ORIGINAL ARTICLE

Karyotyping, dermatoglyphic, and sweat pore analysis of five families affected with ectodermal dysplasia

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ABSTRACT

Background: Hereditary ectodermal dysplasia is a genetic recessive trait characterized by hypohydrosis, hypotrichosis, and hypodontia. The affected individual show characteristic physiognomy like protruded forehead, depressed nasal bridge, periorbital wrinkling, protruded lips, etc. There is marked decrease in sweat and salivary secretion. Due to skin involvement palm and sole ridge patterns are disrupted. **Aim:** In this study an attempt has been made to classify the affected members according to the degree of penetrance by pedigree analysis and also study karyotyping for cytogenetics, dermatoglyphic analysis for the various ridge patterns and variations in the number of sweat glands by sweat pore analysis in affected individuals. **Materials and Methods:** A total of five families who were affected with ectodermal dysplasia were considered. Pedigree analysis was drawn up to three generation by obtaining history. Dermatoglyphics and sweat pore analysis was done by obtaining palm and finger print impression using stamp pad ink. Karyotyping was done by collecting 3–5 ml peripheral blood. Karyotyping was prepared using lymphocyte culture. Chromosomes were examined at 20 spreads selected randomly under ×100 magnification. Results were analyzed by calculating mean values and percentage was obtained. **Results:** Karyotyping did not show any abnormalities, dermatoglyphic analysis and sweat pore counts showed marked variations when compared with normal. Moreover, pedigree analysis confirmed the status of the disease as that of the recessive trait. **Conclusion:** Large number of affected patients needs to be evaluated for dermatoglypic analysis. Genetic aspect of the disease needs to be looked into the molecular level in an attempt to locate the gene locus responsible for ectodermal dysplasia and its manifestation.

Key words: Dermatoglyphic, ectodermal dysplasia, karyotyping, pedigree analysis, sweat pore analysis

INTRODUCTION

Ectodermal dysplasia (ED) is a disease characterized by a classical triad of hypohydrosis (lack of sweat glands), hypotrichosis (skin abnormalities), and hypodontia (abnormal/less number of teeth).[1,2] The condition is thought to occur in 1 in 100,000 births. Clinically ED may be divided in two types hypohidrotic/anhidrotic type and hidrotic type. The hidrotic form of ED usually spares the sweat glands, can affect hair, nail and is inherited as an autosomal dominant trait.[3] Hypohydrotic form has classical triad and is inherited as an X-linked recessive trait. Males are usually affected and females are carriers.[2] However, the anhidrotic variant of ED may occur in families without any history of this disease due to gene mutations.[4] This disorder is characterized by aplasia or dysplasia of tissues of ectodermal origin and occasionally mesodermally derived tissues.[3] The most common complains of individuals with ED is dental abnormalities and facial appearance, and other being lack of sweating due to decrease number or absence of sweat glands. Thus early recognition of disease is critical since hyperthermia secondary to diminished sweating capacity may result in permanent brain damage or even death.[5]

Often in hereditary diseases like Turner's disease and Down's syndrome, palmar and plantar ridges may be abnormal, which is also true for diseases involving skin

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appendages.[6] To study ridge patterns dermatoglyphic studies of hand and sole printings are done. Dermatoglyphic study is basically concerned with epidermal ridge that form system of lines, parallel in small fields on the surface of stratum corneum. The pores of sweat gland duct lie along the center of ridges. The normal ridge configuration may be altered or dysplastic in hereditary diseases. Several authors have reported changes in ridges of palms and soles of ED patients,[5-7] which may be due to diminished number of sweat glands.

One of the most common and widely used methods for identifying chromosomal abnormality is by karyotyping. Various authors have studied karyotyping in patients with Turner's syndrome,[8] Cri-du-chat syndrome[9] and found changes at chromosomal level. As ED is also a hereditary condition with abnormality in the human genome, karyotyping can be used to locate the abnormal chromosome.

Thus the aim of this study was to classify the families of ED patients in groups of affected, unaffected, and carriers based on the degree of penetrance. For this purpose the parameters like clinical features, pedigree analysis, dermatoglyphic study, sweat pore counts, and karyotyping were done.

