Perioperative Corticosteroid Management for Patients with Inflammatory Bowel Disease

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Abstract: Guidelines on the appropriate use of perioperative steroids in patients with inflammatory bowel disease (IBD) are lacking. As a result, corticosteroid supplementation during and after colorectal surgery procedures has been shown to be highly variable. A clearer understanding of the indications for perioperative corticosteroid administration relative to preoperative corticosteroid dosing and duration of therapy is essential. In this review, we outline the basic tenets of the hypothalamic–pituitary–adrenal (HPA) axis and its normal response to stress, describe how corticosteroid use is thought to affect this system, and provide an overview of the currently available data on perioperative corticosteroid supplementation including the limited evidence pertaining to patients with inflammatory bowel disease. Based on currently existing data, we define "adrenal suppression," and propose a patient-based approach to perioperative corticosteroid management in the inflammatory bowel disease population based on an individual's historical use of corticosteroids, the type of surgery they are undergoing, and HPA axis testing when applicable. Patients without adrenal suppression (<5 mg prednisone per day) do not require extra corticosteroid supplementation in the perioperative period; patients with adrenal suppression (>20 mg prednisone per day) should be treated with additional perioperative corticosteroid coverage above their baseline home regimen; and patients with unclear HPA axis function (>5 and <20 mg prednisone per day) should undergo preoperative HPA axis testing to determine the best management practices. The proposed management algorithm attempts to balance the risks of adrenal insufficiency and immunosuppression.

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Key Words: adrenal insufficiency, corticosteroids, hypothalamic–pituitary–adrenal axis, inflammatory bowel disease, perioperative

Many patients with autoimmune diseases and chronic inflammatory processes, including the inflammatory bowel diseases (IBD), are routinely on corticosteroids at the time of or just before undergoing intestinal surgery. Administering "stress-dose" corticosteroids to patients, even with limited exposure, has become routine perioperative practice at many institutions.[1–5] Despite the ubiquity of this practice, there are actually very little data to support the use of perioperative corticosteroid supplementation for these patients. The historical context from which this practice emerged dates back to the 1950s with the publication of 2 case reports of fatalities presumably related to adrenal crises among corticosteroid-dependent patients who did not receive corticosteroid supplementation postoperatively.[6,7] Since then, additional case reports, primarily from the 1950s to the 1960s, reported postoperative fatalities attributed to adrenal insufficiency among steroid-treated patients, the majority of whom did not receive supplemental corticosteroids perioperatively. Although adrenal insufficiency was suspected because of postoperative hypotension, the majority of the associations were inconclusive with only 3 of the 57 reported cases fulfilling diagnostic criteria for adrenal insufficiency.[8]

There is a wide range of corticosteroid-prescribing patterns that are currently used in the perioperative period, but the few studies that have analyzed the utility of different dosing regimens show minimal differences in outcomes for patients receiving high-dose, low-dose, or no perioperative steroids.[1–5,9–19] Within patients with IBD, corticosteroid supplementation during and after colorectal surgery procedures has been shown to be highly variable, even within individual institutions.[20] However, it is well recognized that corticosteroids have a negative impact on wound healing, glycemic control, and infection that may increase the risk of postoperative morbidity.[21–23] These risks may be further potentiated if other variables associated with increased postoperative morbidity are present, such as aggressive disease behavior, malnutrition, advanced age, and concomitant use of other immunosuppressant drugs.[24] Given these potential risks, a clear understanding of the indications for perioperative corticosteroid administration relative to preoperative corticosteroid dosing and duration of therapy is essential.

In this review, we will outline the basic tenets of the hypothalamic–pituitary–adrenal (HPA) axis and its normal response to stress, describe how corticosteroid use is thought to affect this system, and provide an overview of the currently available data on perioperative corticosteroid supplementation including the limited evidence pertaining to patients with IBD. Based on this

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information, we propose an approach to perioperative corticosteroid management for patients with IBD balancing the risks of adrenal insufficiency and immunosuppression. With these recommendations, we aim to add to the paucity of literature regarding perioperative corticosteroid practice patterns and highlight the need for standardized protocols that ultimately may help reduce unnecessary corticosteroid supplementation and decrease risk of postoperative medical and surgical morbidity in patients with IBD.

