Clinical Therapeutics 46 (2024) e59–e63

Contents lists available at ScienceDirect

Clinical Therapeutics

journal homepage: www.elsevier.com/locate/clinthera

Original Research

The effect of different types of oral or intravenous corticosteroids on capillary blood glucose levels in hospitalized inpatients with and without diabetes

Vaishali Limbachia, MBBS[1,2], Ian Nunney, MSc[2], Daniel J. Page[2], Hannah A. Barton[2], Leena K. Patel, MBChB[1], Georgia N. Thomason[2], Stephan L. Green, BSc[2], Kieran F.J. Lewis, BSc[1], Ketan Dhatariya, MBBS, MSc, MD, MS, FRCP, PhD[1,2,3,*]

[1] *Department of Medicine, Norfolk and Norwich University Hospitals National Health Service Foundation Trust, Norwich, United Kingdom*
[2] *Norwich Medical School, University of East Anglia, Norwich, United Kingdom*
[3] *Elsie Bertram Diabetes Centre–Norfolk and Norwich University Hospitals National Health Service Foundation Trust, Norwich, United Kingdom*

ARTICLE INFO

Key words:
Blood glucose
Corticosteroids
Diabetes
Hospitalised
Hyperglycemia
Inpatient

ABSTRACT

Purpose: This study investigated: (1) the type of corticosteroid associated with the greatest degree of hyperglycemia, assessed using bedside capillary blood glucose monitoring, in hospitalized patients; and (2) the pattern of hyperglycemia throughout the day with the use of each type of corticosteroid.

Methods: This single-center, retrospective study used data from 964 adult inpatients receiving oral or IV corticosteroids. Data on capillary blood glucose concentrations and time taken over 7 days were collected. A mixed model for repeated measures was applied to investigate changes in glucose concentration over time with the use of four different corticosteroids. An autoregressive covariance structure was used to model correlations between repeated measurements.

Findings: Across all 7 days, the mean blood glucose concentration was greater with dexamethasone compared to that with hydrocortisone (mean difference, 16.6 mg/dL [95% CI, 8.1–24.8] [0.92 mmol/L (95% CI, 0.45–1.38)]) or prednisolone (mean difference, 20.0 mg/dL [95% CI, 14.2–25.7] [1.11 mmol/L (95% CI, 0.79–1.43)]). The mean blood glucose concentration was greater with methylprednisolone compared to that with hydrocortisone (mean difference, 23.9 mg/dL [95% CI, 11.3–36.4] [1.33 mmol/L (95% CI, 0.63–2.02)]), and with methylprednisolone versus prednisolone (mean difference, 27.4 mg/dL [95% CI, 16.4–38.3] [1.52 mmol/L (95% CI, 0.91–2.13)]). There were no significant differences in the patterns of hyperglycemia at six time points of the day with each type of corticosteroid.

Implications: Treatment with oral or IV dexamethasone or methylprednisolone was associated with greater hyperglycemia in comparison to prednisolone and hydrocortisone. More vigorous monitoring and intervention, when necessary, are suggested in adult inpatients receiving corticosteroids, in particular dexamethasone and methylprednisolone.

Introduction

Corticosteroids (also called *steroids* or *glucocorticoids*) are anti-inflammatory medications used in a variety of specialties, including (but not limited to) respiratory medicine, dermatology, endocrinology, hematology, rheumatology, gastroenterology, ophthalmology, and transplant medicine.[1,2] It has been reported that about 0.7% of all adults are receiving corticosteroids at any given time, with up to 1.8% being long-term users, and 2.5% being aged ≥75 years.[3,4] In one report, 12.8% of hospitalized patients were receiving steroids.[5] Based on the findings from the RECOVERY (Dexamethasone in Hospitalized Patients with COVID-19) trial,[6,7] corticosteroids are now also indicated for use in the management of patients with COVID-19 requiring supplemental oxygen.

