

The Risk of Pill Cutter Sharing

To the Editor:

The most frequent cause of secondary adrenal insufficiency is the prolonged exposure to exogenous glucocorticoids (GCs).¹ The source of GCs is most often obvious (oral or parenteral, or even inhaled), but it may be occasionally more difficult to identify, as GCs may be found in “traditional” or “alternative” medicines.² We describe here the case of a patient with secondary adrenal insufficiency due to using the same pill cutter for her medications and her dog’s prednisone.

A 45-year-old woman with history of depression presented to her internist complaining of 4 weeks of decreased appetite, nausea, weakness, and 4-kg weight loss. Symptoms had started about 1 week after her dog died. Her menstrual periods were regular. She weighed 40.9 kg and stood 147 cm (body mass index = 17.6). Blood pressure was normal. The skin and buccal mucosa had no hyper-pigmentation. She was started on megestrol acetate (MA) 625 mg daily. Morning serum cortisol drawn 3 days later was low, at 1.4 $\mu\text{g}/\text{dL}$ (6-26). A week after the initial visit, after holding MA for 1 day, morning cortisol was low, at 0.9 $\mu\text{g}/\text{dL}$, and 30 and 60 minutes after 250 μg of intravenous adrenocorticotropin hormone (ACTH), was 5.1 and 7.0 $\mu\text{g}/\text{dL}$, respectively, diagnostic of adrenal insufficiency. Dehydroepiandrosterone sulfate (DHEAS) was < 15 $\mu\text{g}/\text{dL}$ (25-220), supporting the diagnosis, and plasma ACTH was < 5 pg/mL (6-50), showing that her adrenal insufficiency was secondary to ACTH suppression. Her serum free thyroxine and insulin-like growth factor-I were both normal. Serum screening for synthetic GCs (4 weeks after the diagnosis) was negative. Pituitary magnetic resonance imaging was normal.

The patient denied ever taking oral, topical, or parenteral GCs or any alternative medicine preparation. Her dog had a history of Addison’s disease and had been on prednisone for the past 6 years. She had been administering prednisone to the pet by breaking twice daily a 5-mg prednisone tablet

into quarters, using a pill cutter. Over these years, she had frequently used the same pill cutter to cut her own antidepressant pills.

MA was discontinued, and hydrocortisone was started. The dose was tapered, and patient self-discontinued it after 7 weeks. After an additional 4 weeks, her morning cortisol was 7.1 $\mu\text{g}/\text{dL}$, her ACTH was 12 pg/mL, and DHEAS was 26 $\mu\text{g}/\text{dL}$. She had regained 3.7 kg. Post ACTH serum, cortisol peak 15 weeks after the initial diagnosis was 20.8 $\mu\text{g}/\text{dL}$, showing resolution of adrenal insufficiency.

This patient had secondary adrenal insufficiency, with normal-appearing pituitary, and otherwise normal pituitary function. Although MA can cause adrenal insufficiency,³ and can acutely suppress serum cortisol,⁴ the short length of MA treatment would be very unlikely to cause the adrenal atrophy that would explain the abnormal post-ACTH serum cortisol peak.⁵ Thus, we conclude that her transient adrenal insufficiency was due to exposure to her dog’s prednisone. This fits with her symptoms starting after stopping this practice upon the dog’s death. Although cutaneous absorption of topical GCs can induce adrenal insufficiency,⁶ we suspect that the contamination was oral, by using the same pill cutter for her medication. We conclude that prolonged sharing of a pill cutter with a housemate (pet or human) who is on GC therapy is a risk factor for adrenal insufficiency.

Sritika Thapa, MD

Roberto Salvatori, MD

Division of Endocrinology

Department of Medicine

Johns Hopkins University School of Medicine

Baltimore, Md

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Requests for reprints should be addressed to Roberto Salvatori, MD, Division of Endocrinology, Johns Hopkins University, 1830 East Monument St. #333, Baltimore MD 21287.

E-mail address: salvator@jhmi.edu