The Impact of Corticosteroid Use on the Efficacy and Safety of Checkpoint Inhibitors Across Cancer Types

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Immune checkpoint inhibitors (ICIs) have transformed cancer care by helping the immune system recognize and fight tumours. However, their benefits often come with a downside: immune-related adverse events (irAEs), which are commonly treated with corticosteroids. Although steroids help lower inflammation and relieve symptoms, their immunosuppressive properties have sparked questions about whether they could compromise the effectiveness of immunotherapy. With an emphasis on timing, dosage and the rationale behind prescriptions, this article examines the effects of corticosteroid use on patient outcomes when taking ICIs. Steroid use before immunotherapy, particularly at doses of 10 mg or more of prednisone per day, has been associated with poorer outcomes for cancers such as melanoma and non-small cell lung cancer. However, steroids do not appear to have the same adverse effects when used later to manage irAEs, particularly after treatment has already shown some benefit. Steroid use may even be linked to a higher chance of survival in certain situations, such as renal cell carcinoma. The timing and manner of steroid use, whether high or low doses, early or reactive, make a significant impact. However, more recent methods that reduce the need for steroids, like customized irAE management or alternative antiemetic tactics, provide ways to help patients without sacrificing the immunological response. Although steroids are still an important part of cancer treatment, their use in conjunction with ICIs requires a cautious, customized strategy. Clinicians should always try to balance symptom relief and treatment efficacy when prescribing, considering timing, dosage and purpose.

Keywords

Checkpoint inhibitors, corticosteroid, gastrointestinal cancer, immunotherapy, lung cancer, melanoma, renal cancer

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Immune checkpoint inhibitors (ICIs) act by blocking inhibitory pathways such as programmed cell death protein 1 (PD-1), programmed cell death ligand 1 (PD-L1) and cytotoxic T lymphocyte-associated antigen 4 (CTLA-4), thereby restoring T-cell activity and promoting tumour cell destruction.[1,2] The ability of tumours to evade the immune system is a significant drawback of conventional treatments that immunotherapy has addressed in a revolutionary way. These treatments interfere with the molecular 'shields' that cancer cells use to stifle immune activity by targeting immune checkpoints like CTLA-4, PD-1 and PD-L1. Through natural immune surveillance, this reactivates T cells and restores the body's ability to recognize and eliminate cancerous cells.[1,2]

Although the T-cell inhibitory checkpoints seem to have a long duration and a good safety profile, they do not work for all patients. One of the reasons is the tumour mutation burden and the different mechanisms of action between them. Some of them only downstream the T-cell, and others make it dysfunctional.[3] Other immune checkpoints could also be targeted to achieve a more significant number of patients responding to this kind of treatment. Current research is also exploring other immune checkpoints, such as lymphocyte activation gene-3 (LAG-3) and T-cell immunoglobulin and mucin-domain containing-3 (TIM-3), which could change the current gap in treatment that still exists.[3]

Corticosteroids are widely used in clinical practice due to their anti-inflammatory, immunosuppressive and antiemetic properties. Their indications include severe allergic reactions, asthma, chemotherapy-induced nausea, autoimmune exacerbations, chronic obstructive pulmonary disease and prophylactic use to prevent transplant rejection.[4] Importantly, the anti-inflammatory and immunosuppressive effects of glucocorticoids are dose-dependent, with immunosuppressive effects primarily observed at higher doses.[4]

One of the main applications of corticosteroids in oncology is managing symptoms. They aid in lowering tumour-related pain and inflammation, especially in cases like spinal cord compression and brain metastases.[5] Furthermore, by improving the effectiveness of antiemetic regimens, corticosteroids are commonly used to prevent chemotherapy-induced nausea and vomiting.[6] Corticosteroids can increase appetite and energy levels in patients with cachexia or advanced cancer, improving their quality of life.[7]

Corticosteroids have direct anticancer effects in addition to managing symptoms, especially in haematologic malignancies. As they cause malignant lymphoid cells to undergo apoptosis, drugs like dexamethasone and prednisone are essential to treatment plans for leukaemias and lymphomas.[8] In patients with brain tumours or metastases, corticosteroids are also frequently used to lessen cerebral oedema, which relieves symptoms like headaches and neurological impairments.[9]

Corticosteroids have recently been used to treat immune-related adverse events (irAEs) brought on by ICIs. Although steroids are helpful in reducing these inflammatory toxicities, there are concerns that their immunosuppressive properties may decrease the effectiveness of immunotherapy.[10] This article investigates the effects of corticosteroid use on the efficacy and safety of immunotherapy, specifically checkpoint inhibitors, in relation to various cancer types. This article examines whether the immunosuppressive effects of corticosteroids interfere with the benefits of checkpoint inhibitors, how they affect treatment outcomes and their role in managing irAEs.