Corticosteroid use has been associated with numerous side effects, including steroid-induced hyperglycemia and steroid-worsened diabetes.[8] These events are thought to occur through increasing insulin resistance and decreasing production and secretion of insulin.[9–11] The effects of corticosteroid use on glucose concentration may vary depending on the type of corticosteroid; for example, hyperglycemia

* Address correspondence to: Prof. Ketan Dhatariya, Elsie Bertram Diabetes Centre, Norfolk and Norwich University Hospitals NHS Foundation Trust, Norwich, Norfolk, UK NR4 7UY.
E-mail address: ketan.dhatariya@nnuh.nhs.uk (K. Dhatariya).

https://doi.org/10.1016/j.clinthera.2023.11.013
Accepted 16 November 2023

may develop a few hours after prednisolone is taken and may then wear off, whereas more prolonged hyperglycemia may occur with the use of dexamethasone, which has a longer half-life.[10]

Hyperglycemia is known to increase the risks for morbidity and mortality in patients admitted to the hospital, including those with newly diagnosed hyperglycemia.[12,13] Currently, evidence suggests that corticosteroid-induced hyperglycemia and diabetes are dose-dependent effects.[14–17] The development of corticosteroid-induced hyperglycemia and diabetes may be dependent on the duration of the treatment course.[16] In contrast, lower doses used for longer durations may be associated with hyperglycemia, suggesting that the effect may be dose-cumulative.[18] The Joint British Diabetes Societies for Inpatient Care (JBDS) guideline recommends capillary blood glucose monitoring in all adult inpatients receiving oral or IV steroids to screen for and manage hyperglycemia.[8]

Few studies have compared the effects of individual corticosteroids on the risk for hyperglycemia, and the pattern of hyperglycemia throughout the day, in hospitalized patients. The risk for hyperglycemia is important in situations in which those at risk for steroid-induced hyperglycemia or diabetes could be monitored at specific times of the day to ensure the rise in glucose is not missed and/or that steroids with a lower risk for hyperglycemia could be prescribed.

A single-center, retrospective study of data from adult inpatients on oral or IV steroids was conducted to investigate the following: Is any particular type of corticosteroid associated with a greater degree of hyperglycemia, assessed by bedside capillary blood glucose monitoring?; and, Is there a pattern of hyperglycemia at each point of the day with each type of corticosteroid?

Materials and Methods

This retrospective study was conducted at Norfolk and Norwich University Hospitals National Health Service (NHS) Foundation Trust (Norwich, UK). Data from February 10, 2021 (the start of recording of point-of-care capillary blood glucose measurements in the hospitals' electronic pathology system), and September 10, 2021, were collected. Using an electronic medicine-prescribing and -administration system, all adult inpatients receiving an oral or IV steroid during this period were identified. Data on patients who were in-hospital on an adult ward and receiving an oral or IV corticosteroid were included. The following data were collected: age and sex; pre-admission diabetes status; pre-admission corticosteroid use status; type, dose, and route of administration of corticosteroid on admission; capillary blood glucose concentrations; and times of glucose measurements. Data from the first corticosteroid administration until the end of the corticosteroid course, up to 7 consecutive days, were collected. Because the JBDS guideline recommends maintaining a glucose concentration of 108 to 216 mg/dL (6.0–12.0 mmol/L) in inpatients, *hyperglycemia* was defined as two readings of >216 mg/dL (>12.0 mmol/L) in 24 hours.

Patients aged <18 years, outpatients, those in the emergency department, and those on corticosteroids that were not administered orally or IV were excluded from the study.

This study was registered with the Clinical Audit and Improvement Department of the Norfolk and Norwich University Hospitals NHS Foundation Trust (registration DIAB_TW_23-24_P04). This study was considered a service-improvement exercise, and the project did not require approval for ethics or research governance.

