Quantitative Analysis of Digitopalmar Dermatoglyphics in Forty Primary Hypertrophic Osteoarthropathy Male Patients

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Abstract: By the quantitative dermatoglyphic analysis we have made research in 25 variables in number of epidermal ridges on palms and fingers in forty male patients with primary hypertrophic osteoarthropathy: on all ten fingers, on five fingers separately and their sum altogether, between triradii a-b, b-c and c-d on one hand and both palms, their sum on one and both palms and atd angles on one and both hands and their sum altogether. Obtained data were compared with control group of 200 healthy men from the Zagreb region in Croatia. Statistically significant differences to control by t-test we have found in 13 variables in the sense of increasing of number of epidermal ridges on second right finger their sum on five and both hands, then first, second, third, fourth and fifth finger left hand, then between triradii a-b of left hand. Decreased were atd angles on right and left palm, and both in degrees. Accordingly a polygenic system identical in some loci to polygenic system predisposing to male patients with primary hypertrophic osteoarthropathy might be found responsible for dermatoglyphic pattern development

1. Introduction

Hypertrophic osteoarthropathy or Touraine-Solente-Gole disorder, is a syndrome of the clubbing of the digits, (1) (Picture 1 and 2), periostitis of the long, tubular, bones, (the tibia is almost invariably involved at epiphysis, which distinguishes it from secondary form that typically spares the epiphysis) and arthritis (2-9) The etiology of primary hypertrophic osteoarthropathy is unknown (10). The disease is classified either as a primary (hereditary or idiopathic, PHO, or secondary, which usually has underlying disease, (cardiopulmonary, malignancies or paraneoplastic syndrome SHO. The three recognized forms of PHO are: (1) complete (clubbing, pachydermia, periostitis, in 45 % men and 18 % women), incomplete, no pachydermia, (in 50 % men and 71 % women) and (3) fruste form (prominent pachydermia with few skeletal manifestations, 5% men and 12 % women (11). PHO was first described by Friedreich 1868, as a ‘excessive growth of bone of the entire skeleton’(12) Then, Touraine-Solente-Gole described
PHO as the primary form of bone disease hypertrophic osteoarthropathy in 1935 (13), and distinguished its three known forms. This disease affects relatively more men than women (ratio around 7:9:1) (14,15). HPO represents 3% of all cases of HPO (16). Its prevalence in the general population is not exactly known, but according to Jajić I, Jajić Z, is 0.16% (17). The other skin features are, coarse skin, oily skin, eczema, thick hand and skin, leonine facies, furrowing cutis verticis gyrate (18), increased secretion of sebum, seborrhoic hyperplasia, keloid formation, thickening of ears, lips and tongue, then bone and joints features, acroosteolysis, myelofibrosis, thick toe and finger bones, widening of bone formation, arthralgia and joint effusion, hyperhidrosis, eye features, dropping eyelids, muscle discomfort and decreased facial and pubic hair (3), and it is known in animals too, its secondary form, in horse, cow, sheep, dog, cat, fowl (19). To curing PHO is by the use of non-steroidal anti-inflammatory drugs (NSAIDS), corticosteroids and infliximab for the joints symptoms, then bisphosphonates for the bone formation (they inhibit bone osteoclastic resorption) and skin manifestations by retinoids and colchicines and surgical care (20). Of our interest, is of course PHO, and its genetic background (21). With the genetics in HPO, we will start by Jajić’s finding of 44 % HLA B12 positive antigen in 75 patients affected by (22). Then, autosomal recessive PHO-2 is caused by homozygous or compound heterozygous mutation in the SLC2A1 gene on chromosome 3q22.1-q22.2. (23). PHO-1 is caused by homozygous mutation in the HPGD gene on chromosome 4q34 (24). In a review of 68 published families, 204 patients, Castori et al. (2005) found that 37 families showed autosomal dominant inheritance, 31 autosomal inheritance and in severe phenotype is often shown a mutation linked to the X chromosome (25).