MATERIALS AND METHODS

The present study was conducted at KLE VK Institute of Dental Sciences, Belgaum and National Institute of Mental Health and Neuro-Sciences (NIMHANS), Bangalore. Institutional ethical clearance was obtained to carry out the study. Clinically confirmed patients from five unrelated families were taken as study group and unaffected patients from the same families were taken as control group. After obtaining informed consent from each individual a thorough clinical evaluation was done and history for at least two to three generations was taken. The individuals were considered affected after a thorough clinical history.

Pedigree analysis

Pedigrees were drawn up to three generations or as far as the patients and families could remember. Standard denominations like Roman numerical for the designations of the generation and Arabic numerical for the designation of the affected person in a generation were used. Probands were marked and other significant features were also noted. Similarly standard rules were followed to signify various relations and the designations of the affected and unaffected individual.

Dermatoglyphs

Dermatoglyphic studies were carried out using hand imprints. The hand impressions were taken using stamp pad ink. The subjects were asked to smear their hands with ink and impressions were taken on white sheet. The imprints were evaluated using hand magnifying lens and results recorded in terms of type of epidermal ridges at finger tips, position of tri radii line, and "atd" angle, (atd angle is an angle formed by the tri axial radius and the tri radii at the base of the index finger (a) and little finger (d)). The tri radii is denoted by 't' and is present at the base of hand. Normally the angle is around 45°. Angle more than 50° is denoted as "t" and intermediate as 't' but narrow hands does not always show higher 'atd' angle. Comparisons were drawn between affected and unaffected individuals from the same families keeping in mind the age group of the patient.

Sweat pore counts

Sweat pores were counted from impressions taken from six fingertips (selected randomly) of affected individuals and compared with those of unaffected individuals.

Impressions

Impressions were made by painting a thin film of 2% cellulose acetate in acetone on each fingertip followed by 0.35% of crystal violet. When this dried two to three coatings of 2% cellulose acetate was again applied. After drying the coating was stripped off using cellulose adhesive tape and impressions were mounted on a micro slide using a clear nail varnish with the impression side upward. To preserve the impressions it was covered with cover slip.

The sweat pores were counted at ×35 magnification of stereomicroscope fitted with ocular micrometer. Six counts along 0.32 cm of epidermal ridge were made on each impression. The results were recorded as sweat pores per cm of the ridge.

Karyotyping

Nearly 3–5 ml of peripherial blood was collected and stored at 4°C for further manipulation. The karyotype was prepared using lymphocyte culture.

Culture setup

The cultures were set up using autoclaved vials and other armamentarium like syringes, test tubes, micropipettes, and phenyl agglutinin wells. The chemicals used for the preparation of media for cell culture were: TC 199/RPMI (culture media)–5 ml, human AB serum/fetal calf serum – 1 ml, heparin – 1 drop (0.2–0.4 microl), streptomycin – 1 drop, penicillin – 1 drop, phenyl agglutinin M – 100–200 microl. Nearly 5–6 ml of media was taken into a sterilized vial and 1 ml of whole blood was added to it. The cultures were incubated at 37°C for 72 hours.

Harvesting of culture

At 70 hours, 0.2 ml of 0.2% colchicines was added to arrest the cell growth. The cultures were incubated at 37° for 120 min,

transferred then to precleaned tubes and centrifuged at 1000 rpm for 10 min. Supernatant was discarded. Nearly 6–8 ml of preincubated potassium chloride was added (0.75 M, pH 7.0). Again incubated for 14 min and centrifuged at 1000 rpm for 10 min. Supernatant was discarded and 3–4 ml of fixative added (3 parts of methanol and 1 part of glacial acetic acid), mixed to avoid clumping of cells. Re-centrifuged and fixative added (repeated twice). Final suspension obtained was kept at 4°C for overnight. The suspension was dropped on to prechilled slide from a height of 1 foot keeping the slide tilted at an angle of 45°C. Suspension was scattered on the slide by blowing gently and then dried on hot plate (45–60°C) for 1–2 min. The slides were kept in incubator overnight for aging before staining.

Staining procedure

The mature slides were placed in Trypsin solution for 80–90 seconds, then the slides were stained with Giemsa solution for 15–20 min. The slides were mounted with DPX and kept overnight before analysis.

