Communications

Myopathic Presentation of Thyroiditis

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"Few present-day physicians, aside from endocrinologists, know that hypothyroidism may cause profound myopathy," wrote Fessel in 1968.[1] Even fewer physicians would appreciate a connection between thyroiditis and myopathy. A case of this association is presented in an attempt to alert primary care physicians to this potential presentation of a treatable disorder.

Case Report

A 30-year-old male tombstone carver, previously well, presented with a complaint of weakness of two weeks' duration. The weakness involved the thighs, calves, and upper arms, but not the hands or feet. He also noted that he had intermittent cramping of the calves and that his arms and legs sometimes "went to sleep." There was no previous history of these symptoms. He denied weight loss, fever, neck pain, recent acute illness, or anorexia.

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Past medical history revealed no significant illness or surgery. He took no medications chronically. He smoked about ten cigarettes daily and drank an occasional beer, but denied problem drinking. His family history was significant for asthma and an unknown neuromuscular disorder that confined his sister to a wheelchair (there were no other family members so affected). There was no family history of thyroid disease. In his work he was exposed often to a lacquer thinner, but there was no history of exposure to lead or other heavy metals.

Physical examination revealed a muscular individual with a weight of 199 lb, blood pressure of 102/78 mmHg, and a regular pulse of 60 beats/min. There was no palpable goiter or any neck tenderness. Skin and hair appeared normal. Deep tendon reflexes were 2+ at the knees, 2+ at the biceps, 3+ at the triceps; relaxation phase appeared normal. Pinprick sensation was everywhere intact. Testing of multiple muscle groups revealed good strength, graded 5/5, without demonstrable weakness of proximal musculature; there was no peripheral edema or notable muscle tenderness to palpation.

Initial laboratory studies revealed a normal urinalysis, complete blood count, and sedimentation rate. A chemical profile showed an elevated creatine phosphokinase (CPK) of 2,805 U/mL (nor-

mal, 0 to 140 U/mL); repeated the following day, it was 2,942 U/mL. Five other elevated items were noted: SGPT 60 U/mL (normal, 6 to 37 U/mL), SGOT 84 U/mL (normal, 10 to 30 U/mL), lactic dehydrogenase (LDH) 356 U/mL (normal 109 to 193 U/mL), cholesterol 340 mg/dL (normal, 115 to 252 mg/dL), and creatinine 1.62 mg/dL (normal 0.7 to 1.5 mg/dL).

A diagnosis of myopathy, type and cause unspecified, was made; a symptomatic medicine was begun and bed rest recommended. Thyroid function studies were obtained, revealing a depressed T_4 of 0.6 μg/dL (normal, 4.5 to 12 μg/dL) and T_3 by radioimmunoassay of 35 ng/dL (normal, 80 to 220 ng/dL); thyroid stimulating hormone (TSH) was greater than 40 μU/mL (normal 0 to 10 μU/mL). The patient was begun on L-thyroxine, and thyroid antibody studies were obtained. Antimicrosomal antibody was not found; however, antithyroglobulin antibody was very significantly positive in a titer of 1:10,485,760. A radioactive iodine scan prior to treatment showed uptake at 24 hours to be less than 2 percent.

Two weeks later the patient returned, taking L-thyroxine and feeling better. His CPK level was 1,340 U/mL and his weight 198 lb. His thyroid gland was felt to be about 3 cm across (total diameter), not unusually firm, and without discrete masses, nodules, or tenderness.

He was then lost to follow-up until about five months later, when, on return, his weight was 194 lb. His thyroid gland was again not enlarged and was without discrete masses. Serum studies at this time revealed a CPK of 170 U/mL, normal cholesterol (224 mg/dL), T_4 of 4.7 μg/dL, T_3 resin of 28.9 percent (normal, 22 to 34 percent), T_7 of 1.4 (normal, 1.3 to 3.4), but again, a TSH of greater than 40 μU/mL. He avowed taking his medicine faithfully. His dose of L-thyroxine was increased slightly. He was then lost to further follow-up.

Discussion

Primary hypothyroidism (ie, not secondary to pituitary dysfunction) is a not uncommon diagnosis for the primary care physician. Thyroiditis is likewise not uncommon, whether the patient presents as euthyroid or in a dysfunctional hormonal state. The provocative feature of the case above is the association of the triad of thyroiditis, hypothyroidism, and myopathy. As a presentation, myopathy usually leads one to consider primary muscular or neuromuscular disorders, but since the late 1800s there has been awareness of a relationship between hypothyroidism and myopathy, with numerous reports from the first half of this century documenting this connection.[1]

During a two-year period, Fessel[1] discovered that 5.6 percent of individuals seen in a community hospital setting for acquired myopathic disorders had hypothyroidism as a presumed cause; furthermore, muscular symptoms were the first to be observed in these particular cases. Pain was not usually a prominent feature, and muscular symptoms responded to therapy with thyroid hormone. He felt that prevalence of muscular disorder with hypothyroidism was "common" and outlined two basic types: (1) hypertrophy with or without myotonia, and (2) atrophy.

