stimulating hormone -0.01 mIU/l (normal 0.3–4.5), thyroglobulin antibody – 86.4 IU/ml (normal 0–20), and thyroid peroxidase antibody -- 134 IU/ml (normal 0–15). Other investigations revealed serum prolactin -- 4 ng/ml (normal 0–15), LH 2.6 IU/l (normal 0–7), FSH 4.6 IU/l (normal 2–10) total testosterone 345 ng/dl (normal 300– 1100), and estradiol 16.5 μ g/dl (normal 5–25). Figure 2 showed markedly increased thyroid gland vascularity with inferno pattern suggestive of thyrotoxicosis.[1] Increased uptake on pertechnate scan confirmed the diagnosis and other serology, and biochemistry results were normal. He was diagnosed to be a case of thyrotoxicosis with gynecomastia, and treated with carbimazole (30 mg/day), and propranolol (40 mg/day). Free thyroid hormones returned to normal after 2 months and gynecomastia showed gradual regression.

Gynecomastia, as a cutaneous feature of thyrotoxicosis, is seen in 10–40% of cases. However, gynecomastia as a presenting feature of thyrotoxicosis is reported in less than 10 cases till date.^[2] Gynecomastia develops in thyrotoxicosis

Bilateral gynecomastia: A rare presentation of thyrotoxicosis

Sir,

Gynecomastia is an uncommon cutaneous feature of thyrotoxicosis, and is due to altered balance between estrogens and testosterone. The incidence of gynecomastia in thyrotoxicosis is decreasing over the past two decades due to early diagnosis of the condition. A 33-year-old man was referred to our department for evaluation of progressive gynecomastia involving both breasts for over 6 months duration. He denied history of decreased secondary sexual characters, galactorrhea, erectile dysfunction, and decreased libido. There was no history of drug intake and the patient gave history of 3 kg weight loss over past 6 months. He denied other symptoms of thyrotoxicosis. Physical examination revealed bilateral gynecomastia with no galactorrhoea [Figure 1]. Thyroid gland was not enlarged and ophthalmic examination was normal. His secondary sexual characteristics, testis, fundi, visual fields, and other systemic examination were all normal.

His thyroid hormonal profile revealed total triiodothyronine – 258.6 ng/dl (normal 60–175), total thyroxine- 24.4 µg/dl (normal 4–11.5), thyroid



Figure 1: Bilateral gynecomastia

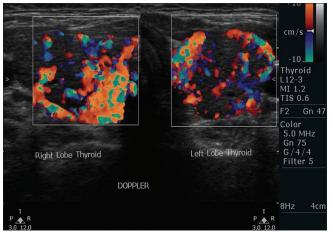


Figure 2: USG thyroid showing inferno pattern

due to physiological increase in estrogen in comparison to testosterone. The factors responsible for this altered sex hormone ratio are increase in sex hormone binding globulin, increased peripheral aromatization of androgens, increased glandular secretion of estrogens, and elevated LH.^[3] Other associated etiologies like hyperprolactinemia and hypogonadism also contribute toward gynecomastia in thyrotoxicosis.^[4] The gynecomastia resolves completely after control of thyrotoxicosis, with complete reversal of histological changes. Our case highlights the uncommon presentation of a common disease, and highlights the need to screen for thyrotoxicosis in all cases of bilateral gynecomastia.

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