SCOPE OF THE PROBLEM

The majority of patients with Crohn's disease will require at least 1 surgery during the natural history of their disease, usually due to stricturing or penetrating sequelae. Approximately, 30% to 40% of patients with ulcerative colitis will also require surgery most commonly for medically refractory disease, but dysplasia or fulminant colitis are also indications for colectomy.[25] Many patients with IBD will also require corticosteroids during the course of their disease, and approximately, 30% to 40% of patients with moderate to severe disease will become steroid-dependent.[26–28] Once corticosteroid therapy has commenced, approximately 20% to 30% of patients with ulcerative colitis and 35% to 38% of patients with Crohn's disease will require surgery within 1 year.[26,27] At the time of surgery, many patients with IBD will either have recently discontinued or remain on a range of steroid doses from low- (less than prednisone 5 mg or equivalent daily) to supraphysiologic (prednisone dosing greater than 10 mg daily) depending on disease severity and corticosteroid-tapering schedules.[29] Although the ideal scenario is to perform elective surgery on patients who have successfully discontinued corticosteroids for a prolonged period of time to minimize potential peri- and post-operative morbidity, the reality is that a sizeable percentage of patients with IBD will be on variable corticosteroid dosing at the time of surgery because of an inability to successfully discontinue therapy due to the expedited need for surgical intervention. Although the majority of patients with IBD undergoing elective surgery will be on the lower end of the dosing spectrum, the fact remains that prescribing practices regarding the most appropriate approach for preoperative corticosteroid management is variable with no consensus regimens to guide practitioners. Individual patient factors, physician preferences and experience, and disease activity each play a role, making the concept of a "standard" regimen difficult to achieve. Because of this variability, the perioperative response of any individual undergoing surgery for IBD is all the more complex.

THE HPA AXIS

Normal Function

The HPA axis is a hormonal system that responds to systemic stressors by stimulating the production and release of cortisol from the adrenal glands (Fig. 1). When the body encounters a stressor (e.g., surgery), the hypothalamus is activated to release corticotropin-releasing hormone. Corticotropin-releasing hormone acts on the anterior pituitary to stimulate the release of adrenocorticotropin hormone (ACTH), which in turn activates the adrenal glands to release cortisol into the blood stream. Cortisol acts to improve vascular tone, increase the availability of glucose in the blood, and retain intravascular volume. The HPA axis has a negative feedback system, such that increased circulating cortisol levels suppresses both corticotropin-releasing hormone release from the hypothalamus and ACTH release from the pituitary gland, thereby self-regulating the amount of cortisol released.[30]

Effects of Surgery

In unstressed individuals with normal adrenal function, the basal secretion of cortisol is approximately 5 to 7 mg/m^2 per day (8.5–12 mg/d in a patient with average body surface area).[31] During a minor surgery or illness, e.g., an examination under anesthesia or ileostomy reversal, cortisol secretion increases up to 5-fold to approximately 50 mg/d[32] (http://www.uptodate.com/contents/the-surgical-patient-taking-glucocorticoids/abstract/14). In patients undergoing a major surgical stress, e.g., a subtotal colectomy, the secretion of cortisol is even greater, increasing up to 75 to 100 mg/d. In rare instances, the cortisol secretion rate can reach between 200 and 500 mg/d, usually in the face of emergent surgery or severe trauma. In most instances, basal cortisol levels normalize by postoperative day 5.[33]

