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Utility of Hormonal Assay in Gynecomastia: A Retrospective Study

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Abstract

Aims: Gynecomastia is a common breast condition in males occurring due to varied etiological factors. Evaluation of such patients includes detailed medical history, clinical examination, specific blood tests, imaging and tissue sampling. Endocrinological workup forms the main part of investigation, the role of which is lacking in current literature. The objective of this study is to assess the productivity of such evaluation in current practice.

Methods: A five year retrospective study was conducted of patients presenting to department of Plastic Surgery at Tertiary care hospital were review with respect to Endocrinological results, treatment and cost.

Results: The mean age at presentation being 21.48 years (Range 15–30 years) with mean history of complaints since 1.84 (SD 0.84) years at presentation. 46.15% (n = 24) patients were overweight and 36.53% (*n* = 19) patients being obese. Unilateral case were 14 % (*n* = 7) and bilateral being 86% (*n* = 42). The median body mass index is 28.47. Four patients had positive family history with 7 of those patients having unilateral presentation. 1 patient was diagnosed with secondary gynecomastia due to anabolic steroids.

Conclusions: Gynecomastia in young is more commonly idiopathic in nature and Endocrinology evaluation should be judiciously used when indicated by proper history taking and thorough clinical examination.

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Keywords: Gynecomastia; Endocrinology; Evaluation.

Introduction

Gynecomastia is characterized by benign male breast development. It is the most common breast condition in male, usually bilateral. One third of males are affected with gynecomastia during their lifetime, majority during adolescence. Pseudogynecomastia is common in obese men, and consists of fat deposition without glandular proliferation. Majority of such cases are idiopathic in nature but it is critical to recognize underlying pathology to direct treatment. Gynecomastia in adolescent and young men in particular leads to significant embarrassment and social impediments leading to decreased participation in physical activities, social activities. Treatment of gynecomastia is based on severity and underlying pathology. Most of pubertal gynecomastia being idiopathic is managed by counselling and or with estrogen blocking medications. Liposuction and subcutaneous mastectomy remains the main stray of management in patients with psychosocial embarrassment or high grade gynecomastia.[1–4]

Disruption of testosterone and oestrogen balance has been attributed to pathogenesis of gynecomastia[5], hence endocrinology evaluation of patients forms the first line of investigations in such patients. It is however, currently unknown what role endocrinology evaluation has in assessment of adolescent and young men when it's idiopathic in nature. The objective of the study is to evaluate the role of such investigations in clinical practice.

Materials and Methods

A retrospective observational study conducted from January 2015 to till June 2018 at tertiary care hospital of patients presenting to department of Plastic Surgery. Data was extracted from medical records stored in Hospital information system, an online data system management. Patients diagnosed with Gynecomastia were included in the study. Patients with incomplete data were excluded. Total of 63 patients fulfilled the inclusion criteria, 11 patients had incomplete data hence excluded. Endocrinological evaluation included Testosterone levels, Lutenizing hormone, Follicular stimulating hormone, prolactin and thyroid stimulating hormone and clinical parameters were assessed (Table 1).

Table 1: Patient database. BMI: Body Mass Index, LH: Luteinizing Hormone, FSH: Follicle Stimulating Hormone, TSH: Thyroid Stimulating Hormon.

