NOVATEUR PUBLICATIONS
JournalNX- A Multidisciplinary Peer Reviewed Journal
ISSN No: 2581 - 4230
VOLUME 8, ISSUE 1, Jan. -2022

THE ROLE OF DIET IN THYROID DISEASE – CASE REPRESENTATION

M.Shirley Lillian
M.sc (PhD), Food Science and Nutrition
Nirwan University, Rajasthan Jaipur

ABSTRACT:

Patients with abnormalities of thyroid gland function or structure come to medical attention for several reasons. They present with symptoms attributable to physiologic effects of increased or decreased plasma concentrations of thyroid hormone (hyperthyroidism or hypothyroidism, respectively). They may also present with symptoms related to localized or generalized enlargement of the gland (diffuse goiter, multinodular goiter, or single thyroid nodule).

Keywords: Hashimoto's thyroiditis, Graves' disease, goiter, and thyroid nodules.

INTRODUCTION:

Thyroid disorders are the most common endocrine problem next to diabetes mellitus. Thyroid disorders affect women more compared to men. Thyroid glands secrete, store, and release triiodothyronine (T3) and thyroxine (T4). The hormone T4 gets converted into T3 at the tissue level and produces its effect. Iodine is necessary for the production of thyroid hormones. Iodine in food is trapped by the thyroid gland and is utilized in hormone production—pituitary and hypothalamus control thyroid gland hormone secretion. TRH from the hypothalamus modulates through the pituitary to produce TSH, which controls thyroid hormone production. If T4 and T3 are low, TSH levels increase to stimulate the thyroid gland to secrete more hormones. T3 and T4 hormones have a profound effect on the body. Almost all the tissues are stimulated, and body metabolism is increased. T3 and T4 affect the cardiovascular system, GI tract, brain, metabolism, weight, bone, etc. With increased T4 and T3, tachycardia, diarrhea, hyperglycemia, cholesterol-lowering, increased infant growth rate, normal brain development, and sexual function. Undiagnosed hypothyroidism in infants affects physical and bony growth and damages brain growth. If untreated, it leads to permanent damage.

Thyroid functions are affected by congenital absence of thyroid glands, autoimmune thyroid disease, surgical removal, infiltrative diseases, and after radiation to the neck. Drugs like amiodarone, lithium, interferon-alpha, and interleukin 2 prevent thyroid glands from making hormones and causing hypothyroidism.

Pituitary damage by tumor radiation or surgery can affect thyroid glands and cause secondary hypothyroidism.

For brain maturation and brain function, thyroid hormone is necessary. Thyroid diseases like hypothyroid can cause lethargy, hyporeflexia, depression, memory impairment, weight gain, dry skin, and constipation, along with dyslipidemia. Hyperthyroidism produces weight loss, tremors, irritability, and hyperreflexia. Glucose intolerance can also be caused by hyperthyroidism.

CASE REPRESENTATION:

A 55-year-old housewife presented to the emergency department with complaints of excessive drowsiness decreased oral intake, and a complete inability to open her eyes for three days. She also had difficulty getting up

from the squatting position in the last three years, combing her hair, and dressing. These difficulties were gradually progressive with no diurnal variations or periodic fluctuations. She complained of increased sensitivity to cold, dryness of skin, increased hair loss, hoarseness of voice, and easy fatigability. She had no history of intake of any prescribed or over-the-counter medications. She also had no history or family history of any major illnesses.

Her detailed physical examination revealed periorbital puffiness and dry, coarse skin with a lemon-yellow tinge. She suffered from extreme psychomotor retardation and only responded to simple motor commands. Complete bilateral ptosis was present, although cranial nerve function examination was normal with no facial abnormality. External ocular movements were normal in all directions. No tremors or fasciculations were elicited. Pupillary reaction and pupil size were normal bilaterally.

Muscle bulk of the upper and lower limbs was normal. Power in the proximal muscle groups in the upper and lower limbs was decreased (3/5). However, it was normal in the distal group of muscles (5/5). Deep tendon reflexes, although present, showed a delay in the relaxation phase. There was no sensory deficit in any limb. The pulmonary, cardiovascular, and abdominal systems examination was unremarkable and did not reveal any abnormality.