Chromosomal analysis

For analysis chromosomes were examined at 20 spreads selected randomly under ×100 magnification. Ten of these were selected on the basis of clarity of chromosomal structure and banding pattern and analyzed under ×1000 magnification. The findings were recorded in terms of number or structural changes (normal or any change).

RESULTS

In the study group (affected) there were six males and nine females with mean age of 13.66 years. In control group (unaffected) there were seven males and three females with mean age being 35.8 years [Tables 1–3] [Figures 1 and 2]. The patients showed most of the clinical features considered typical of the disease. The various features noted were hypohidrosis, lanugo type hair, negligible eyebrows and eyelashes, sparse body hair, characteristic physiognomy like protruded lips, high forehead, etc., and other features like dry hair, pigment around eyes, disturbance in salivary and lacrimal secretions. Maximum number of patients showed presence of all the features taken in account [Table 4]. In dental abnormalities four out of nine patients showed only peg shaped laterals, three showed peg shaped central and lateral incisor. Three patients showed hooked molars. Presence of teeth ranged from 0 to 12 [Table 5] [Figures 3 and 4].

In pedigree analysis 60% of the families showed history of consanguineous marriages in more than one generation [Table 6].

Pedigrees were drawn for all five families following standard rules and denominations. In five families there were total of twelve members affected, out of these two members BII3 (family B, generation II, member 3) and DII3 died in infancy. The reasons given were high grade fever of unknown origin. The subjects were considered affected after thorough history from the mothers.

Table 1: Total showing affected and unaffected individual in five families studied

No. of cases	Affected (Study group)	unaffected (control group)
Total no. of cases	9	10
No. of males	6	7
No. of females	3	3

Table 2: Showing age distribution among affected individuals (Study group)

Age (years)	Male	Female	Total
0–5	3	2	5
6–12	-	1	1
13–16	-	-	-
Above 16	3	-	3
Totals	6	3	9

Table 3: Showing age distribution among unaffected individuals (control group)

Age (years)	Male	Female	Total
0–5	-	1	1
6–12	1	-	1
13–16	-	-	-
Above 16	5	3	8
Total	6	4	10

Table 4: Depicting the various clinical features and the percentage of affected individuals

Clinical features	No. of patients affected (9)	Percentage
Hypohidrosis	9/9	100
Lanugo type hair	9/9	100
Negligible evebrows and eyelashes	8/9	88.8
No body hair	9/9	77.7
Sparse hair	8/9	88.8
Dental abnormalities	9/9	100
Characteristic physiognomy	9/9	100
Protruding forehead	9/9	100
Saddle nose	9/9	100
Protruding lips	9/9	100
Other features		
Dry skin	9/9	100
Reduced salivary secretion	9/9	100
Hoarseness	2/9	22.2
Pigment around eyes	7/9	77.7
Allergy	-	-
Reduced lacrimal secretion	-	-
Increased lacrimal secretion	1/9	11.1

