HORMONAL ANALYSIS AND DELAYED HYPERSENSITIVITY REACTIONS IN IDENTICAL TWINS WITH SEVERE ACNE

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Abstract. Identical twins aged 17 and another pair aged 21 are described. One pair had febrile ulcerative conglobate acne; the other, cystic acne. The location of acne, the type of the lesions and the course of the disease were very similar in the two twins of each pair. The testosterone levels of the 17-year-old pair varied and their acne was in the active stage, while the 21-year-old pair had high testosterone levels and their acne was abating. The 17-year-old pair had negative Mantoux reactions and they reacted negatively to DNCB sensitization. The authors suggest that acne skin may have a certain genetically determined local factor, e.g. hormone receptor, which gives rise to acne in a certain hormonal situation.

Key words: Acne cystica; Acne conglobata; Identical twins; Delayed hypersensitivity; Hormonal analyses

When we examine the aetiological factors in acne, we cannot ignore the role of heredity. Hans Nierman (8) compiled the existing data on acne in identical twins in his book "Zwillingsdermatologie" in 1964. By 1964, altogether 95 pairs of identical twins with acne had been reported. In 97.9% of the cases, both twins were affected. The same work includes a review of 131 dizygotic twin pairs, 54.2% with acne in both twins. Severe acne in identical twins has been described by Hazen (3), one pair with concordance, and by Pena (11), also one pair with concordance.

Severe, acne, often feverish acne, is a relatively rare disease. Cases of ulcerative conglobate acne have been described by Pautrier (10), Kelly & Burns (4), Ström (14), Lippert & Post (7) and Palatsi (9).

In examining the aetiology of this state, Palatsi (9) and Rajka (12) demonstrated that the patients had a weakness in their cell-mediated immunity. In the aetiological studies, much attention has been paid to the hormonal relations in acne. Recently, it was found that even high serum testosterone levels do not show any definite correlation to the manifestation of acne (5, 6) and the hormonal influences on the sebaceous activity are very complex. The skin itself has steroid metabolism. Sansone et al. (13) noted that the acne skin converts testosterone to dihydrotestosterone 2-20 times faster than does the normal skin. Bonne et al. (1) suggested that there are androgen receptors in acne skin, but not in normal skin.

The genetic character of acne has not been sufficiently emphasized during recent years and we therefore wish to present two pairs of identical twins with severe acne, examined in our clinic in 1977.

CASE REPORTS

Case 1
17-year-old male identical twins who had been active athletes and were in good physical trim. Mild acne in symmetrical area appeared when they were 15 years old, became exacerbated 2 years later and turned into febrile ulcerative conglobate acne. Purulent lesions appeared in symmetrical areas on the chest (Fig. 1), on the back (Fig. 2), fewer lesions on the face and upper arms. The lesions tended to be necrotic and one of the twins developed ulcerations on the chest and back. Both youths were feverish, about 37.3-38°C, for several weeks. Leukocytosis, elevated E.S.R., and somewhat elevated alkaline phosphatase levels were the abnormal laboratory values. Both patients were given tetracycline (long-term treatment, 250 mg/day), local benzoylperoxide creams and the one with ulcerations also received prednisolone for 2 weeks in the febrile phase of the disease. The therapeutic outcome was only moderate, and the disease is still active in both brothers.

Case 2
21-year-old male identical twins, who had recessive hereditary aminoaciduria, aspartylglucosaminuria, or AGU-disease. Both had had cystic acne for about 5 years, and the disease was now abating. Cysts and abscesses had been particularly frequent on the temporal areas of the face, and papules and pustules on the cheeks (Fig. 3). Both also had folliculitis and scars symmetrically on the anterior and posterior surfaces of the thighs. The chest and back were asymptomatic. Occasionally the patients had had fever and leukocytosis and cysts had been lanced and treated with antibiotics.

LABORATORY FINDINGS

Urinalyses were normal; Waaler-Rose, latex, and anti-nuclear antibodies were negative.
Fig. 1. Identical twins aged 17. Location of acne on the chest.

DELAYED HYPERSENSITIVITY REACTIONS
The 17-year-old twins were tested with the following antigens: Tuberculin (PPD) 10, 1.0, 0, and 0.1 TU/ml, allergic extract trichophytin and oidiomycin “0” (Hollister Stier Laboratories) in dilutions 1:50. The reactions were read at 48 and 72 hours and proved negative. Dinitrochlorobenzene (DNCB) sensitization was made with 10% DNCB in acetone and the challenge test with 0.1% DNCB was carried out 2 weeks later, but the results proved negative.

The delayed reactions of the older twins were not examined.

HORMONE ANALYSES
Hormonal analyses were carried out in the hormone laboratory of our hospital. The hormones were assayed by routine commercial radioimmunoassay methods. The se-
Fig. 3. Identical twins aged 21. Acne cysts symmetrically on the temporal areas and on the chin.