In the clinical laboratory, the CPK level has been noted as a useful marker of hypothyroid myopathy. Graig and Smith[2] studied euthyroid controls and hyperthyroid and hypothyroid subjects; they found excellent correlation between the level of CPK activity and thyroid dysfunction. The relationship between serum enzyme levels and thyroid activity was found to be inverse, and serial studies reflected response to appropriate therapy.

Histologic studies have also been performed. Determined by electron microscopy, muscle tissue from patients with hypothyroidism shows mitochondrial changes, excessive glycogen deposits, and various abnormal subsarcolemmal deposits.[3] The mechanism of subcellular changes remains speculative.

Etiology of thyroid hypofunction is usually not mentioned in the studies cited, but thyroiditis (specifically, autoimmune, or Hashimoto's thyroiditis) is a well-documented cause of hypothyroidism.[4] Yet, a myopathic presentation would not necessarily be quickly associated with thyroiditis, and though an association with myasthenia gravis has been described with respect to autoimmune thyroiditis, myopathy (in a more general sense) is

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not mentioned as a finding of thyroiditis in two standard thyroid texts consulted.[5,6] One explanation for this omission is probably that, when thyroid diagnoses are being considered, myopathy draws attention to the hypothyroidism per se rather than the thyroid inflammatory process resulting in the hypofunctional state. This is especially true in the absence of a goiter, which specific finding might more likely provoke thoughts of thyroiditis.

In the patient presented, the diagnosis of primary hypothyroidism was substantiated by the depressed levels of serum T_4 and T_3 and by the elevated TSH. The diagnosis of autoimmune thyroiditis (probably the "atrophic variant"[6]) is supported by the markedly elevated antithyroglobulin antibody level in this clinical setting; Volpé[6] notes that thyroglobulin antibody titers of over 1:20,000 may be found only in chronic lymphocytic (autoimmune) thyroiditis or in Graves' disease.

The diagnosis of myopathy in the absence of a muscle biopsy was supported by the patient's elevated serum CPK and his subjective, focal paresthesias and weakness, severe enough to disturb him in his work with heavy tombstones. (As to the lack of objectively demonstrable weakness on examination, it should be emphasized that the patient was quite muscular and of significantly greater strength than his examiner, so that relatively subtle signs of muscle weakness may have passed unnoticed.)

In summary, the patient discussed presented with thyroiditis and resulting hypothyroidism; apart from laboratory abnormalities, the only indication of his actual endocrine disorder was myasthenic (myopathic) in nature. Furthermore, subjective strength returned and laboratory studies normalized with thyroid hormone treatment alone. It is not known, of course, had treatment not been instigated, when other signs and symptoms of hypothyroidism would have developed; it is presumed his myopathy would have worsened, consistent with findings of the authors cited above.

Thus, myopathic symptoms, as in this patient, may signify a hypothyroid disorder, including thyroiditis, and not only thyroiditis but also myopathy may be the first (and only) nonlaboratory indicator of the dysthyroid state. These facts should be

borne in mind as the primary physician considers the clinical conundrum of thyroid disease and when he or she is called upon to evaluate the patient with myasthenic symptoms.

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A Four-Year Experience with Hemoccult Testing Kits in a Family Medicine Center

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The importance of screening for colorectal cancer is widely accepted and has been reviewed in this journal.[1] The Hemoccult* slide test is perhaps the best screening method for colon cancer. It is also used to determine whether gastrointestinal blood loss is occurring in symptomatic patients. This report presents four years of experience using prepared kits given to patients in a family practice clinic.

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Methods

A standardized method of Hemoccult screening was introduced in the Family Medical Center at the University of Washington in June 1978. The population served at this residency training site (18 residents, 10 faculty) includes 7,100 patients, most of whom live in the surrounding university community of middle-class predominance. Thirty-two percent of the active registered patients are over the age of 35 years, with a female-to-male ratio of 1 to 6. Forty percent of total patient visits (1982) were from patients over the age of 35 years, with a female-to-male ratio of 2 to 1. The clinic screening protocol for adults over the age of 40 years includes Hemoccult testing every one to two years. Kits are prepared that contain three Hemoccult cards, applicators, instructions (including a recommended diet), and a return envelope. Attached