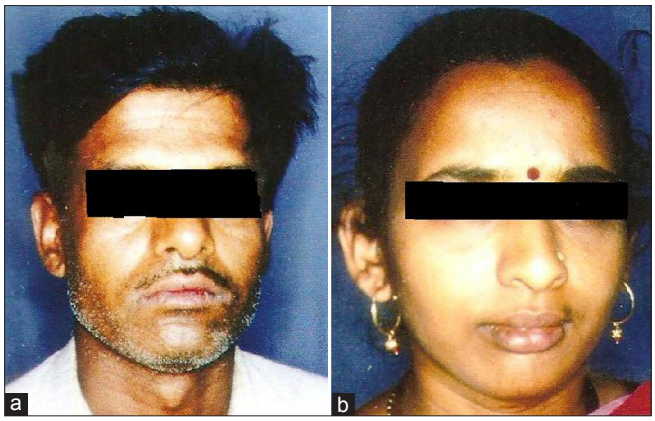

Figure 1: (a and b) Photograph showing unaffected parents of a family

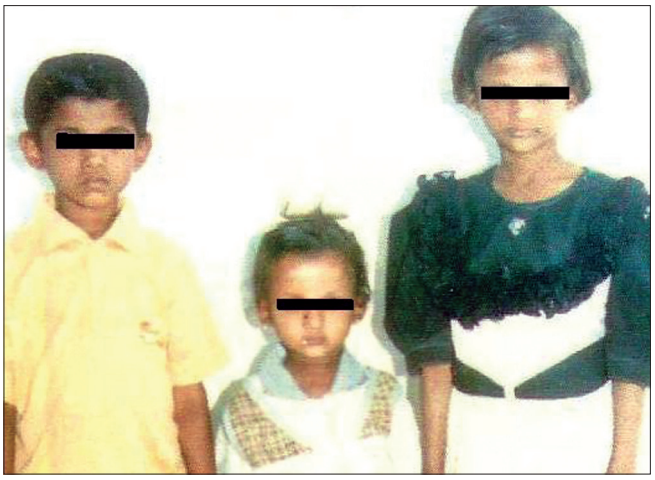

Figure 2: Photograph showing affected siblings of the same family

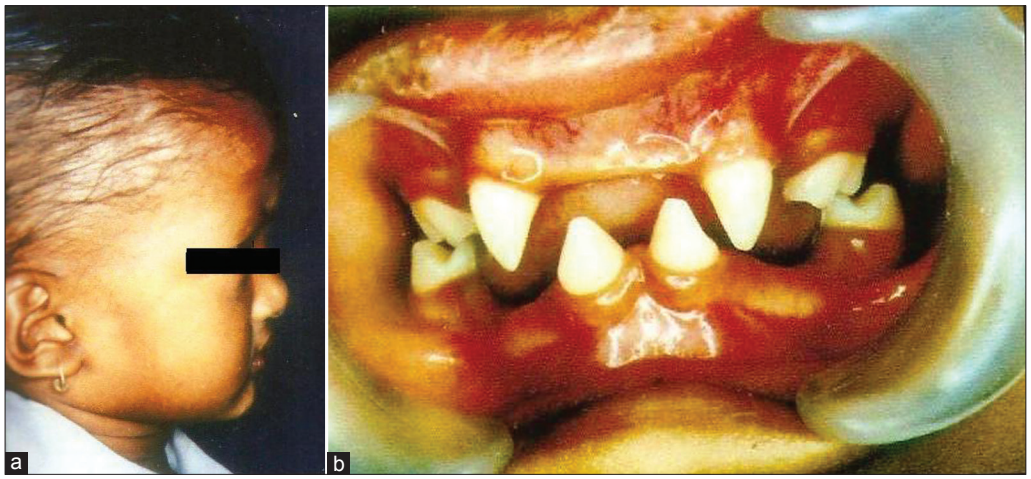

Figure 3: (a) Photograph showing lateral view of the affected individual with sparse hair, saddle nose, frontal bossing (b) Intraoral photograph of the same individual showing dental abnormalities

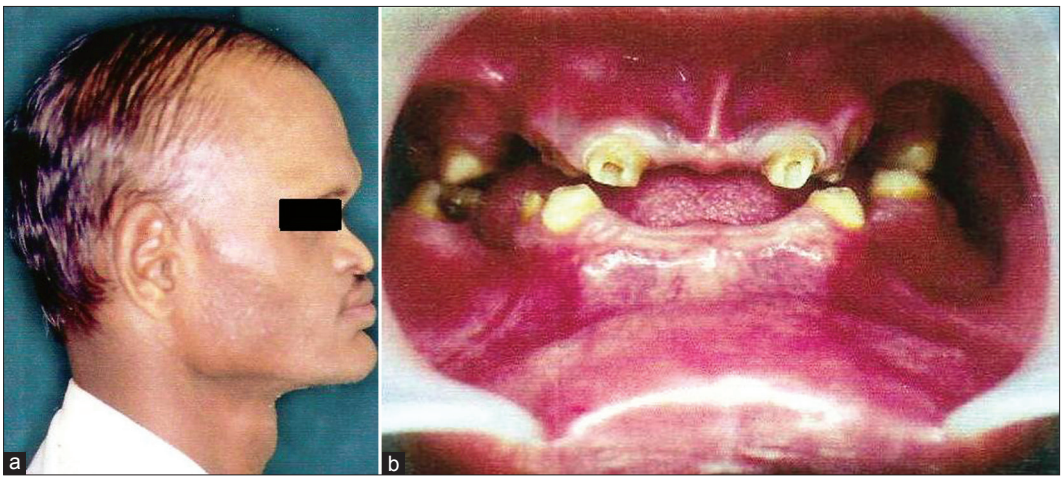

Figure 4: (a) Photograph showing lateral view of the affected adult individual with sparse hair, saddle nose, frontal bossing (b) Intraoral photograph of the same individual showing dental abnormalities

