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Dermatoglyphs and Brachial Plexus Palsy

Svetislav Polovina[1], Miljenko Cvjetičanin[2], Jasna Miličić[3] and Tajana Polovina Prološčić[4]

[1] Polyclinic for Physical Medicine and Rehabilitation »M. Stojčević-Polovina«, Zagreb, Croatia
[2] Private Clinic for Physical Medicine and Rehabilitation, Zagreb, Croatia
[3] Institute for Anthropological Research, Zagreb, Croatia
[4] Department of Physical Medicine and Rehabilitation, University Hospital Osijek, Croatia

A B S T R A C T

Perinatal brachial plexus palsy (PBPP) is a handicap quite commonly encountered in daily routine. Although birth trauma is considered to be the major cause of the defect, it has been observed that PBPP occurs only in some infants born under identical or nearly identical conditions. The aim of this study was to test the hypothesis of genetic predisposition for PBPP. It is well known that digito-palmar dermatoglyphs can be used to determine hereditary roots of some diseases. Thus, we found it meaningful to do a study analysis of digito-palmar dermatoglyphs in this disease as well, conducting it on 140 subjects (70 males and 70 females) diagnosed with PBPP. The control group was composed of fingerprints obtained from 400 adult and phenotypically healthy subjects (200 males and 200 females) from the Zagreb area. The results of multivariate and univariate analysis of variance have shown statistically significant differences between the groups observed. In spite of lower percentage of accurately classified female subjects by discriminant analysis, the results of quantitative analysis of digito-palmar dermatoglyphs appeared to suggest a genetic predisposition for the occurrence of PBPP.

Key words: *dermatoglyphs, brachial plexus palsy, perinatal, genetic predisposition*

Introduction

Perinatal brachial plexus palsy (PBPP) has been traditionally classified into three types: upper plexus palsy (Erb's) affecting the C5, C6, and +/– C7 nerve roots, lower plexus palsy (Klumpke's) affecting the C8 and T1 nerve roots, and total plexus palsy[1,2]. PBPP is a handicap quite commonly encountered in daily routine. The incidence of PBPP in Croatia is 3.4/1000 newborn [3]. In Sweden it increased significantly from 1.4/1000 in 1980 to 2.3/1000 in 1994, while in Netherlands it is estimated 4.6/1000 newborn[4,5]. Although the etiology of PBPP varies, birth trauma (caused by high birth weight, vertex presentation, shoulder dystorcia) is considered to be the major cause of the defect[6–9]. Gherman and collaborators noted that brachial plexus injury may be unrelated to manipulations performed at the time of delivery and can be associated with cesarean delivery. They concluded that such palsies appear to be of intrauterine origin and more likely to persist[10]. The incidence of Erb's palsy in Pennsylvanian population is similar to that of other studies and has remained unchanged over the past 30 years, even as cesarean rate has risen from 5 to 20 %[11]. This would suggest that there is some genetic or intrauterine influence for perinatal expression of brachial plexus palsy. Any analysis to confirm these suspicions could be helpful for reduction all factors that may possibly influence damage of the brachial plexus in intrauterine development.

Dermatoglyphs are patterns made by epidermis on fingers, palms and soles. They are completely formed by 21st week of intrauterine development. The dermatoglyphic pattern of human palms and soles are individually unique and unchangeable during the life time. These are highly hereditary determined, although the exact way of inheritance is still unknown. Therefore, dermatoglyphs are informative for understanding the genetic status, as well as early disturbances in intrauterine development[12–14].

It has been observed that PBPP occurs only in some infants born under identical or nearly identical conditions, giving rise to a hypothesis on the possible genetic susceptibility of the disorder. In a number of Egyptian families with high consanguinity rate several members in successive generations were found to have PBPP[15]. In

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some cases with PBPP the patient had a history of decreased right arm movement detected by fetal ultrasound at 18 to 20 week of gestation, which coincide with development of dermatoglyphs[12,16]. With all above mentioned there is reason to assume that it would be useful to test the hypothesis of genetic predisposition for PBPP. The circumstances the development of the peripheral nerves and the development of the dermatoglyphs are the same, leading to the situation where the disturbances in the phase of embryological development could have various influences on the evolution of the palsy, simultaneously be reflected on the dermatoglyphic patterns. This reflection can be tested using the comparative analysis of dermatoglyphs of the digito-palmar complex in all of PBPP patients and healthy control groups.

Materials and Methods

Analysis of digito-palmar dermatoglyphs was examined in 140 subjects (70 males and 70 females), with PBPP. Among our patients, there were number of close relatives (three pairs consisting of – two sisters, a brother and a sister, and a mother and a daughter)[17]. Fingerprints were obtained from 400 adult and phenotypically healthy subjects (200 males and 200 females) from the Zagreb area, was used as a control group[18]. The digito-palmar prints were taken and analyzed according to the Cummins and Midlo (1961), Schauman and Alter (1976), Miličić et al. (1989) methods[12,14,19].

