STUDY OF DYSHIDROSIS

RATAN SINGH, DEVINDER KAUR, M. PARMESWARAN

Summary

85 cases of dyshidrosis were studied in detail. Vesicular fluid from 19 cases were examined for total protein, pH, fungus and other organisms and for organisms alone from additional 15 cases. Cases of clinical tinea-pedis were examined for fungus in KOH. Most freshly developed vesicles from 22 cases were biopsied and serial sections studied.

Only 2 proved cases of tinea-pedis were seen but there was no association with dyshidrosis. Hyperhidrosis was present in 10.6% cases only. The mean values of total protein and pH of vesicular fluid were 4.48 mg% and 8.58 respectively. No fungal elements were seen in the dyshidrotic vesicles. Histological examinations of dyshidrotic vesicles showed that the earliest vesicle was spongiotic in origin. Serial section did not reveal any definite relationship of the sweat duct to the vesicle.

This study is in disagreement with the Sudoral theory and the concept of "id eruption of tinea-pedis" in the causation of dyshidrosis. Dyshidrosis as a manifestatton of endogenous eczema is advocated.

In 1873, Tilbury Fox first described dyshidrosis as a primary affection of the sweat glands and the ducts with associated hyperhidrosis, analogus to miliariasis of palms and soles. Hutchinson in 1876, not convinced by Tilbury Fox's theory, just named the disorder with morphological term "Cheiropompholyx" or the "vesicles of the hands". Since then numerous investigators have for many years studied the problem whether the disease is a primary, secondary or even an incidental affection of the sweat ducts (Peck[4], Whimster[5], Sulzburger & Baer[6], Devine[7], Wilson & Thackray[8], and Simons[9].) Dyshidrosis being a common problem in the tropics also, and its etilogy still unsettled, the authors were prompted to undertake this study.

Department of Dermatology & Venereology
Maulana Azad Medical College and
Associated Irwin and G. B. Pant Hospitals,
New Delhi-1

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Material and Method

The material of this study is formed by 85 patients of dyshidrosis who attended the skin and V.D., Out-patient of Irwin Hospital, New Delhi, during a period of two years. A detailed clinical history was taken and a thorough clinical examination of all systems conducted and recorded on a specially prepared proforma for the purpose. A particular reference was made to seasonal variations, associated hyperhidrosis, occupation and hobbies, incidence of allergic manifestations (like bronchial-asthma, urticaria atopy etc., both in the patient himself and the family), relationship to emotions, hormones, drugs and gastro-intestinal tract disturbances, any evidence of focal sepsis in ear-nose-throat, respiratory and genito-urinary systems and finally the distribution and characteristics of the lesions.

Vesicular fluid was examined in 19 cases for total protein (Copper

sulphate specific gravity method, Harrison[10]), pH (litmus paper method) and fungus (KOH preparation). Vesicular fluid from additional 15 cases (total 34 cases) was cultured for various organisms.

Most freshly developed vesicle availble was biopsied in 22 cases and serial sections (Haemotoxylin and Eosin stain) were examined.

Only three cases of clinical tinea-pedis were seen in 85 patients of Dyshidrosis and were subjected to KOH examination for fungus. Routine blood (T.L.C., D.L.C, Haemoglobin % and E.S R.), stool and urine examinations were done in every case.

Observations and Results

The age and sex distribution of patients with dyshidrosis and their relationships to seasons are shown in tables 1 and 2 respectively. 80 out of 85 cases (94.6%) fell between 11 and 50 years of age, with maximum incidence (37.6%) for both sexes in the third decade of life. There were 37 males and 48 females ; the male and female sex ratio being 8.7 : 11.3.

TABLE 1
Age and sex distribution of patients with dyshidrosis: Total cases 85

Age	Male 37 cases (43.5%)	Female 48 cases (56.5%)
0—10 years	0	0
11—20 years	6	10
21—30 years	14	18
31—40 years	5	7
41—50 years	8	12
51 – 60 years	2	1
61 years and above	2	0

52 out of 85 cases (61.3%) had seasonal relapses and / or aggravation of dyshidrosis ; 45 out of these 52 cases (53%) were affected mainly in summer and rainy season (April to September) and the remaining 7 cases (8.2%) in winter (November to February).

TABLE 2
Cases of dyshidrosis showing relationship to season. Total cases 85.

