywords** Medicine management/ Steroids/Dexamethasone

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## In this article...

- Why steroids such as dexamethasone are used in palliative care and how they work
- How these are used, including dosages and duration of treatment
- Their adverse effects and why and how steroids might be withdrawn

# Using steroids in adult palliative care: an introduction for nurses

## Key points

**Steroids are prescribed for a bewilderingly wide range of indications in palliative care**

**Dexamethasone is used in up to half of all palliative care patients**

**Steroid usage varies significantly between units and prescribers and doses are often arbitrary**

**There is little evidence to support steroid use, but their continued use is inevitable**


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**Abstract** Nurses new to adult palliative care – in any setting – soon discover that steroids are frequently prescribed, often for a bewilderingly wide range of indications and despite limited supportive evidence. For many, their use can remain something of a mystery. Steroid use varies significantly between palliative care units and prescribers, and doses are often arbitrary. This article introduces both newcomers and more experienced health professionals to the reasons steroids are so commonly used in palliative care, why and how they might be withdrawn, and how nurses – through education and monitoring – might help patients and their families to recognise and beware of the many potential adverse effects.

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There are many steroids, including sex hormones such as oestrogen and testosterone, and anabolic steroids, which are sometimes misused – infamously in athletic or body-building contexts – to increase muscle mass. In palliative care, however, the focus is on corticosteroids ('corticoids'), which are produced naturally by the cortex of the adrenal glands or created synthetically for medical use. A subgroup of these, known as glucocorticoids, have anti-inflammatory effects and play a role in the metabolism of carbohydrates, fats and proteins (Wilcock et al, 2022). Synthetic glucocorticoids are commonly used in clinical practice to mimic the action of natural (endogenous) steroids (Joint British Diabetes Societies for Inpatient Care (JBDS-IP), 2021).

Dexamethasone and prednisolone are synthetic glucocorticoids frequently used in palliative symptom management. Dexamethasone is over seven times stronger than prednisolone (2mg dexamethasone = 15mg prednisolone) and has a much longer duration of action (36-54 hours versus 12-36 hours, respectively). Oral dexamethasone reaches peak plasma concentration after 1-2 hours, with a plasma half-life of 4.5 hours. Dexamethasone causes less fluid retention than prednisolone and requires fewer tablets, making it a preferred option in palliative care – although prednisolone may be equally effective and is often favoured in respiratory disorders (Wilcock et al, 2022; Hardy et al, 2021). Dexamethasone is used in up to half of all palliative care patients, most often in courses lasting 5-7 days (JBDS-IP, 2021).

## Uses

### Anti-inflammatory

In advanced cancer, glucocorticoids generally work by reducing the oedema surrounding tumours (Khadka et al, 2023). Dexamethasone is highly anti-inflammatory and particularly useful when fluid retention is unwelcome – such as in cases of cerebral oedema (NICE, 2025a). Corticosteroids may be more effective than non-steroidal anti-inflammatory drugs (NSAIDs), but long-term steroid use can

cause more adverse effects compared with NSAIDs (Twycross et al, 2021).

By reducing peri-tumour oedema, steroids may relieve pressure on adjacent nerves and alleviate obstruction in hollow organs and passages, such as the lungs and ureters. In the gastrointestinal (GI) system, this may allow improved opening of lumen and hopefully improved bowel patency. For instance, in the initial management of inoperable bowel obstruction, subcutaneous dexamethasone – typically 6.6-13.2mg daily – may be given for 5-7 days alongside an antacid or proton pump inhibitor (PPI) such as omeprazole by daily infusion.

**Analgesic**

Evidence is limited, but corticosteroids can provide rapid – if sometimes short-lived – relief from pain caused by tumours in confined spaces. This includes brain and pelvic tumours, kidney and liver capsule pain and in cancer-related neuropathic pain, such as spinal cord or nerve compression. In cases of bone pain, including pathological fractures and acute vertebral collapse, oral dexamethasone 4-8mg daily may be beneficial, especially when pain is accompanied by limb weakness, or while awaiting other treatments such as radiotherapy. In ureteric colic caused by tumour-related occlusion, oral dexamethasone 8mg daily may offer relief, with or without concurrent NSAIDs. Corticosteroids may also alleviate pain that is only semi-responsive to opioids, such as muscular or soft tissue infiltration pain. In cases of tenesmus due to tumour irritation of the rectum, oral dexamethasone 8mg daily may be helpful in pain reduction (Wilcock et al, 2022; Twycross et al, 2021; World Health Organization, 2019; Lim et al, 2017; Haywood et al, 2015). Dexamethasone may also reduce radiation-induced inflammation (Hardy et al, 2021).

