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PSORIASIS DETECTION METHODOLOGY DISCUSSION AND COMPARISON TO DIFFERENT SKIN PARAMETERS

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ABSTRACT

Psoriasis is a common inflammatory skin disease characterized by abnormal keratinocyte inflammation and differentiation that has a major impact on patients' quality and way-out of life. IL-36γ, a member of IL-36 cytokine family, is highly expressed in psoriasis and plays an important role in inflammation response and colour to cell differentiation. Several studies have linked psoriasis with metabolic syndrome (MS), but only a few of them have used population specific criteria for diagnosis of MS as observed reading the other papers. None of the previous studies have explored whether MS is more frequent in psoriasis when compared to another chronic inflammatory disease of skin or not. The concept of Sense of Coherence (SOC), the main concept in the theory of salutogenesis may help explain the substance of our growth as human being. There is a need of further investigation of Sense of Coherence (SOC), the central concept of salutogenesis and its relationship with long-term sickness such as psoriasis.

KEYWORDS: Psoriasis Detection, Colour Identification, Concept of Salutogenesis, Sense of Coherence, MS, Psoriasis Detection Methodologies.

INTRODUCTION

Psoriasis is a common, chronic skin disease with colour differentiation, affecting approximately 2%-3% of the population. The disease is usually characterised with raised, well-demarcated, discoloured erythematous plaques with adherent silvery scales. The scales are formed as a result of hyper-proliferation and aberrant differentiation of keratinocytes and infiltration of inflammatory cells, such as dendritic cells, macrophages, and T cells in the dermis and neutrophils, with some T cells in the epidermis. The inflammatory infiltrate indicates that cytokines plays an important role in the pathogenesis of psoriasis. Here interesting therapeutic targets could be identified, such as drugs targeting TNF-α, IL-17, IL-22, IL-23 and GM-CSF [1,2,3,4,5].

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However, though these are pre-dominant, the exact mechanisms regulating these abnormal differentiation and immunological dysfunction remain largely unknown. Psoriasis is now considered as a systemic inflammatory disease in world researches. The chronic inflammatory nature of psoriasis has been suggested to be a contributing and potentially independent risk factor for development of MS as seen studied in researches.Several thorough studies has shown an association between psoriasis and MS [8,9,10]. Most of these were of cross sectional design or image processed. There have been only a few case control studies addressing the issue to be detected. Only a few studies from India and very few from one other country have used the revised population specific criteria for diagnosis of MS. Control groups in the previous case control studies were either healthy people or persons with various skin diseases and henceforth we thought that comparing the association of MS in psoriasis with another chronic and often recurrent inflammatory skin disease such as eczema would be more informative. As the lifestyle changes, effect of chronic inflammation of the skin can be expected to be comparable in both groups, and hence, further difference observed between the groups can be more directly attributed to the disease process of psoriasis detection and analysis [14,15,16,17,18].

DIFFERENT MODEL DISCUSSIONS

The current analysis represent that disclosure to IL-36γ persuade differential inhibition, up-regulation and pro-inflammation cytokines secretion .In the new decade, the major problem for illumination of Psoriasis remained arguable counting the auto-immune origin of the swelling process, the congruity of cutaneous versus systemic factors as well as the part of genetic and the surrounding effect on the focalization of the disease. Past studies of Psoriasis had conveyed that pronouncement of IL-36γ is up-regulated in colonic epithelial form sufferers with Inflammatory Bowel Disease (IBD). But in the Lung the pronouncement of Il-36γ is heavily induced in different models of Asthma that can be initiated by bronchial epithelium exposed to Viral infection, smoke, or inflammatory cytokines. From the recent analysis of Psoriasis it is found that only one exposure to IL-36γ lead to the growth and spread of Psoriasis skin Lesion [1,2,3,6,7,10]

The epidermis tissue regenerate itself within the body which is purposed to protect the inner parts of the body. System Keratinocyte differentiation is important for the emergence of the entire epithelial fencing. The non-adaptive epidermal fencing and the abnormal

Keratinocyte are the main cause for several skin disease such as Psoriasis ,basal and squamous skin Cancer and atopic dermatitis. The main embryonic feature of Psoriasis are enlargement of epidermis-acanthosis, amplification of the cornified layer Parokenotosis [12,15,20].

Alongside the provocative characteristics, Psoriasis lesions manifest the peculiar symptoms of altered epidermal differentiation. In Psoriasis the whole Keratinization procedure is not finished because of the accelerated Keratinization process is entrusted by untimely cell death. Past studies have revealed t6hgat the abnormal differentiation of Keratinocytes is the reason of pathological aspect of Psoriatic lesions. Then before time differentiation signs that are demonstrated in Psoriasis are small proline-rich proteins (SPPR), Cystanine A and transglutaminase 1 and the later differentiation sign are profilaggrin and loricrin.

The proper inspection of Psoriasis had conveyed that the unusual differentiation of Keratinocyte results in adversity of the skin barrier function. It is revealed in our Research that the treated HaCaT cell IL-36γ can stop the differentiation of Kerationocyte that results in

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decreased regulation of filaggrin, involurin, Keratin 1 and Keratin 5 at mRna levels. Moreover IWP-2 can brick the reaction of IL-36γ on HaCaT cells [4,7,8,11,15,18].

The swelling of skin that is noticed in Psoriasis is the outcome from the various interactions of K,C and T cells, antigen presenting cells (APC) , fibroblast and endothelial cells. In Psoriasis the Keratinocyte eagerly takes part in forming multi-molecular grid harmonized by cytokines. Psoriatic Keratinocytes are important origin of ant-imicrobial peptides, including IL-36γ, β-defensive and Psoriasin, which manifest the effects of Chemotaxis and shape-immune cell function. They are also reciprocate to dendritic cell and T cell derived cytokines including interferons, TNF interlukin-17 and many more things [1,2,5,7,12.13].

In gross, the present Research had manifested that Haca T cells and IL-36γ subdue the consequence of differentiation and encourage the inflammatory response of Psoriasis. These results depicts that IL-36γ may constitute the potential target for the treatment of Psoriasis.

CONCLUSION

The underlying conclusion that can be drawn from this review paper is that the Psoriasis is a chronic skin disease which is caused by over active immune system. It is characterised by rashes, dryness, fissures, small bump, thickness or redness of skin. The appearance of Psoriasis is very awkward. The main cause of Psoriasis is disclosure to IL-36 that causes differential inhibition of cells and pro-imflammation cytokine secretion. This happens because the differentiation of epidermis tissue is not possible due to untime cell death which causes Psoriatic inflammation. The IL-36 can be treated further and may be depicted as a measure for the treatment of Psoriasis [7,15,16,17]

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