Adrenal Insufficiency

Adrenal insufficiency (AI) is characterized by a loss of normal adrenal function and is divided into primary and secondary forms based on the cause of dysfunction. Primary AI is caused by direct adrenal gland damage. In the developed world, the most common etiology of primary AI is autoimmune adrenalitis.[34] Secondary AI is characterized by reduced secretion of ACTH and is most commonly caused by the abrupt cessation of systemic corticosteroid supplementation.[34] In general, secondary AI only affects the secretion of cortisol and adrenal androgens, whereas in primary AI, there is also a deficit of mineralocorticoids (i.e., aldosterone). The minimum corticosteroid regimen that can cause secondary AI is currently unknown. Neither the dose nor the duration of treatment can accurately predict its development, although it is commonly believed that several weeks of systemic corticosteroid supplementation are required before adrenal gland function is suppressed.[35,36]

Both primary and secondary AI can develop acutely or more slowly over a period of weeks to months. Patients with acute AI may present with significant cardiovascular impairment that mimics septic shock, including elevated cardiac output and low systemic vascular resistance.[37] Acute AI can lead to serious consequences, including myocardial infarction and death. Chronic AI, which is more insidious, may present with fatigue, anorexia, nausea, weight loss, diarrhea, and/or abdominal pain. Patients with secondary AI tend to have milder symptoms than those with the primary form and frequently do not present with symptoms until they encounter a stressor, which under normal circumstances would stimulate the HPA axis to release cortisol.[38] For example, patients with secondary AI may become hypotensive, tachycardic, or hypoglycemic in the postoperative period as their body is

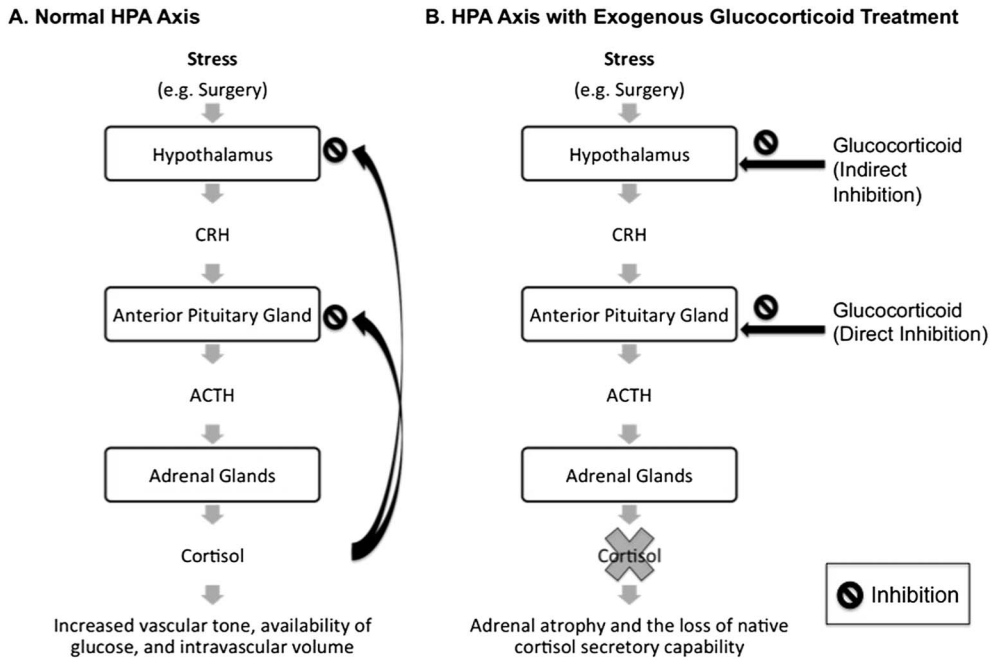

FIGURE 1. The HPA axis. The HPA axis is a hormonal system that responds to systemic stressors by stimulating the production and release of cortisol from the adrenal glands. When circulating cortisol levels are high, both ACTH and CRH release are suppressed by means of a negative feedback system (A). Exogenous corticosteroid treatment also suppresses the release of ACTH by acting on the pituitary directly (direct inhibition) and the release of CRH from the hypothalamus (indirect inhibition) (B). Chronic corticosteroid supplementation leads to adrenal atrophy and the loss of native cortisol secretory capability because the production and release of cortisol from the adrenal glands are suppressed over extended periods of time.

unable to compensate for the stress associated with surgery.[34] The basis behind perioperative corticosteroid dosing for patients with AI is to preemptively increase the corticosteroid supply to help overcome the potential stress-related insults of surgery. However, this practice is often presumptive as many patients with IBD do not have proven AI, and the risk of AI is anticipated at the discretion of the gastroenterologist or surgeon rather than objectively determined preoperatively.