The analysis comprised a total of 18 quantitative variables of digito-palmar dermatoglyphics: finger ridge-counts on the right and left hand: FRCR1, FRCR2, FRCR3, FRCR4, FRCR5, FRCL1, FRCL2, FRCL3, FRCL4, FRCL5; palmar ridge counts on the right and on the left hand: a–b rc R, b–c rc R, c–d rc R, a–b rc L, b–c rc L, c–d rc L and the atd angles: atd R and atd L. The quantitative traits of digito-palmar dermatoglyphs were analyzed using descriptive statistics, multivariate and univariate analysis of variance and discriminant analysis.

Results

The results of descriptive statistics based on the comparison 18 quantitative variables of digito-palmar dermatoglyphs from PBPP patients groups (male and female) with their control groups are presented in Table 1 for males and Table 2 for females.

Multivariate analysis of variance (Table 3) showed statistically significant difference between male control group and male patients with PBPP ($F=2.21064$; $p<0.004$), as well as statistically significant difference between healthy females and female patients with PBPP ($F=2.27947$; $p<0.003$).

Univariate analysis of variance (Table 4) enabled the identification of variables, tracing the greatest contribution to this heterogeneity between the investigated groups: in males: FRCL1 ($F=4.8679$; $p<0.05$), c–d rc L ($F=4.31446$; $p<0.05$), atd R ($F=12.182$; $p<0.001$) and in

TABLE 1
DESCRIPTIVE STATISTICS FOR QUANTITATIVE DERMATOGLYPHIC TRAITS IN PATIENTS WITH BRACHIAL PLEXUS PALSY – PBPP (N=70) AND HEALTHY CONTROLS (N=200) – MALES

	PBPP		Healthy control	
Variables	X	SD	X	SD
FRCR 1	19.79	6.44	19.37	5.63
FRCR 2	11.31	7.12	11.41	7.27
FRCR 3	11.83	6.41	11.99	6.58
FRCR 4	15.33	5.89	16.16	6.15
FRCR 5	13.21	4.97	13.63	5.16
a–b rc R	40.51	6.64	41.85	6.86
b–c rc R	27.77	6.00	28.59	5.78
c–d rc R	36.34	7.12	37.94	5.98
atd R	51.57	9.32	47.42	8.27
FRCL 1	18.10	6.44	16.19	6.14
FRCL 2	11.60	6.88	10.76	6.78
FRCL 3	12.53	6.25	11.78	6.37
FRCL 4	16.16	5.63	16.24	6.17
FRCL 5	13.46	4.45	13.49	4.60
a–b rc L	42.03	5.13	43.58	7.05
b–c rc L	28.51	6.10	28.73	5.72
c–d rc L	34.54	8.16	36.62	6.84
atd L	48.86	9.10	47.86	7.70

TABLE 2
DESCRIPTIVE STATISTICS FOR QUANTITATIVE DERMATOGLYPHIC TRAITS IN PATIENTS WITH BRACHIAL PLEXUS PALSY – PBPP (N=70) AND HEALTHY CONTROLS (N=200) – FEMALES

	PBPP		Healthy control	
Variables	X	SD	X	SD
FRCR 1	20.14	4.56	17.23	5.56
FRCR 2	13.89	6.09	11.62	6.55
FRCR 3	14.43	4.66	11.44	5.31
FRCR 4	16.96	4.65	15.78	5.72
FRCR 5	14.19	4.64	12.70	4.84
a–b rc R	41.00	5.93	41.03	6.02
b–c rc R	29.06	4.75	27.31	6.01
c–d rc R	38.84	5.92	36.70	6.43
atd R	49.07	10.71	46.87	8.67
FRCL 1	17.59	4.99	14.80	5.76
FRCL 2	13.16	5.60	10.87	6.88
FRCL 3	14.09	5.18	11.58	5.72
FRCL 4	16.70	4.20	15.13	5.25
FRCL 5	14.46	4.21	12.26	4.81
a–b rc L	42.40	4.87	41.82	5.90
b–c rc L	28.89	4.95	26.90	5.67
c–d rc L	37.66	8.54	36.34	6.86
atd L	50.23	11.95	47.70	8.39

females: FRCR1 (F=14.848; p<0.001), FRCR2 (F=6.010; p<0.05), FRCR3 (F=16.611; p<0.001), FRCR5 (F= 4.6109; p<0.05), FRCL1 (F=12.382; p<0.001), FRCL2 (F=5.668; p<0.05), FRCL3 (F=9.512; p<0.005), FRCL4 (F= 5.131; p<0.05), FRCL5 (F=11.558; p<0.001), b–c rc R (F= 5.002; p<0.05), c–d rc R (F=5.374; p<0.05), b–c rc L (F= 6.090; p<0.05).

