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Research Article

Gynecomastia in German soldiers: etiology and pathology

Gynäkomastie bei Soldaten der Deutschen Bundeswehr: Ätiologie und Pathologie

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Abstract

Background: We found a high incidence of patients with gynecomastia in the German Ministry of Defense Guard Battalion in Berlin. For this reason, we conducted the present study to investigate etiological and pathological aspects of this condition.
Methods: Within six years, a total of 211 patients underwent surgery for gynecomastia. We compared this group of patients with a control group of healthy males without signs or symptoms of gynecomastia. The two groups were matched for median age.
Results: The groups showed significant differences ($p<0.05$) in serum testosterone, free triiodothyronine (fT3), LH (luteinizing hormone) and prolactin levels and in body mass index (BMI). In addition, there was a highly significant correlation between left-sided gynecomastia and membership in the Guard Battalion.
Conclusions: We found differences in hormone blood levels between gynecomastia patients and a control group. Moreover, gynecomastia was predominantly seen on the left side in guard soldiers. A possible explanation is the mechanical impact of the carbine against the left side of the body during rifle drills.

Keywords: gynecomastia, male breast, soldiers' disease

Zusammenfassung

Soldaten der Deutschen Bundeswehr im Wachbataillon in Berlin zeigten eine hohe Inzidenz an Gynäkomastien. Dies veranlasste uns zur Durchführung der vorliegenden Studie. In einem Zeitraum von sechs Jahren führten wir 211 Operationen an Patienten mit Gynäkomastie durch. Dieses Kollektiv wurde mit einer altersangepassten Gruppe von Männern ohne Gynäkomastie verglichen. Die beiden Gruppen zeigten signifikante Unterschiede in folgenden Parametern: Serum-Testosteron, freies Tetrajodthyronin (fT4), Prolaktin, Lutenisierendes Hormon (LH) und im Body-Mass-Index (BMI). Ferner zeigte sich eine hochsignifikante Korrelation zwischen der Tätigkeit als Wachsoldat und einer linksseitigen Gynäkomastie. Eine mögliche Erklärung für diese erhöhte Inzidenz unilateraler Gynäkomastie bei Wachsoldaten liegt in den repetitiven Karabineranschlägen an die linke Brust beim Ausführen des formalen Protokolls im Wachdienst.

Schlüsselwörter: Gynäkomastie, männliche Brust, Soldaten-Krankheit

Introduction

Cases of gynecomastia have been reported for as many as 2600 years. Paul of Aegina (AD 625–690), for example, provided detailed descriptions of patients with this condition [1]. Since then, therapeutical options [1], [2], [3], [4], [5], [6], [7], [8], [9], [10], [11], [12] and hypotheses regarding the pathogenesis of gynecomastia [1], [3], [4], [5], [6], [8], [12], [13], [14], [15], [16], [17], [18], [19], [20] have been repeatedly discussed in the scientific literature.

The present study was conducted to investigate the increased incidence of left-sided gynecomastia in members of the German Ministry of Defense Guard Battalion who perform ceremonial duties in Berlin. We carried out a controlled prospective study to obtain significant evidence for or against the hypothesis that gynecomastia can be induced by the mechanical impact of the carbine against

the left side of the body during drills. In accordance with the relevant literature, we analyzed clinical parameters and hormone levels and compared the patients with a control group. Differences were assessed for statistical significance.

Methods

In 2010 we retrospectively collected over a six and a half year period a total of 221 soldiers from military units including the Guard Battalion and other facilities in and around Berlin who presented to their unit surgeons with clinical manifestations of gynecomastia. At the same time, we established a control group of healthy males without signs or symptoms of gynecomastia. The two groups were matched for median age. The unit surgeons were instructed to complete a questionnaire covering all relevant aspects. Patients with a histologically confirmed diagnosis of gynecomastia were followed until discharge from hospital.

