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Report on Scientific Meeting

Management of Psoriasis: Quo Vadis?

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Venue:	Saloon III & IV, Hyatt Regency Hotel, Tsim Sha Tsui, Hong Kong
Speaker:	Prof. Christopher Griffiths Professor of Dermatology and Associate Dean for Research, Faculty of Medical and Human Sciences, The University of Manchester, United Kingdom
Organiser:	The Hong Kong Society of Dermatology and Venereology

Genetic study of psoriasis found an association of psoriasis with loci HLA-C allele & ERAP1. HLA-CW0602 is associated with early-onset psoriasis and guttate psoriasis. Stratification of psoriasis vulgaris according to phenotype (e.g. thickness of plaque, size of plaque, nail involvement or not) will help to predict the prognosis of disease.

Generalized pustular psoriasis is now believed not to be psoriasis itself but another disease entity associated with psoriasis. It is a sudden onset multisystem disease and responds poorly to current therapy. Studies found a deficiency of interleukin-36 receptor antagonist which allows continuous inflammation in generalized pustular psoriasis.

Early-onset psoriasis (age <40 years old) accounts for 75% of cases with psoriasis. Late-onset psoriasis (age >40 years old) accounts for 25% of cases. HLA-CW0602 is associated with early onset psoriasis. Epidermal Langerhans cell function is different between early and late-onset psoriasis. There are more CD2 cells in late-onset psoriasis. Hence early-onset and late-onset psoriasis are different diseases, such as the different types of diabetes mellitus. However, there are no clinical trials yet to address the efficacy of medication in the treatment of late-onset psoriasis.

Problem sites in the management of psoriasis include scalp and nails. Scalp psoriasis is still difficult to treat despite progress with new steroid-containing shampoos and scalp preparations containing a dual combination of calcipotriol and steroid. Anti-TNF and systemic treatment are helpful but topical treatment is not useful in nail psoriasis.

Factors predicting the response and toxicity to therapy include clinical phenotype of psoriasis, genetic makeup (personal genome), immune biomarker, HLA-CW0602. Pharmacogenetic factors that affect treatment outcome include genetic variation in efflux transport influence outcome of methotrexate (e.g. ABCC1 is associated with good response to treatment), and TNFAIP3 polymorphism which affects the response to TNF blockade in psoriasis.

One study compared the outcome of treatment between etanercept and ustekinumab (anti-interleukin 12, 23) for moderate to severe psoriasis with the following results: 68% and 74% achieved PASI 75 at week 12 in two groups of patients receiving ustekinumab 45 mg and 90 mg respectively, while 57% achieved PASI 75 in the etanercept group (50 mg 2x/wk). Patients who failed to respond to etanercept still responded to ustekinumab. Etanercept 0.8 mg/kg/wk improved psoriasis in children (age 4-12 years old). Biologics cannot produce a cure. Long term use of anti-TNF may develop antibody and decrease efficacy. Etanercept can be used intermittently. Ustekinumab can be used intermittently and restarted with good response. It is uncertain whether systemic treatment of psoriasis decreases co-morbidity (e.g. decrease cardiovascular risk).

Psychosocial aspect is important in the management of psoriasis. There are a few good studies on the effectiveness of cognitive behavourial therapy (CBT) which showed an enhanced response to therapy in psoriasis and decrease anxiety. In reality, there is a market gap in the management of mild to moderate psoriasis with no significant recent advance in management.

Learning points:

There is a lot of unmet need in the management of psoriasis which requires further exploration. Psychosocial aspect is important in management of psoriasis.