CORTICOSTEROIDS

General Overview

Corticosteroids modulate inflammatory cytokine response by inhibiting the production and activity of proinflammatory cytokines (interleukin [IL]-1, IL-2, IL-3, IL-6, interferon-γ, and tumor necrosis factor–α), chemokines, eicosanoids, bradykinin, and migration inhibitory factor.[39–41] They also affect venous smooth muscle tone by activating target genes in the vasculature to help maintain endothelial function and integrity, reduce the release of nitric oxide from the vascular endothelium, prevent vasodilation through endothelin-1 expression, and sympathetically-mediated venoconstriction.[42–46]

Different corticosteroids have variable anti-inflammatory and HPA axis suppressive effects. They also have variable serum half-lives, ranging from 90 minutes (i.e., hydrocortisone) to many hours (i.e., dexamethasone). Physicians should familiarize themselves with the concept of "hydrocortisone equivalency" when prescribing and interpreting the effects of corticosteroids (Table 1). Budesonide is a corticosteroid with a high anti-inflammatory effect, approximately 200 times higher than hydrocortisone but generally thought to have minimal systemic activity because of a rapid, extensive (85%–90%) first pass liver metabolism that produces metabolites with minimal or no biological activity.[47,48] However, after 5 days of administration, budesonide controlled ileal release capsules, in both standard (9 mg/d) and higher doses (15 mg/d), affected plasma cortisol less than a moderate (20 mg/d) dose of prednisolone but more than placebo, showing a potential to cause secondary AI.[49]

TABLE 1. Corticosteroid Equivalency Table

Corticosteroid	Relative Potency	Equivalent Dose, mg	Biological Half-Life, h
Hydrocortisone	1	20	8–12
Cortisone	0.8	25	8–12
Prednisone	4	5	18–36
Prednisolone	5	4	18–36
Dexamethasone	25–50	0.5	36–54

Adapted from Salvatori.[34] Adaptations are themselves works protected by copyright. So in order to publish this adaptation, authorization must be obtained both from the owner of the copyright in the original work and from the owner of copyright in the translation or adaptation.

Effects on HPA Axis

As noted above, the most common cause of secondary AI is the abrupt or rapid cessation of exogenous corticosteroids use.[34] As a result, the production and release of cortisol from the adrenal glands is suppressed, eventually leading to adrenal atrophy and the loss of native cortisol secretory capability (http://www.uptodate.com/contents/the-surgical-patient-taking-glucocorticoids/abstract/33).[50] After a prolonged (weeks or more) course of exogenous corticosteroid treatment is stopped, affected patients often display signs of AI of varying degrees, depending on their stressors. The HPA axis will eventually recover, but the timing of recovery is highly variable, ranging from weeks to several months, depending on the formulation, dose, and duration of corticosteroid therapy, concomitant medications that affect corticosteroid metabolism, and patient-specific characteristics.[51–53]

Effects on Postoperative Course

The adverse effects associated with perioperative corticosteroid supplementation may be significant. Corticosteroid use has been associated with a 5- to 18-fold increased risk in surgical-related complications depending on the dose.[22,23] The immunosuppressive effects of corticosteroid supplementation place patients at increased risk for secondary infections and frequently cause hyperglycemia, hypertension, and fluid retention that may interfere and prolong postoperative recovery.[23,54–57]

Within the IBD population, patients receiving corticosteroids preoperatively have a greater likelihood of postoperative medical and surgical morbidity, including infections and septic shock.[22,23,58,59] Postoperative anastomotic leaks are significantly increased by corticosteroids, presumably because of impaired wound healing and increased tissue friability.[60–62] Additionally, corticosteroid use has been linked to an increased risk of serious infections and mortality among patients with Crohn's disease.[63] As a result, the potential benefit of using perioperative corticosteroids in patients with IBD with previous or recent exogenous corticosteroid exposure must be weighed against the potential risks with the aim of limiting treatment to those that most likely will require it.