The following parameters were assessed for each patient: year of data acquisition, last name, first name, guard soldier yes/no, age, gender, body height, body weight, body mass index (BMI), duration of symptoms, previous surgery (if yes, when and where performed), pain on palpation of the right/left breast, diameter of gynecomastia/swelling on the right/left side in cm, secretion, signs and symptoms of inflammation, radiography of the sella turcica in the presence of clinical evidence of abnormalities or hormone changes, medications, weight of resected tissue from the right/left breasts, histological diagnosis, malignancy, presentation to the urological department for a testicular examination.

Hormone studies: follicle-stimulating hormone (FSH), luteinizing hormone (LH), alpha-fetoprotein (AFP), beta-human chorionic gonadotropin (beta-HCG), carcinoembyronic antigen (CEA), prostate-specific antigen (PSA), estrogen, prolactin, progesterone, testosterone, thyroid-stimulating hormone (TSH), free triiodothyronine (fT3), free thyroxine (fT4), sodium, potassium, calcium, chloride, aspartate aminotransferase (ASAT), alanine transaminase (ALAT), gamma-glutamyltransferase (GGT), lactate dehydrogenase (LDH), alkaline phosphatase (AP), amylase, lipase, glucose, creatinine, urea, bilirubin, total protein, and albumin.

The Mann-Whitney rank sum test for independent samples was used to determine if there were statistically significant differences between the gynecomastia group and the control group. A p-value <0.05 indicated statistical significance.

Laboratory results were compared with reference data obtained from the German Society of Clinical Chemistry and Laboratory Medicine. The control group consisted of healthy males between 20 and 24 years of age. The gynecomastia group and the control group were matched for median age.

Results are expressed as medians and percentiles, which are represented by box plots. Means and standard deviations are not reported. Microsoft Excel 2007 was used for statistical analysis.

Results

Our results confirmed the existence of a correlation between left-sided gynecomastia and membership in the Guard Battalion in Berlin. A comparison of gynecomastia patients revealed a highly significant difference between patients from the Guard Battalion and patients from other military units and facilities. Figure 1 shows the absolute numbers of gynecomastia patients from the Guard Battalion in Berlin (n=35) and the other gynecomastia patients (n=186) whose left breast was not exposed to mechanical impact. It clearly reveals that the majority of patients from the Guard Battalion underwent surgery for left-sided gynecomastia. A comparison of the laboratory values and other results obtained for the guard soldiers and the control group showed major differences ($p<0.05$) in terms of age, height, weight, body mass index (BMI), testosterone, luteinizing hormone (LH), prolactin, and free thyroxine (fT4). In the following, the five last-mentioned parameters will be analyzed in more detail. The median BMI was 23.99 in the gynecomastia group and 23.49 in the control group ($p<0.0462$) (Figure 2) which is most probably without clinical relevance. The gynecomastia patients had a testosterone level of 4.65 ng/ml compared to a level of 6.56 ng/ml in the control group (Figure 3). Reference values were obtained from the German Society of Clinical Chemistry and Laboratory Medicine and range between 2.4 ng/ml and 12 ng/ml. The p-value was less than 0.0000011. The median LH level was 3.84 mIU/ml in the gynecomastia group and 3.2 mIU/ml in the control group (Figure 4). Normal values range between 2 mIU/ml and 10 mIU/ml. The difference was statistically significant ($p<0.0455$). The gynecomastia patients had a median prolactin level of 9.5 ng/ml compared to 8.2 ng/ml in the control group (Figure 5). The difference was again statistically significant ($p<0.0326$). Reference values for prolactin range from 1.61 ng/ml to 18.77 ng/ml. The median fT4 level was 1.06 in the gynecomastia group and 1.2 in the control group (Figure 6). This difference between the two groups was significant ($p<0.0139$). Reference levels range between 0.71 ng/dl and 1.85 ng/dl.