S No	Age in years	Duration in years	Family history	BMI kg/m²	Side	Testosterone in ng/dl	LH in IU/L	FSH in mIU/ml	Prolactin ng/ml	TSH in mIU
1	27	2	nil	28.25	Bilateral	334.8	5	7.09	5.78	0.68
2	23	1	nil	27.8	Right	232.83	6.38	8.54	5.44	4.46
3	25	2	nil	36.3	Bilateral	335.72	7.35	6.1	7.2	2.15
4	19	3	nil	26.2	Bilateral	309.36	5.68	7.45	10.15	3.24
5	18	3	nil	27.4	Bilateral	353.61	2.96	2.7	6.93	0.92
6	16	2	nil	26.76	Bilateral	227.89	3.97	6.76	10.01	1.29
7	17	2	yes	24.5	Left	245.18	3.57	3.22	3.46	2.32
8	19	3	nil	27.32	Bilateral	585.61	4.73	5.48	6.31	2.49
9	26	4	nil	23.74	Bilateral	349.89	3.44	4.69	5.15	1.94
10	16	1	nil	25.83	Bilateral	258.29	3.84	6.74	8.64	2.73
11	23	1	nil	28.12	Bilateral	260.39	4.36	3.74	6.44	1.24
12	17	1	nil	26.36	Bilateral	177.37	4.21	3.07	5.93	1.36
13	21	2	yes	23.4	Right	104.4	5.72	5.25	6.41	2.45
14	22	2	nil	24.28	Bilateral	228.72	3.28	4.63	6.04	1.48
15	18	1	nil	30.16	Bilateral	314.45	7.42	9.7	6.26	2.39
16	23	3	nil	28.42	Bilateral	361.36	4.28	7.15	4.15	1.82
17	30	2	nil	29.24	Bilateral	546.72	1.61	3.02	3.23	2.12
18	24	0.5	nil	34.83	Bilateral	349.82	2.65	5.16	5.26	0.9
19	18	2	nil	27.11	Bilateral	302.7	4.17	4.72	6.18	2.01
20	26	1	nil	33.15	Bilateral	426.14	1.73	2.49	6.8	1.15
21	18	2	nil	28.52	Bilateral	571.83	2.65	2.5	9.12	2.22
22	22	1	nil	31.72	Bilateral	522.9	7.69	7.21	5.47	3.01
23	20	2	nil	26.14	Left	173.69	4.27	6.26	5.28	2.74
24	22	1	nil	33.25	Bilateral	441.16	3.37	2.55	6.5	2.88
25	15	2	nil	31.45	Bilateral	558.51	6.42	9.37	10.31	1.82
26	20	1	nil	33.82	Bilateral	442.51	6.05	4.32	7.12	1.28
27	24	1	nil	32.74	Bilateral	724.27	4.12	6.26	9.14	2.13
28	25	2	nil	26.52	Bilateral	384.26	5.44	7.38	11.14	1.74
29	23	2	nil	23.18	Bilateral	624.4	3.56	3.37	8.82	0.86
30	27	1	yes	31.42	Right	206.39	4.93	5.03	6.44	2.02
31	19	2	nil	34.66	Bilateral	518.21	6.77	5.23	7.12	3.14
32	26	1	nil	27.06	Bilateral	411.73	2.82	4.68	7.38	2.67
33	20	1	yes	29.64	Bilateral	542.72	3.76	4.11	6.54	1.84
34	23	3	nil	36.42	Bilateral	477.83	5.25	6.64	8.84	1.35
35	25	2	nil	26.61	Bilateral	374.95	4.74	5.15	4.7	4.67
36	18	2	nil	30.72	Bilateral	426.36	6.82	3.48	8.16	2.13
37	25	1	nil	27.44	Bilateral	330.45	5.5	6.12	7.11	0.94
38	29	1	nil	32.76	Bilateral	518.35	8.28	7.49	5.63	1.16

S No	Age in years	Duration in years	Family history	BMI kg/m²	Side	Testosterone in ng/dl	LH in IU/L	FSH in mIU/ml	Prolactin ng/ml	TSH in mIU
39	21	2	nil	28.15	Bilateral	585.61	4.73	5.48	6.31	2.49
40	29	1	nil	27.37	Left	619.04	4.04	4.54	8.66	1.73
41	16	2	nil	33.45	Bilateral	367.14	8.93	5.73	9.14	0.72
42	22	1	nil	27.83	Bilateral	375.38	3.82	2.82	7.32	3.42
43	20	1	nil	30.76	Bilateral	937.52	9.82	8.73	6.18	2.15
44	18	2	nil	36.12	Bilateral	648.2	8.26	4.63	4.34	1.57
45	25	2	nil	34.33	Right	278.64	4.89	5.27	10.31	3.22
46	23	1	nil	26.62	Bilateral	673.25	6.53	3.68	8.36	2.17
47	17	2	nil	29.77	Bilateral	838.52	2.34	8.22	7.74	2.31
48	21	1	nil	28.16	Bilateral	375.35	8.34	3.64	8.91	1.42
49	21	1	nil	31.24	Bilateral	282.73	6.57	5.16	6.47	3.12
50	15	3	nil	33.9	Bilateral	862.27	2.83	6.34	13.26	2.05
51	17	2	nil	32.18	Bilateral	572.47	4.63	7.46	8.12	0.66
52	23	2	nil	30.63	Bilateral	712.23	6.48	7.26	11.56	2.11