**Anti-emetic**

Corticosteroids have anti-emetic effects, and daily oral dexamethasone 4-16mg (or subcutaneous 3.3-13.2mg), may help to address refractory nausea and vomiting in cancer patients. Dexamethasone can be used once or twice daily, either alone or in combination with ondansetron or levomepromazine, or with metoclopramide if nausea and vomiting is attributed to cytotoxic drugs or delayed gastric emptying (Wilcock et al, 2022; Twycross et al, 2021). Evidence supports steroid use when nausea is chemotherapy-related, but is otherwise weak (Hardy et al, 2021).

For nausea and vomiting caused by raised intracranial pressure, NICE (2021) recommends adding oral dexamethasone 8-16mg daily to cyclizine for up to one week.

**Appetite**

Corticosteroids, such as oral dexamethasone 2-4mg daily or oral prednisolone 15-30mg daily, can increase appetite; however, this effect may also be attributable to placebo response (Hardy et al, 2021; Watson et al, 2016). When used for more than two weeks, prednisolone may be less likely than dexamethasone to cause muscle catabolism, and for patients with a prognosis of several months, progestogens such as megestrol acetate (Megace) may be more appropriate – though progestogens are more expensive than steroids and carry similar risks of significant adverse effects, including thromboembolism. Regardless of the medication, any improvements are temporary, and any weight gain is probably due to increased fluid and fat retention, and, particularly in inactive patients, indicative of catabolism of skeletal muscle (Wilcock et al, 2022; Twycross et al, 2021).

*"Corticosteroids can provide rapid relief from pain caused by tumours in confined spaces"*

**Breathlessness**

Although supportive evidence is scarce, dexamethasone may reduce airway obstruction and improve breathlessness caused by lung tumours (Hardy et al, 2021; Healthcare Improvement Scotland, 2019; Watson et al, 2016). Oral dexamethasone 4-8mg daily should elicit improvement within days; if not, dexamethasone should be stopped. While long-term use of oral steroids is not supported by evidence, short-term administration of at least 4mg dexamethasone daily may improve lung function (Watson et al, 2016). Steroids may also have a role in managing breathlessness associated with other conditions encountered in palliative care (Wilcock et al, 2022; Hardy et al, 2021).

**Brain tumour/metastases**

Though optimal dosage remains debated, 4-16mg of dexamethasone daily may reduce cerebral oedema and raised intracranial pressure, and alleviate resulting symptoms such as headache, nausea, neurological impairment and delirium caused by brain tumours or cerebral metastases (Wilcock et al, 2022; Hardy et al, 2021; Twycross et al, 2021; Healthcare Improvement Scotland, 2019; Wilkinson et al, 2017), though severe cerebral oedema may take 2-3 weeks to respond (NICE, 2025a). As patients with increased intracranial pressure typically have risk factors for GI bleeding, NICE (2025a) advises gastric protection with a PPI.

Dexamethasone should be continued subcutaneously if previously given orally in the terminal phase if withdrawal might result in agitation or worsening headaches, and to minimise the risk of seizures (NHS England, 2025).

**Palliative care emergencies**

Unless contraindicated, patients with malignant spinal cord compression or cauda equina syndrome should receive a loading dose of oral dexamethasone 16mg as soon as possible to reduce oedema and hopefully postpone spinal cord ischaemia. Oral dexamethasone 8mg daily should continue until surgery or until radiotherapy is started, then reduced and discontinued over 1-2 weeks after surgery or radiotherapy is completed. If any neurological deterioration occurs during this tapering off, the dose should be increased back to the previously effective level and maintained at that dose for two weeks before attempting reduction again (NICE, 2023a; Wilcock et al, 2022; Hardy et al, 2021).

When severe symptoms are caused by cancer-related superior vena cava obstruction (SVCO), emergency treatment (despite little supporting evidence) typically involves giving oral dexamethasone 16mg daily to alleviate peri-tumour oedema and the resulting compression. When SVCO presents as the first sign of cancer, high-dose corticosteroids are not given (unless severe symptoms intervene) until a biopsy is performed, as they might delay histological diagnosis. If further treatment is not possible, or SVCO is not responsive to chemotherapy or radiotherapy, oral dexamethasone 16mg daily can continue to be given, and a stent inserted into the SVCO (Hardy et al, 2021; Twycross et al, 2021; Healthcare Improvement Scotland, 2019).