HPA TESTING AND PATIENT STRATIFICATION

In general, a patient's likelihood for having secondary AI after exogenous corticosteroids use can be predicted based on the dose of treatment they received. Patients can be classified into one of the 4 categories (in prednisone equivalents)[34,64]:

1. Patients with normal adrenal function (documented serum cortisol >18 μg/dL).
2. Patients with low risk of adrenal suppression: patients who have been taking exogenous corticosteroids of any dose for less than 3 weeks or chronic prednisone ≤5 mg daily.
3. Patients with probable adrenal suppression: patients who are currently taking prednisone ≥20 mg daily for 3 weeks or more or patients with a Cushingoid appearance.
4. Patients with intermediate or unclear adrenal suppression: patients with a history of prolonged (>3 wk) corticosteroid exposure (prednisone ≥20 mg daily) during the past year or patients with sustained use of prednisone 5 to 20 mg daily.

Patients who fall into categories 2 or 3 likely do not require further HPA axis testing because their adrenal function is presumed based on the dosing and timing of their corticosteroids regimen. However, the adrenal status of patients who fall into category 4 is unclear and this subgroup may benefit from preoperative investigation of HPA axis status. AI can be ruled out using laboratory measures in a stepwise approach (Fig. 2). Using these measures in certain patients may be useful to identify those who would likely benefit from perioperative corticosteroid treatment rather than adopting an empiric practice based on case-by-case physician discretion.

The initial step in AI testing is to obtain a serum cortisol and ACTH level between 6 and 8 AM, when (in patients who follow a normal wake–sleep cycle) cortisol levels peak.[34] If a patient's morning serum cortisol level is >18 μg/dL, they can be classified as having normal adrenal function. However, if a patient's morning serum cortisol level is <3 μg/dL, they can be classified as having AI.[64] Assuming that the etiology of their AI is exogenous corticosteroid administration, the ACTH levels

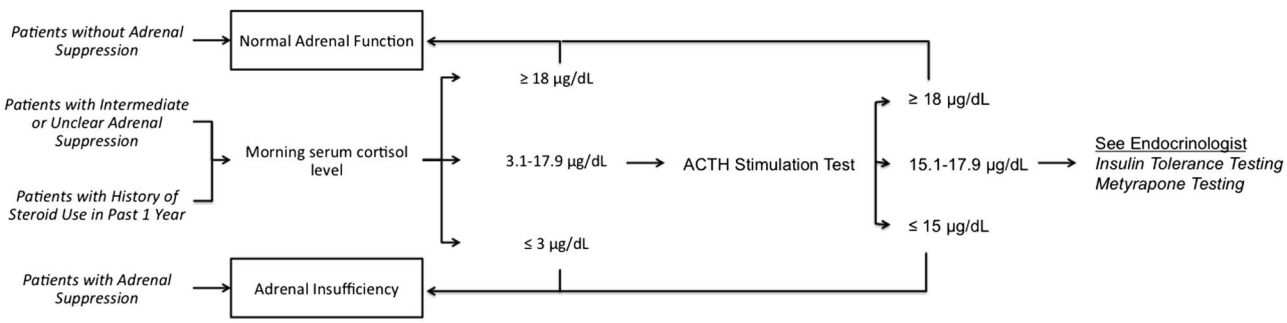

FIGURE 2. Stepwise approach to classifying adrenal function in corticosteroid-dependent patients. Patients' low risk of adrenal suppression: Patients who have been taking exogenous corticosteroids of any dose for <3 weeks or prednisone (≤5 mg daily or equivalent) for any duration. Patients with intermediate or unclear adrenal suppression: patients taking sustained prednisone dosing between 5 and 20 mg/d. Patients with history of corticosteroid use in past 1 year: patients with a long-term (i.e., >3 wk) history of prednisone ≥20 mg/d in the past year. Patients with adrenal suppression: patients who are currently taking prednisone ≥20 mg/d for 3 weeks or more and patients with a Cushingoid appearance.

should be inappropriately low or normal. From a clinical practice standpoint, if patients are not able to have their blood drawn early in the morning (e.g., outpatient preoperative testing before an elective surgery), an ACTH stimulation test (described below) can be done as the first test.

