STUDY OF COMPOSITION OF SWEAT IN DERMATOLOGICAL DISORDERS

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Summary

Sweat electrolytes and urea levels were studied in a variety of dermatological conditions and 15 normal subjects. This study demonstrated significantly raised levels of Sweat urea in Tinea infections and pustular folliculitis. Sweat sodium and chloride levels were elevated significantly in psoriasis, hyperhidrosis, tinea infections and pustular folliculitis. Sweat potassium levels were normal in tinea infections while significantly elevated levels of sweat potassium were noticed in psoriasis, hyperhidrosis and pustular folliculitis. The elevated levels of sweat urea in tinea (fungal) infections after extended study may be used as a laboratory parameter in the differential diagnosis of tinea infections.

Introduction

The sweat gland is a versatile, evolved, and organised excretory channel for the removal of waste products from the body. Sweat glands which are normally described as merocrine are sub-divided into apocrine and eccrine. In human beings, the eccrine glands dominate and number between 2 to 5 millions.

Normally sweat is hypotonic and has a specific gravity of 1.002 — 1.003 and its pH ranges between 4.2 and 7.5. The chief factor that influences the pH is lactic acid excretion. Normal range of sodium is 15 to 60 mEq /L as reported by Arthur Rook[1]. Chloride concentration is equivalent with sodium and varies in parallel fashion. The chloride concentration ranges between 32.9 to 63.6 mEq/L. The sodium and chloride concentrations are influenced by exercise, diet, temperature and rate of sweating[2]. The potassium content of the sweat has a normal range of 0 to 5 mEq/L[3]. It is believed that chloride has the relationship with potassium in the ratio of 9 : 1. The mean urea level of sweat is 68 mg%[4], and is always higher than blood urea with quotient of sweat urea Nitrogen/ BUN 2 : 1. The mechanism of urea formation could be selective uptake from blood with reabsorption of water or new formation of urea in sweat glands[2]. The observation of large amounts of arginine by Hier et al[5]., and the observation of ornithine by Rothman and Sullivan[6] suggest that possibly urea is manufactured in sweat gland by the action of the enzyme arginase which splits arginine into urea and ornithine.

Sweat also contains creatinine, ammonia, aminoacids, glucose, choline, iron, calcium, magnesium sulphate and

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Received for publication on 6—11—1975

phosphate. Sweat is characterised by the absence of proteins and bicarbonates.

Alteration in sweat chemistry has been studied by some workers and it is of great importance to note that raised levels of electrolytes form the diagnostic index of fibrocystic disease of pancreas. Changes in electrolytes may be found in Cushing's disease, Addison's disease, Nephrosis, Congestive cardiac failure and after administration of hormones like Aldosterone and ADH[2]. Sweat in hyperhidrosis demonstrates the elevated levels of electrolyte and normal urea levels confirming the earlier opinion that electrolytes excretion is directly proportional to the rate of sweating and NPN excretion decreases with the increase in the rate of sweating[7]. However, knowledge of sweat chemistry in common skin diseases is scanty.

In the light of the above knowledge an endeavour is made in the present study to evaluate the levels of sodium, potassium, chloride and urea in the sweat in common dermatological conditions like psoriasis, hyperhidrosis, tinea infections and pustular folliculitis and identify whether sweat analysis forms a formidable tool of laboratory diagnosis in dermatological conditions.

Material and Methods

Patients attending the Dermatology Department of Government General Hospital, Guntur were chosen for this study at random. Patients belonged to both sexes and were not in receipt of any medication. A group of 15 healthy individuals were chosen from the staff as control group.

Face, front and back of neck of subjects were cleaned at first with soap and tap water and then with distilled water and areas were wiped gently with clean towel. Sweat secretion was induced by thermal stimulation and mild exercise without influence of any drug. The sweat from the skin of forehead, face and neck was collected and transferred to a clean test tube through a fine capillary tube. This sample was used to analyse sodium, potassium chloride and urea levels. Sodium and potassium were estimated by Lange Flame Photometer. Chloride was estimated by the titrimetric method of Vanslyke[8] and urea is estimated by the micro-diffusion method of Conway[9].