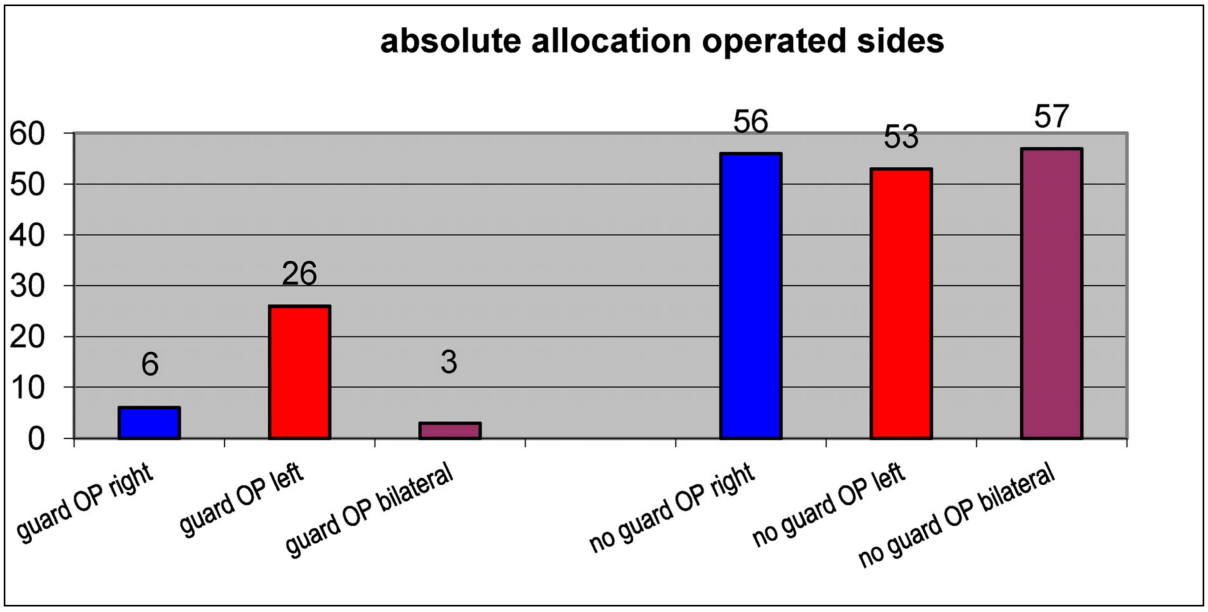

Figure 1: Distribution of right-sided, left-sided and bilateral gynecomastia in patients from the Guard Battalion (n=35) and patients from other military units or facilities (n=186, p<0.0001)