Impact on immunotherapy efficacy

Although corticosteroids help to reduce irAEs and control inflammation, there are concerns that they might also inhibit the body's immune response against tumours, especially when used with ICIs. There have been conflicting findings from studies looking at how they affect critical clinical outcomes like overall survival (OS), progression-free survival (PFS) and overall response rates (ORRs). According to research, how corticosteroids affect immunotherapy mostly depends on the dosage, timing and purpose of use.[10–12]

Goodman et al. provided an in-depth analysis of how corticosteroids interact with ICIs, showing that the impact depends on how, when and why the steroids are used.[11] Their study found that patients who were on steroids, especially doses of 10 mg or more of prednisone daily, just before or at the start of immunotherapy often had worse outcomes. In comparison to patients who were not on steroids at the time, these patients generally had lower response rates (RRs), shorter OS and cancers that progressed more quickly. Steroids used to treat cancer-related problems such as brain metastases, exhaustion or appetite loss were found to have a detrimental effect.[11]

However, the treatment did not appear to lose its efficacy when steroids were used to treat irAEs after patients had already begun to benefit from ICIs.[11] This highlights an important point: why steroids are being used makes a big difference.

Immunosuppressive mechanisms of corticosteroids

Corticosteroids work by suppressing the immune response through both non-genomic and genomic mechanisms. Following their passive diffusion into cells, they attach to intracellular glucocorticoid receptors to form a complex that moves to the nucleus and inhibits important transcription factors like nuclear factor kappa-light-chain-enhancer of activated B cells and activator protein 1, thereby downregulating the transcription of pro-inflammatory genes such as interleukin (IL)-1α and IL-1β. This action changes the function of several immune cell types and decreases the synthesis of inflammatory cytokines. Both innate and adaptive immune responses are dampened by corticosteroids, which also reduce circulating lymphocytes, monocytes, basophils and eosinophils; cause neutrophilic leukocytosis through demarginating, prevent macrophage activation, inhibit fibroblast proliferation; and disrupt matrix metalloproteinases and arachidonic acid pathways.[4,11]

These immunosuppressive effects can disrupt critical antitumour immune responses in the context of cancer, impacting immune-mediated tumour cell death, lymphocyte trafficking and tumour antigen release. Crucially, IL-2-mediated effector T-cell inactivation (a crucial element of checkpoint inhibitor efficacy) is one of their main effects in patients receiving immunotherapy. Therefore, even though glucocorticoids are good at controlling inflammatory toxicities, their widespread immunosuppressive effects may lessen the effectiveness of ICIs, so their use should be used carefully and sparingly.[13]

Corticosteroids in specific cancer types

Melanoma

The impact of corticosteroid use on managing irAEs in patients with melanoma receiving ICIs was evaluated in a large multicentre cohort by Verheijden et al.[12] The study demonstrated that higher peak doses of corticosteroids were significantly associated with worse survival outcomes. Specifically, compared with a 40 mg prednisolone equivalent dose, a peak dose of 80 mg was associated with an adjusted hazard ratio (HR) of 1.14 (95% confidence interval [CI]: 1.01, 1.29; p<0.05) for PFS and 1.29 (95% CI: 1.12, 1.49; p<0.001) for OS. The association was even stronger at a 160 mg dose: HR=1.42 (95% CI: 1.03, 1.95) for PFS and 1.97 (95% CI: 1.36, 2.85) for OS.[12] Notably, cumulative corticosteroid dose was not associated with impaired survival, suggesting that peak dosing rather than total exposure may be the critical factor. These findings support the need to re-evaluate current irAE management strategies, particularly advocating for minimizing peak corticosteroid doses when clinically feasible.[12]

However, there is conflicting evidence. For example, Horvat et al. investigated how corticosteroid use might affect ORR in patients with melanoma undergoing anti-PD-1 treatment.[14] Their study found no appreciable impact on ORR from the short-term use of corticosteroids to treat irAEs. This is consistent with other studies, such as one by Lev-Ari et al., which also demonstrated that systemic steroids used to treat irAEs in patients with melanoma did not correlate with worse disease outcomes, such as OS, PFS or ORR.[15]