2. Materials and methods

Dermograms of forty primary hypertrophic osteoarthropathy male patients were analysed in keeping with instruction provided by Miličić et al methods (26), and the most of patients with complete form according to Martinez-Lavin, Matucci-Cerinčić, Jajić and Pineda classification (5). Results were compared with 200 dermograms of phenotypically normal men from the Zagreb area, obtained from the Institute of Anthropology in Zagreb, Croatia (27). Student t-test was used to test statistically significant differences in the ridge count between patients and controls. Digitopalmar prints were taken by use of finely granulated silver-gray powder onto transparent, adhesive tape (28). Dermatoglyphic analysis should be strictly separated according to sex, because of the great impact of sex chromosomes and sex hormones on dermatoglyphic traits (29,30). Even significant sex differences have been found within control groups (27). The following 25 traits were examined by the quantitative dermatoglyphic analysis, as it has shown on Picture 4 and tables 1-3.

1. FRD1 ridge count on the first finger of the right hand, 2. FRD2 ridge count on the second finger of the right hand, 3. FRD3 ridge count on the third finger of the right hand, 4. FRD4 ridge count on the fourth finger of the right hand, 5. FRD5 ridge count on the fifth finger of the right hand, 6. TFRCD total ridge count on the all five fingers of the right hand, 7. a-b rcD ridge count between triradii a-b of the right hand, 8. b-c rcD ridge count between triradii b-c of the right hand, 9. c-d rcD ridge count between triradii c-d of the right hand, 10. TPR rcD ridge count between all triradii a-b, b-c, and c-d of the right hand all together, 11. atd D atd angle on the
right palm in degrees. 12. FRL1 ridge count on the first finger of the left hand, 13. FRL2 ridge count on the second finger of the left hand, 14. FRL3 ridge count on the third finger of the left hand, 15. FRL4 ridge count on the fourth finger of the left hand, 16. FRL5 ridge count on the fifth finger of the left hand. 17. TFRCL ridge count on all five fingers on the left hand, 18. a-b rcL ridge count between triradii a-b on the left hand, 19. b-c rcL ridge count between triradii b-c of the left hand, 20. c-d rcL ridge count between triradii c-d on the left hand, 21. TPR rcL ridge count between triradii a-b, b-c and c-d, all together on the left palm., 22. atd L atd angle on the left palm in degrees, 23. TFRC total ridge count on all ten fingers of the palms, 24. TPRC bilateral ridge count between all triradii of the palms, 25. ATDDL bilateral sum of palmar atd angles in degrees.

3. Results

Results are tabularly presented in Tables 1-3. Statistically significant differences to control were found in 13 varibales, in the sense of increasing number of epidermal ridges: on second finger, on all five right toger and in decreasing atd angle on right palm in degrees, (presented by variable FRD2, TFRC and Atd D in Table 1), then on first, second, third, fourth and fifth finger and between triradii c-d rcL and TFRLC on the all five fingers left, in the sense of increasing number of epidermal ridges and decreasing atd angle in degrees (presented by variables FRD1, FRD2, FRD3, FRD4, FRD5, TFRCD, a-b rcD, b-c rcD, c-d rcD, TPR rcD, Atd D, FRL1, FRL2, FRL3, FRL4, FRL5, TFRCL, a-b rcL, b-c rcL, c-d rcL, TPR rcL, Atd L, TFRC, TPRC, ATDDL).

Table 1. Quantitative properties of right hand digitopalmar dermatoglyphics in patients and controls

