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Dermatoglyphic Profile in 22q Deletion Syndrome

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Abstract

A genetic subtype of schizophrenia has been described in 22q11 Deletion syndrome. Previous studies have described an excess of dermatoglyphic alterations in schizophrenia, such as low a–b ridge counts (ABRCs), a high frequency of ridge dissociations, and increased dermatoglyphic fluctuating asymmetry. Little is known however, about the dermatoglyphic profile of 22qDS subjects showing psychotic symptoms and its similarity to the previously reported anomalies in schizophrenia. We studied the palmar dermatoglyphics of 22 subjects with 22qDS of predominantly Caucasian origin, 15 of whom had psychotic illness, and in 84 healthy controls of similar ethnicity. We observed higher values for total ATD angle in cases than in controls ($P=0.04$). In addition, there was an excess of radial figures in the hypothenar area in cases, especially in the left hand. Interestingly, greater fluctuating asymmetry, determined by the absolute difference between right and left ABRC, was observed in 22qDS subjects compared to controls ($P=0.05$). However, no differences were found for ABRCs and frequency of dissociations. Despite the small sample size, the palmprints analyzed suggest the existence of an altered dermatoglyphic profile in 22qDS, involving: (i) ATD angle amplitude, (ii) presence of radial loops in the hypothenar area, and (iii) an increment of fluctuating asymmetry. The first two features are similar to those found in other genetic syndromes associated with low IQ, while high levels of fluctuating asymmetry have often been reported in schizophrenia.

Chromosome 22q11 Deletion syndrome (22qDS) is due to microdeletions of chromosome region 22q11.2, involving multiple genes [Karayiorgou et al., 1995; Bassett and Chow, 1999; Bassett et al., 2001]. 22qDS comprises several genetic syndromes, including velocardiofacial syndrome and DiGeorge syndrome. The most common phenotypic consequences of this deletion are, among others, (1) learning disabilities, (2) palatal anomalies, (3) typical facial features, (4) congenital cardiac defects, and (5) psychiatric disorders. The estimated prevalence of the deletion 22q11.2 is 1/4,000 [du Montcel et al., 1996], which usually occurs as a sporadic mutation, but approximately 10% of cases are inherited from transmitting parents, who often have a less severe phenotype [Demczuk and Aurias, 1995].

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A minority of patients with schizophrenia (2%) are affected by 22qDS [Karayiorgou et al., 1995]; although this prevalence is higher in subpopulations of psychotic patients with comorbid developmental delay [Bassett and Chow, 1999]. At least 25% of patients with 22qDS develop schizophrenia [Murphy et al., 1999]. 22qDS could, therefore, represent a more homogeneous genetic subtype of schizophrenia [Bassett and Chow, 1999]. This subtype may serve as a model for the neurodevelopmental hypothesis of schizophrenia [Lewis and Murray, 1987; Bassett et al., 2001] and could aid in delineating etiologic and pathogenetic mechanisms of the disorder.

The epidermal ridges that appear on the surface of the hand by the end of the 24th week of prenatal life are called dermatoglyphic patterns. They share origins with the CNS, as both neural tissue and epidermis are embryonic ectodermal derivatives and the critical period of their respective embryological–fetal developmental takes place during the second trimester of intrauterine life [Rakic, 1988; Babler, 1991]. Despite their high but varying levels of heritability [Holt, 1968a], part of the dermatoglyphic morphology may be influenced by intrauterine environmental factors acting during the period of ridge differentiation [Babler, 1991].

Developmental stability is the extent to which an individual develops according to a specified ontogenic program in the presence of disruptive factors [Waddington, 1957]. Developmental instability, therefore, represents the degree to which an individual's developmental trajectory is perturbed. It can be measured qualitatively by the presence of minor physical anomalies and quantitatively by the levels of fluctuating asymmetry of paired bilateral traits [Palmer and Strobeck, 1986]. Dermatoglyphics are suitable bilateral traits to analyze the fluctuating asymmetry of an individual [Opitz and Utkus, 2001]. Since dermatoglyphic characteristics remain unchanged over lifetime, the presence of abnormalities in the patterned tracteries of fine ridges in fingers and palms may constitute evidence of a prenatal insult in the first months of pregnancy.