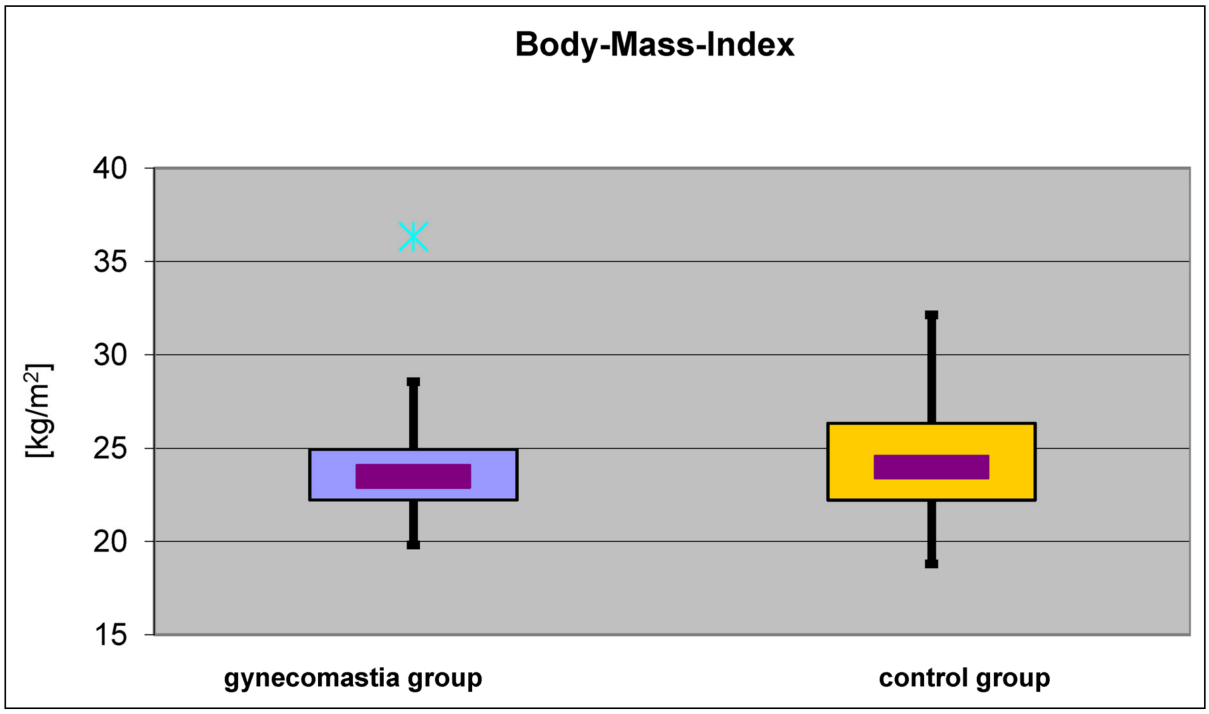

Figure 2: Distribution of body mass index (BMI) scores in the control and gynecomastia groups

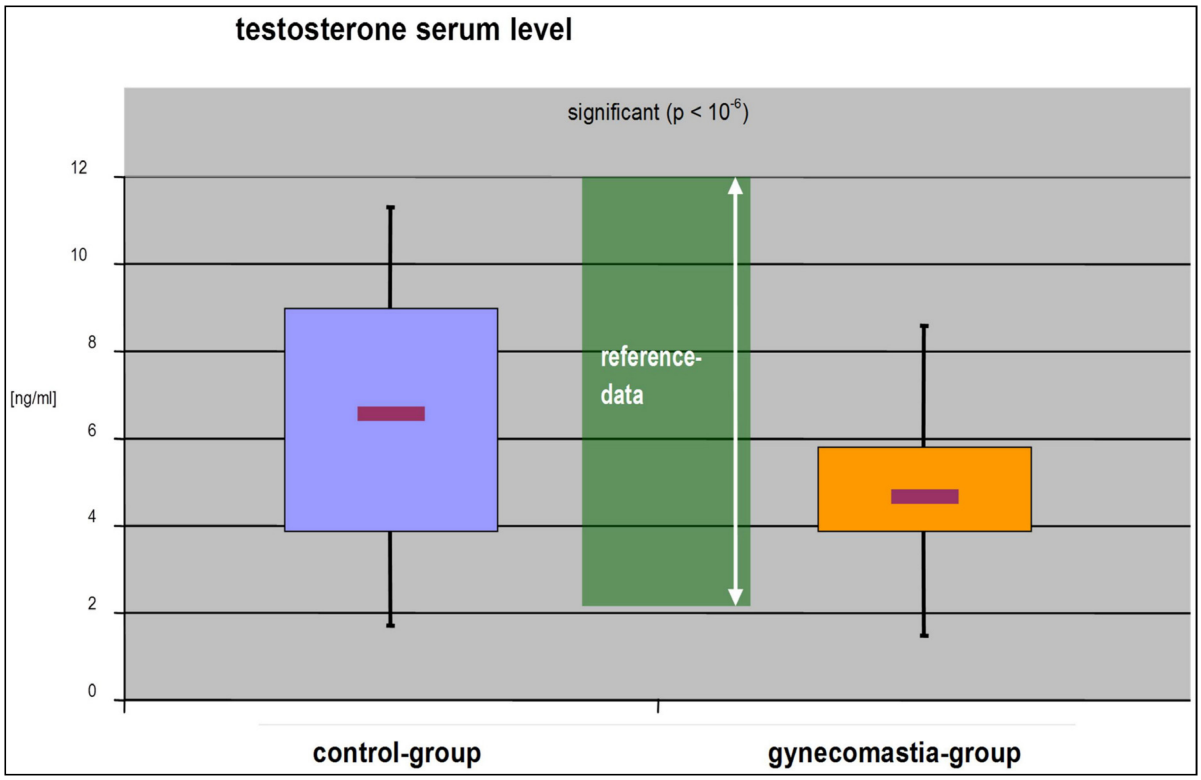

Figure 3: Testosterone reference range and distribution of serum testosterone levels in the control and gynecomastia groups

