Letters to the Editor

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This series is coordinated by Kenny Lin, MD, MPH, Associate Deputy Editor for *AFP* Online.

Hyperthyroidism Caused by Thyroid Hormone Therapy

Original Article: Hyperthyroidism: Diagnosis and Treatment

Issue Date: March 1, 2016

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TO THE EDITOR: Dr. Kravets provides a good review of hyperthyroidism; however, the article did not discuss what is likely the most common cause of hyperthyroidism: overuse and misuse of thyroid hormone. Many physicians prescribe thyroid hormone therapy for various reasons, such as promoting weight loss in overweight or obese patients, even if not indicated.[1] Thyroid-stimulating hormone or other appropriate thyroid testing should always be performed to verify the need for exogenous thyroid hormone replacement.

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IN REPLY: I would like to thank Dr. Scherger for making an important point about the role of exogenous thyroid hormone in causing hyperthyroidism. Iatrogenic hyperthyroidism may be intentional (e.g., patients with thyroid cancer who have clinicopathologic parameters that warrant thyroid-stimulating hormone suppression to prevent cancer recurrence) or inadvertent (e.g., an excessive dose of thyroid hormone prescribed for hypothyroidism).

In patients with no evidence of hypothyroidism, it is inappropriate for physicians to prescribe thyroid hormone therapy to treat obesity or depression. Some patients ingest exogenous thyroid hormone surreptitiously (thyrotoxicosis factitia); others may ingest exogenous thyroid hormone unintentionally when taking contaminated dietary supplements. In addition, there have been cases of accidental ingestion of animal thyroid tissue in hamburgers after the thyroid tissue was inadvertently removed and ground up with neck muscle in a slaughterhouse.[1]

When exogenous hyperthyroidism is suspected but cannot be confirmed by patient history, a radioactive iodine uptake of less than 1% and a low serum thyroglobulin level may serve as diagnostic clues. In some patients with thyrotoxicosis factitia, the serum thyroglobulin level may not be suppressed because of a goiter or antithyroglobulin antibodies. In these cases, one can consider measuring fecal thyroxine (T_4) levels. The T_4 levels were shown to be twice the normal value in patients with Graves disease and increased 12- to 24-fold in patients with thyrotoxicosis factitia.[2]

Unintended iatrogenic hyperthyroidism in patients with hypothyroidism who receive thyroid hormone therapy can be prevented by monitoring thyroid-stimulating hormone levels at least annually and adjusting the dose as needed. Physicians must adhere to the principles of beneficence and nonmaleficence when prescribing thyroid hormone therapy.[3] Thyrotoxicosis factitia should be treated by discontinuing exogenous thyroid hormone therapy, providing patient education, and referring for psychiatric evaluation when appropriate.

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Exercise Is Effective Therapy for Chronic Nonbacterial Prostatitis and Chronic Pelvic Pain Syndrome

Original Article: Common Questions About Chronic Prostatitis

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TO THE EDITOR: The authors of the article on chronic prostatitis effectively summarize the practical dilemmas that arise when managing this complex disease. Although the authors related this disease to other chronic pain syndromes, including fibromyalgia and irritable bowel syndrome, they did not highlight exercise as a useful therapy for chronic nonbacterial prostatitis/chronic pelvic pain syndrome (CNP/CPPS) despite its proven benefit in chronic pain syndromes. *American Family Physician* articles addressing fibromyalgia and irritable bowel syndrome highlight exercise as a valid treatment option with evidence ratings of A and B, respectively.[1,2] For CNP/CPPS specifically, a randomized, double-blind study demonstrated significant benefit with aerobic exercise in both pain and quality-of-life scores, and another study suggested that exercise can also lower the risk of CNP/CPPS.[3,4]

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3. Giubilei G, Mondaini N, Minervini A, et al. Physical activity of men with chronic prostatitis/chronic pelvic pain syndrome not satisfied with conventional treatments—could it represent a valid option? The physical activity and male pelvic pain trial: a double-blind, randomized study. *J Urol.* 2007;177(1):159-165.
4. Zhang R, et al. Physical activity and chronic prostatitis/chronic pelvic pain syndrome. *Med Sci Sports Exerc.* 2015;47(4):757-764.

IN REPLY: I appreciate Dr. Meyers bringing this study[1] to my attention. The researchers surveyed 20,918 male professionals on lifestyle and symptoms over 22 years (surveys were performed in 1986, 1988, and 2008). The 2008 survey included symptoms suggestive of CNP/CPPS; men with this condition at the start of the surveys in 1986 were excluded. The surveys also included questions on tobacco use, hypertension, duration of weekly screen time in 1988, amount of physical activity, and type of physical activity. Based on symptoms, 689 men developed CNP/CPPS over the study period. There was a strong inverse relationship between the intensity and duration of physical activity and the incidence of CNP/CPPS. No other comorbidities or potential confounding factors were included in the surveys.

Table 3 of our article mentioned another study that randomized 103 men with CNP/CPPS to aerobic exercise or to a stretching routine. Aerobic exercise showed statistically significant but modest clinical benefits.

Although more research is needed to clarify the extent of benefit, I agree that the new study highlighted by Dr. Meyers strengthens the argument that exercise is effective in the prevention and treatment of CNP/CPPS.

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1. Zhang R, et al. Physical activity and chronic prostatitis/chronic pelvic pain syndrome. *Med Sci Sports Exerc.* 2015;47(4):757-764.

Corrections

Incorrect diagnostic information. The article "Diabetes Mellitus: Screening and Diagnosis" (January 15, 2016, p. 103) contained incorrect information regarding diagnostic testing for diabetes. The first paragraph of the "Diagnostic Testing" section (p. 106) should have read as follows: "The diagnosis of diabetes can be made when classic signs and symptoms of hyperglycemia are associated with a single random plasma glucose measurement of 200 mg per dL (11.1 mmol per L) or greater. Alternatively, the diagnosis can be made with an A1C level of 6.5% or greater, a fasting plasma glucose level of 126 mg per dL (7.0 mmol per L) or greater, or a two-hour plasma glucose level of 200 mg per dL or greater during an oral glucose tolerance test with 75-g glucose load *(Table 2*[17]*)*; however, testing should be repeated on a subsequent day to confirm the diagnosis.[1,17] If testing results do not match the clinical picture or are inconsistent, repeat testing or testing with another modality may be helpful.[17]" These changes affected Question 2 in the January 15, 2016, issue of the CME Quiz. The question has been deleted from the online assessment to avoid confusion. The online version of the article has been corrected. ■