Non-small cell lung cancer

Ricciuti et al. investigated the effect of baseline corticosteroid use on the efficacy of ICIs in 650 patients with non-small cell lung cancer (NSCLC).[16] At the initiation of immunotherapy, 93 patients (14.3%) received ≥10 mg of prednisone daily. These patients had significantly shorter median PFS (2.0 versus 3.4 months; HR=1.36 [95% CI: 1.08, 1.73; p=0.01]) and median OS (4.9 versus 11.2 months; HR=1.68 [95% CI: 1.30, 2.17; p<0.001]) compared with those receiving <10 mg.[16]

Significantly, the indication for corticosteroid use modified its impact on outcomes. Patients receiving corticosteroids for cancer-related palliative reasons had markedly worse outcomes (mPFS: 1.4 months; mOS: 2.2 months) compared with those on corticosteroids for non-cancer-related reasons (mPFS: 4.6 months; mOS: 10.7 months) or to those not on corticosteroids (mPFS: 3.4 months; mOS: 11.2 months).[16] In multivariable analysis, corticosteroid use for cancer-related palliation was independently associated with increased mortality, for OS, HR=1.60 (95% CI: 1.07, 2.39; p=0.02), while non-cancer-related corticosteroid use

showed no significant association, for OS, HR=0.91 (95% CI: 0.47, 1.79; p=0.79).[16]

These findings highlight that although baseline corticosteroid use is often associated with worse outcomes in patients with NSCLC receiving ICIs, the negative impact is primarily seen in those using steroids for cancer-related palliative reasons.[16] This points to the underlying severity of the disease, rather than the immunosuppressive effect of corticosteroids alone, as a key factor influencing outcomes.

Arbour et al. examined the impact of baseline corticosteroid use on the efficacy of PD-1 and PD-L1 inhibitors in patients with advanced NSCLC, using data from 640 patients treated at Memorial Sloan Kettering Cancer Center and Gustave Roussy Cancer Center.[10] Of these, 90 patients (14%) received ≥10 mg of prednisone or equivalent at the start of immunotherapy. The most frequent indications for corticosteroid use were dyspnoea (33%), fatigue (21%) and brain metastases (19%).[10]

In a multivariable analysis adjusted for smoking history, performance status and brain metastases, baseline corticosteroid use (≥10 mg) was significantly associated with worse outcomes: the HR for PFS was 1.31 (95% CI: 1.03, 1.67; p=0.03), and for OS, the HR was 1.66 (95% CI: 1.28, 2.16; p<0.001).[10]

These findings suggest that baseline corticosteroids may impair the effectiveness of ICIs, likely due to their immunosuppressive effects. The authors advocate for the judicious use of corticosteroids at the start of PD-L1 therapy, especially in patients who may otherwise be candidates for immunotherapy.[10]

In a study by Wang et al., the effectiveness of ICIs was examined in the timing and dosage of glucocorticoid use in 130 patients with advanced NSCLC.[17] Overall, glucocorticoid use was linked to a higher ORR, according to the authors with HR=3.07 (95% CI: 1.31, 7.21; p=0.010). Interestingly, patients who started glucocorticoids early during immunotherapy had worse outcomes than those who received glucocorticoids at baseline, prior to the start of ICI treatment (ORR: 65.4% versus 30.8%, p=0.024). Additionally, glucocorticoid use was associated with more prolonged PFS, with HR=0.37 (95% CI: 0.17, 0.78, p=0.009), according to multivariate Cox analysis. However, neither the length of time nor the dosage of glucocorticoids significantly impacted ORR or PFS. The timing made a difference, immune markers like neutrophil levels and the CD4+/CD8+ T-cell ratio were influenced by when the steroids were given. The authors concluded that glucocorticoid use, especially when started at baseline, may improve ICI efficacy, allaying previous concerns that steroids would generally impair immunotherapy results.[17]