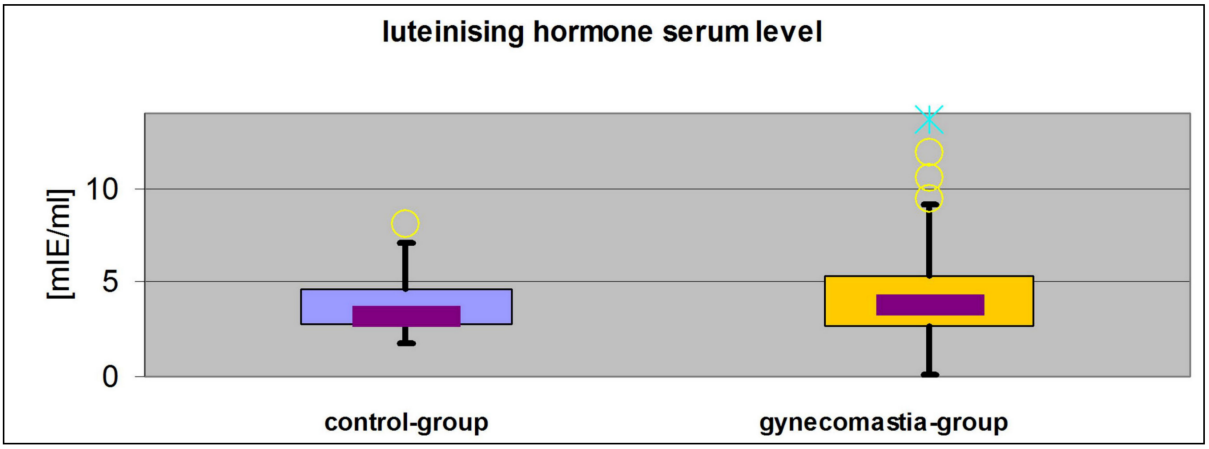

Figure 4: Luteinizing hormone (LH) reference range and distribution of serum LH levels in the control and gynecomastia groups

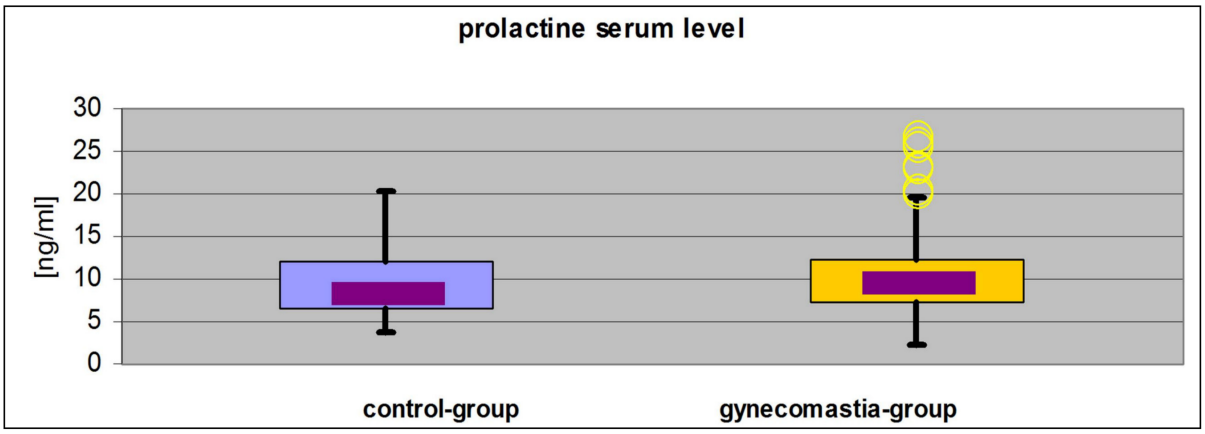

Figure 5: Prolactin reference range and distribution of serum prolactin levels in the control and gynecomastia groups

