Gynecomastia As The Initial Presentation Of Thyrotoxicosis – A Case Report

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Citation

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Abstract

Gynecomastia is a well recognized association with hyperthyroidism. The reported percentage is widely variable. Some studies have report the percentage to be close to 40% [1,2] while others report it to be about 10% [3,4]. This difference is apparently due to the difference in the criteria used to define gynecomastia. But, thyrotoxicosis presenting with gynecomastia as the initial manifestation is very rare. Only about 10 case reports are found in literature. We present the case of a 26 year old man with thyrotoxicosis who had presented initially with gynecomastia.

INTRODUCTION

Gynecomastia is a well recognized association with hyperthyroidism. The reported percentage is widely variable. Some studies have report the percentage to be close to 40% [1,2] while others report it to be about 10% [3,4]. This difference is apparently due to the difference in the criteria used to define gynecomastia. But, thyrotoxicosis presenting with gynecomastia as the initial manifestation is very rare. Only about 10 case reports are found in literature.

We present the case of a 26 year old man with thyrotoxicosis who had presented initially with gynecomastia.

CASE REPORT

The patient is a 26-year-old white male who presented with bilateral breast swelling, more pronounced on the right, for about 3.5 months. It was associated with some pain in both breasts. There was no bleeding or fluid discharge from the nipple area. He denied noticing any lumps or bumps over the breast area, around the axillary region or around his testicles. Further questioning revealed that he also had complaints of increased sweats, which had been present over the last few months. In addition he was also jittery and anxious and occasionally had some palpitations too. He did not have any major weight loss or recent history of cold/viral infections or any pain in the neck area. He was not taking any medications or over the counter herbs or steroids. He did not have any difficulties with his sexual functions. He had no other significant past medical history.

On examination, he was anxious and jittery and had a heart rate of 100. His thyroid gland was diffusely enlarged and was soft in texture with no appreciable nodularity. There was no inguinal lymphadenopathy or bruit. He had normal testicular size with no appreciable nodules. His penis was normal in size without any ulcerations or lesions. Axillary and pubic hair distribution was normal and there was no inguinal lymphadenopathy. Breast examination revealed diffusely enlarged breasts bilaterally. Glandular tissue could be palpated bilaterally underneath the nipple area. No nodularity could be appreciated bilaterally and no fluid could be milked from the nipple areas. There was no axillary lymphadenopathy.

Complete blood count, metabolic profile and liver functions were essentially normal. Thyroid functions revealed grossly reduced TSH and increased free T3 and T4 (Table-1).

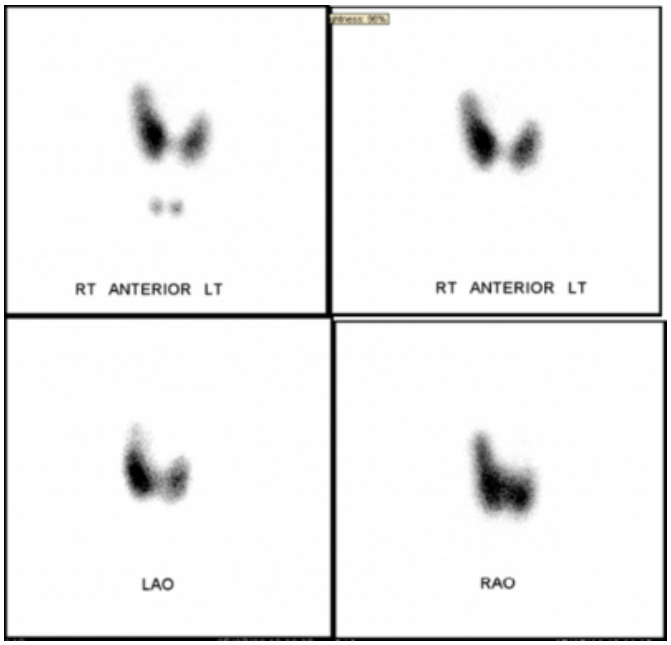
Figure 1

Thyroid Function Tests	
TSH	<0.01 (0.40 - 4.50) mU/L
Free T3	>2000 (230 – 420) pg/dL
Free T4	7 (0.8 - 1.8) ng/dL

Thyroid uptake study revealed slight enlargement of right upper lobe and homogeneous uptake in both lobes. There were no hot or cold nodules. Six hour uptake was computed at 95.8% (normal range 10-25%) and twenty-four hour