**Other**

Dexamethasone 2-4mg daily may help address fatigue, improve mood (although placebo might work equally well), and general wellbeing, with doses of 2-8mg daily for hiccups or minor haemoptysis (Sandford et al, 2023; Hardy et al, 2021; Healthcare Improvement Scotland, 2019; Watson et al, 2016). In lymphangitic carcinomatosis

(despite little supporting evidence) and lymphangitis, 8-16mg oral dexamethasone may be required daily (Hardy et al, 2021; Healthcare Improvement Scotland, 2019; Wilkinson et al, 2017).

Oral dexamethasone or rectal prednisolone may help reduce discharge from a rectal tumour. Dexamethasone is also used to manage complications such as chemotherapy fibrosis or pneumonitis. For skin irritation or itching caused by inflammation (after infection has been excluded), a short course of oral dexamethasone 2-4mg daily or oral prednisolone 10-20mg daily for one week may be effective.

In Hodgkin's lymphoma, oral dexamethasone 4mg daily may ease paraneoplastic sweating and fever. When lymphoedema results from cancerous infiltration of lymph nodes, oral dexamethasone 8mg daily may reduce peri-tumour inflammation sufficiently to ease lymphatic obstruction. If swelling is reduced, a maintenance dose of oral dexamethasone 2-4mg daily can continue indefinitely, provided the benefits are considered to outweigh the risks (Wilcock et al, 2022; Twycross et al, 2021).

Dexamethasone may also be used as part of hormone therapy to shrink or inhibit tumours in prostate cancer (NICE, 2025c), and as part of anti-cancer treatment in breast cancer (University Hospitals Sussex NHS Foundation Trust, 2023) and haematological malignancies (NHS Northern Cancer Alliance, 2018).

## Administration

Oral dexamethasone is available as tablets, soluble tablets and oral solution (Wilcock et al, 2022). Both dexamethasone and prednisolone can be given as a single morning dose to minimise disturbance of the circadian rhythm of cortisol secretion (Joint Formulary Committee, no date) and reduce suppression of the hypothalamic-pituitary-adrenal axis (Cardenas-Mori and Lewis-Ramos, 2020). However, if tablet burden or injection volumes are problematic, dexamethasone doses can be split into morning and lunchtime doses – with or after food. Doses administered after 2-4pm increase the likelihood of insomnia, but even with morning-only doses, a Z-drug, such as zopiclone, or a benzodiazepine may be needed to alleviate agitation or insomnia (Wilcock et al, 2022), although Hardy et al (2021) could find no evidence to link steroids with insomnia. Dexamethasone can be given at any time in an emergency, including intravenously.

For practical purposes, 4mg of oral dexamethasone is considered equivalent to approximately 3.3mg or 3.8mg of subcutaneous dexamethasone, depending on the formulation used. Both formulations – 3.3mg/ml or 3.8mg/ml – can be used, allowing straightforward conversions (Wilcock et al, 2022) (Table 1).

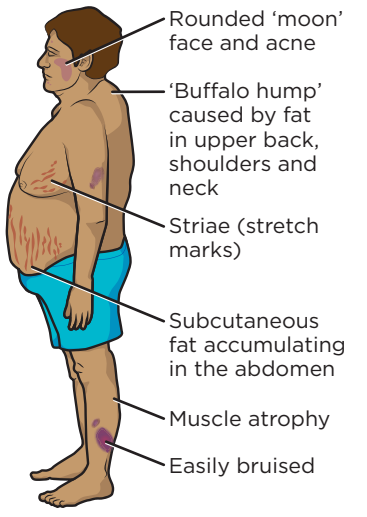
Table 1. **Dexamethasone doses**

| Oral dose | Subcutaneous equivalent | Injection volume |
|---|---|---|
| 1mg | 0.825 or 0.95mg | 0.25ml |
| 2mg | 1.65 or 1.9mg | 0.5ml |
| 4mg | 3.3 or 3.8mg | 1ml |
| 8mg | 6.6 or 7.6mg | 2ml |
| 16mg | 13.2 or 15.2mg | 4ml as 2x2ml |

If a patient cannot swallow oral dexamethasone, or is prevented by nausea or vomiting, it can be given by continuous subcutaneous infusion by syringe driver, but it does not mix well with other drugs at therapeutic doses. Instead, given its lengthy duration of action, dexamethasone is better given by subcutaneous injection each morning (Wilcock et al, 2022). For subcutaneous doses equivalent to >8mg, injections should be administered at two sites, as the recommended maximum volume for a single subcutaneous injection is 2ml (Twycross et al, 2021).