The other affected subjects were AIII 1,3, BII 6,8, BIII 3,5, CIV 1,2, DII 8, and E1 V1. Of all these BIII3 (male10 years) was not present for clinical examination but was considered affected on the basis of clinical history provided by other members of the family. Three of these five families had history of consanguineous marriage.

Table 5: Showing dental abnormalities in each affected individual

Case no.	No. of teeth present	Peg shaped central incisor/ lateral incisor	Hooked molars	Dentures
AIII 1	9	Both incisor affected	Present	Partial
AIII 3	8	Both incisor affected	Present	Partial
BII 6	6	Only lateral incisor affected	_	Complete
BII 8	8	Only lateral incisor affected	_	Complete
BIII 5	4	Only lateral incisor affected	_	Complete
CIV 1	4	Only lateral incisor affected	_	Partial
CIV 2	0	-	_	_
DII 8	12	-	Present	Complete
EIV 1	5	Both incisor affected	_	_

Table 6: Depicting the number and percentage of consanguineous and nonconsanguineous marriages

Total no. of families	Consanguineous marriages	Percentage	Non consanguineous marriages	Percentage
5	3	60	2	40

From the nine examined study subjects, six were males giving a preponderance of males over females (77.7%). The affected males varied in age from 4 to 38 years, with the mean age being 17 years.

For the three females in the study group the age variation was from 2 to 10 years giving a mean age of 7 years. The variations in clinical features of the three females were quite distinct. The youngest one (2 years) BIII 5 showed marked skin abnormalities but her hair color and texture appeared normal. The partial absence of deciduous dentition can also be attributed to delayed dentition as the central and lateral incisors were present. The lateral incisor showed typical peg shaped crowns, radiographs for the further confirmation was not possible as the permission for the same was not given by the child's parents.

The second affected female (9 years) AIII 3 had severe hypodontia and remarked features of sparse hair, dry skin, and abnormal facies. The child had mixed dentition with permanent incisors but crowns showed abnormal shape. The present molar showed hooked cusps. The Orthopantomogrh (OPG) of the patient showed absence of any successor teeth. Only canines and second premolars were present in upper arch whereas the lower arch showed presence of second premolar only.

The third female (10 years) EIVI had facies typical of ED and hypodontia. The deciduous dentition had only one molar. The proband showed dystrophic nails but her hair looked normal and dark in color with no areas of alopecia.

The affected males all showed typical facies and sparse, thin hair, hypodontia was marked in all the members with only one or two teeth present in an arch. The youngest patient (AIII 3, 4 years) showed absence of permanent dentition on an OPG whereas in deciduous dentition only incisors were present. There was no history of caries, extraction or premature exfoliation. The older subject BII 6 and 8 DII 8 showed only the presence of incisors and one molar. When present, teeth were malformed and widely spaced. Another feature of clinical examination was that all the subjects had halitosis. The subjects also showed dry and parched lips, coated tongue and thin, dried oral mucosa. There was decreased secretion of saliva with dryness of the mouth.

The younger patients CIV 1, 2 complained of high grade fevers of unknown origin. The episodes of fever and discomfort increased in hot weather. Two members in the study group CIV 2 (3 year male) and BIII 5 (2 years female) were not checked for the paucity of sweat pores as they were not ready to undergo the required test.