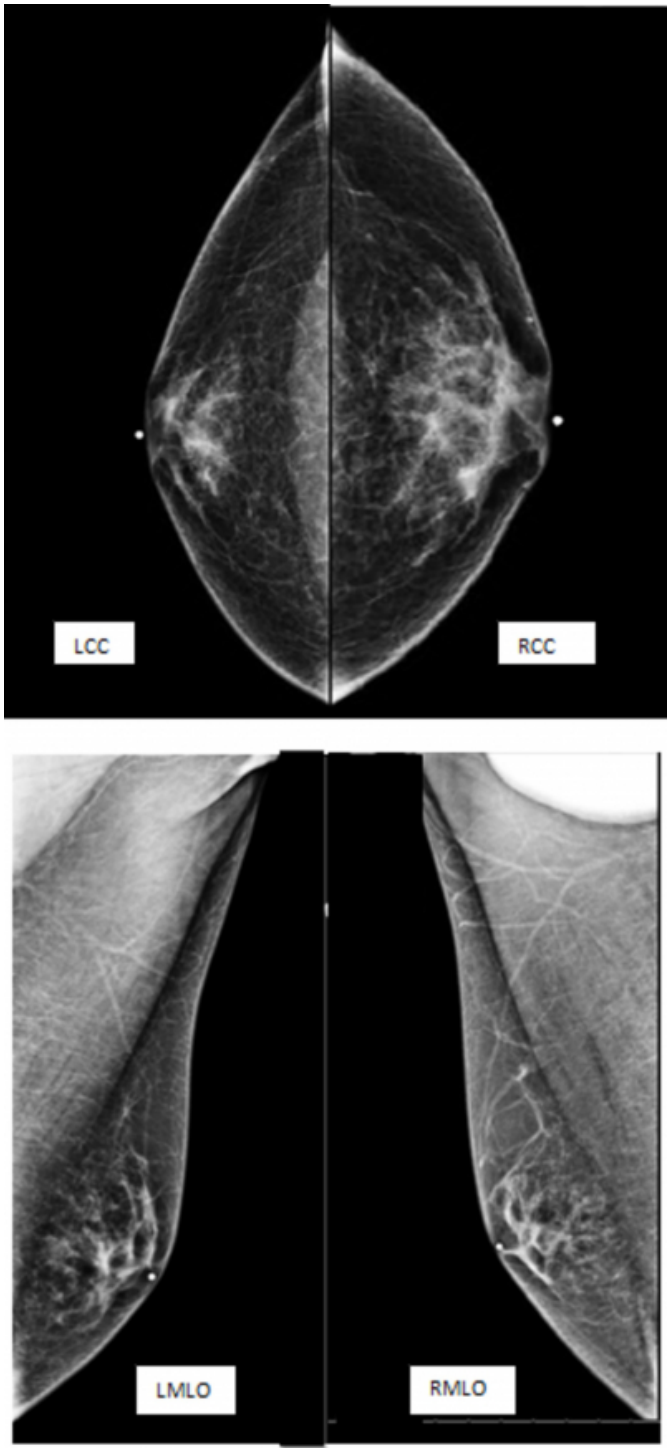
uptake was computed at 102.5% (normal range 10-40%) (Figure -1)

Figure 2



Mammography showed increased density in the retro-areolar area bilaterally, representing ductal prominence secondary to gynecomastia. No evidence of tumor mass density was seen. No abnormal calcifications were seen. Figures – 2 and 3

Figure 3



Tests of endocrine functions revealed elevated LH, estradiol and a grossly elevated testosterone levels. But free testosterone was in fact reduced and sex hormone binding globulin (SHBG) was abnormally elevated. Table- 2

Figure 4

Tbl2. The patient was sent for a radio Iodine ablation of the thyroid.

Testosterone	2286 (241- 827) NG/DL
Luteinizing Hormone	12.4 (1.5 - 9.3) MIU/ML
Follicle Stimulating Hormone	3.2 (1.6 - 8.0) MIU/ML
Estradiol	81 (13-54) PG/ML
HCG	< 2 (<5)
Percentage free testosterone	0.47 (1.50 - 2.20 %)
SHBG	166 (7 – 49) nmol/L

DISCUSSION

Gynecomastia is a benign proliferation of the glandular tissue of the male breast. It could be diagnosed on exam as a palpable mass of tissue at least 0.5 cm in diameter. This proliferation is brought about by an increase in the ratio of estrogen to androgen activity. Gynecomastia could be physiological or pathological. Physiologic gynecomastia, which resolves spontaneously in most cases, is common in infant and adolescent boys. Various etiologies of gynecomastia in an adult patient is shown in table- 3

Figure 5

Etiology	Percentage
Persistent pubertal gynecomastia	25 percent
Drugs	10 to 25 percent
No detectable abnormality	25 percent
Cirrhosis or malnutrition	8 percent
Hypogonadism	primary (8 percent), secondary (2 percent)
Testicular tumors	3 percent
Hyperthyroidism	1.5 percent
Chronic renal insufficiency	1 percent

In hyperthyroidism, hepatic production of SHBG is increased, leading to a high blood level of SHBG [5]. This in turn reduces the concentration of free testosterone. The total testosterone and serum LH levels are often elevated, contributing to increased estradiol relative to testosterone production by Leydig cells. There is also enhanced aromatization of testosterone to estradiol and of androstenedione to estrone in extraglandular tissues. Gynecomastia results from the combination of decreased free androgen levels combined with the overproduction of estrogens.

Our patient clearly had thyrotoxicosis, from his very low TSH (<0.01) and elevated free T3 (>2000) and T4 (>7). His total testosterone was grossly elevated (2286), but percentage of free testosterone was reduced (0.47) due to elevated elevated SHBG (166). There was also an increase in LH and estradiol. All these findings correlate with the pathophysiology of gynecomastia in hyperthyroidism.

CONCLUSION

Hyperthyroidism is known to be the cause of about 1.5% of adult gynecomastia. An increase in the ratio of estrogens to androgens is believed to be the cause of glandular proliferation in gynecomastia, which is brought about in a hyperthyroid patient by the increase in SHBG, causing the reduction of free testosterone. Although it is very rare for hyperthyroidism to present with gynecomastia as the initial manifestation, making the diagnosis is very important as it is a completely reversible condition.

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