Şen et al. examined the results of 90 patients with advanced NSCLC who received ICIs either alone or in conjunction with chemotherapy in this retrospective single-centre study.[18] Assessing the effect of corticosteroid use (defined as ≥10 mg of prednisone or its equivalent) and irAEs on treatment efficacy was the primary goal. Forty-five patients (50%) experienced irAEs, and the time-dependent nature of irAE development was considered using landmark analysis. Patients with irAEs had much better outcomes: median PFS was 25.9 months versus 8.4 months with HR 2.54 (95% CI: 1.52, 4.25; p<0.001), and median OS was 52.1 months compared with 14.4 months in those without irAEs with HR=2.71 (95% CI: 1.55, 4.73; p<0.001).[18] Patients who experienced irAEs also responded better to treatment, with an ORR of 60% compared with 33.3% in those without irAEs (p=0.011). Most patients (76%) did not need steroids, but about a quarter (24%) did, mainly to help manage their irAEs. OS (26.5 versus 28.7 months; HR=1.14 [95% CI: 0.63, 2.08; p=0.652]) and PFS (16.9 versus 13.5 months; HR=0.99 [95% CI: 0.57, 1.74; p=0.997]) did not differ statistically significantly between the steroid and non-steroid groups.[18] Additionally, survival outcomes were not significantly impacted by the severity or quantity of irAEs experienced by a patient. These findings suggest that the incidence of irAEs is a good predictor of improved response to ICIs and that using corticosteroids to treat these events does not impair treatment efficacy.[18]

Renal cancer

Lefort et al. studied patients treated with corticosteroids and nivolumab to determine whether corticosteroid use during immunotherapy influences outcomes in patients with metastatic clear cell renal cell carcinoma (mccRCC).[19] Among 665 individuals enrolled, 113 (17%) received corticosteroids during treatment, primarily to manage irAEs.[19] The median starting dose of corticosteroid administration was 60 mg prednisone equivalent (interquartile range: 40–80 mg), and it started 21.6 weeks after the start of nivolumab. In a 12-week landmark analysis, corticosteroid exposure was associated with significantly improved 12-month PFS (61.1% versus 41.6%; HR=0.63 [95% CI: 0.45, 0.88; p=0.007]) and OS (85.6% versus 73.5%; HR=0.57 [95% CI: 0.40, 0.81; p=0.002]). The OS advantage remained at 24 weeks, even though the PFS benefit was not maintained (HR=0.61 [95% CI: 0.40, 0.94; p=0.02]).[19] Multivariable analysis adjusting for clinical confounders confirmed that corticosteroid use did not negatively impact survival. These results imply that corticosteroids may not impair, and may even be linked with improved, survival in patients with mccRCC receiving nivolumab when used primarily to treat irAEs.

Gastrointestinal cancer

A study by Shen et al. examined whether corticosteroid premedication impacts dual immune checkpoint blockade's (dICB) efficacy in hepatocellular carcinoma (HCC) mouse models.[20] The researchers administered anti-CTLA-4 and anti-PD-1 antibodies following either low (10 µg) or high (200 µg) doses of dexamethasone using an orthotopic BNL 1MEA.7R.1 HCC model in Bagg albino laboratory-bred/c mice and a subcutaneous Hepa1-6 hepatoma model in C57 black 6 mice. Dexamethasone was found to have no significant impact on the antitumour effects of dual dICB in either the orthotopic or subcutaneous tumour model.[20] Single-cell RNA sequencing of tumour-infiltrating lymphocytes revealed that corticosteroid premedication did not affect the dICB-induced depletion of exhausted CD4+ or CD8+ T cells. Dexamethasone reduced the growth of effector memory CD8+ T cells. However, it did not affect lymph node T-cell priming, IFN-γ production or the cytotoxic function of tumour-infiltrating CD8+ T cells.[20] These results suggest that even when given in high doses, corticosteroids do not seem to interfere with how well dual dICB works in HCC. This means it might be possible to use steroids to manage or prevent irAEs without taking away from the benefits of the treatment.[20]

Higher peak doses of corticosteroids used to treat irAEs, however, were associated with worse PFS and OS across several cancer types, including gastrointestinal cancers, according to Verheijden et al.[12] Although corticosteroids are useful in treating irAEs, overusing them might interfere with how well ICIs work by distrupting the tumour environment and suppressing the immune response.

Management of corticosteroid dosage and timing

A careful, individualized approach is required when administering corticosteroids to patients receiving ICIs. The advantages of immunotherapy may occasionally be hampered by the use of steroids, even though they are frequently required to treat irAEs. According to Schneider et al.'s 2021 American Society of Clinical Oncology (ASCO) guidelines, the goal is to control side effects without compromising the treatment's effectiveness.[21]

There are a few key things to keep in mind. Timing matters – starting steroids too early, especially within the first month of ICI treatment, has been linked to worse outcomes.[22] When it is safe, delaying steroid use can help preserver the benefits of immunotherapy.[22] How long and how much you use also matters – longer courses or higher doses have been associated with lower survival rates. Therefore, it is usually recommended to use the lowest effective dose for the shortest duration necessary to get symptoms under control.[12,22,23]

Equally important is limiting the adverse effects of steroids. Long-term steroid use can lead to complications, especially in people with diabetes or weakened immune systems. For this reason, it is important to taper the dosage gradually when feasible and take infection prevention measures.[21] Since irAEs can affect different organs, involving a team of specialists often gives patients the safest, most personalized care. Ultimately, clinical judgement, collaboration and careful consideration of patient needs are needed to maintain ICI efficacy while managing adverse events.

