Microbial Flora in Stable Chronic Plaque Psoriasis (A random study of 12 patients)

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الخلاصه :

يعتبرداء الصدفية مرض مزمن ذاتيالمناعه ينشأ من اسباب متعددة العوامل يعتقد ان للاخماج دور مهم في نشوء و تسبب المرض وانها ربما تتأثر بالجراثيم المؤالفه في آفة الصدفيه ان هذه الدراسه صممت لتقييم الجراثيم المؤالفه لدور مصابين بداء الصدفيه الصدفيحي المزمنه من المرضى (4،33%) اظهروا وجود بكتريا العنقوديه الذهبيه حين كانت بكتريا سالبه صبغه الكرام تمثل من المرضى(83,4%) الامر الذي يثير امكانية تأثر الصدفيه بها من خلال السموم الداخليه التي تأتي من هذه البكتريا نتساءل عن إمكانية الاستفاده من المظلحيواتيه ضد سالبه صبغه الكرام لدى مرضى الصدفية الدراسه اظهرت وجود بكتريا الخيطيه الشعاعيه وبنسبه (41,7%) ما يثير احتماليه تأثر الصدفيه بها كما انها يمكن ان تشكل بعض الخطر على المرضى

Introduction:

Psoriasis vulgaris is a common, chronic, recurrent, inflammatory disease of the skin characterized by round, erythematous, dry scaly plaques of various sizes. The lesions have predilection for he scalp, nails and extensor surfaces of limbs, elbows &knees.

Psoriasis is characterized by three main pathogenic features: abnormal differentiation, keratinocyte hyperproliferation & inflammation .Accelerated epidermopoisis has been considered to the fundamental pathologic event in psoriasis (1).

The cause for that is still unknown, however, psoriasis is thought be to an auto-immune disorder influenced by some inheritance interplaying with acquired environmental factors (i.e. stress, hypocalcaemia, hormonal disturbances, infections).Due to the strong association between microbial
skin flora &infections influencing psoriasis, we should focus on this flora & its change.

The normal skin of healthy individuals is highly resistant to invasion by the wide variety of bacteria to which it is constantly exposed (2).

Bacteria are unable to penetrate the keratinized layers of normal skin &when applied to the surface, rapidly decrease in number (3).

The nature & relative importance of the factors thought to be involved in
this local resistance to bacterial multiplication & to infection are not clear (4). The presence of natural antibacterial sebaceous secretions may be a factor in the bacterial elimination from the skin .Streptococci appear to be particularly sensitive (3). The role of circulating immunoglobulin,cellular immunity &delayed hypersensitivity in the defense coetaneous mechanism is under intense investigation. However , the greater frequency with which a specific cutaneous & mucous membrane mycotic infection ,candidiasis occurs in patients with severe combined immunodeficiency suggests a relationship (2).

The relative dryness of normal skin contributes to the marked limitation of growth of bacteria, especially gram negative bacilli with their higher moisture requirements (Escherichia coli , pseudomonas, proteus). Whereas application of 10^6 p .aerogenosa alone on normal skin application lesion , the presence of a similar inoculum under dressings that increased local skin hydration led to superficial popular & pustule infection (5).

Bacterial interference (the suppressive effect of one bacterial strain or species on colonization by another) exerts a major influence on the overall composition of the skin flora . Profound changes in these bacterial interactions may be effected by the use of antibiotics topical steroid (2).

The association of streptococcal pharyngitis with initiation or exacerbations of gutter psoriasis is –well- established, but the mechanism is unknown .recent evidence suggests that staphylococcus aurous and streptococci secrete large family of serotoxins that are super antigens, producing massive T- cell activation (1)

Rosenberg et al has published extensively on the aggravation of psoriasis by activation of the alternate pathway of complement by malasicia ovalis, or by intestinal yeasts, or by endotoxins produced

by gram – negative bacteria in the gut. He believed this helps to explain:-
*seborrhea localization (M. ovals effect)
*Diaper area lesion (Candida albicans)
*excitation by typhoid vaccine (which contains endotoxin)
*Intestinal enterobacteriacea may cause relapse or exacerbation.
*Severity of psoriasis in alcoholics who are not well protected of end toxins in to the circulation by their damaged kupffer cells.
(6, 7, 8)

Aim of study:

The study was carried out to evaluate microbial flora &its changes in (random sample) of patients with psoriasis vulgasis, so as to be a modest basis for further detailed elaborated studies.