In dermatoglyphics the affected individuals showed a number of variations like presence of flattened and vestigial palmer ridge patterns [Figure 5]. The epidermal ridges showed increase in number of arches in the study group by 10% as compared with the control group [Table 7]. The affected individuals also showed vestigial patterns especially with the 'C' and 'D' tri-radii. The study group showed increase in number of arches (10%) as compared with that of control group (0%). The number of radials decreased from 12% in control group to 5.5% in study group. There were no significant changes observed in number of ulnars whorls [Table 7]. The study group showed a mean 'atd' angle of 45.35° (range 42–49.5°) as compared with control that showed mean 'atd' angle of 42.05° (range 34.5–50°). Mean 'atd' angle for study group being 45.36° and 44.47° for controls, respectively. Although the females in study group showed a slightly higher mean 'atd' angle of 43° as compared with controls with mean angle of 39.5° the difference was not considered significant [Tables 8 and 9].

Karyotypes of the affected individuals did not show any abnormal patterns [Figure 8]. The sweat pore counts of the individual in the study group showed a decreased count as compared with the control group. The study group showed a mean range of 1.8–9.0 pores/cm as compared with control group, which showed higher range of pores like 23.75–46.76 pores/cm. The females in the study group showed a slightly higher degree of counts, maximum value being 9.0 pores/cm compared with the males, with a maximum value being 3.33 pores/cm [Tables 10 and 11] [Figures 6 and 7].

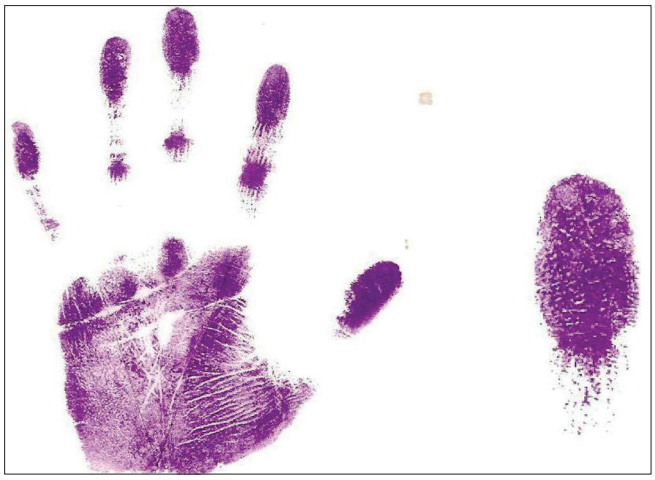

Figure 5: Photograph showing loss of ridge clarity in affected individual by dermatoglypics

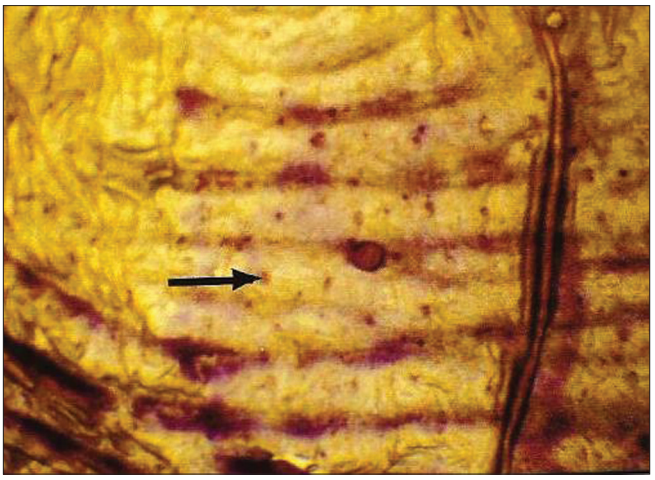

Figure 6: Photograph showing sweat pore on the finger of unaffected individual (under stereomicroscope)

Figure 7: Photograph showing scaly surface and sweat pore on the finger of unaffected individual (under stereomicroscope)