TABLE 3
MULTIVARIATE ANALYSIS OF VARIANCE FOR THE QUANTITATIVE DERMATOGLYPHIC TRAITS BETWEEN PATIENTS WITH BRACHIAL PLEXUS PALSY – PBPP AND HEALTHY CONTROLS

	F	P	df
Males	2.21064	0.004	18; 251
Females	2.27947	0.003	18; 249

TABLE 4
UNIVARIATE ANALYSIS OF VARIANCE FOR THE QUANTITATIVE DIGITO-PALMAR DERMATOGLYPHIC TRAITS BETWEEN PATIENTS WITH BRACHIAL PLEXUS PALSY – PBPP AND HEALTHY CONTROLS

Variables	F-males	F-females
FRCR 1	0.255	14.848***
FRCR 2	0.010	6.010*
FRCR 3	0.029	16.611***
FRCR 4	0.955	2.640
FRCR 5	0.351	4.619*
a–b rc R	2.000	0.044
b–c rc R	1.031	5.002*
c–d rc R	3.339	5.374*
atd R	12.182***	2.908
FRCL 1	4.867*	12.382***
FRCL 2	0.798	5.668*
FRCL 3	0.733	9.512**
FRCL 4	0.010	5.131*
FRCL 5	0.003	11.558***
a–b rc L	2.855	0.526
b–c rc L	0.068	6.090*
c–d rc L	4.314*	1.554
atd L	0.796	1.703

*p<0.05, **p<0.01, ***p<0.005

We also analyzed the differences between investigated groups using discriminant analysis. It confirmed a correct classification in 68.52% of male patients (65.7% boys with PBPP and 69.5% healthy control), while in females 65.30% (60.3% girls with PBPP and 67% healthy control) were correctly classified (Table 5).

Discussion

With the knowledge of the existing literature we have found only few studies describing similar problematic. Loesch et al. (1990) compared hand locomotor function and body structure with epidermal ridge patterns[20]. Their data showed significant correlation between hand locomotor function and dermatoglyphic characteristics, especially in men. In the second study of Philpot et al. (1995) reported a case of an infant with congenital symmetrical weakness of the upper limbs and abnormal dermatoglyphs on both palms with poorly expressed transversal crease[21]. The finding of abnormal dermatoglyphs indicates a possibility of a prenatal start of extremity weakness. The authors linked this clinical finding to possible drug toxicity in the first trimester of pregnancy.

As far as we know, there are no other data connecting about the clinical state of PBPP with dermatoglyphs. Thus, our results are not further comparable to the other findings in the known literature The methods of descriptive statistics in our study did not show observable differences in ridge counts of boys, whereas girls were found to have a greater number of finger ridges. The total number of ridges for all ten fingers TRC in males was TRC= 141.03 for healthy controls and TRC=143.31 for PBPP, while in the case for females it was TRC=131.38 in healthy controls and TRC=155.59 in PBPP girls this difference being statistically significant (F=14.49; p< 0.001). The possible differences between male and female subjects with PBPP and their control groups were determined variables. As the multivariate analysis of variance produced statistically significant differences between the observed groups, each original variable was also subjected to a univariate analysis of variance. In the case of boys with PBPP, a statistically significant difference from the control group was found for the FRCL1, c–d rc L and

TABLE 5
THE RESULT OF DISCRIMINANT CLASSIFICATION BETWEEN THE GROUP OF PATIENTS WITH BRACHIAL PLEXUS PALSY – PBPP AND HEALTHY CONTROLS

Males	N	Correctly classified	%	Incorrectly classified	%
Patients PBPP	70	46	65.70	24	34.30
Healthy controls	200	139	69.50	61	30.50
Total of correctly classified			68.52		
Females	**N**	**Correctly classified**	**%**	**Incorrectly classified**	**%**
Patients PBPP	68	41	60.30	27	39.70
Healthy controls	200	134	67.00	66	33.00
Total of correctly classified			65.30		

atd R original variables. We wish to emphasize that in the case of girls with PBPP, a statistically significant difference from the control group was confirmed for almost all finger variables: FRCR1, FRCR2, FRCR3, FRCR5, FRCL1, FRCL2, FRCL3, FRCL4, FRCL5, as well as for the palmar variables: b–c rc R, c–d rc R and b–c rc L variables. Our results indicate that PBPP reflects more changes on dermatoglyphs of the girls than of the boys, and confirm the existence of genetic susceptibility to PBPP together with well known risk factors for development of the disease.

Literature data show that finger variables are mostly polygenically determined, while palmar variables are more susceptible to external effects[22,23]. Furthermore, it is documented that women are less susceptible to changes in dermatoglyphic characteristics in comparison to men[22,23]. Arrieta et al. (1991) investigated genetic component of variables a–b rc, b–c rc and c–d rc in healthy persons and concluded that in healthy men, concerning the c–d rc findings, there is a stronger influence of environment while in healthy women all variables a–b rc, b–c rc and c–d rc have a strong genetic component that affect their phenotypic expression[24].