Statistical Analysis

Descriptive statistics are presented as absolute numbers (%), means (SD), or medians (interquartile range; IQR). A mixed model for repeated measures was applied to investigate changes in glucose concentration over time, comparing four different steroids. An auto-regressive covariance structure was used to model correlations between repeated measurements. The repeated-measures model was adjusted for age, sex, and pre-admission diabetes and/or steroid use status. The effects of the four steroids were compared using data from day 1, day 7, and across all 7 days. To ensure similar intervals between timepoints, the mean of the glucose concentrations measured every 4 hours was calculated, resulting in up to six glucose measurements, and, over the 7 days, up to 42 glucose measurements. Results are presented with 95% CIs and P values, with $P < 0.05$ deemed to be statistically significant. Data from days with no recorded capillary blood glucose concentrations were excluded from the analysis.

Results

During the data collection period, 3260 inpatients received oral or IV corticosteroids (range of capillary blood glucose readings per day, 1–24). Data from 964 patients with available day-1 blood glucose measurements were included in the analysis. The distribution of corticosteroid types on day 1 of administration were: prednisolone, 55.3% ($n = 533$); dexamethasone, 28.0% ($n = 270$); hydrocortisone, 11.9% ($n = 115$); and methylprednisolone, 4.8% ($n = 46$). The median (IQR) total daily doses of the corticosteroids administered over the course of the 7 days were: prednisolone, 19.3 mg (8.0–30.0); dexamethasone, 6.9 mg (6.0–12.0); hydrocortisone, 48.0 mg (28.6–100.0); and methylprednisolone, 80.0 mg (68.6–500). A summary of baseline characteristics of the 964 inpatients can be found in Table 1.

Of the patients who underwent blood glucose monitoring on day 1 of taking corticosteroids, 36.0% ($n = 347$) were identified as having hyperglycemia. Of those, 48.7% ($n = 169$) had hyperglycemia on day 1, and in 51.3% (n = 178), hyperglycemia developed after day 1.

On comparison of capillary blood glucose concentrations between those who were new to corticosteroids and those who were taking corticosteroids before admission, mean blood glucose concentrations on day 1 were similar (both, 167 mg/dL [9.3 mmol/L]; $P = 0.72$) and the difference in mean blood glucose levels on day 7 was not statistically significant (176 vs 187 mg/dL [9.8 vs 10.4 mmol/L]; $P = 0.24$).

The mean glucose concentration on day 1 was significantly greater in the subgroup with a preexisting diagnosis of diabetes compared to those who did not (194 vs 140 mg/dL [10.8 vs 7.8 mmol/L]; mean difference, 52 mg/dL [95% CI, 45–61] [2.9 mmol/L (95% CI, 2.5–3.4)]; $P < 0.0001$). The mean glucose concentration on day 7 was significantly greater in the subgroup with a preexisting diagnosis of diabetes compared to those who did not (207 vs 157 mg/dL [11.5 vs 8.7 mmol/L]; mean difference, 52 mg/dL [95% CI, 36–67] [2.9 mmol/L (95% CI, 2.0–3.7)]; $P < 0.0001$). The blood glucose concentrations in the subgroup with pre-existing diabetes compared to the subgroup without are shown in Figure 1.

The differences in day-1 glucose concentrations between the different types of corticosteroids were not statistically significant. The difference in glucose concentrations on day 7 was statistically significant between those on dexamethasone compared to those on prednisolone (difference, 28.8 mg/dL [95% CI, 9.0–48.6] [1.57 mmol/L (95% CI, 0.46–2.67)]; $P = 0.0349$). Table 2 shows, across all 7 days, the persistence of the statistical significance of the differences in glucose concentrations between dexamethasone and hydrocortisone ($P = 0.0007$), dexamethasone and prednisolone ($P < 0.0001$), hydrocortisone and methylprednisolone ($P = 0.0011$), and methylprednisolone and prednisolone ($P < 0.0001$). Those on dexamethasone and methylprednisolone had the highest mean blood glucose concentrations. The mean blood glucose levels across all 7 days with the use of the steroids were: methylprednisolone, 194 mg/dL (10.8 mmol/L); hydrocortisone, 171 mg/dl (9.5 mmol/L), dexamethasone, 187 mg/dL (10.4 mmol/L); and prednisolone, 167 mg/dL (9.3 mmol/L).