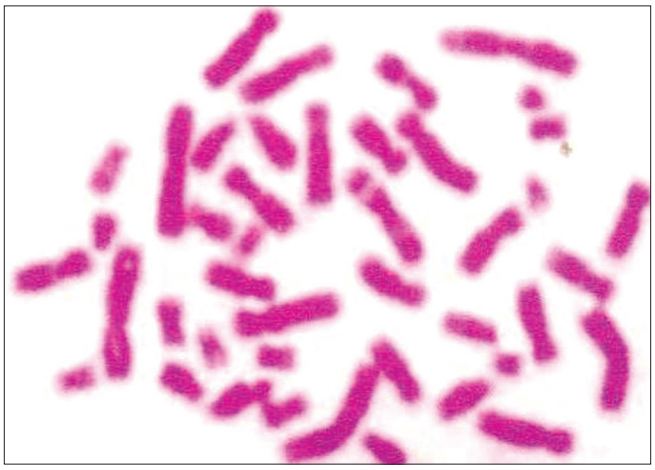

Figure 8: Photograph showing normal appearing chromosomes on karyotyping

DISCUSSION

ED is a genetic disorder characterized by aplasia or dysplasia of tissues of ectodermal origin and occasionally of mesodermal origin. The most common type of ED is "hypohidrotic" ED.[4] Other two varieties being the anhidrotic and hidrotic variety.[10]

Two types of inheritance for this disorder are postulated. An X-linked recessive gene in families where only males are affected and an autosomal dominant inheritance where females are also affected.[11] Although the evidence of autosomal dominant inheritance remains inconclusive, X-linked recessive inheritance is well accepted.[2,3,6,11] Thus keeping in mind the hereditary inheritance, the present study was carried out to check for pedigree analysis, aberrations in chromosomal structures of affected members.

A total of twelve members were affected in five families. Out of which three families had a history of consanguineous marriage (60%). This explains the predominance of a recessive trait as more the inbreeding among families and higher the risk of manifestation of recessive trait. But till date the mode of transmission of this disease has been subject of controversy. Cockayne in an extensive review of the subject recognized two types of inheritance for this condition. He postulated a sex linked recessive gene in families where males only were affected. In the families where females were involved he called it autosomal dominant gene.[11]

Kerr *et al*. felt that the inheritance in all the families reviewed by Cockayne was consistent with sex linkage. Results of thermally induced sweating and histological examination of the skin sections in carriers and controls indicate quantitative deficiencies of sweat gland in the carriers. Thus they concluded that the syndrome is sex linked and that the mildly affected females are heterozygous.[12]

Reports of partially affected females showing various manifestations of the gene are common and usually are concern

Table 7: Percentage of expression of ridge patterns in control and study group

Group studied	Whorls	Arches	Ulnars	Radials	Total
Percentage of expression in control group	28	0	60	12	100
Percentage of expression in study group	30	10	54.4	5.5	99.9

Table 8: Showing "Atd" angle in control group

Age	Sex	Atd angle	
		Left hand (in degrees)	Right hand (in degrees)
4	F	40	44
8	M	47	53
24	F	37	40
26	M	42	46
30	M	41	43
32	F	48	40
38	M	49	48
42	M	35	36
68	F	34	35
76	M	46	37

Table 9: Showing "Atd" angle in study group

Age	Sex	Atd angle	
		Left hand (in degrees)	Right hand (in degrees)
2	F	Not evaluated	
3	M	50	45
4	M	49	50
5	M	50	48
9	F	42	44
10	F	No ridges found on palms	
22	M	47	37
30	M	45	43
38	M	42	45

Table 10: Mean number of sweat pore counts in the control group

Age	Sex	Mean pore counts/cm
4	F	28.75
8	M	23.75
24	F	43.0
26	M	46.76
28	F	30.83
30	M	26.66
30	M	25.33
40	M	37.66
60	F	43.66
72	M	33.1

Table 11: Mean number of sweat pore counts in the study group

Age	Sex	Mean pore counts com
2	F	7.90
3	M	2.34
4	M	2.83
5	M	2.0
9	F	8.11
10	F	9.0
22	M	1.8
30	M	3.33
38	M	2.33

relatives of known male patients. Partial anadontia is reported most commonly but some females have only hypotrichosis. Intolerance to heat is occasionally noted as the only component of the syndrome. These manifestations are similar to our finding among females, with fewer manifestations in females.[12] This also suggests that the disease is of recessive trait. All these findings are in consistent with our finding thus confirming the transmission of recessive trait

Another significant clinical manifestation in the present study was presence of unilateral epiphora in one of the elder male patient (DII8). Beckerman reported similar finding of bilateral epiphora caused by lacrimal drainage anomalies. Anhidrotic ED, however, is only occasionally associated with ophthalmic complications. Most present with lacrimal gland hypoplasia, deficient lacrimation, corneal dystrophies, lenticular changes, chorioretinal changes, strabismus, and myopia.[13]

An outstanding feature of ED is the absence/decreased number of sweat pore counts in affected individuals. The study group showed a marked degree of decrease in sweat pore counts, (mean 4.30, range 2.0–9.0 pores/cm) as compared with the control group, (mean 34.28, range 23.75–46.83 pores/cm. In the study group variations were seen among males and females. The males showed a decreased number of sweat pores (2.0–3.33 pores/cm) as compared with females (8.11–9.0 pores/cm).