Our findings also revealed significant differences in total ridge count TRC in the group of girls with PBPP in comparison to the control group for most variables.

Discriminant analysis allowed the allocation of individual entities to particular groups to be predicted. Accurate classification was recorded in 68.52% and 65.30% of male and female subjects, respectively. We believe that the percentage of accurately classified female subjects would bee higher if we were able to single out in the male and female subjects with real birth trauma as the cause of PBPP.

In spite of a lower percentage of accurately classified female subjects by discriminant analysis, the results of multivariate and univariate analysis of variance appeared to suggest a genetic susceptibility for the occurrence of PBPP, especially in girls. Furthermore, some epidemiological studies showed higher incidence for PBPP in girls. Mandić et al. (1957), Zancoli et al. (1981), Greenwald et al. (1984) and Stojčević-Polovina et al. (1986) showed in their studies that the percentage of female patients with PBPP is higher than male patients (55%, 61%, 56%, 55% respectively)[3,25–27].

It is a well known fact that the main cause of PBPP is obstetrical trauma. On the other hand, the empiric fact that PBPP is more frequent in some families seems to reveal that genetic predisposition could contribute to the development of PBPP [28]. Furthermore, it is important to acknowledge the fact that there were three pairs of relatives in our sample (two sisters, brother and sister, mother and daughter) with PBPP.

Many authors applied the analysis of dermatoglyphs of digito-palmar complex to estimate the hereditary foundation of some diseases, but they could not answer the question of the connectedness between the changes in dermatoglyphs and certain specific disease[29–34]. Schaumann and Kimura (1991) connected the distortions in whorl ridges with intrauterine disturbances in early pregnancy[35]. Knowledge of the embryological developmental stages clarifies this happening through the fact that the peripheral nervous system as well as epidermis develops from the ectoderm, while the supportable vertebral tissue, from the mesoderm. Consequently, the embryogenesis and morphogenesis of dermatoglyphs and peripheral nervous system happened simultaneously[36]. The formation of first spinal cord starts at the eighth and a half gestational week, which is the exact period in which the secondary volar pads on fingers vanish. With tenth and a half gestational weeks starts involution of primary volar pads and differentiation of epidermal ridges. At that time, the reflex of grasping has been already formed [37]. As the peripheral nervous system as well as dermatoglyphs develops at the same time, possible damages that led to aberrant dermatoglyphs could be the same stressors for the peripheral nervous system, as well.

Although our results indicate genetic predisposition in some patients, analysis of dermatoglyphs does not allow the allocation of individual entities to particular risk group of children.

However, our results are significant in the sense that the recognition of possible heredity in the background of PBPP might contribute to the prevention of this handicap in newborns by pointing to the need of more detailed family history anamnesis to be taken on routine check-ups of pregnant women. Above all it also orientates toward further investigations aimed at a targeted reduction of all factors that may possibly entail damage to the brachial plexus in intrauterine development.

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S. Polovina

Polyclinic for Physical medicine and rehabilitation »M. Stojčević-Polovina«, Kosirnikova 14, Zagreb, Croatia

DERMATOGLIFI I KLIJENUT BRAHIJALNOG SPLETA U DJECE

S A Ž E T A K

Klijenut brahijalnog spleta u djece ulazi u red češćih hendikepa s kojim se susrećemo u svakodnevnoj praksi. Trauma u porodu smatra se najčešćim razlogom njenog nastanka. Činjenica da kod određenog broja novorođenčadi rođenih u istim ili približno istim uvjetima samo jedan njihov dio zadobije leziju brahijalnog spleta pobudila je pretpostavku o eventualnoj genetskoj predispoziciji oboljenja. Kako je analiza digito-palmarnih dermatoglifa već primjenjivana u procjeni nasljedne osnove nekih bolesti, u našem istraživanju izvršeno je ispitivanje digito-palmarnih dermatoglifa u 140 ispitanika s kljenuti brahijalnog spleta, i to 70 muškog i 70 ženskog spola. Kao komparativna skupina poslužili su otisci 400 odraslih i fenotipski zdravih osoba zagrebačke regije i to 200 muškaraca i 200 žena. Učinjene multivarijantna i univarijantna analizea varijance pokazale su da postoje statistički značajne razlike između promatranih skupina. Iako je diskriminacijskom analizom, kojom je moguće prognozirati pripadnost pojedinih entiteta pojedinim grupama, dobiven nešto niži postotak točno klasificiranih ispitanica, rezultati ukazuju da postoji određena genetska predispozicija za nastajanje kljenuti brahijalnog spleta.