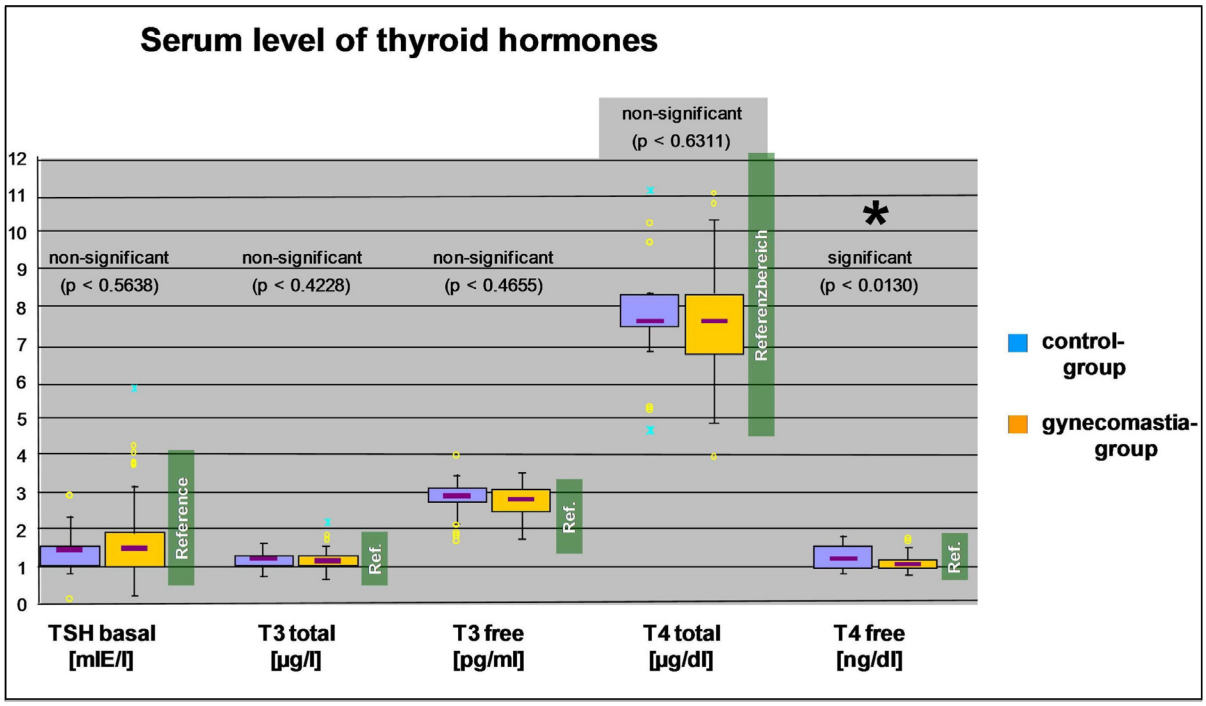

Figure 6: Distribution of thyroid hormone levels. The only significant difference was found for free T4 (p<0.013). (TSH = thyroid-stimulating hormone, T3 = triiodothyronine, T4 = thyroxine)

Discussion

We conducted this study in order to investigate the question of whether there is an increased incidence of left-sided gynecomastia in members of the German Ministry of Defense Guard Battalion in Berlin. A possible explanation is the mechanical impact of the carbine against the left side of the body during the drills that these soldiers regularly perform as part of their ceremonial duties. Our study demonstrated that the difference between patients from the Guard Battalion and other patients was highly significant (p<5.2*10E-22). A review of the literature revealed only a few older publications in which gynecomastia was attributed to a fundamentally mechanical cause, for example a thoracic trauma [21]. The hypotheses that were established in those publications were, however, not further investigated. As a result, there are no experimental studies identifying possible mechanisms at the cellular level that might induce gynecomastia mechanically. Our findings may now provide new impetus for further research into cytocontractile elements.

Other reports of gynecomastia that can be found in the literature describe the well-known physiological causes of this condition [3], [14], [18], [22]. In our study, almost all cases of gynecomastia were found in an age group that is not typically affected. For this reason, the presence of a malignant tumor must always be excluded [3], [14], [18], [22] or drug-induced gynecomastia must be considered [16], [23].