The pattern of glucose variability across each 24-hour period showed a periodic variation (Figure 1). The mean blood glucose concentrations were lowest between 4 and 8 AM. The mean blood glucose concentrations over the 7 days were highest between 4 and 8 PM, except on days 2 and 6 in the subgroup without a pre-existing diagnosis of diabetes.

Table 1
Baseline characteristics of the adult inpatients on corticosteroids who had blood glucose monitoring on day 1. Data are given as number (%) of patients unless otherwise specified.

Characteristic	Hydrocortisone (n = 115)	Prednisolone (n = 533)	Dexamethasone (n = 270)	Methylprednisolone (n = 46)	Overall (n = 964)
Age, mean (SD), y	66.7 (18.4)	73.6 (14.7)	68.1 (15.4)	59.4 (19.5)	70.5 (16.1)
Sex					
Male	60 (52.2)	259 (48.6)	148 (54.8)	19 (41.3)	486 (50.4)
Female	55 (47.8)	274 (51.4)	122 (45.2)	27 (58.7)	478 (49.6)
Previous diagnosis of diabetes	52 (45.2)	235 (44.1)	90 (33.3)	11 (23.9)	388 (40.2)
Steroids before admission	54 (47.0)	307 (57.6)	54 (20.0)	3 (6.5)	418 (43.4)

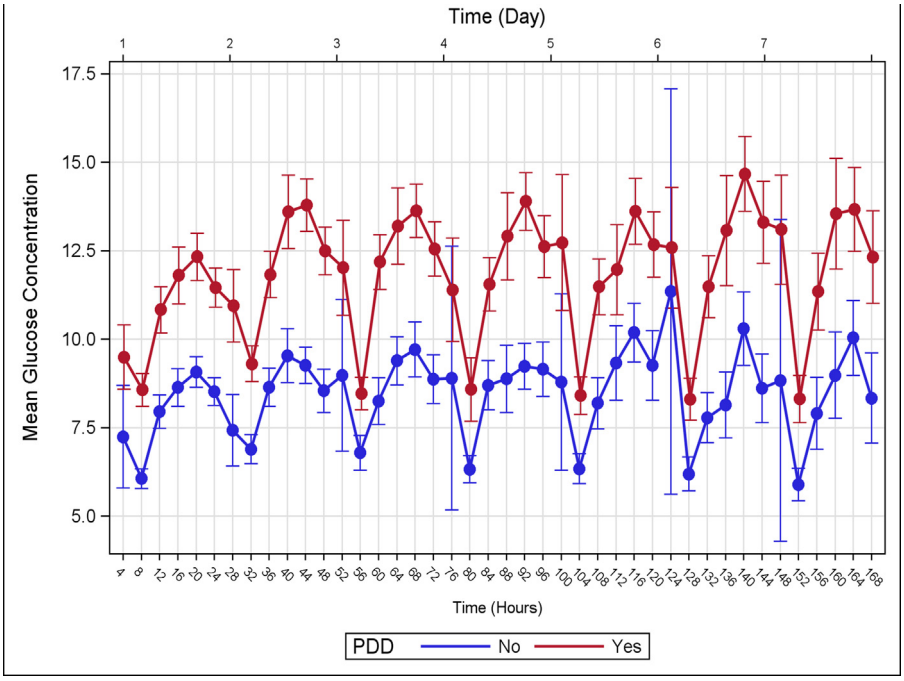

Figure 1. Mean (95% CI) blood glucose concentrations (in mmol/L), as measured q4h over 7 days in patients with and without a preexisting diagnosis of diabetes (PDD).

Table 2
Mean differences in blood glucose concentrations (in mmol/l) of different steroid classes with 95% confidence interval on day 1, day 7, and all seven days. In bold are the statistically significant differences.

