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KELOIDS: A STUDY OF THE IMMUNE REACTION TO SEBUM

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SUMMARY

A controlled study of the "immune reaction to sebum' pathogenesis of keloids was carried out at the University College Hospital, Ibadan, Nigeria; on 22 humans using a homogenate solution of liquid paraffin and vernix caseosa from neonates. The 22 human volunteers comprised two groups, 11 of which were keloid formers and 11 non keloid formers. 0.1cc of sterile liquid paraffin was injected intradermally into the anterior aspect of the left forearm. The mean reaction in keloid formers was 11.55mm while in non keloid formers, it was 8.18mm. Although a higher reaction was demonstrated in this study, in keloid formers, this was found not to be significant ($p > 0.05$), thus suggesting that sebum may not be an important factor in the pathogenesis of keloid.

INTRODUCTION

Over the last four decades, there have been many investigations on the immunological pathogenesis of keloids. Glucksmann and Mowlem(1,2), in a clinical and histological studies implicated detached portions of skin left behind in a wound. Oluwasanmi(3) suggested melanin and altered collagen, Mukherjee(4), cutaneous antigens, Nunzi *et al*(5), haematoporphyrins from blood products that leak into the dermis, while Abdalla-Osman *et al*(6) and Yagi *et al*(7) suggested sebum. Yagi *et al*(7) obtained a significantly low recurrence rate when they proceeded surgical excision by desensitisation of the keloid former with sebum.

To substantiate this mode of management, and the sebum autoimmune theory of the pathogenesis of keloids, the reaction of the keloid former with that of the non keloid former to a homogenised mixture of vernix caseosa (90% sebum) in paraffin oil was compared.

MATERIALS AND METHODS

Twenty two humans comprising two groups of 11 each and selected by convenience method of non random sampling were included in the study. A group consisted of keloid formers while the other consisted of non keloid formers. A keloid former was taken to be one that had at least one clinically obvious lesion. Those in the non keloid former group had no positive past medical history of the lesion and did not have any clinical evidence of the presence of the lesion. Each individual had 0.1cc of sterile liquid paraffin injected intradermally into the anterior aspect of the right forearm as a control, while 0.1cc of the homogenate was injected also intradermally into the anterior aspect of the left forearm.

The homogenate consisted of a mixture of liquid paraffin and vernix caseosa. vernix caseosa is a secretion obtained from the body surface of neonates. It consists mainly of sebaceous gland secretions (sebum) as well as epithelial debris. The sebum was contained to a 6% concentration (WV) in the homogenate.

The reaction which was the diameter of the wheal that developed, was measured in millimetres after 48 hours. The differences between the readings of both forearms obtained from the keloids formers were compared with those obtained from non keloid formers using the Student's t test. The level of significance was taken to be $p < 0.05$.

RESULTS

The readings obtained, are shown in Tables 1 and 2. The mean reaction in keloid formers was 11.55mm while in non keloid formers, it was 8.18mm. Although this value was higher in keloid formers than in nonkeloid formers, when compared, the difference was not statistically significant $p = 0.13$ (Table 3).

Table 1

Keloid formers

Name	Age	Sex	Site of keloid	RF (mm)	LF (mm)	Reaction (mm)
O.O.	21	F	R ear lobe	0	7	7
A.O.	18	F	R ear lobe	2	14	12
O.E.	21	F	Neck	3	10	7
E.U.	21	F	Presternal	0	12	12
O.N.	20	F	Breast, chest	3	12	9
O.N.	32	M	Occipital, scalp	0	8	8
F.O.	35	F	Presternal	0	30	30
A.K.	19	M	Cheek, presternal	0	12	12
O.B.	22	F	R hand dorsum	2	9	7
B.A.	20	M	Chest, back, buttock	0	14	14
R.U.	28	M	Jaw, upper limb	0	9	9
Total						127
Mean						11.55

Table 2

Non keloid formers

Name	Age	Sex	RF (mm)	LF (mm)	Reaction (mm)
A.D.	24	F	2	9	7
O.F.	31	M	0	9	9
R.A.	17	F	4	8	4
A.F.	31	F	0	5	5
A.J.	45	M	0	10	10
A.O	21	M	0	10	10
A.A.	23	M	1	12	11
A.R.	27	M	1	12	11
A.J.	24	M	0	9	9
A.U.	21	F	1	9	8
A.U.	20	M	1	7	6
Total					90
Mean					8.18

Table 3

Student's t test: reaction to homogenate, keloid formers vs non keloid formers

Table values											Total
7	12	7	12	9	8	30	12	7	14	9	127
7	9	4	5	10	10	11	11	9	8	6	90

$t = 1.59$

$p = 0.13$

DISCUSSION

A lot of work has been performed to suggest the immunologic basis of keloid formation. Whereas a delayed hypersensitivity reaction to cutaneous antigens has been demonstrated, it is not yet clear which cutaneous antigen the reaction is directed against. While an antinuclear antibody directed against fibroblasts in keloid tissue has been substantiated(8), cutaneous antigens such as melanin, blood products, and sebum have only been suspected(3,5,6)

Kischer *et al*(9) found localised increase in IgA, IgG and IgM in keloid tissue compared to normal skin. However an investigation of the general immune reactivity revealed a significantly higher serum level of IgM and C3 but lower IgA and C4 in keloid formers than non keloid formers(10). The serum IgG was similar in both groups. Cohen *et al*(11) also demonstrated a greater alpha globulin level in keloids and hypertrophic scar tissue than in normal skin and scars.

Yagi *et al*(7) obtained gratifying results when they combined surgical excision of keloid with desensitisation using sebacious material. Their method of treatment was based on the observation of the association of sebacious glands with keloid tissue, and the demonstration of a significant reaction to sebum in keloid formers. Abdalla-Osman(6) also showed that there was a significantly higher reaction in keloid formers than in non keloid formers to sebum. Although a higher reaction rate is demonstrated in this study, this is found not to be significant thus suggesting that sebum may not be an important factor in the pathogenesis of keloid.

Keloid has been observed not to occur in areas where there are no sebacious glands such as the genitalia, the palm, and the sole(7). It is to be noted that the central part of the face, where there is a preponderance of sebacious glands, almost never forms keloid(12). This clinical note is further evidence of the invalidity of the sebum autoimmune pathogenesis of keloid formation.

More research particularly *in vivo* studies is required to establish the aetiology and pathogenesis of keloid. The aetiology is possibly multifactorial.

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