<table>
<thead>
<tr>
<th>Variable</th>
<th>Patient group n x SD</th>
<th>Control group n x SD</th>
<th>Risk p</th>
</tr>
</thead>
<tbody>
<tr>
<td>FRD1</td>
<td>40 20.70 6.33</td>
<td>200 19.38 5.63</td>
<td>0.185</td>
</tr>
<tr>
<td>FRD2</td>
<td>40 14.68 6.54</td>
<td>200 11.42 7.27</td>
<td>0.009</td>
</tr>
<tr>
<td>FRD3</td>
<td>40 13.80 6.42</td>
<td>200 11.99 6.58</td>
<td>0.111</td>
</tr>
<tr>
<td>FRD4</td>
<td>40 18.08 5.09</td>
<td>200 16.16 6.15</td>
<td>0.066</td>
</tr>
<tr>
<td>FRD5</td>
<td>40 15.15 4.79</td>
<td>200 13.64 5.16</td>
<td>0.087</td>
</tr>
<tr>
<td>TFRCD</td>
<td>40 82.35 23.14</td>
<td>200 72.57 24.65</td>
<td>0.021</td>
</tr>
<tr>
<td>a-b rcD</td>
<td>40 41.40 5.83</td>
<td>200 41.85 6.86</td>
<td>0.698</td>
</tr>
<tr>
<td>b-c rcD</td>
<td>40 28.35 4.76</td>
<td>200 28.58 5.87</td>
<td>0.814</td>
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<tr>
<td>c-d rcD</td>
<td>40 39.08 4.82</td>
<td>200 37.94 6.07</td>
<td>0.267</td>
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<td>TPR rcD</td>
<td>40 108.63 10.16</td>
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<tr>
<td>Atd D</td>
<td>40 45.00 6.89</td>
<td>200 47.43 8.27</td>
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Table 2. Quantitative properties of left hand digitopalmar dermatoglyphics in patients and controls

<table>
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<tr>
<th>Variable</th>
<th>Patient group n x SD</th>
<th>Control group n x SD</th>
<th>Risk p</th>
</tr>
</thead>
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<tr>
<td>FRL1</td>
<td>40 18.95 6.23</td>
<td>200 16.20 6.14</td>
<td>0.010</td>
</tr>
<tr>
<td>FRL2</td>
<td>40 14.03 6.23</td>
<td>200 10.76 6.78</td>
<td>0.005</td>
</tr>
<tr>
<td>FRL3</td>
<td>40 14.83 6.23</td>
<td>200 11.78 6.37</td>
<td>0.006</td>
</tr>
<tr>
<td>FRL4</td>
<td>40 18.30 5.46</td>
<td>200 16.25 6.17</td>
<td>0.038</td>
</tr>
<tr>
<td>FRL5</td>
<td>40 15.25 4.19</td>
<td>200 13.50 4.60</td>
<td>0.026</td>
</tr>
<tr>
<td>TFRCL</td>
<td>40 81.35 23.81</td>
<td>200 68.47 23.88</td>
<td>0.002</td>
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<tr>
<td>a-b rcL</td>
<td>40 42.78 5.22</td>
<td>200 53.48 7.05</td>
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<td>b-c rcL</td>
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<tr>
<td>c-d rcL</td>
<td>40 39.23 5.40</td>
<td>200 36.60 7.00</td>
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<tr>
<td>TPR rcL</td>
<td>40 109.63 10.96</td>
<td>200 109.02 14.79</td>
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<tr>
<td>Atd L</td>
<td>40 45.35 7.54</td>
<td>200 47.86 7.70</td>
<td>0.009</td>
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</table>

Table 3. Quantitative properties of digitopalmar dermatoglyphics on both hands in patients and controls

<table>
<thead>
<tr>
<th>Variable</th>
<th>Patient group n x SD</th>
<th>Control group n x SD</th>
<th>Risk p</th>
</tr>
</thead>
<tbody>
<tr>
<td>TFRC</td>
<td>40 163.70 45.99</td>
<td>200 141.03 47.44</td>
<td>0.006</td>
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<td>TPRC</td>
<td>40 218.45 20.37</td>
<td>200 217.94 27.19</td>
<td>0.910</td>
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<tr>
<td>ATDDL</td>
<td>40 90.35 13.55</td>
<td>200 96.28 14.30</td>
<td>0.046</td>
</tr>
</tbody>
</table>

The areas of quantitative analysis of digitopalmar dermatoglyphics on palm and fingers

Picture 3

The areas of quantitative analysis of digitopalmar dermatoglyphics on palm and fingers

