Relative adrenal insufficiency (RAI) is defined as the inability to produce a cortisol level needed to control the inflammatory stress response. Severe illness activates the hypothalamic-pituitary-adrenal axis, which results in a six-fold increase in cortisol. Features suggesting RAI relate to cortisol’s well-established intracellular role in energy production, suppressing excess inflammation, and maintaining hemodynamic stability. On review of the literature, acute ileus has been described previously as a presenting feature of adrenal insufficiency in sick preterm infants and also in oncology and ulcerative colitis patients with long-term steroid use. It has never been described for patients with arthritides. This case is the first report in the literature of adrenal insufficiency presenting solely as prolonged ileus due to long-term steroid use for arthritic pain.

A 59-year-old man was referred for prolonged, unrelenting, apparently inexplicable intestinal obstruction. He was admitted two weeks earlier with a diagnosis of rectal bleeding from diverticular disease for which an exploratory laparotomy, sigmoid colectomy, and primary colocolic anastomosis was performed without event. On day four postoperatively, severe abdominal distension was observed and he had not yet passed flatus or feces. Serum electrolytes were within normal limits. An abdominal radiograph suggested intestinal obstruction (dilated loops of large intestine). At a second laparotomy on day four, no mechanical obstruction was found. The bowel was decompressed through an enterotomy in the distal ileum. Three days later, there was again persistent, tense abdominal distension causing tachypnea, tachycardia, and severe discomfort. A laparotomy, both small and large intestines were quite distended with no evidence of mechanical obstruction. The bowel was decompressed through enterotomy and tube suction, and a right transverse colostomy performed. Five days after this, he developed abdominal distension producing respiratory distress despite a patent colostomy; nasogastric aspirate was feculent.

On examination, he was tachypneic with gross abdominal distension. His BP was 135/85 mmHg; pulse, 90/min; respirations, 24/min; and temperature, 36.5°C. He had typical rheumatoid hands [Figure 1] showing bilateral synovitis, tophaceous olecranon bursitis and very thin skin with...
multiple ecchymoses. Steroid use was not volunteered on a routine medication history, but upon specific and repeated questioning of the patient and his relatives, his wife admitted that he had been self-medicating with 10 mg of over-the-counter prednisolone daily for more than 5 years to gain relief from his arthritic pains. On this basis, a diagnosis of RAI was considered and a therapeutic trial of glucocorticoids was instituted with 1 g of intravenous methylprednisolone. Following this, bowel sounds were auscultated 6 hours later and a bowel action experienced the next day. Clinical improvement was dramatic and sustained with 100 mg of intravenous hydrocortisone administered every 8 hours. The patient was fit to be discharged 3 days later on replacement glucocorticoids. A morning cortisol level was 35 μg/dl, which was likely inaccurately elevated as it was measured after one dose of methylprednisolone was given. Histology of the resected sigmoid colon showed diverticular disease with no evidence of inflammatory disease, rheumatoid vasculitis, or malignancy.

**Discussion**

The diagnosis of RAI requires a high index of suspicion. The proposed thresholds are a baseline cortisol level of less than 15 μg/dl (416 nmol/l) and an inadequate rise in cortisol after adrenocorticotropic hormone (ACTH) stimulation of less than 9 μg/dl (250 nmol/l).[1] However, patients have a poor prognosis (mortality 82%) with a baseline cortisol level of more than 34 μg/dl (938 nmol/l).[2] Plasma cortisol levels neither convey free cortisol levels nor indicate corticosteroid action at the tissue level and therefore these thresholds are mere guidelines and the use of glucocorticoid therapy as a rescue strategy is acceptable.[3] Plasma cortisol levels also do not accurately convey pituitary-adrenal axis function in patients treated with long-term glucocorticoid therapy. Thus, even though this patient’s random cortisol level was 35 μg/dl, improvement following glucocorticoid therapy was immediate and telling for this diagnosis. Inflammatory conditions such as rheumatoid vasculitis, which was indeed possible in this case, would have also responded rapidly with glucocorticoids. However, the pathology specimen did not reveal any vasculitic features.

Exogenous glucocorticoid suppresses the production of corticotrophin-releasing hormone and can induce adrenal atrophy, the severity of which is related to the dose and duration of treatment. A minimum of 7.5 mg of prednisolone (30 mg hydrocortisone; 0.75 mg dexamethasone) per day for more than 5 weeks can induce adrenal insufficiency for months after prompt cessation.[4] In our case, the large dose of prednisolone coupled with the long duration made RAI likely. Stelzner et al. also described steroid withdrawal simulating intestinal ileus in a large group of ulcerative colitis patients who were on long-term steroid treatment.[5] It is interesting to note that primary adrenal crisis may also present with an acutely painful and distended abdomen requiring surgical intervention. However, this was not thought to be the case in our patient. The contradiction of the patient’s prompt response to high-dose steroid despite having a high serum cortisol (35 μg/dl) may be explained by either an inaccurate level or steroid desensitization.[7] The serum cortisol was drawn after the first dose of methylprednisolone was given which is known to give a falsely elevated serum cortisol. On the other hand, in critically ill patients, failure to maintain hemodynamic stability was speculated to be due to impaired glucocorticoid-adrenergic receptor coupling and downregulation of adrenergic receptors. High cytokine levels during high stress states (e.g., sepsis) can induce corticosteroid resistance.[8] In both scenarios, the normal adrenal response becomes inadequate to control inflammation.

Self-medication with prednisolone is not uncommon in our experience. Since it is cheap and highly efficacious in a wide range of arthritides, it is an attractive option for analgesia. However, it becomes dangerous when available over-the-counter and used in an unregulated manner. For example, a recent study in India showed that the prevalence of self-medication was 31.3% with 87% of users unaware of side effects.[9] The diagnosis of RAI was delayed as the patient failed to recognize the relevance of self-medication. Medication histories volunteered by patients are commonly inaccurate with reported frequencies as high as 54%.[9] Moreover, many patients do not readily admit to steroid use when they obtain it without prescription. However, once previous glucocorticoid use has been identified, clinical suspicion of adrenal insufficiency is warranted.[6]

RAI is infrequently diagnosed due to its vague and controversial diagnostic criteria. It should be considered, however, in any critically ill patient who is unresponsive to treatment. Even though our patient had no evidence of shock or electrolyte abnormalities, the inexplicable, unresolving ileus proved to be an unusual presentation of RAI. The diagnosis would not have been made if there was not a high index of suspicion of self-medication with steroids. In countries where these drugs are available over the counter, RAI should be considered in critically ill patients who do not seem to respond to conventional treatment.

**References**

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