Results

The sweat electrolytes and urea levels observed in fifteen normal subjects (control groups) and ten patients of psoriasis, ten patients of hyperhidrosis, six patients of tinea infection, and five patients of pustular folliculitis are presented in Tables 1 and 2.

The mean sweat sodium in control group was found to be 122.6 ± 15.1 mEq/L while in subjects of psoriasis, hyperhidrosis, tinea infection, pustular folliculitis, it was found to be 192.4 ± 34 mEq/L, 174.9 ± 32.2 mEq/L, 165 ± 23.9 mEq/L and 263.6 ± 33.3 mEq/L respectively. Sweat sodium level thus demonstrated statistically significant rise in all the diseases studied.

The mean sweat chloride in control group was observed to be 82.7 ± 29.1 mEq/L while values of 138.5 ± 25.3 mEq/L, 120.9 ± 30.6 mEq/L, 108.5 ± 16.9 and $128\ 6 \pm 29.3$ mEq/L were encountered in psoriasis, hyperhidrosis, tinea infection and pustular folliculitis respectively demonstrating the significant elevation in all, along with elevated sodium levels.

Mean sweat potassium in control group was found to be 11.9 ± 3.1 mEq/L while in psoriasis, hyperhidrosis, tinea infection, pustular folliculitis 21.8 ± 13.8 mEq/L 19 ± 8.8 mEq/L, 11.9 ± 4.4 mEq/L and 22.6 ± 6.1 mEq/L were observed respectively. Tinea infection is

TABLE 1

Sweat composition in control group and psoriatic subjects.

Investigation	Control (N = 15)	Psoriasis (N = 10)	Statistical significance (P. Value)
1. Sweat Sodium (mEq/L)	122.6 ± 15.1* (92 to 147) †	192.4 ± 34 (133 to 256)	0.001
2. Sweat Potassium (mEq/L)	11.9 ± 3.1 (8 to 17)	21.8 ± 13.8 (12 to 60)	0.05
3. Sweat Chloride (mEq/L)	82.7 ± 29.1 (43 to 130)	138.5 ± 25.3 (90 to 174)	0.001
4. Sweat urea (mg%)	57 ± 20.9 (24 to 93)	81.9 ± 43.2 (22 to 150)	NS

* Mean ± Standard deviation † Range NS = Nil Significant.

TABLE 2

Sweat composition in hyperhidrosis, tinea infection, pustular folliculitis.

Investigation	Hyperhidrosis (N = 10)	Statistical significance (P. Value)	Tinea infection (N = 6)	Statistical significance (P. Value)	Pustular folliculitis (N = 5)	Statistical significance (P. Value)
1. Sweat Sodium (mEq/L)	174.9 ± 32.2 * (100 to 280) †	0.001	165 ± 23.9 (139 to 201)	0.001	263.6 ± 33.3 (149 to 391)	0.001
2. Sweat Potassium (mEq/L)	19.2 ± 8.8 (9 to 40)	0.05	11.9 ± 4.4 (5 to 16)	NS	22.2 ± 6.1 (14 to 30)	0.01
3. Sweat Chloride (mEq/L)	120.9 ± 30.6 (81 to 165)	0.001	108.5 ± 16.9 (80 to 126)	0.01	128.6 ± 29.3 (100 to 166)	0.01
4. Sweat Urea (mg%)	64 ± 24 6 (44 to 120)	NS	135 ± 78.6 (60 to 285)	0.02	96.8 ± 40.9 (27 to 135)	0.05

* Mean ± S.D., † Range NS = Nil Significant.

characterised by normal levels as seen in control group while the other three disorders showed significantly elevated levels of sweat potassium.

Mean sweat urea in control group was observed to be 57 ± 20.9 mg% while the values of 81.9 ± 43.2 mEq/L in psoriasis, 64 ± 24.6 hyperhidrosis, 135 ± 78.6 in tinea infection and 96.8 ± 40.9 in pustular folliculitis were observed. There is significant rise in tinea infection and pustular folliculitis and of these, tinea has shown more significantly elevated level of sweat urea.