Low doses of dexamethasone (0.5-1mg over 24 hours) are sometimes added to a continuous subcutaneous infusion to reduce site reactions (Wilcock et al, 2022), but Dickman and Schneider (2016) advise against this, recommending instead that dexamethasone be injected directly into the infusion site.

Fig 1. **Cushing's syndrome**

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## Adverse effects

The risk of undesirable effects from corticosteroids increases with both duration of treatment and dosage. Courses lasting less than two weeks are generally tolerated well, other than possible hyperglycaemia, however, adverse effects become more common with continued use beyond 3-4 weeks (NICE, 2025a; Hardy et al, 2021).

While specific risk factors are largely unidentified, older people are more likely to experience adverse effects from corticosteroids (NICE, 2025a). These can include hunger; ophthalmic complications such as glaucoma, cataracts and central serous chorioretinopathy (accumulation of fluid under the retina); and dose-related insomnia. Psychiatric disturbances are also well-documented (Hardy et al, 2021; Healthcare Improvement Scotland, 2019). Other potential adverse events include sodium and water retention, oedema, hiccups, hepatic or renal impairment, hypertension and non-fluid weight gain (Wilcock et al, 2022), negative nitrogen balance and hypokalaemia, which can precipitate hepatic encephalopathy (Joint Formulary Committee, no date).

High-dose glucocorticoids given for eight weeks or more cause the characteristics of Cushing's syndrome (Fig 1) in approximately 30-70% of patients, including a plethoric, rounded 'moon' face, acne, striae, hirsutism, lipodystrophy and delayed wound healing (Wilcock et al, 2022).

Patients prescribed systemic dexamethasone for more than three weeks and/or high doses should receive a Steroid Treatment Card (Wilcock et al, 2022), which warns carers and health professionals that missing doses or experiencing illness puts the patient at risk of adrenal crisis, and advises on emergency treatment (Joint Formulary Committee, no date).

### Immunosuppression

Long-term glucocorticoid use increases both susceptibility to infections (including oral thrush), and the potential severity of those infections. Corticosteroids may also obscure the signs and symptoms of serious

illnesses and infections. For instance, conditions such as peritonitis may present atypically, with patients remaining apyrexial despite infection.

Severe viral or bacterial infections, including septicaemia, afebrile neutropenic sepsis, and pneumocystis pneumonia, may become advanced before being diagnosed. Incipient or dormant infections, such as tuberculosis, may become activated. Also, unless they are already immune, patients are at increased risk of developing severe measles or chickenpox if exposed, and immunoglobulin therapy may be indicated.

Patients should be advised to avoid contact with individuals with shingles, due to increased risk of varicella-zoster virus transmission. Systemic infection is a contraindication to initiating oral corticosteroids – unless their use is likely to be lifesaving and the patient is receiving antimicrobial therapy (Wilcock et al, 2022).

**Diabetes**

Glucocorticoids affect carbohydrate metabolism by increasing gluconeogenesis and glucose release from the liver, thereby raising blood glucose levels. They also antagonise the effect of insulin and oral antidiabetic drugs, decrease the uptake and utilisation of glucose and so destabilise glycaemic control in patients with diabetes. In non-diabetic individuals, corticosteroids can precipitate the onset of steroid-induced diabetes – possibly within a few days (Wilcock et al, 2022; Hardy et al, 2021; JBDS-IP 2021). High-dose steroids taken for long periods can cause significant hyperglycaemia, presenting with symptoms including polyuria, polydipsia and fatigue, increasing the patient's vulnerability to infections and acute complications of hyperglycaemia, such as diabetic ketoacidosis (JBDS-IP, 2021; Watson et al, 2016).

Little evidence exists to guide the management of patients with glucocorticoid-induced hyperglycaemia, but blood glucose should be monitored before starting and while taking medium to high doses of dexamethasone, especially in at-risk patients (Wilcock et al, 2022).