If a patient's morning cortisol level is inconclusive (i.e., between 3.1 and 17.9 μg/dL), additional information is needed. Patients with inconclusive morning cortisol levels can undergo dynamic testing to determine the status of their adrenal function. The most conventional of the dynamic function test is the ACTH stimulation test, where a dose of 1 to 24 ACTH is injected intravenously (IV) or intramuscularly (IM), and the serum cortisol level is measured at 30 and/or 60-minute postinjection. There are 2 variations to this test: a conventional dose version (administered ACTH dose 250 μg IV or IM) or a low-dose version (administered ACTH dose 1 μg IV).[34] For the 1 μg dose, the cortisol peak generally occurs at 30 minutes, therefore obtaining only a 60-minute value would generate false positives. In the conventional 250 μg dose, ACTH can also be administered IM, and the peak cortisol is expected to occur at 60 minutes, making the test easier to perform in the ambulatory setting (IM injection and single blood draw 1 h later).[65] For both versions, the poststimulation serum cortisol should rise to >18.1 μg/dL in patients with normal adrenal function. A poststimulation cortisol level of <15 μg/dL is diagnostic for AI.[66] Although initial reports suggested the low-dose ACTH stimulation test may be more sensitive for mild AI than the conventional-dose version, a meta-analysis of the tests found no difference between the 2.[66,67]

PERIOPERATIVE MANAGEMENT OF STEROID-EXPOSED PATIENTS

Despite the widespread use of perioperative corticosteroids in clinical practice, there is limited literature on perioperative corticosteroid regimens, which consist primarily of small cohort studies, case reports or series, and few randomized controlled trials.[10–13,15,16,18,19,68,69] In one of the earlier studies investigating perioperative stress dosing of corticosteroids, patients exposed to steroids for at least 14 days during the 4 weeks before surgery were given pre- and peri-operative cortisone, followed by a tapering regimen of IV cortisone over the postoperative 5-day period with 1 documented episode of AI responsive to additional IV corticosteroids. Patients whose corticosteroid exposure occurred more than 4 weeks before surgery were not given additional corticosteroid, and no episodes of AI occurred.[68] Another study reported no differences in supplemental postoperative corticosteroid requirements among patients with rheumatoid arthritis on long-term steroids who had versus had not received additional preoperative hydrocortisone dosing.[2] Results from several small cohort studies and 1 randomized controlled trial from the transplant population, where patients are often maintained on low-dose corticosteroids as part of their immunosuppressive regimen, showed no additive hemodynamic benefits with the use of high-dose perioperative corticosteroids compared with continuation of the patients' baseline regimen.[10,12–14,70] A randomized controlled trial of patients taking prednisone dosed 7.5 mg daily or higher for several months and with documented secondary AI showed no increased episodes of hemodynamic instability among patients randomized to receive saline versus IV cortisol, in addition to their usual daily dose of prednisone during their major surgeries.[9] Another randomized trial investigated organ transplant patients maintained on prednisone 5 to 10 mg daily in a double-blind crossover design where patients were given placebo or hydrocortisone 100 mg IV before either 1 of their 2 gingival surgeries. There were no clinically meaningful hemodynamic changes suggestive of AI, although some patients had evidence of HPA suppression based on ACTH levels checked postprocedurally. Therefore, the authors concluded that maintaining preoperative corticosteroid dosing for these patients was sufficient.[14]