Study Drugs	Day 1			Day 7			All 7 Days		
	Mean Difference, mmol/L	95% CI	P	Mean Difference, mmol/L	95% CI	P	Mean Difference, mmol/L	95% CI	P
Dexamethasone									
vs hydrocortisone	0.49	–0.26 to 1.25	0.7348	1.63	0.08 to 3.18	0.2211	0.92	0.45 to 1.38	0.0007
vs prednisolone	0.23	–0.31 to 0.76	0.9565	1.57	0.46 to 2.67	0.0349	1.11	0.79 to 1.43	<0.0001
vs methylprednisolone	–0.55	–1.66 to 0.55	0.9064	0.11	–1.7 to 1.93	1.0000	–0.41	–1.02 to 0.21	0.7216
Hydrocortisone									
vs prednisolone	–0.27	–0.95 to 0.41	0.9697	–0.06	–1.39 to 1.27	1.0000	0.20	–0.22 to 0.61	0.9283
vs methylprednisolone	–1.05	–2.26 to 0.16	0.4285	–1.51	–3.66 to 0.63	0.6634	–1.33	–2.02 to –0.63	0.0011
Methylprednisolone vs prednisolone	0.78	–0.32 to 1.88	0.6562	1.45	–0.4 to 3.31	0.5501	1.52	0.91 to 2.13	<0.0001

The CIs of greatest mean blood glucose levels overlapped at all time points on each day, except between 4 and 8 AM. Figure 2 shows the mean blood glucose concentrations at each 4-hour interval on days 1 and 7 with each corticosteroid class. The differences in concentrations between corticosteroid classes at different points of the day were not significant.

Discussion

In the present study, bedside point-of-care capillary blood glucose concentrations rose significantly in adult inpatients receiving oral or IV steroids during the first week after initiation. Increases in glucose concentrations were greatest in those receiving dexamethasone or methylprednisolone. The increase in glucose was greater in patients with preexisting diabetes than in those without it.

Uncontrolled high blood glucose concentrations in hospitalized patients have been associated with increased harm, however that is measured.[19] Conditions associated with hyperglycemia include (but are not limited to) immunosuppression, oxidative stress, increased risk for thrombosis, and the precipitation of ischemic events.[20,21] These conditions can lead to increased morbidity or mortality, and increased overall hospital care–related costs in patients with hyperglycemia, with or