As expected from our review of the literature, an analysis of the laboratory values revealed significant differences between the gynecomastia patients and the control group in terms of BMI, testosterone, LH, prolactin, and fT4. High body weight is associated with increased aromatase activity in fat cells, which, in turn, leads to an increased conversion of testosterone to estrogen [3], [13], [20] and thus to a feminization of males. This explains why there is an increased incidence of gynecomastia among patients with high body weight and thus with elevated levels of estrogen. At the same time, it should be noted that it is not easy to differentiate gynecomastia from lipomastia. Many studies describe simple palpation as a method of distinguishing these two conditions. In patients with gynecomastia, palpation is reported to reveal the presence of firm tissue especially in the region of the anterior axillary fold [15], [19]. Further criteria and especially parameters for an objective differentiation between the two conditions are not mentioned. Experience on the part of the examining physician plays an important role in diagnosis as does a subjective evaluation of symptoms on the part of the patient.

Against this background, it is not surprising that gynecomastia patients had lower testosterone levels than the control group. This can be explained by increased aromatase activity in patients with high body weight. The presence of an estrogen-secreting tumor must, however, always be considered and a routine examination of the external genitalia, including both inspection and palpation, is indispensable. In addition, another possibility is the presence of peripheral testosterone receptor resistance, which, however, usually becomes clinically manifest during childhood or puberty. Anti-androgen therapy, which is used, for example, in the management of patients with prostate carcinoma, should be taken into consideration, too. In this context, it is important to note that testosterone binds to sex hormone-binding globulin (SHBG) in the circulating blood. This binding can be competitively re-

versed by certain medications, as a result of which testosterone levels may be too high or too low.

Routine diagnostic tests usually include the measurement of luteinizing hormone (LH) levels. In our study, however, patients with gynecomastia showed higher LH levels than the control group, which cannot be explained at this stage. Although an elevated LH level theoretically leads to an increased production of testosterone, we found a higher rate of gynecomastia. A possible explanation is an increase in the aromatization of testosterone in the presence of obesity. This would be associated with elevated estrogen levels that might explain the rate of gynecomastia.

Especially the ratio of LH to testosterone and beta-HCG may play an important role in differential diagnosis (e.g. in association with germ cell tumors).

Prolactin is not directly involved in the pathogenesis of gynecomastia. Our review of the literature revealed, however, that patients with an elevated prolactin level showed an increased incidence of gynecomastia [6]. In patients with increased prolactin levels, the presence of a prolactinoma of the pituitary gland should always be excluded by diagnostic imaging, for example magnetic resonance imaging (MRI) or computed tomography (CT). Empirical evidence suggests, as does our study, that patients with elevated levels of prolactin have a higher risk of developing gynecomastia. The literature, however, does not offer a pathogenetic explanation.

Hyperthyroidism, as evidenced by a decreased thyroid-stimulating hormone (TSH) level, is associated with a direct stimulation of peripheral aromatase by free T4 and T3. As a result, aromatization of testosterone to estrogen increases. In addition, hyperthyroidism increases SHBG, which binds free testosterone. Since bound testosterone is biologically ineffective, there is a relative lack of testosterone. This explains the increased incidence of gynecomastia in patients with hyperthyroidism. In our study, gynecomastia patients had lower fT4 levels than the control group. Their fT3 levels were, however, higher. This possibly reflects a less relevant etiogenetic role of thyroid function in the development of gynecomastia in the patient population investigated here.

Our review of the literature revealed that gynecomastia was almost always associated with an imbalance between estrogen and testosterone, which was attributed to different pathomechanisms [12], [13], [17]. Our study supports the hypothesis that gynecomastia can be induced by mechanical trauma. Excessive estrogen action relative to testosterone action then leads to an enlargement of or an increase in tubular structures in the region of the breast [3], [4], [6]. There is general agreement that increased hyalinization and an increased amount of collagen fibers are seen within two to four years after the occurrence of gynecomastia. Once this transformation process is completed, conservative treatment such as the regulation of the hormone status or estrogen deprivation therapy is unlikely to be successful and surgical resection of excess breast tissue is the only effective treatment [7], [8], [11].

Notes

Competing interests

The authors declare that they have no competing interests.

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