The 14th Regional Conference of Dermatology (Asian-Australasian)

reported by Dr. L. Y. Chong

Date:	26-30 July, 2000
Venue:	Shangri-La Hotel Kuala Lumpur, Malaysia
Organizer:	Dermatological Society of Malaysia

Update on Newer Phototherapy Regimes

Speaker: Dr. J. Hawk

The speaker had talked on different types of phototherapy regimes, with regard to their efficacy and possible adverse effects.

It is known that the inflammatory activity localised at the region of the epidermal basal layer is responsible for many of the dermatoses treated by phototherapy. This area however is also most susceptible to the damage by ultraviolet light, leading to skin aging and cancer. Therefore, it appears that the more effective is the treatment, the more adverse effects may arise. In general, a cumulative dosage of 1000-1500 J/cm^2 or 150-200 sessions of phototherapy increases cancer risk in fair skin.

Over the past decades, different type of ultraviolet (UV) lamps had been developed. These include broad-band UVB, UVA, narrow-band UVB and UVA$_1$. The broad-band UVB penetrates the skin inefficiently. It is absorbed predominately by the DNA in superficial keratinocytes, causing sunburn and yet without fully treating the sites of the disease activity. However the basal cells are largely spared and therefore it has less long term side effect. The UVA on the other hand penetrates deeply in the skin, but it is not well absorbed by DNA alone unless psoralen is present at all depths in the skin. It is relatively more effective than broad-band UVB, but has more long term adverse effect. Similarly the narrow-band UVB can also penetrate to the basal layer because it is not efficiently absorbed in upper epidermis. Its better efficacy would likely cause greater adverse effects as well. According to the speaker, it seems less effective than PUVA but possibly with less long term adverse effect. But further studies are needed with regard to these. The UVA$_1$ (340-400 nm) can penetrate far into deeper layer of dermis. It is poorly absorbed by DNA and is absorbed primarily by non-DNA substances before being re-emitted. It probably causes secondary DNA damage through free radical and singlet oxygen activity. It is a new regime, therefore the efficacy and long term adverse effect are still uncertain.

The speaker then mentioned about the approach in using these different regimes: how to use and what to expect? For broad-band UVB, it can be used in treating superficial disease such as guttate psoriasis. One would expect to have mild response and low long term adverse effect. For narrow-band UVB, it can treat deeper disease with moderate response and with some long term side effects. For systemic PUVA, it can be used for deep disease. One would expect good response but also significant long term adverse effect. For immersion PUVA, it can be used in those unsuitable for systemic PUVA. Again, one would expect good response but some long term effect, although up to now, it has not yet been documented to have significant long term adverse effect. Finally, UVA$_1$ works on different purposes in treating deep dermal lesions. Very large dose is needed in order to have therapeutic effect and little is known on its long term adverse effect.

In conclusion, it seems unlikely that any phototherapy regime is totally safe. Nevertheless, phototherapy is an excellent tool if appropriately used.

Learning points:

In general, a cumulative dosage of 1000-1500 J/cm^2 or 150-200 sessions of phototherapy increases cancer risk in fair skin.

Risks of Phototherapy Revisited

Speaker: Dr. P. Gritiyarangsan

The increased risk of non-melanoma skin cancers (NMSC) has been well-documented in Caucasian patients who have received PUVA. However, there is very limited data in Oriental patients. The risk factors of NMSC include total accumulative dose of the UVA received, co-carcinogens (such as superficial X-ray therapy, arsenic, etc.) and the skin type of the patients. According to the experience of the speaker, co-medication with methotrexate with phototherapy only marginally increases risk of squamous cell carcinoma, therefore this combination actually can be used in treating severe psoriasis.

The speaker also mentioned the strategies that can reduce the risk of skin cancer in phototherapy. These include limiting the number of treatment sessions or cumulative dosage; shielding the areas where are prone to skin cancer (face, genitalia); reducing the exposure to sunshine; taking cautions to patients who have exposed to other carcinogen such as arsenic; and using combination therapy to increase efficacy and reduce risk of carcinogenesis.

Debate: Systemic Steroids Should be Used for Early Management of Stevens-Johnson Syndrome

Speakers: T. Ganesapillai (proposer)
P.S. Friedmann (opposer)

For

Stevens-Johnson syndrome is caused by immunological process, giving rise to significant morbidity and mortality. Systemic steroid can decrease the oedema, necrosis, scarring, etc. that occur as a result of this immunological reaction. It may prevent further progression of the disease process. In addition, Stevens-Johnson syndrome is an acute disease that is stressful to the body, in which the endogenous steroid may be insufficient. The exogenous steroid can therefore replace the deficiency. Furthermore, the incidence of mucosal scarring seems to be increased in those who have not received systemic steroid, and this may result in blindness if ocular complication occurs. The proposer also mentioned that the circulating antibodies can be decreased by using systemic steroid. Finally, he stated that many peer-reviewed big textbooks in dermatology had mentioned that early use of steroid was important, and it was often the patients' preference to receive the treatment as well.

Against

The opposer started by saying that scientific proof was essential in evidence-based medicine. Currently, lots of workers think that erythema multiforme differs from Stevens-Johnson syndrome/toxic epidermal necrolysis, as they are different disease processes. The use of systemic steroid in Stevens-Johnson syndrome is based on poor quality citation from uncontrolled small-scale studies. There is no good evidence that steroid can modify the process of this disease. With regard to the mucosal scarring, it is often the result of secondary infection that is preventable. He also mentioned that in Stevens-Johnson syndrome/toxic epidermal necrolysis, the keratinocytes expressed the Fas receptor (CD95) (suicidal receptor) and died by themselves (apoptosis). It is known that steroid cannot interfere this dying process. Finally the opposer stressed that critical analysis was needed when reading the literatures and even the peer-reviewed big textbook might be out-of-date.

The debate was voted by a show of hands from the audience. It seemed that the supporters were mainly from the eastern countries, while the opposers were mainly from the western countries, showing that there were different views on this topic between the east and the west.

Learning points:

In the issue of use of steroid for early Stevens-Johnson Syndrome, critical analysis was important when reading the literatures as even the peer-reviewed big textbook might be out-of-date.