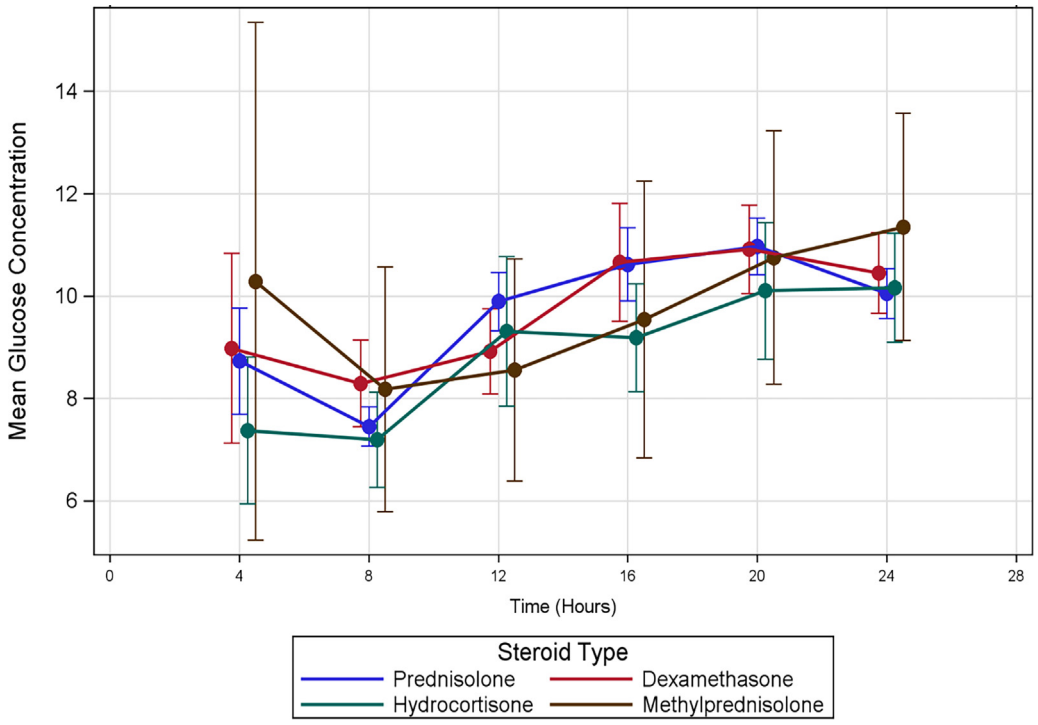

Figure 2. Mean (95% CI) blood glucose concentrations (in mmol/L), as measured q4h on day 1 (*A*) and day 7 (*B*) of treatment with corticosteroids in patients with diabetes.

without a preexisting diagnosis of diabetes.[21–23] In a study by Umpierrez et al,[13] those with newly diagnosed hyperglycemia had a longer length of hospital stay, were more likely to need admission to the intensive care unit, and were less likely to return to their baseline premorbid state. The importance of controlling glucose concentrations in inpatients has been discussed elsewhere.[19,24]

The evidence that corticosteroid use leads to hyperglycemia is compelling.[25,26] Inpatient corticosteroid use has increased since the COVID-19 pandemic.[7,27] However, the RECOVERY trialists have yet to publish data on the impact of dexamethasone on glucose concentrations. To the best of our knowledge, no work has assessed the effects of corticosteroids with more potent anti-inflammatory activity on the differential hyperglycemic effect in inpatients. In the present study, hyperglycemia was more prevalent in patients receiving dexamethasone or methylprednisolone as opposed to prednisolone or hydrocortisone. This finding most likely was attributable to the strength and half-life of the corticosteroid.[10] With less potent corticosteroids, such as hydrocortisone, the half-life is shorter (eg, 8 hours), in comparison to that of dexamethasone (36–54 hours),[8] suggesting that dexamethasone has a longer-lasting, greater anti-inflammatory effect; however, when administered acutely, its use may increase endogenous hepatic gluconeogenesis and reduce peripheral glucose uptake and insulin secretion.[10]

Currently, guidelines and recommendations on monitoring capillary blood glucose do not differentiate by corticosteroid type, despite differences in the potency and formulation of each type.[8] The findings from the present study can help to refine current guidelines in that they suggest a benefit of more vigorous blood glucose monitoring in patients receiving dexamethasone or methylprednisolone, especially if the treatment duration is prolonged. In the present study, the differences in blood glucose concentrations at various time points across the week were not significant between types of corticosteroid. Therefore, blood glucose monitoring is recommend regardless of the corticosteroid type, as per the guideline produced by the JBDS: "Prior to lunch or evening meal, or alternatively 1–2 hours post lunch or evening meal."[8] The present findings suggest that capillary glucose need not be monitored routinely between 4 and 8 AM.

The strengths of the present study included that the comprehensive database included all adult inpatients administered a corticosteroid for at least a week over the 7-month data-collection period. A sample size analysis was not done; however, the sample size was sufficient for the detection of an association between types of corticosteroid and blood glucose concentrations. Future work will look at the impact of various glucose-lowering agents on the management of patients with corticosteroid-induced hyperglycemia.

Limitations of the present single-center, retrospective study included that the study did not differentiate between hyperglycemia from preexisting, suboptimally controlled diabetes and new-onset, steroid-induced hyperglycemia. Because only data on the total daily dose on each day of corticosteroid treatment were collected, it could not be differentiated whether hyperglycemia occurred with once-daily or multiple daily doses of steroids. Because data up to only day 7 of corticosteroid use in inpatients were collected, the impact of a longer treatment course could not be determined. Despite there being national guidelines on the management of patients with steroid-induced hyperglycemia or steroid-worsened diabetes,[8] because glucose concentrations were suboptimal, the guidelines may not have been followed. Data on ketones were not collected, as they are not electronically recorded at the institution. The findings do not take into account the agents used for managing patients with hyperglycemia. It could not be determined whether those on the corticosteroids associated with the lowest increase in glucose concentrations were treated more aggressively with glucose-lowering agents. The demographic of the population served by the hospital is predominantly White; thus, differences potentially related race or ethnicity could not be determined.