Friedman et al[11] performed 35 major orthopedic surgeries among 28 patients receiving chronic steroids (mean dose of prednisone 10 mg daily) without administration of stress-dosed corticosteroids and noted no clinical evidence of AI, and 24-hour urine cortisol measurements suggested appropriate endogenous corticosteroid response to operative stressors. In another study of corticosteroid-exposed patients undergoing major surgery, over 50% of patients had evidence of impaired adrenocorticoid responses based on serum cortisol measurements at 1 hour after surgery began.[71] All patients who had been taking doses of prednisone 12.5 mg or greater for more than 6 months, 10 mg or greater for more than 2 years, or 7.5 mg or greater for more than 5 years had evidence of impaired adrenocortical response perioperatively. Hypotension occurred in almost 25% of study patients, but all episodes occurred among patients with normal or only slightly impaired adrenal function. These findings suggest adrenal impairment may be relatively common among chronic corticosteroid recipients, but AI is unlikely to be the primary etiology for peri- or post-operative hypotension, the most common impetus to prescribe additional corticosteroids for this group of patients.[71] In a systematic review of the available perioperative corticosteroid literature, Marik and Varon[72] concluded patients receiving therapeutic doses of maintenance corticosteroids do not require stress-dose corticosteroids perioperatively as long as their usual dose is continued.

Within the IBD population, there is also limited data on the impact of chronic corticosteroid exposures in perioperative setting. Knudsen et al reported perioperative outcomes for 95 patients with IBD who underwent 250 operations during an 11-year study period. All patients with IBD had preoperative corticosteroid exposure, with the majority of patients prescribed prolonged courses and hypotensive episodes occurred during 12% of surgeries. Although hypotension occurred more frequently among patients with IBD with more recent steroid exposure, the significance was negated when stratified for disease severity, and preoperative steroid dosing did not consistently prevent hemodynamic instability for these patients.[73]

Zaghiyan et al conducted a randomized trial investigating the use of the high-dose (IV hydrocortisone 100 mg 3 times daily, followed by taper) versus low-dose (IV hydrocortisone equivalent to preoperative dosing or no corticosteroid dosing for patients who were not taking steroids at the time of surgery) perioperative

corticosteroids for 92 corticosteroid-treated patients with IBD undergoing colorectal surgery. The authors found no differences in the incidence of postoperative postural hypotension between groups but did report a nonstatistically significant trend toward more infectious complications in the high-dose treated patients (16% versus 4%; $P = 0.11$). Based on these findings, they suggest that a low-dose approach to perioperative corticosteroids supplementation is appropriate in corticosteroid-treated patients with IBD undergoing major colorectal surgery.[16] Aytac et al[15] reported that although the use of stress-dose corticosteroids did not negatively impact early postoperative outcomes in their retrospective cohort study of 235 corticosteroid-treated patients with ulcerative colitis undergoing restorative proctocolectomy, there were no cases of intraoperative adrenal crises among patients with IBD even if no stress-dose steroids were given. Similarly, Zaghiyan et al reported no significant improvement in postoperative hemodynamic stability with the use of high-dose perioperative corticosteroids compared with low-dose or no corticosteroids in their 2 retrospective review of 97 and 49 patients with IBD treated with corticosteroids within the past year, respectively.[18,19] However, no objective measures to document potential AI, such as pre- or postoperative ACTH or cortisol measurements were performed in any of these studies. Variations in definitions of chronic or recent steroid exposure relative to time of surgery and low versus higher steroid dosing impact the ability to synthesize these results into a standardized algorithm based on the current literature.