Rum testosterone was assayed by the method described by Hammond et al. (2). Specific attention was paid to the serum testosterone values. The 17-year-old pair gave several samples during 4 months of observation. The initial values were 4.5 and 12.7 nmol/l, after two months 13.5 and 21 nmol/l and 17 and 22 nmol/l after 4 months. The 21-year-old boys gave only two samples, which were 21.2 and 24.3, and 22.0 and 23.2 nmol/l. After 4 months observation the younger boys had still leukocytosis and elevated E.S.R. (40 and 36) and elevated serum alkaline phosphatase though their clinical condition was better.

DISCUSSION

Particular attention in these two pairs of identical twins is drawn to the similarity of location of the

Table 1. Laboratory findings

<table>
<thead>
<tr>
<th>Blood and serum analysis</th>
<th>Normal value limits</th>
<th>17-year-old twins with ulcerative conglobate acne</th>
<th>21-year-old twins with cystic acne</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Hb</td>
<td>135–165 g/l</td>
<td>106</td>
<td>128</td>
</tr>
<tr>
<td>Leukocytes</td>
<td>4–10×10⁹/l</td>
<td>14.3</td>
<td>8.9</td>
</tr>
<tr>
<td>E.S.R.</td>
<td></td>
<td>88</td>
<td>38</td>
</tr>
<tr>
<td>ALT</td>
<td>&lt;40 U/l</td>
<td>14</td>
<td>16</td>
</tr>
<tr>
<td>y-GT</td>
<td>&lt;50 U/l</td>
<td>9</td>
<td>8</td>
</tr>
<tr>
<td>Alkaline phosphatase</td>
<td>60–250 U/l</td>
<td>520</td>
<td>507</td>
</tr>
<tr>
<td>IgA</td>
<td>1.5–5.2 g/l</td>
<td>4.7</td>
<td>3.9</td>
</tr>
<tr>
<td>IgM</td>
<td>0.3–1.4 g/l</td>
<td>2.6</td>
<td>3.1</td>
</tr>
<tr>
<td>IgG</td>
<td>8.0–19.0 g/l</td>
<td>18.5</td>
<td>16.8</td>
</tr>
<tr>
<td>AST</td>
<td>&lt;200</td>
<td>&lt;200</td>
<td>&lt;200</td>
</tr>
</tbody>
</table>

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Table II. Hormone analyses
Pathological values in italics

<table>
<thead>
<tr>
<th>Hormone</th>
<th>Normal value limits in men under 60 years</th>
<th>17-year-old twins with ulcerative conglobate acne</th>
<th>21-year-old twins with cystic acne</th>
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</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>s-Testosterone</td>
<td>11-55 nmol/l, mean 25</td>
<td>4.5</td>
<td>12.7</td>
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<tr>
<td>s-Estradiol</td>
<td>0.05-0.23 nmol/l, mean 0.14</td>
<td>0.24</td>
<td>0.24</td>
</tr>
<tr>
<td>s-Progesterone</td>
<td>0.22-1.7 nmol/l, mean 0.86</td>
<td>1.75</td>
<td>2.1</td>
</tr>
<tr>
<td>s-FSH</td>
<td>2-24 U/l, mean 18</td>
<td>15.6</td>
<td>8.6</td>
</tr>
<tr>
<td>s-LH</td>
<td>8-39 U/l, mean 18</td>
<td>6.5</td>
<td>9.3</td>
</tr>
<tr>
<td>s-Prolactine</td>
<td>2-14 µg/l, mean 7</td>
<td>4.6</td>
<td>0.9</td>
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<tr>
<td>dU 17-KGS</td>
<td>32-81 µmol/d</td>
<td>77.5</td>
<td>75.0</td>
</tr>
<tr>
<td>dU 17-KS</td>
<td>32-105 µmol/d</td>
<td>30.0</td>
<td>96.9</td>
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</table>

acne, its type and the course of the disease in the two individuals. One pair had intensely inflammatory acne of the ulcerative conglobate type, while the other had cystic acne.

Hormonal analyses showed that the 21-year-old youths had already reached a high serum testosterone level and their acne was abating. The hormonal situation of the 17-year-olds was not equally stable. testosterone values varied and their disease was active.

Delayed hypersensitivity reactions were examined only in the younger twins. The reactions were weak, which may be attributed to the active phase of the disease. This result supports the previous findings that delayed sensitivity is altered in febrile acne conglobata (9, 12).

In the light of these cases we cannot avoid posing the question what characteristic it is that is inherited and causes acne. Acne appears in areas rich in sebaceous glands. It is possible that acne patients have in these areas a genetically determined factor, which triggers acne in a certain hormonal situation. We cannot disregard the role of heredity in the location, number, and anatomical structure of sebaceous glands. But there may be other, more specific factors, such as the quality or quantity of the hormone receptors. Sansone & Reisner (13) pointed out the possibility that the local steroid metabolism is altered in acne-affected area. All these questions require further elucidation.

REFERENCES
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12. Rajka, G.: On cell-mediated immunity in Acne cong-
13. Sansone, G. & Reisner, R. M.: Differential rates of conversion of testosterone to dihydrotestosterone in acne and in normal human skin. A possible pathogene-

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