Her baseline investigations revealed a hemoglobin level of 11 g/dl with a normal cystic, typical chromic picture on peripheral blood smear with normal serum ferritin levels, vitamin B12, and folic acid. Renal and liver function analyses along with serum electrolytes were within normal limits. Blood sugar levels and HbA1c were also normal. The lipid profile was deranged with increased serum triglyceride and cholesterol levels. Further investigations revealed a state of primary hypothyroidism. Her CPK levels were raised to 410 U/L (60-174 U/L). Serum cortisol and Serum ACTH levels were normal.

The electromyographic study revealed a myopathic pattern of weakness in the proximal muscle groups of both upper and lower limbs. Repeated nerve stimulation for ocular muscles was negative for myasthenia gravis. Acetylcholine receptor and anti-skeletal antibody levels were not elevated. Cranial MRI and abdominal ultrasound were normal. Contrast-enhanced computed tomography of the chest ruled out any evidence of thymoma. Ultrasound of the thyroid gland revealed small lobes and isthmus with normal blood flow on color doppler. Echocardiography showed grade I diastolic dysfunction with normal left ventricular ejection fraction. Detailed ophthalmic examination ruled out any local, infectious, inflammatory, or aponeurotic causes of ptosis.

The patient was started on 100 µg (1.6 µg/Kg body weight) of thyroxine daily. After one week of treatment, the patient dramatically improved, with complete recovery of ptosis and significant improvement in her consciousness level. Her muscle power also improved, although some residual weakness was still persistent. After two weeks of hospitalization on the same treatment, the patient was discharged and was advised regular monthly follow-up. Her thyroid function tests were repeated every two months with necessary titration of the dose until her free T4, and TSH levels were restored to normal range. Her sense of well-being improved gradually, with a definitive improvement in muscle power. After six months of follow-up, all her symptoms about hypothyroidism had resolved, and she had no active complaint.

IODINE: I recommended amounts of iodine foods, including the following: **Fish (such as cod and tuna)**, seaweed, shrimp, and other seafood, which are generally rich in iodine. Dairy products (such as milk, yogurt, and cheese) are significant iodine sources in American diets.

VEGETABLES: Blueberries, tomatoes, bell peppers, and other foods rich in antioxidants can improve overall health and benefit the thyroid gland. Eating foods high in B vitamins, like whole grains, may also help.

CONCLUSION:

This is further supported by the fact that the patient completely recovered with thyroxine replacement therapy alone.

REFERENCES:

1) Duyff RF, Van den Bosch J, Laman DM, Van Loon BJ, Linssen WH. Neuromuscular findings in thyroid dysfunction: A prospective clinical and electrodiagnostic study. J Neurol Neurosurg Psychiatry. 2000;68(6):750-5. PMID: 10811699.
2) Rao S, Katiyar B, Nair K, Misra S. Neuromuscular status in hypothyroidism. Acta Neurol Scand. 1980;61(3):167-77. http://dx.doi.org/10.1111/j.1600-0404.1980.tb01479.x.
3) Unnikrishnan AG, Menon UV. Thyroid disorders in India: An epidemiological perspective. Indian J Endocrinol Metab. 2011;15(2): 78–81. http://dx.doi.org/10.4103/2230-8210.83329.
4) Woeber KA. Update on the management of hyperthyroidism and hypothyroidism. Arch Intern Med. 2000;160(8):1067-71. http://dx.doi.org/10.1001/archinte.160.8.1067.
5) Singh SK, Singh KK, Sahay RK. Images in medicine: Hashimoto's thyroiditis with orbitopathy and dermopathy. J Postgrad Med. 2000;46(4):286-7. PMID: 11435659.
6) Jayakumar RV. Hypothyroidism. J Indian Med Assoc. 2006;104(10): 557-60,562. PMID: 17380819
7) Hallengren B. Hypothyroidism-clinical findings, diagnosis, therapy. Thyroid tests should be performed on broad indications. Lakartidningen. 1998;95(38):4091-6. PMID: 9772803.
8) Barohn R. Metabolic myopathies. In: Cecil RL, Goldman L, eds. Textbook of Medicine. 21st ed. Orlando, Fla: Harcourt Health Sciences; 2000.
9) Miyake I, Hiromatsu Y. Hypothyroid myopathy. Ryoikibetsu Shokogun Shirizu. 2001;(36):294-7. PMID: 11596395.
10) Lo YL, Ho SC, Koh LK, Kooh DH. EMG myokymia as a cause of ptosis in hypothyroidism. Eur J Neurol. 2003;10(1):87-90. http://dx.doi.org/10.1046/j.1468-1331.2003.00544.x.