3. Results

Results are tabularly presented in Tables 1-3. Statistically significant differences to control were found in 13 varibales, in the sense of increasing number of epidermal ridges: on second finger, on all five right toger and in decreasing atd angle on right palm in degrees, (presented by variable FRD2, TFRC and Atd D in Table 1), then on first, second, third, fourth and fifth finger and between triradii c-d rcL and TFRLC on the all five fingers left, in the sense of increasing number of epidermal ridges and decreasing atd angle in degrees (presented by variables FRD1, FRD2, FRD3, FRD4, FRD5, TFRCD, a-b rcD, b-c rcD, c-d rcD, TPR rcD, Atd D, FRL1, FRL2, FRL3, FRL4 and FRL5), and TFRLC, c-d rcL and Atd L in Table 2), then variable TFRC in the increasing number of epidermal ridges on the fingers of both hands all together, then decreasing atd angles on both hands (presented by variables TFRC and ATDDL in Table 3).
4. Discussion

To the best of our knowledge there is not even one paper dealing with dermatoglyphics in HOP except our conference presentation (31), and an attempt in palmistry (32). That is why we could not compared our research with some to. As we mentioned before there is a genetic influence in that disorder (21-25). Quantitative traits of dermatoglyphics (as a one of genetic method), are under a strong genetic influence by a few main and great number of modification gene (33). Besides, primary hypertrophic osteoarthropathy is interesting from the differential diagnostic standpoint to other seronegative spondyloarthropathies, for example, in psoriatic classical arthritis subgroup (statistically significant differences in 11 variables totally between them - seven variables on the right fingers and palm, and three on the left and only one on both, were found), not only in clinical sense and curing, but in syndrome loci on fourth chromosome. Namely, in our research of rheumatic diseases, with skin and nail manifestations, as a syndrome algodystrophicum or complex regional pain syndrome on 4p12 (34), psoriatic arthritis 4q27 (35), psoriasis 4q31 (36), we have HPO locus on 4q34. Moreover, on 4q22-23 is a locus for SMARCAD1 gen which deletes a dermatoglyphic drawing on fingers and palm and plants surfaces, Picture 4. What is a connection among them is the question of big challenge, because we performed the research by dermatoglyphics, and the cardinal gen which deletes drawing is on the same chromosome (37), Picture 5. On the sixth chromosome of HLA region we have further connections of the genetic loci for the same diseases and syndromes what is known long ago. For the algodystrophy syndrome characteristic are the next loci: A3, A29, B62, DR13, DR15, DQ1, DQ8 (38). Psoriatic arthritis, A2, B8, B27, B38, DR7, DQ1 (39), psoriasis, B13, B57 (B17), Cw6 (40). On the end, the most interesting thing is, that by very cheap genetic method, dermatoglyphic research, it seems possible to find genetic differences among the rheumatological (rheumatoid arthritis, (41), ankylosing spondylitis (42), Reiter syndrome (43), psoriatic arthritis (44, 45), and other diseases, (46-49).

5. Conclusion

It seems that polygenic system, by a few main and great number of modification genes responsible for development

The fourth human chromosome

![Genetic loci SMARCAD1, algodystrophy syndrome, psoriatic arthritis, psoriasis and primary hypertrophic osteoarthropathy in the 4 chromosome](Picture 4)
Adermatoglyphia caused by SMARCAD1 gene (in chromosome 4p22-23, taken over from the reference 37 of dermatoglyphics is identical in some loci with polygenic system for liability to primary hypertrophic osteoarthropathy. This genetic method may be used to diagnostic, preventive and even prognostic purposes.

6. Ethics

There is not any danger for the patients from this kind of research. Dermatoglyphic analysis, which is one of genetic method, is with out any harmful consequence for sick persons. The procedures are in accordance with ethical standards in scientific research at Croatian Medical Association’s Codex of Medical Ethic and Deontology, and Helsinki Declaration of World Medical Association, Edinburg, 2000.

7. Conflicts of interest

There is no conflicts of interest among the authors.

8. Acknowledgments

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9. References