Discussion

In the present study control group has shown mean sweat urea value of 57 ± 20.9 mg% which is slightly less than mean value of 68 mg% obtained by Lobitz[4]. Sweat sodium is 122.6 ± 15.1 mEq/L which is higher than already reported levels of 15-60 mEq/L[1]. Sweat chloride level of 82.7 ± 29.1 mEq/L was also found to be higher than previously recorded level of 32.9 to 63.6 mEq/L. Sweat potassium level is 11.9 ± 3.1 mEq/L which is also higher than the value reported previously (vide introduction).

These variations may be due to various factors influencing sweat composition such as temperature, exercise and rate of secretion. It was already pointed out that the increased rate of sweating increases expulsion of sweat electrolytes and decreases NPN expulsion[2,7].

In the present study of 10 cases of psoriasis, the levels of sodium, and chloride showed steep rise while sweat urea did not show any statistically significant increase and sweat potassium has shown significant rise when compared to control group. In psoriasis the area of lesion is anhidrotic or hypohidrotic while surrounding area happens to be hyperhidrotic. The increased salt (Sodium Chloride) concentration observed in present study depicts true hyperhidrosis. The elevated sweat potassium level may be due to the scales in psoriasis which increase sweat loss of potassium and they add up to sweat pottassium level being themselves rich in potassium.

Subjects of hyperhidrosis (Ten) has shown similar results as shown by psoriatic subjects but for slightly lower mean levels compared to psoriatic levels and are in conformity with previous findings[7]. The patients did not present any evidence of internal pathology such as thyrotoxicosis, hypoglycemia or carcinoid syndrome. Hyperhidrosis is an effect rather than cause. The findings support the already known fact that the electrolyte concentration in sweat increases with the increase of rate of secretion and BUN decreases.

Patients of tinea infection (6) showed significant rise in levels of sweat urea, sodium and chloride levels while potassium levels were same as that of control group. The cases included tinea versicolor and tinea circinata. The alterations in two electrolytes is presumed to be due to increased rate of secretion. The significant rise of sweat urea levels is very characteristic and needs extended study to be utilised as a tool of laboratory diagnosis of tinea infection. The increased sweat urea is possibly contributed by hyperkeratinisation and increased protein break down. The chemical milieu constituted by various aminoacids of scales of tinea infection may act as substitute for urea production by the action of arginase. The presence of citrulline, aspertic acid, ornithine and arginine which participate in Kreb's – Hanslett cycle suggest the production of urea in the sweat glands of skin.

Five cases of pustular folliculitis studied has shown highly significant increase in electrolytes and slightly significant rise of urea level. The electrolyte increase may be due to generalised hyperhidrosis associated with this infection. Sweat urea level rise may be due to protein break down and abnormal proteins that are produced by causative bacteria.

Acknowledgments

We acknowledge our gratitude to all the volunteers and patients for their cheerful endurance and co-operation in the study. Thanks are also due to Sri R. V. Anjaneyulu for secretarial assistance.

REFERENCES

1. Rook A and Ebling FJG; Text Book of Dermatology 2nd Ed, Blackwell Scientifiic Publications, 1972.
2. Rothmann S: Physiology and Biochemistry of the Skin, 1954.
3. Randall: Volume and composition of blood plasma gastrointestinal secretions and sweat, S Clin North America, 32:3, 1952.
4. Lobitz WC and Osterberg AE: Chemistry of palmar sweat, Arch Derm and Syph 56: 462, 1947.
5. Hier SW, Cornbleet T and Bergeim O: The aminoacids of human sweat J Biol Chem, 166:327, 1946.
6. Rothman S and Sullivan MB: Amino Acids on the normal Skin Surface, J Invest Derm, 13:319, 1949.
7. Fitzpatrick: Dermatology in General Medicine, Blackiston Publication, 1971.
8. Vanslyke DD: Determination of Chloride J Biol Chem 58:523, 1923.
9. Conway E: Microdiffusion Analysis and Volumetric error Princeton, NJ, Van Nostrand, 1960.