Dexamethasone taken each morning often causes a late afternoon or early evening rise in blood glucose levels. For this reason, glucose monitoring should ideally occur before an evening meal or, if this is not possible, 1-2 hours post-prandially (JBDS-IP, 2021; Healthcare Improvement Scotland, 2019). In patients approaching the end of life, routine glucose monitoring can be stopped (Maslen, 2019).

**Musculoskeletal**

Significant musculoskeletal effects can result from long-term (beyond a few weeks) dexamethasone use at high doses (over 4mg daily), though such effects are possible at lower doses, with dexamethasone more prone to their causation than prednisolone. Glucocorticoids increase protein breakdown and reduce protein synthesis, especially in muscle. Although glucocorticoids may temporarily alleviate weakness, prolonged use can lead to myopathy characterised by atrophy of respiratory and limb muscles. This may cause reduced mobility and compromise respiratory function and limb muscle strength – the latter typically involving increasing difficulty standing from sitting (Ritter et al, 2023; Wilcock et al, 2022; Hardy et al, 2021; Cardenas-Mori and Lewis-Ramos, 2020; Watson et al, 2016).

QUICK FACT **50%** The glucocorticoid dexamethasone is used in up to half of all palliative care patients

Corticosteroids impair calcium metabolism by reducing absorption, increasing its excretion and antagonising the effects of oral calcium supplements (Healthcare Improvement Scotland, 2019). The resulting bone catabolism can cause osteopenia (low bone density), osteoporosis, fracture and avascular necrosis (Hardy et al, 2021; Twycross et al, 2021); Healthcare Improvement Scotland (2019) recommends osteoporosis prophylaxis with bisphosphonates when dexamethasone is prescribed for more than three months.

**Drug interactions**

Taking dexamethasone alongside warfarin significantly increases the International Normalised Ratio (INR), heightening the risk of bleeding. It is recommended that INR should be checked weekly for 2-3 weeks when corticosteroids are started or the dose is changed in this patient group (Wilcock et al, 2022). When used in combination with anticoagulants (such as heparin or warfarin), alcohol, urokinase and streptokinase, dexamethasone increases the risk of GI ulceration and haemorrhage (Hardy et al, 2021). Phenobarbital, carbamazepine, phenytoin and rifampicin reduce the effect of dexamethasone, and larger doses (at least double) are needed when used concurrently, while dexamethasone also disrupts phenytoin plasma levels, complicating seizure control. Larger doses of corticosteroids are also needed if prescribed alongside diuretics and antihypertensives (Wilcock et al, 2022).

Conversely, anti-thyroid agents, oral contraceptives, clarithromycin and antifungals such as itraconazole can markedly increase the effect of corticosteroids (NICE, 2025a; Hardy et al, 2021; Healthcare Improvement Scotland, 2019). Magnesium trisilicate may reduce the absorption of corticosteroids, so they should be administered at least two hours apart (Twycross et al, 2021).

Oral corticosteroids can interact with digoxin, and prescribing beta-2 agonists, such as salbutamol alongside corticosteroids, increases the risk of hypokalaemia (NICE, 2025a; Wilcock et al, 2022).

Corticosteroids may also affect the response to immunotherapy (Cardenas-Mori and Lewis-Ramos, 2020). The antibody response to vaccines may be reduced (Medicines and Healthcare products Regulatory Agency, 2019) and live vaccines should be avoided (Wilcock et al, 2022).

**NSAIDs**

Corticosteroids increase gastric acid production, leading to common symptoms such as indigestion, dyspepsia and heartburn, and may be accompanied by abdominal distension. The risk of stomach ulceration from corticosteroid use alone is small, hence PPIs are not routinely recommended for gastric protection. However, taking corticosteroids alongside NSAIDs, including aspirin, increases the risk of gastric ulceration 15-fold, and also elevates the likelihood of other serious GI complications, such as bleeding, silent perforation and oesophageal ulceration. Therefore, patients taking dexamethasone alongside NSAIDs should receive a prophylactic PPI, H2 blocker (such as famotidine) or misoprostol.

Dexamethasone should be prescribed with caution in patients with active peptic ulceration or a history of GI bleeding or ulceration, and only if anticipated benefits likely outweigh the risks (NICE, 2024; Wilcock et al, 2022; Hardy et al, 2021; Twycross et al, 2021; Cardenas-Mori and Lewis-Ramos, 2020; Watson et al, 2016).