PROPOSED APPROACH TO IDENTIFYING PATIENTS WITH IBD WHO MAY REQUIRE PERIOPERATIVE STEROIDS

Many patients with IBD have been exposed to steroids for extended durations of time, often with repeated courses to treat moderate-to-severe disease frequently in combination with other immunosuppressant agents. Because of progressive or refractory disease, many of these patients will ultimately require surgery, and a sizeable percentage of patients will have recent or current steroid exposure preoperatively. Because of limited presently available evidence, there is currently a lack of standardized recommendations to help regulate the prescribing practices of perioperative corticosteroids in patients with IBD.[20] A large part of the problem likely stems from the high variability in preoperative corticosteroid use that is observed within this population. Patients with IBD may take anywhere from ≤5 mg of prednisone (or equivalent) up to 75 mg per day depending on their disease activity, which makes establishing a single standard of care more challenging.[29] Based on our understanding of HPA axis function, review of above-mentioned limited available data on the efficacy of perioperative corticosteroid dosing, and the premise that the adrenal function of corticosteroid-dependent patients with IBD can be predicted based on the dose and duration of their corticosteroid regimens, we propose the following management strategy for perioperative corticosteroid supplementation in patients with IBD (Fig. 3). However, the paucity of randomized controlled trials or larger prospective cohort studies on the topic does not allow for formal evidence-based recommendations.

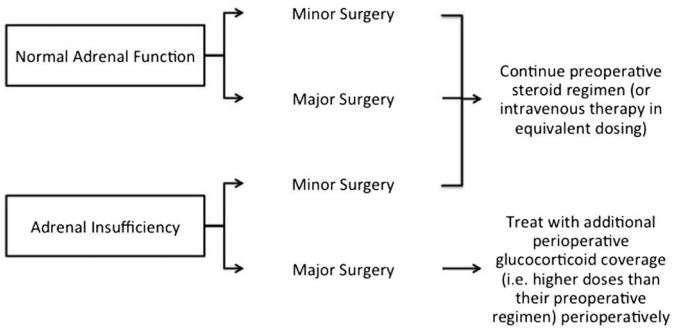

FIGURE 3. Management strategy for perioperative corticosteroid supplementation in patients with IBD (minor surgery: loop ileostomy reversal; major surgery: laparotomy, laparoscopy, colectomy, and proctectomy).

1. Patients without adrenal suppression: these patients do not require extra corticosteroid supplementation in the perioperative period.
2. Patients with adrenal suppression: patients taking supraphysiologic (>20 mg prednisone per day) corticosteroids dosing for at least 3 weeks preoperatively should be assumed to have HPA axis suppression will likely need additional corticosteroid supplementation in the perioperative period when undergoing major surgery. These patients should be treated with additional perioperative corticosteroid coverage (i.e., higher doses than their preoperative regimen) perioperatively. Patients with presumed adrenal suppression undergoing minor surgery (e.g., local excision, examination under anesthesia, ileostomy reversal) should not require additional treatment aside from preoperative steroid dosing.
3. Patients with intermediate or unclear adrenal suppression: patients with intermediate or unclear HPA axis function (prednisone 5–20 mg daily) should be considered for preoperative HPA axis testing to determine whether they fall under the guidelines for group 1 or 2, as mentioned above.

CONCLUSIONS

The use of perioperative corticosteroids in corticosteroid-treated IBD patients is common, based on early case reports of intraoperative adrenal crisis after abrupt corticosteroids withdrawal and observation that corticosteroids have a significant effect on vascular tone and cardiovascular stability.[6,7] However, recent data suggest that additional corticosteroid supplementation in the perioperative period may be unnecessary and may serve only to increase the risk of poor wound healing and infectious complications.[16,22,23] Preoperative corticosteroid use in the IBD population is common, but current prescribing practices are highly variable, likely because of a lack of randomized controlled

data and a wide range of preoperative treatment regimens.[20] Additional evidence-based prospective data are needed to help guide the multidisciplinary IBD teams regarding pre-, peri- and postoperative corticosteroids management, especially regarding appropriate corticosteroids tapering for chronic corticosteroid-exposed patients. We propose a patient-based approach to perioperative corticosteroid use of the IBD population based on an individual's historical use of corticosteroids, the type of surgery they are undergoing (anorectal operation versus laparotomy), and HPA axis testing when applicable. Stratifying patients with IBD using more objective parameters based on physiology may reduce the potentially unnecessary exposure to additional perioperative corticosteroids and decrease the possibility of steroid-associated postoperative morbidity.

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