The bittersweet of steroid therapy

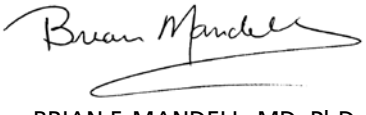
Every physician—general internist, subspecialist, or surgeon—has had to deal with complications of glucocorticoid therapy. Infections may be the most worrisome in terms of risk of death, as these drugs can both open the door to opportunistic organisms and delay the diagnosis by blunting the signs and symptoms of infection-associated inflammation. But the metabolic ravages of long-term steroid therapy can also plague patients and their physicians.

For many of those long-term effects, such as osteoporosis, cushingoid features, skin fragility, and cataracts, all we can do is hope that they don't occur, since there is little we can do to screen for or prevent them. We have previously discussed steroid-associated osteoporosis in the *Journal*,[1] and strategies for preventing it have been proposed by specialty societies.[2] For other complications such as hypertension, weight gain, and glucose intolerance, we can offer common-sense protective suggestions, monitor for them, and intervene if they occur.

On page 748 of this issue, Dr. M. Cecilia Lansang and Ms. Leighanne Kramer Hustak[3] discuss the management of steroid-induced adrenal suppression and diabetes. They offer practical management suggestions but also point out that the evidence base for our treatment decisions is surprisingly limited.

Nearly all patients chronically receiving high-dose glucocorticoid therapy develop glucose intolerance, but knowing when that is happening is not always easy. In patients destined to develop type 2 diabetes, the laboratory or clinical signs of hyperglycemia appear only when the pancreas can no longer maintain the insulin production necessary to overcome peripheral insulin resistance. Steroid-induced diabetes is characterized by increased gluconeogenesis, insulin resistance, and excessive postprandial surges, so fasting glucose levels are not sensitive for this clinical syndrome.

The degree and duration of the chronic hyperinsulinemia and hyperglycemia dictates the risk of microvascular complications and thus will be linked to duration of steroid therapy (unless the steroid is unmasking preexisting mild diabetes). Although issues surrounding tight control of blood glucose levels in the acute setting remain unresolved, I believe that even short-term significant steroid-induced hyperglycemia should be prevented when reasonably possible, at the least keeping in mind the additive ill effects of hyperglycemia and steroid therapy on the risk of nuisance infections such as oral and vaginal candidiasis and urinary tract infections that, in the setting of high-dose steroid therapy, can rapidly turn nasty.



BRIAN F. MANDELL, MD, PhD
Editor-in-Chief

1. **Dore RK**. How to prevent glucocorticoid-induced osteoporosis. Cleve Clin J Med 2010; 77:529–536.
2. **American College of Rheumatology Ad Hoc Committee on Glucocorticoid-Induced Osteoporosis**. Recommendations for the prevention and treatment of glucocorticoid-induced osteoporosis: 2001 update. Arthritis Rheum 2001; 44:1496–1503.
3. **Lansang MC, Hustak LK**. Glucocorticoid-induced diabetes and adrenal suppression: how to detect and manage them. Cleve Clin J Med 2011; 78:748–756.

doi:10.3949/ccjm.78b.11011

Downloaded from www.ccjm.org on April 25, 2024. For personal use only. All other uses require permission.