A number of studies have considered the importance of dermatoglyphics as markers of prenatal disturbance in developmental disorders of unknown origin, including idiopathic mental retardation [Rosa et al., 2001]. In the last several years, schizophrenia has been well characterized from the dermatoglyphic point of view. A great number of dermatoglyphic features can be analyzed, but some have special interest for investigation of psychiatric disorders, including dermatoglyphic indices that may reflect intrauterine environmental influences, such as: (i) total finger ridge count (TFRC), (ii) ridge dissociations, and (iii) fluctuating asymmetry in palmar a–b ridge count (ABRC).

Several authors have reported decreased TFRC and/or ABRC values in schizophrenia [Fañanás et al., 1990, 1996a; Turek, 1990; Fearon et al., 2001]. There are also several studies in the literature describing an excess of dermatoglyphic abnormalities, such as ridge dissociations and abnormal palmar flexion creases in schizophrenic patients [Raphael and Raphael, 1962; Markow and Wandler, 1986; Fañanás et al., 1996b; Rosa et al., 2000a, 2002]. Likewise, several studies of developmental instability in schizophrenia have indicated an increase in minor physical anomalies [O'Callaghan et al., 1995; Lane et al., 1997] and greater levels of fluctuating asymmetry in bilateral measurements of dermatoglyphic

variables compared to controls [Markow and Wandler, 1986; Bracha et al., 1992; Reilly et al., 2001].

The goal of this study was to characterize the dermatoglyphic profile of 22qDS subjects, and determine its degree of similarity with the previously described results in schizophrenia. On the other hand, we considered the possible resemblance between 22qDS-dermatoglyphic profile and the ones described in other syndromes caused by chromosomal anomalies which also present with mental retardation. From that point of view the interesting dermatoglyphic variables to explore could be: (i) the magnitude of ATD angles, (ii) the presence of unusual frequencies of patterns in the different palmar areas of the hand [Schaumann and Alter, 1976], and (iii) the presence of abnormal palmar flexion creases [Rosa et al., 2001, 2002].

The case group comprised 22 subjects (nine males, 13 females) with a mean age of 30.27 years ($SD = 8.71$; range = 17–47), affected by 22q11 Deletion syndrome. The subjects were carriers of a microdeletion in the 22q11.2 chromosome region detected by fluorescence in situ hybridization (FISH) using a standard probe. The sample was collected in the Clinical Genetics Research Program (CAMH, Canada) from multiple sources, including referrals from several psychiatric centers, and through a study screening for 22q11.2 deletions in adults affected by tetralogy of Fallot. The majority of the 22qDS subjects were white Canadians ($n = 21$) with European origins; one subject was Asian. Fifteen subjects had psychotic symptoms, meeting criteria for DSM-IV schizophrenia or schizoaffective disorder. Fifty percent of the subjects had mental retardation ($IQ < 70$) and the rest ($n = 11$) presented learning disabilities but of borderline or low average IQ. Eight patients were affected both by psychosis and mental retardation. The participants had given written informed consent after having received a complete description of the study. All of them agreed to have their palmprints taken using a non-inky method. The study was approved by the Research Ethics Board of CAMH.

The control sample comprised 84 healthy people (39 males, 45 females), aged 18–40 years (mean = 22.63, $SD = 4.34$). Controls were all white individuals collected in Spain and Toronto (Canada). All of them had no personal or family history of psychiatric illness or mental retardation.

According to previous studies, we selected two quantitative variables of special interest for a patient population with both mental retardation and psychosis: (i) total ABRC and (ii) total ATD angle. Two qualitative anomalies were analyzed according the dermatoglyphic reports in schizophrenia: (i) the presence of dissociations in palmar areas, and (ii) the presence of abnormal palmar flexion creases [for further details see Rosa et al., 2000a]. Additionally, the figure patterns in the hypothenar area of the palm were studied, given the interest of these variables in disorders with mental retardation.

The ABRC is a measure of the second interdigital area of the palm. It is calculated by counting the number of ridges between the triradius 'a,' at the base of the index digit, and triradius 'b,' at the base of the middle finger (see Fig. 1). The total ABRC is determined by adding both left and right counts [for further details, see Fañanás et al., 1996a]. From this metric trait, we obtained the fluctuating asymmetry (FA), determined here as the absolute

difference between the right and left ABRCs ($FA = |RABRC - LABRC|$) [Palmer and Strobeck, 1986]. The ATD angle is formed by the palmar triradii 'a,' 't,' and 'd.' Adding the corresponding left and right hand ATD angles, we obtained the total ATD angle (see Fig. 1).