Discussion

The clinical use of corticosteroids in patients receiving ICIs presents a nuanced challenge. Corticosteroids are the cornerstone of managing irAEs; on the other hand, they carry the potential to dampen the antitumour effects of immunotherapy. According to the ASCO guidelines, grade 3 irAEs should be managed with high-dose corticosteroids (prednisone 1–2 mg/kg/day or equivalent), followed by a taper over at least 4–6 weeks. If symptoms fail to resolve within 48–72 hours, additional immunosuppressive agents, such as infliximab, may be warranted.[21] The guidelines emphasize the importance of using the lowest effective dose for the shortest possible duration to minimize adverse steroid-related outcomes, especially in patients with diabetes, immunosuppression or other comorbidities.[21]

Multiple studies have examined how the timing and dose of corticosteroids influence ICI effectiveness. Albarrán et al. analysed data from 475 patients with advanced solid tumours.[23] They found that patients who received corticosteroids within 30 days before initiating ICIs had significantly lower RRs (20.3%) than those who did not (36.7%). A similar reduction in efficacy was observed in patients who received corticosteroids during the first 90 days of treatment. Notably, both higher cumulative exposure and even low-dose corticosteroid use were associated with poorer disease control. However, corticosteroid administration beyond 6 months of ICI initiation did not affect outcomes negatively, highlighting a critical time-dependent vulnerability window.[23]

Byron et al. conducted a systematic review that further supported these findings.[24] Across eight retrospective studies, mostly involving melanoma and NSLC, early corticosteroid use, particularly at doses ≥10 mg prednisone equivalent, was linked to reduced OS, shorter PFS and lower RR. Notably, the indication for corticosteroid use appeared to influence its impact.[24] When used for cancer-related symptoms or brain metastases, steroids were more likely to interfere with treatment efficacy. Conversely, when administered for irAE management, the detrimental effect seemed less pronounced, though not absent.[24]

These concerns extend to other immunotherapies as well. The National Comprehensive Cancer Network (NCCN) guidelines address corticosteroid use in the context of chimeric antigen receptor (CAR) T-cell therapy, particularly for cytokine release syndrome.[25] Despite concerns that corticosteroids may impair CAR T-cell expansion, they remain a key therapeutic option. Once symptoms improve, early corticosteroid tapering is recommended to minimize immunosuppressive effects.[25] These results show the importance of timing, dosage and indication when corticosteroids are used to determine the effectiveness of immunotherapy. While corticosteroids remain essential for managing irAEs, their use outside that context, especially early in treatment, should be approached with caution.[23–25] Personalized corticosteroid management plans that balance symptom control with the preservation of immunotherapeutic efficacy are essential. Close coordination among oncology care teams is crucial to tailor steroid use appropriately and continually reassess its necessity throughout treatment.

Conclusion

This study sheds light on the complex, and still evolving, understanding of how corticosteroids affect irAEs during treatment with ICIs. Steroids remain the standard approach for managing moderate to severe irAEs, but there is a genuine concern that their immunosuppressing effects could interfere with the effectiveness of immunotherapy. Retrospective research has so far yielded conflicting results; some indicate that high-dose or early steroid use may be associated with poorer cancer outcomes, while others show little to no effect, mainly when steroids are used reactively rather than prophylactically. One of the most significant obstacles is the great diversity in study designs, cancer types, steroid timing, dosage and outcome measures, all of which make it challenging to reach definitive conclusions. Additionally, there is a dearth of prospective data and a lack of consensus regarding the best way to taper steroids or when to look at alternatives. This highlights how important it is to practice steroid stewardship, striking the right balance between reducing adverse events and maintaining immunotherapy's capacity to combat cancer.

Future research should focus on finding the safest and most effective ways to use steroids, paying close attention to the correct dose, timing and duration; so, we can manage irAEs without getting in the way of treatment. For patients who need both immunotherapy and steroids it is essential to understand the long-term impact, not only just on how long they live but also on how they feel every day and whether the treatment continues to work overtime. More research is needed to create more functional, individualized guidelines that assist physicians in using steroids in cancer treatment more safely and effectively by following patients over an extended period and learning from actual experiences. ❑

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