Results

A total of 63 patients presented to department of plastic surgery over the duration of study of which 40 patients underwent surgery, Liposuction and 12 patients were managed conservatively, 11 patients excluded due to incomplete details.

The mean age at presentation being 21.48 years (Range 15–30 years) with mean history of complaints since 1.84 (SD 0.84) years at presentation. 46.15% (n = 24) patients were overweight and 36.53% (n = 19) patients being obese. Unilateral case were 14% (n = 7) and bilateral being 86% (n = 42). The median body mass index is 28.47. Four patients had positive family history with 7 of those patients having unilateral presentation. 1 patient was diagnosed with secondary gynecomastia due to anabolic steroids.

Discussion

Gynecomastia is a multifactorial condition associated with imbalance between estrogen and androgen. The estrogen/androgen imbalance is attributed to raised levels of estrogen secreted by the testes or adrenal glands, extraglandular aromatization of estrogen precursors, decreased estrogen degradation, exposure to estrogen-like chemicals or exogenous estrogens and use of drugs that cause displacement of more estrogen than androgen from Sex Hormone-Binding Globulin (SHBG).[6] Most of the estrogens (80%) are produced by peripheral conversion of two precursors, androstenedione and testosterone, respectively in estrone and estradiol, under the influence of the enzyme aromatase, which plays a pivotal role in male secretion of estrogens. Aromatase activity increases both with age and with elevation of the body mass index.

According to Braunstein[7], almost two-thirds of the patients have physiological GM (approximately 25%), no underlying detected abnormality (idiopathic, approximately 25%) or drug induced breast development (up to 20%). The frequencies of some of the remaining causes have been estimated as follows: Cirrhosis, 8%; primary hypogonadism, 8%; testicular tumors, 3%; secondary hypogonadism, 2%; hyperthyroidism, 1.5%; and renal disease, 1%.

A detailed history, with attention given to age, medications, duration and onset of breast enlargement, symptoms of tenderness or pain, recreational drug use and anabolic steroid use, is crucial. Serum assaying of the following hormones: Testosterone, free (bioavailable) testosterone, estradiol, hCG, LH, FSH, prolactin, T3, T4 and TSH. Testosterone, free (bioavailable) testosterone and LH are routinely advised to determine the aetiology.[8,9]

In our retrospective study of three and half years in 52 patients, patients presented were adolescent and young men with average age of presentation being 21.48 years with onset of complaints of mean duration of about 1.84 years, only 0.02% of patients had abnormal sex hormone profile. However, positive blood workup did not change the management of the patients underwent liposuction for correction. These sex hormone assays did not yield any additional information to the management of the patient in rest 99.98% of cases. Considering the rising cost of endocrinological evaluation and poor yield in gynecomastia cases especially in adolescent and young men, these investigations

should be ordered in clinically relevant cases like unilateral presentation, positive family history, and history of anabolic steroid usage meticulous clinical exam suggesting secondary cause.

Conclusion

Gynecomastia in young is more commonly idiopathic in nature and Endocrinology evaluation should be judiciously used when indicated by proper history taking and thorough clinical examination.

Key Message: Gynecomastia affects two third of adolescent and young male, majority of which are idiopathic in nature, and patients undergo routine endocrine evaluation. Endocrinology evaluation should be judiciously used when indicated by proper history taking and thorough clinical examination.

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