**Adrenal suppression/insufficiency**

Dexamethasone doses >1mg daily can cause adrenal suppression, which can persist for a year or more after discontinuation. Prolonged corticosteroid use reduces production of natural steroids, supressing the patient's physiological stress response during episodes of other illness, trauma, infection or surgery (NICE, 2025a; Ritter et al, 2023; Wilcock et al, 2022). Patients who have received >5mg of prednisolone

(or dexamethasone equivalent) daily for four or more weeks require an increase in steroid dose (or, if discontinued within the past three months, its reintroduction) during significant episodes of physiological stress, which might otherwise cause symptoms of adrenal insufficiency and 'pseudo-rheumatism'. In severe cases, patients may develop hypoadrenal crisis characterised by profound weakness, malaise and potentially life-threatening hypotension (NICE, 2025a; Wilcock et al, 2022). Acute adrenal insufficiency may also occur following sudden withdrawal of corticosteroids after three or more weeks of use, along with a worsening of the underlying condition (NICE, 2025a).

### Withdrawal

Dexamethasone trials should be time-limited, typically ranging from three days to 2-4 weeks (NICE, 2025b; NICE, 2023b; Wilcock et al, 2022; NICE 2021; Cardenas-Mori and Lewis-Ramos, 2020), and titrated to the lowest effective dose (Twycross et al, 2021) as soon as the patient's condition allows – provided that doesn't precipitate a deterioration (Watson et al, 2016). Over time, symptom relief from dexamethasone diminishes but adverse effects increase. Therefore, dexamethasone doses should ideally decrease after a week and stop after 2-4 weeks. However, without other treatment, patients may suffer symptom recurrence, so its use may need to continue indefinitely (Wilcock et al, 2022).

Given the paucity of evidence to support safe recommendations for glucocorticoid withdrawal, and the unpredictability of individual reactions, there is considerable uncertainty and variability of practice, and many conflicting guidelines (Hardy et al, 2021).

Hardy et al (2021) advise that if no clinical benefit is evident, dexamethasone should be withdrawn within two weeks. However, they caution against abrupt cessation if the daily dose has exceeded 4mg for more than five days. In contrast, lower-dose regimens (such as <4mg daily for ≤2-3 weeks) can be discontinued abruptly, provided the patient:

- Has not received repeated recent courses (particularly in those exceeding three weeks);
- Has not taken additional evening doses (which increases the risk of adrenal insufficiency);
- Is not currently taking steroids within a year of previous long-term use (months or years);
- Does not have other known risk factors for adrenal insufficiency;
- Is not at risk of severe symptom recurrence (NICE, 2025a).

For patients with any of the above risk factors, doses should be reduced more gradually (NICE, 2025a).

High doses can be tapered initially using the following approaches: Reduce by up to 50% every 3-5 days until reaching 2mg daily; reduce by 2-4mg every 5-7 days, or taper by 0.5mg on alternate days, weeks, or months. As the dose approaches physiological levels, approximately 1-2mg dexamethasone daily, the pace of tapering should slow further, reducing by 0.5-1mg daily every 5-7 days, or reducing every 2-4 weeks, potentially extending over many weeks to allow time for adrenal function to recover (NICE, 2025a; Wilcock et al, 2022; NICE, 2021; Twycross et al, 2021; Watson et al, 2016).

If withdrawal symptoms emerge, or if the underlying symptoms being treated recur, the steroid dose should be increased temporarily before reattempting withdrawal more slowly. In dying patients who are unable to take oral medications, subcutaneous dexamethasone can be given to avoid distress and agitation associated with withdrawal, though the risk of withdrawal-related distress within the first 24 hours is highly unlikely (NICE, 2023b; Healthcare Improvement Scotland, 2019).

## Conclusion

In palliative care, the use of steroids is ubiquitous, although the evidence base for steroid use and dosing is limited. They have a multitude of uses and can be very beneficial – at least in the short term. However, given their many potentially serious adverse effects and the harmful consequences associated with rapid withdrawal, the safe and effective use of glucocorticoids requires a cautious approach. This means considering the potential risks versus the benefits, for symptoms likely responsive to their use, and stopping if no benefits are apparent, or if unacceptable toxicities intervene. For nurses, it also means monitoring for adverse effects and informing patients about the purpose and risks of steroid therapy. **NT**

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