Conclusions

In this retrospective study of the types of corticosteroid that lead to greater degrees of hyperglycemia, over a 7-day period, dexamethasone or methylprednisolone use was associated with greater hyperglycemia in comparison to prednisolone or hydrocortisone. These findings could help to raise awareness about which steroids require more vigorous glu-

cose monitoring. In agreement with the UK guideline on managing patients with corticosteroid-induced hyperglycemia or worsened diabetes, routine blood glucose monitoring before lunch or the evening meal, or 1 to 2 hours after lunch or the evening meal, in hospitalized patients on oral or IV corticosteroids is still recommended. Mean capillary blood glucose levels in patients receiving any steroid were much greater in those with a preexisting diagnosis of diabetes. Therefore, in patients with a preexisting diagnosis of diabetes, testing four times a day is recommended.

Declaration of Competing Interest

The authors have indicated that they have no conflicts of interest with regard to the content of this article.

Author Contributions

K.D. and V.L. were involved in the conception and design of the study. V.L., D.J.P., H.A.B., L.K.P., G.N.T., S.L.G., and K.F.J.L. were involved in the data collection the study. K.D., V.L., and I.N. were involved in the analysis and interpretation of the results. V.L. wrote the first draft of the manuscript. All of the authors edited, reviewed, and approved the final version of the manuscript.

K.D. is the guarantor of this work and, as such, had full access to all of the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

