

CASE SERIES: COMPENSATED HYPOTHYROIDISM PRESENTED AS SIMPLE GOITRE

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ABSTRACT

A lady of age 23 years from Babukhan, Rangpur came to our centre for estimation of thyroid hormones (tri-iodothyronine, T_3 & thyroxine, T_4) and thyrotropin (thyroid stimulating hormone, TSH) on 4 Oct. 2001. She had high TSH, but normal T_3 & T_4 which is known as compensated hypothyroidism. She had a small goitre and was given thyroxine tablets 50 to 100 micrograms (mcg) per day, but her TSH level was found to be very high on 19 Feb. 2002, most probably due to irregular thyroxine intake (Table 1). She had a fine needle aspiration cytology (FNAC) from her neck mass on 30 April 2002 which revealed benign nature of her goitre. She stopped thyroxine in January 2003 and her goitre aggravated as confirmed by thyroid scintigraphy using technetium ($Tc-99m$) under computerized gamma camera on 26 July 2003 (Fig. 1). She was advised to start thyroxine again.

TABLE 1 : Hormones levels

Date	T3 nmol/L	T4 nmol/l	TSH mIU/L
4 Oct. 01	1.8	76	11
19 Feb. 02	2.8	65	100

Normal ranges : T3 = 0.8 - 3.16 nmol/L

T4 = 64.5 - 152 "

TSH = 0.3 - 6 mIU/L

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Fig.1 Scintigraphy showing enlarged thyroid.

DISCUSSION

When iodine deficiency tends to become increasingly severe, it is evidenced first by a drop in T_4 along with a stationary serum T_3 level or even a compensatory rise, owing to the stimulation by TSH. Although the effects of this situation on certain target organs are still insufficiently documented, the individual does not seem to be clinically affected, and retains euthyroidal status. This is known as compensated hypothyroidism.¹ Thyroxine is used in diffuse non-toxic goitre, initially 50-100 mcg daily, preferably before breakfast, adjusted in steps of 50 mcg every 3-4 weeks until normal metabolism is maintained (usually 100-200 mcg/day); where there is cardiac disease, initially 25 mcg/day or 50 mcg on alternate days, adjusted in steps of 25 mcg every four weeks.² The combination of normal free thyroxine index (FTI) with modest elevation in TSH is considered diagnostic of subclinical hypothyroidism and consistent with a failing thyroid gland. Serum T_3 may be normal in subclinical or mild hypothyroidism. Titrating and fixing the long-term replacement dose of thyroxine is best done on clinical grounds. TSH may also be used to titrate dosage; a persistently elevated TSH indicates lack of adequate thyroxine replacement except in rare patients with longstanding primary hypothyroidism or

TSH-secreting pituitary tumors who may still secrete TSH despite becoming euthyroid. TSH determinations may also be falsely elevated in rare patients exposed to rabbits who develop antibodies to the rabbit immunoglobulin used in the radioimmunoassay,³ but our patient had high TSH due to inadequate replacement of thyroxine.

REFERENCES

1. Thilly CH, Bourdoux P, Contempre B, Swennen B, Strategies for combating endemic goitre and iodine deficiency disorders (IDD). 1988 No.175/176 Children in the tropics. International Children's Centre, Paris pp. 5-68.
2. British National Formulary No. 32 Sept. 1996, p. 297. British Medical Association and Royal Pharmaceutical Society of Great Britain, London.
3. Safrit HF. Thyroid disorders. In Fitzgerald PA (ed.): Handbook of clinical endocrinology. 1986 Jones Med. Pub. Greenbrae, CA pp. 122-169.