	No. of cases	Percentage
Seasonal variation not seen	33	38.7
Seasonal variation observed	52	61.3
Relapses and/or aggravation in summer and rainy season (April to September)	45	53.0
Relapses and/or aggravation in winter (November to February)	7	8.2

62 cases (73%) belonged to middle income group ; 20 cases (23.5%) to poorer class and only 3 (3.6%) to higher income group. Itching was a prominent symptom in all the patients.

Only 10.6% (9 cases, 5 males and 4 females) had associated hyperhidrosis.

40% cases (9 males and 25 females, a total of 34) showed definite worsening of dyshidrosis with emotional upsets. 22 cases had constipation ; dyspeptic symptoms in 17 cases and dysentery in 1 ; other associated disorders are shown in table 3 : no definite relationship with dyshidrosis could be observed in any. Of 48 females, 10 had menstrual irregularity and 4 menopausal symptoms, but there was no association with dyshidrosis.

TABLE 3
Cases of dyshidrosis with associated disorders

	Nos.
Chronic Bronchitis	3
Enlarged Tonsils	4
Caries Teeth	8
Pyorrhoea	13
Gingivitis	12
Infective Eczema	1
Total	31

The duration of dyshidrosis on first visit and the number of earlier relapses are shown in tables 4 and 5 respectively.

TABLE 4

Duration of disease on first visit.
(Total cases 85)

	No. of cases	Percentage
Less than six months	23	27.0
6—12 months	16	18.8
1—5 years	28	33.0
5—20 years	18	21.3

TABLE 5

Number of relapses before first visit.
(Total cases 85)

	No. of cases	Percentage
First attack	22	25.8
2—5 relapses	27	31.8
6—15 relapses	31	36.5
Continuous process; no periodicity	5	5.9

The distribution of the lesions is shown in table 6. In only 3 cases (3.5%) vesicles were on erythematous bases. All the vesicles were deep seated and resembled sago-grains, with exception in only one case, where vesicles appeared more superficial.

TABLE 6

Distribution of lesions of dyshidrosis.
(Total cases 85)

	No. of cases	Percentage
Bilateral and symmetrical	81	95.3
Unilateral	4	4.7
Both palms and fingers only	65	64.7
Both soles and toes only	18	21.2
Both palms and soles	16	18.8
Lesions extending on the dorsa of hands and feet	33	38.8

The biochemistry of vesicular fluids studied in 19 cases of dyshidrosis is shown in table 7. The total protein and pH values were found to be 4.48 mgm% and 8.58 respectively.

The KOH preparation of 19 vesicular fluids whose biochemistry was studied and also of those from 4 unilateral cases of dyshidrosis, were negative for fungus. Two out of three cases of clinical tinea-pedis were positive for fungus (KOH preparation); but their dyshidrotic vesicular fluids were negative for fungus (KOH preparation).

TABLE 7

Biochemistry of vesicle fluid of dyshidrosis.
(Total 19 cases)

	Total protein in mgm%	pH
Mean	4.48±3(0.148)	8.58±3(0.108)
Range: Maximum / Minimum	5.20 — 3.96	9.5 — 7.5

One case showing total protein 1.8 mgm% and pH 6.0 has been accounted for separately.

Culture of vesicular fluids from 34 cases (table 8) revealed staphylococcus pyogenes in 8, β-hemolytic streptococcus in 2, a mixture of organisms contaminant in 1, and no growth in 18 cases (53%).

TABLE 8

Culture of vesicle fluids (34 cases)

	No. of cases	Percentage
Staph. Pyogenes	8	—
B. Hemoly. Streptococcus	2	—
Mixture of organisms	5	—
Contaminant	1	—
No growth	18	53

The routine examination of urine and stool did not reveal any significant finding except in 7 samples of stool (Hook worm ova-2 cases; Thread worm ova-1 case; Giardia-1 case; E. Histolytica cyst-1 case, and E. Coli cyst-2 cases). The total leucocyte count was within normal range in all except 6 cases where it was above 11,000/c.m.m.; 2 out of these were grossly secondarily infected. Mean haemoglobin and ESR values were 10.5% (range 13.8G%) and 11 mm/Ist hour (range 24-6 mm/Ist hour) respectively.

Histologic examination of serial sections from 22 cases (including 4 cases with associated hyperhidrosis) revealed vesicles in upper epidermis in all specimens, with varying degree of spongiosis around the vesicle in some. Most of the closely set vesicles were separated only by thin septa of epidermal cells. Inflammatory infiltrate of very mild degree was observed in the dermis in few cases (Fig. 1). Spongiotic vesicle in very early stage of development was seen in 3 cases (Fig. 2). In none of serial sections examined, was observed any relation of sweat duct to the vesicle; except in one case (Fig. 3) where a distended sweat duct was seen close to a vesicle.