References

1. Coutinho AE, Chapman KE. The anti-inflammatory and immunosuppressive effects of glucocorticoids, recent developments and mechanistic insights. *Molecular and Cellular Endocrinology*. 2011;335:2–13.
2. Hodgens A, Sharman T. Corticosteroids [StatPearls online]. Available at: https://www.ncbi.nlm.nih.gov/books/NBK554612/. Accessed November 6, 2023.
3. Fardet L, Petersen I, Nazareth I. Prevalence of long-term oral glucocorticoid prescriptions in the UK over the past 20 years. *Rheumatology*. 2011;50:1982–1990.
4. Bénard-Laribière A, Pariente A, Pambrun E, Bégaud B, Fardet L, Noize P. Prevalence and prescription patterns of oral glucocorticoids in adults: a retrospective cross-sectional and cohort analysis in France. *BMJ Open*. 2017;7:e015905.
5. Narwani V, Swafe L, Stavraka C, Dhatariya K. How frequently are bedside glucose levels measured in hospital inpatients on glucocorticoids? *Clinical Medicine*. 2014;14:327–328.
6. World Health Organization. Corticosteroids for COVID-19. 2020. Available at: https://www.who.int/publications/i/item/WHO-2019-nCoV-Corticosteroids-2020.1. Accessed November 6, 2023.
7. The Recovery GroupDexamethasone in hospitalized patients with Covid-19—preliminary report. *New England Journal of Medicine*. 2021;384:693–704.
8. Roberts A, James J, Dhatariya K. on behalf of the Joint British Diabetes Societies for Inpatient Care group. Management of hyperglycaemia and steroid (glucocorticoid) therapy: a guideline from the Joint British Diabetes Societies (JBDS) for Inpatient Care group. *Diabetic Medicine*. 2018;35:1011–1017.
9. Kwon S, Hermayer KL. Glucocorticoid-induced hyperglycemia. *American Journal of the Medical Sciences*. 2013;345:274–277.
10. Perez A, Jansen-Chaparro S, Saigi I, Bernal-Lopez MR, Miñambres I. Gomez-Huelgas R. Glucocorticoid-induced hyperglycemia. *Journal of Diabetes*. 2014;6:9–20.
11. Geer EB, Islam J, Buettner C. Mechanisms of glucocorticoid-induced insulin resistance. *Endocrinology & Metabolism Clinics of North America*. 2014;43:75–102.
12. Golubic R, Caleyachetty R, Barber TM, Adler A. Glucocorticoid-induced hyperglycaemia and diabetes: call for action. *Diabetic Medicine*. 2022;39:e14843.
13. Umpierrez GE, Isaacs SD, Bazargan N, You X, Thaler LM, Kitabchi AE. Hyperglycemia: An independent marker of in-hospital mortality in patients with undiagnosed diabetes. *Journal of Clinical Endocrinology Metabolism*. 2002;87:978–982.
14. Gurwitz JH, Bohn RL, Glynn RJ, Monane M, Mogun H, Avorn J. Glucocorticoids and the risk for initiation of hypoglycemic therapy. *Archives of Internal Medicine*. 1994;154:97–101.
15. Suh S, Park MK. Glucocorticoid-induced diabetes mellitus: an important but overlooked problem. *Endocrinology and Metabolism (Seoul)*. 2017;32:180–189.
16. Ambery P, Adamsson Eryd S, Jenkins-Jones S, et al. The absolute risk of incident type 2 diabetes following exposure to systemic corticosteroids in selected steroid-related and phenotypic groups. *Diabetes, Obesity and Metabolism*. 2022;24:2222–2231.
17. Wu J, Mackie SL, Pujades-Rodriguez M. Glucocorticoid dose-dependent risk of type 2 diabetes in six immune-mediated inflammatory diseases: a population-based cohort analysis. *BMJ Open Diabetes Research & Care*. 2020;8:e001220.
18. Dobravc Verbič M, Gruban J, Kerec Kos M. Incidence and control of steroid-induced hyperglycaemia in hospitalised patients at a tertiary care centre for lung diseases. *Pharmacological Reports*. 2021;73:796–805.
19. Pasquel FJ, Lansang MC, Dhatariya K, Umpierrez GE. Management of diabetes and hyperglycaemia in the hospital. *Lancet Diabetes & Endocrinology*. 2021;9:174–188.
20. Chaudhuri A, Umpierrez GE. Oxidative stress and inflammation in hyperglycemic crises and resolution with insulin: implications for the acute and chronic complications of hyperglycemia. *Journal of Diabetes and its Complications*. 2012;26:257–258.
21. Clement S, Braithwaite SS, Magee MF, et al. Management of diabetes and hyperglycemia in hospitals. *Diabetes Care*. 2004;27:553–597.
22. Zhu L, She Z-G, Cheng X, et al. Association of blood glucose control and outcomes in patients with COVID-19 and pre-existing type 2 diabetes. *Cell Metabolism*. 2020;31:1068–1077 e1063.
23. Kerr M. Inpatient Care for People with Diabetes: the Economic Case for Change. 2013. Available at: http://webarchive.nationalarchives.gov.uk/20130513172211/http://www.diabetes.nhs.uk/document.php?o=3034. Accessed November 6, 2023.
24. Dhatariya K, Mustafa OG, Rayman G. Safe care for people with diabetes in hospital. *Clinical Medicine*. 2020;20:21–27.
25. Breakey S, Sharp SJ, Adler AI, Challis BG. Glucocorticoid-induced hyperglycaemia in respiratory disease: a systematic review and meta-analysis. *Diabetes, Obesity and Metabolism*. 2016;18:1274–1278.
26. Walters JA, Tan DJ, White CJ, Gibson PG, Wood-Baker R, Walters EH. Systemic corticosteroids for acute exacerbations of chronic obstructive pulmonary disease. [Cochrane Database of Systematic Reviews online]. 2014. Available at: http://onlinelibrary.wiley.com/doi/10.1002/14651858.CD001288.pub4/full#pdf-section. Accessed November 6, 2023.
27. Rhou YJ, Hor A, Wang M, et al. Dexamethasone-induced hyperglycaemia in COVID-19: glycaemic profile in patients without diabetes and factors associated with hyperglycaemia. *Diabetes Research and Clinical Practice*. 2023;194:110151.