USANA Technical Bulletin

Hyperthyroidism

Description

- The thyroid is a gland located slightly below the larynx in front of the trachea. The three primary hormones produced by the thyroid gland are thryoxine (T_4), triiodothyronine (T_3), and calcitonin. Thryoxine (T_4) and triiodothyronine (T_3) maintain the level of metabolism in the cells that is optimal for their normal function. Calcitonin regulates the calcium and phosphate levels in the blood.
- Hyperthyroidism is characterized by excessive secretion of the thyroid hormones. This increases the basal metabolic rate, causing an increased demand for food to support this metabolic activity.[1]

Causes

- Because there are many types of hyperthyroidism, this disease has many causes, not all of which are known. Graves disease is thought to be caused by genetic and immunologic factors. In a person with latent hyperthyroidism, excess iodine or stress may precipitate clinical hyperthyroidism.[2]

Types

- There are various forms of hyperthyroidism: Graves disease, toxic adenoma, thyrotoxicosis factitia, functioning metastatic thyroid carcinoma, TSH-secreting pituitary tumor and subacute thyroiditis.

At Risk

- Those with a family history of thyroid problems have an increased risk of developing hyperthyroidism. Disease onset is usually between the ages of 30 and 40 years.

Prevention and Management

- Persons with hyperthyroidism may need to avoid excess iodine.
- Supportive measures include adequate nutrients and vitamins.[2]
- Vitamin E may protect the hyperthyroid heart against lipid peroxidation by mechanisms that may be independent of its function as an antioxidant.[3]
- In experimental animals, folate helped bring total cholesterol to near normal.[4]

Sources of Additional Information

- http://medhlp.netusa.net/general/THYROID.TXT

Abstracts

Viswanathan G; Nair CP. Altered metabolism of folates and lipids in hyperthyroid and hypothyroid rats. Indian J Biochem Biophys 1996 Aug;33(4):311-4. Effect of excess folate on the metabolism of folate and plasma lipids in thyroid stressed animals was studied. Administration of excess dietary folate to hyperthyroid rats did not affect the decreased in vivo histidine oxidation in these animals, but it increased the hepatic tetrahydrofolate content. Hypothyroid rats had higher plasma total cholesterol (TC), high density lipoprotein cholesterol (HDLC) and trigylcerides (TG) with no change in post heparin lipolytic activity (PHLA). In hyperthyroidism both TC and HDLC were decreased in plasma and PHLA levels were elevated. On administration of excess dietary folate to hyperthyroid rats HDLC levels were not altered but PHLA and TC levels became near normal, while TG levels remained elevated.

References

[1] Taber's Cyclopedic Medical Dictionary. 16th ed. Philadelphia:F.A. Davis Company;1985 p 869.
[2] Diseases. Springhouse (PA):Springhouse Publishing;1993. p 1003-6.
[3]Venditti P, De Leo T, Di Meo S. Vitamin E administration attenuates the tri-iodothyronine-induced modification of heart electrical activity in the rat. J Exp Biol 1997 Mar;200 (Pt 5):909-14.
[4] Viswanathan G, Nair CP. Altered metabolism of folates and lipids in hyperthyroid and hypothyroid rats. Indian J Biochem Biophys 1996 Aug;33(4):311-4.