The dermatoglyphic analysis was performed according to Cummins and Midlo [1961] and was conducted by BM, blind to the psychiatric condition of subjects.

For each of the continuous variables, differences between groups were tested using a *t*-test for independent samples. The level of significance for all reported differences was set at $P < 0.05$. The χ^2 -test was used to test for differences between cases and controls for the presence of qualitative variables. Pearson correlations were performed to examine the relationship between ATD angle and ABRC variables.

Complete ABRC and ATD angle data were available for all 22 cases and 84 controls. We found that 22qDS subjects had significantly higher values for total ATD angle compared to controls (22qDS: mean = 90.70, SD = 14.64; controls: mean = 84.51, SD = 12.47, $P = 0.04$). This was mainly due to the frequent distal displacement of axial triradii to the t' or t'' position. When left and right ATD angles were analyzed separately, only the angle in the left hand was significantly higher in cases than controls (22qDS: mean = 45.45, SD = 8.29; controls: mean = 41.99, SD = 6.87, $P = 0.04$). When the patients were grouped according to the presence of mental retardation, we detected a tendency in the magnitude of total ATD angles to be higher in patients than controls (22qDS + MR ($n = 11$): mean = 91.59, SD = 18.02; controls: mean = 84.51, SD = 12.47, $P = 0.09$). The ATD angle in the 22qDS patients with psychotic symptoms was lower than in 22qDS patients with mental retardation (22qDS + SZ ($n = 15$): mean = 88.03, SD = 11.38).

With respect to ABRC 22qDS subjects had higher mean ridge counts than controls, although this finding failed to reach significance (22qDS: mean = 81.32, SD = 12.70; controls: mean = 78.29, SD = 10.64, $P = 0.20$). There was a trend for the left ABRC to be higher in cases than controls ($P = 0.07$). We hypothesized post-hoc that ABRCs may have been higher because of the distal displacement of 't' and consequent higher ATD angles, since the triradius 'a' is in one side of the angle. We, therefore, performed a correlation analysis and found that the higher ATD angle, the higher the ABRC in the 22qDS sample ($r = 0.44$, $P = 0.04$).

Fluctuating asymmetry was higher in cases than controls (22qDS: mean = 5.59, SD = 4.76; controls: mean = 3.40, SD = 2.87, $P = 0.05$). When grouped according to the presence of schizophrenia, significantly higher FA was observed in cases compared to controls (22qDS + SZ: mean = 6.40, SD = 5.53, $P = 0.05$). Differences were significant as well, when subjects were grouped according to the presence of mental retardation (22qDS + MR: mean = 5.64, SD = 4.11, $P = 0.02$). Patients with both mental retardation and psychotic symptoms ($n = 8$) showed a trend to increased FA compared to controls (22qDS + MR + SZ: mean = 6.50, SD = 4.44, $P = 0.07$).

In the qualitative analysis of the palmprints, we observed an excess of radial loops (see Fig. 1) in the palmar hypothenar area (especially in the left hand) of individuals with 22qDS (36.4% of 22qDS subjects vs. 19% of controls, $\chi^2 = 2.98$, d.f. = 2, $P = 0.08$). Unexpectedly,

there were no observable differences between cases and controls in other dermatoglyphic anomalies, such as ridge dissociations or abnormal palmar flexion creases.

To our knowledge, this is the first time that a palmar dermatoglyphic study has been performed in a sample of individuals affected with 22q11 Deletion syndrome. Despite the small sample size, the results of our analysis suggest a characteristic dermatoglyphic profile in 22qDS, involving: (i) ATD angle amplitude, (ii) presence of radial loops in the palmar hypothenar area, and (iii) an increment of fluctuating asymmetry in ABRC. The frequent distal displacement of axial triradii 't' to the t' or t'' positions (in 50% of cases) increased the mean ATD angle in the 22qDS group, restricted to the left hand. Likewise, in the hypothenar area of the left palm, we observed a higher than usual frequency of patterns, perhaps because of the displaced axial triradius 't.' Most of the patterns were of the radial type. The frequency of radial loops, alone or combined with other figures, was 31.8% on either hand, and 36.4% on the left hand. Reviewing available data from other studies, the presence of radial loops in this palmar area is reported to be 16.65% in American whites [Plato et al., 1975] and 14.65% in German males [Cummins and Midlo, 1961], in line with results for our controls.