Patients & Methods:

12 patients(10 male ,2 female) with psoriasis vulgaris with only chronic stable plaque type, other variants of psoriasis (i.e. guttate , erythrodermic , inverse ,psoriasis ,etc) were excluded from this study , these patients were subjected to swabs taken from different site of disease involvement (scalp ,trunk, extremities).The swabs were taken from lesional sites &normal skin to see the microbial flora.

The swabs were cultured on blood agar, MacConky agar & Sabourand dextrose agar.

Results:

These 12 patients as shown in the table were found to have staph. aureus colonization in lesion & normal skin in 4 patients (33.4%) as revealed on blood agar.

MacConky agar showed the presence of the gram negative bacteria (E .coli) in the lesional skin in 10 patients (83.4 %) & in normal skin only in 3 patients (25 %).

This agar also showed the presence of Pseudomonas Pseudomallei in the lesional skin of 3 patients (25 %).

Sabauroed dextrose agar showed the presence of Actinomycetes in lesional skin of 5 patients (41.7 %)& only 3 of then were found to have Actinomycetes in their normal skin.

Table of the patients and results of cultures

Patient no.	age	Sex.	blood Agar		maCconky agar		Sab. Dexa Agar	
			lesion	normal	lesion	normal	lesion	normal
1	45 year	Male	*Staph. auras*	*Staph. auras*	*E .coli*	-ve	Actinomycetes	**Actinomycetes**
2	37y	Male	-ve	-ve	*E .coli*	-ve	Actinomycetes	**Actinomycetes**
3	36y	Male	-ve	-ve	*E .coli* *P. seudmallei*	-ve	-ve	**-ve**
4	35y	Male	-ve	-ve	*E .coli*	-ve	Actinomycetes	**-ve**
5	30y	Male	*Staph. auras*	*Staph. auras*	*E .coli*	-ve	-ve	**-ve**
6	24y	Male	-ve	-ve	*Yesinia clitrobacter*	-ve	-ve	**-ve**
7	21y	Male	-ve	-ve	*E .coli*	*E .coli*	-ve	**-ve**
8	20y	female	-ve	-ve	-ve	-ve	-ve	**-ve**
9	17y	Male	-ve	-ve	*E .coli*	-ve	Actinomycetes	**-ve**
10	15y	Male	*Staph. auras*	-ve	*E .coli* *P.Pseudmallei*	*E .coli*	-ve	**-ve**
11	11y	Male	-ve	-ve	*E .coli*	*E .coli*	-ve	**Actinomycetes**
12	**15y**	**female**	***Staph. auras***	***Staph. auras***	***E .coli***	**-ve**	**-ve**	**-ve**

Discussion:

Psoriasis vulgaris is a chronic inflammatory discarder thought to be influenced largely by G +ve bacteria that are super antigen producing massive T-cell activation, or through end toxins produced by G-ve bacteria, that activate the alternate pathogen of complement.

According to best of our knowledge, there are no even simple study putting some light focus on our own patients wether to be from those influenced through G +ve or G -ve bacteria flora.

Gram +ve bacteria (mainly staph. aurous but not streptococci) were seen in patient but in low percentage (33.4 %); & these might be considered of staph.aurous carriers in their psoriatic lesions and there is no staphylogenic skin problems (i.e. boil, folliculitis, etc) apart from possible exacerbation.

On the other hand, MacConky agar of lesional skin showed colonization by E. coli in (83.4 %) of patient & Pseudomonas Pseudomallai (25 %).This reflects that our patient apparently

complain from aggravation of psoriasis through endotoxins produced by these bacteria.The requirements (i.e. hydration & moisture)could be produced partly by extensive usage of topical ointments chronically by the patient (because ointments exert some occlusive effect on skin with subsequent increased hydration). However, the presence of G-ve bacteria in psoriatic skin lesions of our patients may make one wonders if these patient could get benefit from anti-gram negative antibiotic agent, wither topically or systemically.

The presence of Pseudomonas psedomallai in the lesion even in low percentage (25 %), it may carry some risk on patient, because in human, this bacteria may cause severe glander –like illness which may be fatal, or else it may cause mild febrile illness that is subclinical infection.

Saboraund dextrose agar revealed interestingly Actinomycetes in psoriatic lesions of 5 patients (41.7 %) , this organism may play some role in exacerbation or even carry some risk on patients.

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