The results from our study were found to be in agreement with other studies.[5,11] Crump IA and Davis MD found normal frequency of sweat pores in 1 of the 6 probable heterozygous female subjects, which indicated that diminished sweat pores is not a consistent feature in the carrier female subject. They also found, along with this, a girl with normal sweat pore count with dental abnormalities and another girl having asymmetric counts, both were the off springs of the only mother who had a normal frequency of sweat pores. Thus they suggested that this could be due to influence of the genetic background over the expression of this feature in hypohydrotic ED. They also postulated decrease in number of sweat pores with increase in age,[14] but no such correlation was found in our group. Frias and Smith also showed a total absence of sweat pores in affected males of ED in their study, which was not found in any of the affected members of our study group.[5]

It is a well known fact that the patterns of hands and sole ridges are characteristic of each individual. The imprints of these are called dermatoglyphics. The ridge patterns of hand and feet are laid down during the third month of fetal life and remain unchanged throughout life except for an increase in size and physical growth. The pattern type and detail of ridge can be studied separately using hand imprints. Normal variations of the patterns, which represent mainly hereditary differences, are found between separate populations or members of same population. On the fingertips, derma ridges are arranged in patterns called arches, loops, and whorls. In ED as skin is also affected, characteristic changes can be expected to occur in dermatoglyphic patterns.[6] In the present study, the dermatoglyphic patterns were obtained from study group and control group using stamp pad ink. The study group showed an increase in the number of arches (10%) when compared with control group (0%). The number of radials in study group was 5.5% when compared with control group, which was 12%. There was no significant changes in the number of ulnars (54.4% in the study group and 60% in control) and also in whorls [Table 7]. Jean reported ridge disruption in palms and soles, presence of vestigial patterns or distally displaced tri-radii angle in ED patients. He also reported complex patterns (patterns not arranged in any form) in hypothenar areas. Although disruptions of ridge patterns were seen in three affected males in the palmar areas, no other changes were observed in the present study. The ridges on the hands of affected males looked flattened but this could be the result of excessive crusting due to absence of sweat glands.[7] Julian reported an increase in 'atd' angle in the affected patients[6] but no such significant variations was seen in our study group compared with controls. The males did not show any significant variation in 'atd' angle but females in study group showed a slightly higher mean 'atd' angle compared with controls. One of the affected female (EIVI) children did not show any clear pattern of dermatoglyphs and this could also be due to severe drying of hands. It also made measuring of 'atd' angle impossible.

In various hereditary conditions, cytogenetics studies are done to locate any chromosomal abnormality. Midtbo *et al*. studied the karyotypes of patients with Turners syndrome especially those presenting with malocclusion. They concluded that the mosaic and isochrome for long arm of chromosome showed the same pattern of malocclusion with varying degrees.[8] Similar study by Cockanye postulated on X-linked recessive gene in families where males alone were affected and a typical phenotype of the disease was found.[12] Thus we studied structural aberrations of the X-chromosome using karyotyping. We did not find any changes at chromosomal level in any case of ED. Although Wesser DW has reported an X-chromosome aberration in one reported case. They found consistent abnormality at the same locus on the same chromosome in 8 out of 15 cells studied.[15]

CONCLUSION

The present study of pedigree analysis showed definite pattern of inheritance. The study also confirms the mode of transmission of the disease is recessive trait. Males are affected more commonly than females. Dermatoglyphics showed disruption of dermal ridges due to decrease in number of sweat pores. Slight variation in 'atd' angle was noted when compared with normal individuals, increase in number of arches and decrease in number of radials were also noted. No change was demonstrated at chromosomal level on karyotyping. Thus further studies at the molecular level are needed to locate gene locus showing changes that are responsible for the manifestations of the disease.

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