

Case Report

Hypothyroidism : a treatable cause of erectile dysfunction

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Sexual dysfunction is a common yet ignored part of medical consultation. Thyroid abnormalities is associated with sexual dysfunction most of which rectifies after normalising thyroid levels. This may reduce the need for other hormone replacement .We present a case of inconsumous marriage in a patient of hypothyroidism which was treated with thyroxine replacement.

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Key words : Hypothyroidism, erectile dysfunction, thyroxine replacement, hyperprolactinemia, sexual dysfunction.

A 27 year old male came with his 24 year old wife for consultation for an inconsumous arranged marriage. The marriage was on the verge of breaking point because the husband was unable to achieve adequate erection.

On examination, weight was 83 kg, he was normotensive and physical examination was normal. Patient had appropriate secondary sexual characteristics developed. However he appeared very slow in his response time to any question.

His routine blood investigation came back normal.

Free t3- 0.89Pmol/L (4-8.3), free t4-3.5Pmol/L (12-22), TSH >100 uIU/ml (0.27-4.2) TPO antibodies -positive.

Prolactin -31ng/ml (6-28), Total Testosterone -3.3 ng/ml (3-10.6)

Patient was started on thyroxine 50 mcg every morning and psychosexual counselling was given to the couple.

1st Follow up at 2 weeks - Patient started feeling generally better. His weight reduced by 1 kg. His thyroxine dose was increased to 75mcg.

2nd Follow up at 4 weeks – Weight was 80 kg. His speech and alertness level was within normal limits

Free t3- 4.8Pmol/L (4-8.3), free t4-12.2Pmol/L (12-22), TSH- 80 uIU/ml (0.27-4.2), Prolactin -27ng/ml (6-28).

3rd follow up at 18 weeks – Weight was 77 kg at thyroxine 75mcg.

Free t3 -6.3Pmol/L (4-8.3), free t4 -15.6Pmol/L (12-22), TSH- 5.6mIU (0.27-4.2), Total Testosterone -5.9ng/ml (3-10.6).

He was been able to consummate his marriage. Patient was lost to follow up for 8 months after that.

4th follow up at 1 year – a happy married couple. He is on 75mcg thyroxine. Free t3 -6.9Pmol/L (4-8.3), free t4 -17.2Pmol/L (12-22), TSH- 3.8mIU (0.27-4.2), Prolactin -19ng/ml (6-28), Total Testosterone -7.2ng/ml (3-10.6). To sum up, patient sexual dysfunction was corrected on thyroid replacement which improved the other hormone parameters by itself.

Thyroid Physiology :

Thyroid hormone production is regulated by the Hypothalamus – Pituitary – Thyroid (HPT) axis. Upon secretion from the hypothalamus, Thyrotropin releasing hormone (TRH) binds to re-

- Check thyroid function in every patient with erectile dysfunction.
- Thyroid abnormalities are associated with Hypoactive sexual disorder, Erectile dysfunction and ejaculatory problems.
- Normalise thyroid levels first before treating any other Endocrine abnormality.

ceptors, on the anterior pituitary gland, where Thyroid Stimulating Hormone (TSH) is stored. The released TSH directly stimulates thyroid epithelial cells to signal the release for T3 and T4¹.

The Hypothalamus – Pituitary –Gonadal axis (HPG axis) lies parallel to the HPT axis. The hypothalamus regulates the release of hormones from the anterior pituitary via the gonadotropin releasing hormone (GnRH). The anterior pituitary is responsible for the production of luteinizing hormone (LH) and follicle stimulating hormone (FSH). LH stimulates leydig cells in the testicles to produce testosterone².

FSH works in concordance with LH and directly acts on Sertoli cells and secretes paracrine molecules which are needed for spermatozoa maturation. Physiological changes in the HPT axis leads to changes in the HPG axis. The manner in which these two axis are integrated is not fully understood.

Low levels of T3 and T4 leads to a rise of TRH. TRH directly stimulates the release of TSH and prolactin. Increased prolactin inhibits the release of GnRH. This results in the decrease of LH and FSH affecting the testosterone levels and spermatogenesis³. The direct effect of hypothyroidism, if any on the testis has not been clearly defined

DISCUSSION

Erection is basically a spinal reflex that can be initiated by recruitment of penile afferents but also by visual, olfactory and imaginary stimuli. The generated nervous signals will influence the balance between the contractant and relaxant factors, which control the degree of contraction of penile smooth muscles and, thus, determine the functional state of the penis.

Neurogenic nitric oxide is still considered the most important factor for immediate relaxation of penile vessels and corpus cavernosum. However, endothelially generated nitric oxide seems essential for maintaining erection. Endothelial dysfunction can contribute to ED in several patient subgroups⁴.

In animal models, hypothyroidism or thyroidectomy has been found to cause depletion of Endothelium Derived Relaxant Factor

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(EDRF) thereby causing very feeble contraction of the cavernosum muscle. Thyroxin treatment produced contraction proportionate to the concentrations of PGE1 and Sildenafil; providing evidence that the erectogenic actions of both PGE1 and Sildenafil are possible only in the presence of adequate thyroid hormone level⁵.

In addition, in conditions associated with reduced function of nerves and endothelium, such as aging, hypertension, smoking, hypercholesterolemia and diabetes, circulatory and structural changes in the penile tissues can result in arterial insufficiency and defect muscle relaxation⁴. Hypothyroidism is associated with these conditions.

In a multicentric study of hypothyroid men, the prevalence of Hypoactive Sexual Disorder Delayed ejaculation, and Erectile Dysfunction was seen to be 64.3% and of Premature Ejaculation was 7.1%⁶. By using a validated SHIM 5-item questionnaire, based on the International Index of Erectile Function questionnaire, it has been seen that approximately 80% of patients with thyroid dysfunction had erectile dysfunction(ED), compared with 34% of controls. Erectile dysfunction was severe in 37.5%. After restoration of euthyroidism, 30% of the patients had ED, which was similar to controls⁷.

Autoimmune hypothyroidism in men can be characterized by sexual and mood disturbances⁸. Men with overt hypothyroidism obtained lower scores in all five domains of IIEF-15, while men with subclinical hypothyroidism only in erectile function. L-thyroxine improved erectile function and normalized intercourse satisfaction, orgasmic function, sexual desire and overall satisfaction in autoimmune hypothyroidism, as well as normalized erectile function in subclinical hypothyroidism⁸. Six months of thyroxin improved the BD scores(Beck Depression Inventory-Second Edition - BDI-II) of autoimmune hypothyroid patients.

Screening for thyroid dysfunction in men presenting with ED is recommended, whereas specific treatment for ED should be postponed in such patients for at least 6 months after achieving euthyroidism because the latter might be responsible for ED⁷.

Possible mechanism through which hypothyroidism causes

erectile dysfunction :

Hormonal

Hyperprolactinemia causing low testosterone

Vascular (causing reduced penile blood flow)

Hypertension

Antihypertensives like bblocker

Hypercholesterolemia

Endothelial damage

Neurological

Depression

Reduced spinal reflex

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