Discussion

The markedly high pH (8.58) and the total protein (4.48 mgm%) values of the vesicular fluid are so striking that the Sudora origin of vesicle in dyshidrosis seems most improbable: such high values speak more of eczematous nature of vesicular fluid. In only one case with associated hyperhidrosis, the total protein and pH values of the vesicular fluid were 1.08 mgm% and 6 respectively: the serial sections of the vesicle showed a dilated sweat duct close to the vesicle (Fig. 3) and the low values of total protein and pH were possibly brought about by the admixture of sweat. None of the vesicular fluid showed any fungal element and 53% were sterile on culture, which is consistent with a deep seated eczematous vesicle protected by a thick horny layer.

Study of serial sections did not show any definite relationship of the sweat duct to the vesicle except in one case (vide supra). On the contrary, the spongiotic changes were seen during the very early stage of vesicle formation (Fig. 2). It is conceivable that as the spongiotic vesicle increased in size, it could have pressed upon the epidermal sweat duct causing its dilatation or even rupture in exceptional cases. Thus the association of sweat duct in the pathogenesis of dyshidrotic vesicle could be incidental only.

Associated hyperhidrosis was seen in only 10.6% of cases and cannot be considered as an important etiological association of dyshidrosis, as postulated by Tilbury Fox and his school.

Only 2 cases of dyshidrosis were seen along with tinea-pedis; one of them was treated with griseofulvin; the tinea-pedis was cured, but relapses of dyshidrosis continued to appear. Thus the concept of "Id eruption of tinea-pedis" in the causation of dyshidrosis seems to have been overworked.

The high protein and pH values of the vesicle fluid and the spongiotic origin of the vesicle (Fig. 2) suggest that the dyshidrotic vesicle is eczematous in nature. The detailed study of 85 cases did not suggest the nature of eczematous eruption to be one of contact sensitivity. 17.6% (15 cases) had family history of constitutional allergy (chronic urticaria, bronchial asthma and atopic dermatitis). 28.2% (24 cases) were themselves suffering from chronic urticaria. 7% (6 cases) had dyshidrosis in the family. Further, 4 cases of dyshidrosis, during their follow-up presented with endogenous eczema (3 nummular type and one flexural type). Grosshans et al[11] observed white dermographism and delayed blanch phenomenon in patients of dyshidrosis. These observations certainly speak of much more than 'chance' associations and it can be surmised that dyshidrosis represents an endogenous type of eczema. In the present study, dyshidrosis showed its peak incidence (53%) in hot and humid months, not because of increased sweating in these months, but because like some endogenous eczemas showing seasonal variations, dyshidrosis had its highest incidence in summer season, brought about in some way by environmental conditions, directly or indirectly

Fig. 1

Multiple closely set vesicles, separated by thin septa of epidermal cells, in the upper epidermis. No sweat duct seen. Mild degree of infiltrate in the dermis.

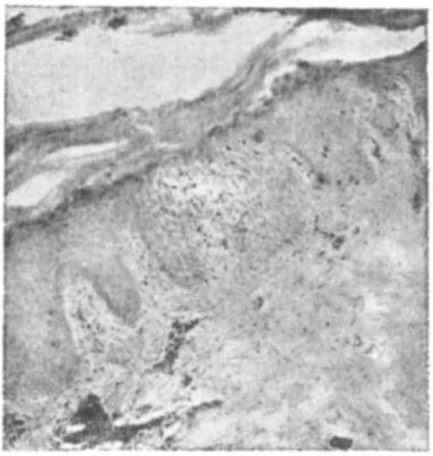

Fig. 2

Spongiosis leading to vesicle formation (very early stage) in the upper epidermis. No sweat duct is seen in the vicinity.

Fig. 3

A vesicle in upper epidermis with a distended sweat duct (S.D) at its bottom. The duct (S.D) can be seen continuing its course in the corneal layer. Mild degree of infiltrate in upper dermis.

affecting the human system by multitudes of factors like food articles, vegetations, metabolites, etc. Being a manifestation of endogenous eczema, the lesions need not be strictly confined to palms and soles. In the present study 38.8% (33 cases) had lesions extending on to the dorsa of hands and feet. Simons[12] reported cases of dyshidrosis where lesions were seen on helix of ears.

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