Precocious Puberty Secondary to Topical Testosterone Transfer: A Case Report

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ABSTRACT

Introduction. Testosterone replacement therapy is the standard of care for androgen deficiency syndrome, and patients and physicians can choose among depot injectable, subcutaneously implanted pellet, transdermal patch, topical gel, and buccal tablet dosage forms. Topical gels have become popular and, although unintentional secondary transfer to a spouse or child is a known hazard, physicians and patients may underestimate the risk.

Aim. We report a case of precocious puberty in a 10-month-old male secondary to transfer of topical testosterone from his father, who was treated for primary hypogonadism.

Results. Once the father's therapy was changed from a topical to a buccal dosage form, the symptoms in his son receded.

Conclusion. The potential for secondary exposure to testosterone—and its consequences—may be underappreciated by patients and by health care providers not involved in managing testosterone replacement therapy. The patient's lifestyle (e.g., contact with children, physical limitations, daily schedule) should be part of the discussion when selecting a method of testosterone replacement therapy. **Cavender RK and Fairall M. Precocious puberty secondary to topical testosterone transfer: A case report. J Sex Med 2011;8:622–626.**

Key Words. Precocious Puberty; Hormone Replacement Therapy; Environmental Exposure

Introduction

The standard of care for androgen deficiency syndrome is testosterone replacement therapy, which is available in many forms: depot injectables, subcutaneously implanted pellets, transdermal patches, topical gels, and buccal tablets (approved by the U.S. Food and Drug Administration [U.S. FDA] in 1953, 1972, 1995, 2000, and 2003, respectively) [1]. Topical gels have become progressively more popular in the primary care arena because of the ease of application, as well as the clinically proven benefits such as stable therapeutic testosterone levels, improvement in metabolic parameters (e.g., blood sugar control in diabetics), and improvement of symptoms commonly expressed by androgen-deficient men (e.g., functional capacity, sexual function, libido, mood, cognition, and bone and body composition). In a competitive whirlwind, the marketing campaigns of the topical gel industry have targeted primary care providers, barraging them with data that demonstrate the ease of administration and testosterone levels achieved without formally educating them about the international testosterone prescribing guidelines [2] and the potential risks, specifically the risk of drug transfer to spouses and/or children (also known as secondary exposure).

Cutaneous transfer of topical testosterone to a spouse or child has long been recognized as a possible complication of this method of administration [3–5], yet cases of secondary transfer are still reported [6,7]. It is standard practice to advise patients to wash their hands following the application of these products and to cover the treated area with clothing. Prior to prescribing a topical testosterone product, it is imperative that physicians be aware of all associated risks and that they conduct a detailed lifestyle and risk assessment for secondary exposure. Moreover, patient circumstances commonly change after the initial assessment, necessitating ongoing lifestyle and

transfer-risk surveillance as well as counseling at each follow-up visit. If a risk of secondary exposure is identified, or if the patient fails to acknowledge transfer precautions, an alternative method of therapy should be employed.

This report describes a case of precocious puberty in a 10-month-old male secondary to transfer of topical testosterone from his father, who was treated for primary hypogonadism.

Case Report

On January 26, 2006, a 32-year-old white male with a past medical history of hypertension presented with the complaint of 1.5 years of progressive generalized sarcopenia with asymmetrical lower extremity atrophy, left greater than right. Symptom onset followed repair of a left anterior cruciate ligament tear and subsequent prolonged immobilization. The patient reported delayed rehabilitation, with generalized decreased muscular strength, tone, and endurance. The patient also reported a 40-lb central weight gain over the 1.5-year period, with associated symptoms of insomnia, fatigue, decreased libido, and mild erectile insufficiency. His score on the Androgen Deficiency in the Aging Male questionnaire was positive for 9 out of 10 variables and the Sexual Health Inventory for Men score was 20 out of 25 points. His International Prostate Symptom Score was 0 out of 35 points, indicating no lower urinary tract symptomatology. His physical exam was significant for generalized lower extremity muscular atrophy, with left greater than right. His genitourinary and prostate exams were normal. His baseline morning hormone laboratory results are shown in Table 1; additionally, glucose, renal and liver function tests, electrolytes, and complete blood count were within normal limits.

The patient was diagnosed with primary hypogonadism, having met both clinical and laboratory criteria, and was counseled regarding pathophysiology and methods of androgen therapy. He subsequently elected topical therapy. On February 8, 2006, following detailed secondary exposure risk counseling and his provision of informed consent, he was started on testosterone 150 mg/mL, 1 mL topically to the shoulder area daily at bedtime. Additionally, he started therapy with anastrozole 0.2 mg sublingually once daily to decrease the supraphysiologic estrone level noted in his baseline laboratory results. Follow-up assessment 2 months later revealed a subtherapeutic response with laboratory results as shown in Table 1 (April 1 data). On April 10, the testosterone dose was increased to 200 mg/mL, 1 mL topically daily at bedtime, with no change in the anastrozole regimen. At the next assessment approximately 6 weeks later, his clinical response had improved and laboratory results were stable (Table 1, May 20 data). His laboratory results for the metabolic panel and complete blood count were also within normal limits.

In early June, approximately 4 months after starting topical testosterone treatment, the patient notified our office that his 10-month-old son had undergone a pediatric endocrinology evaluation secondary to the development of precocious puberty. Further inquiry revealed that the infant had developed progressive penile enlargement

Table 1 Selected laboratory results for father (32 years old)

			Laboratory results (2006)		
Hormone*	Units	Normal range	January 26 (baseline)	April 1	May 20
Total testosterone	ng/dL	241–827	264	369	378
Free testosterone	pg/mL	8.7–25.1	13	15.7	19
Dihydrotestosterone	ng/dL	30–85	—	46	73
Sex hormone binding globulin	nmol/L	13–71	12	—	9
Luteinizing hormone	mIU/mL	1.5–9.3	2.6	—	—
Follicle stimulating hormone	mIU/mL	1.4–18.1	4.7	—	—
Estrone	pg/mL	12–72	69	54	59
Estradiol	pg/mL	3–70	21	9	15
Prostate-specific antigen	ng/mL	0.0–4.0	0.5	0.6	0.6
Dehydroepiandrosterone	μg/dL	120–520	378	—	—
Cortisol	μg/dL	3.1–22.4	8.9	—	—
Thyroid stimulating hormone	μIU/mL	0.350–5.500	4.961	—	—
T4 free	ng/dL	0.61–1.76	0.89	—	—
T3 free	pg/mL	2.3–4.2	3	—	—

*Blood for all laboratory tests was collected in the morning.
— = not tested.