The finding of a distal displacement of the axial triradius and the consequent increase of the ATD angle amplitude in 22qDS is shared by most chromosomal syndromes. It has been previously described in Down syndrome [Plato, 1973; Schaumann and Alter, 1976] and trisomy of chromosome 18 [Walbaum et al., 1966; Penrose, 1969], and in syndromes caused by deletions, such as Cri-du-Chat, 18p- and 18q-syndromes [Schaumann and Alter, 1976]. In some of these cases, the increased ATD angle is accompanied by a high frequency of hypothenar patterns, as in Down- and 18q-syndromes [Schaumann and Alter, 1976]. In Down syndrome however, most of the patterns were of the ulnar type [Plato, 1973], whereas the excess of patterns in the hypothenar area found in 22qDS were radial loops.

In opposition contrast to previous results in schizophrenia, the individuals affected by 22qDS in this sample do not show a low ABRC compared to controls [Turek, 1990; Fañanás et al., 1996a; Fearon et al., 2001]. We also did not observe in our 22qDS patient sample the higher frequency of ridge dissociations reported in schizophrenia.

Recent studies of the ABRC in schizophrenia have considered this dermatoglyphic trait to be the most sensitive to environmental influences, due to the extended period of time over which the area at the base of the 1st and 2nd fingers is configured in the embryo [Bogle and Reed, 1996; Fañanás et al., 1996a]. In syndromes caused by cytogenetic alterations this trait is variable, sometimes increased and sometimes decreased, perhaps because genetic and other influences, such as the hand amplitude, have effects extending over the whole pregnancy.

Genetic effects from the chromosome 22 deletion could have induced the distal displacement of the axial triradius 't' and the consequent increased ATD angles. This would have influenced the displacement of the triradius 'a,' at the base of the index finger, towards the radial side of the palm. The higher values of ABRC in 22qDS subjects with schizophrenia could also be caused this way. Previous results suggesting low ABRC to be

associated with schizophrenia [Fañanás et al., 1990, 1996a; Turek, 1990; Fearon et al., 2001] have been interpreted to be due to intrauterine environmental noise provoking a lower development of the embryonic pads of some patients. In other chromosomal abnormality syndromes, ABRC does not show a consistent pattern, being increased in Turner syndrome [Dallapiccola et al., 1972], not differing in Down syndrome [Holt, 1968b], or appearing decreased in 18q-syndrome [Schaumann and Alter, 1976].

In the current study, 22qDS subjects demonstrated greater dermatoglyphic fluctuating asymmetry in ABRC than controls. This phenomenon has been considered evidence of neurodevelopmental disturbances in prenatal life [Markow and Gottesman, 1989] which could manifest later as behavioral and/or psychiatric disorders. Higher fluctuating asymmetry in 22qDS is consistent with previous findings in schizophrenia [Rosa et al., 2000b; Reilly et al., 2001], and in samples of monozygotic twins showing behavioral discordances [Rose et al., 1987; Bogle et al., 1994]. It is possible that the deletion causing 22qDS may render individuals more vulnerable to adverse intrauterine environmental factors because of a decrease in developmental buffering during intrauterine life. This could be expected for a marker so sensitive but non-specific, indicator of prenatal instability, the fluctuating asymmetry.

In conclusion, the dermatoglyphic profile of 22qDS appears more similar to the results described in other syndromes associated with chromosomal abnormalities and low IQ than in schizophrenia. Future dermatoglyphic case-control studies with larger samples of individuals with 22qDS, with and without schizophrenia, will allow more detailed assessment of the reported preliminary dermatoglyphic profile in 22q11 Deletion syndrome.

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Fig. 1.

Left palm of a 27-year-old woman affected with 22q11 Deletion syndrome, who had both mental retardation and schizophrenia. Palmar a–b ridge count (ABRC) is a measure of the size of the second interdigital area between triradii ‘a’ and ‘b,’ located beneath the bases of the index and middle finger; in this case, the left ABRC is 44. The triradii ‘a,’ ‘t,’ and ‘d’ delimit the ATD angle. Observe the distal displacement of the axial triradius to the t' position. The ATD angle here has a value of 50° . The circled area in the figure shows the usual location of the axial triradius ‘t.’ It is interesting to notice as well the radial loop in the hypothenar area of the palm (see the rectangle). A loop is an open dermatoglyphic pattern where the ridges curve around only one extremity of the pattern; if the loop opens to the radial margin of the palm it is called a radial loop. This individual is a clear example of dermatoglyphic features we commonly observed in whole or in part in the 22qDS sample.