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Research review paper

Insight into immunocytes infiltrations in polymorphous light eruption

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ABSTRACT

Polymorphous light eruption (PLE) which is one of the most common photodermatoses has been demonstrated to be immune-mediated disorder. Resistance to UV-induced immunosuppression resulting from differential immune cells infiltration and cytokines secretion has been highlighted in the pathogenesis of PLE. In this study, we reviewed differential patterns of immune cells infiltrations and cytokines secretion that may contribute to PLE occurrence and development.

1. Introduction

Polymorphous light eruption (PLE) belongs to one of the most common photodermatoses in all skin types, and its morbidity in North America and Europe can reach 10%–20% in young adults(Rhodes et al., 2010). PLE manifesting as multi-shaped lesions such as papule, blister and plaque, at exposed parts with conscious itching, occurs more often in 20–30-year-old female youths and affects physical and psychological health of patients severely (Gruber-Wackernagel et al., 2014, Jong et al., 2008). More than that, the clinical symptoms of PLE are generally continuous progression which may even develop into lupus erythematosus (Millard et al., 2001).

Previously the cause of PLE was thought to be related with genetics (Millard et al., 2000, Millard et al., 2008) and environmental factors (Bissonnette et al., 2012, Nakamura et al., 2016). Polymorphism of glutathione S-transferases (GST) gene family has been brought up to be probably result to PLE development, however no association between the functional polymorphisms of GST isozymes and PLE was found (Zirbs et al., 2013). Thereby immune reaction changes induced by resistance to ultraviolet-induced immunosuppression have been highlighted by many recent studies (Gambichler et al., 2013, Gruber-Wackernagel et al., 2011b, Koulu et al., 2010, Sarchio et al., 2014, Schweintzger and Reginato, 2013, Wolf and Legat, 2010), and some of them have pointed towards differential immune cells infiltration as a critical role in the occurrence and development of PLE (Gambichler et al., 2013, Schweintzger and Reginato, 2013, Wolf and Legat, 2010).

2. Immunocytes infiltration in PLE

Efflux or influx of immunocytes after radiation of ultraviolet (UV) largely results from cytokines which act as critical participant of immune system regulator. (Fig. 1) Studies on cytokines expression of PLE demonstrated that tumor necrosis factor (TNF)-α, interleukin (IL)-4, IL-10, IL-1β and IL-18 were reduced in exposed skin of PLE patients. Among the differential expressed cytokines in PLE, TNF-α secreted by keratinocyte (KC) and mast cell and contributing to langerhans cell (LC) migration, IL-10 secreted by macrophage (Mø) and KC and secretion of IL-4 by neutrophil, indirectly indicate the differential infiltration of immunocytes. Additionally, diverseness in migration patterns and immunocytes proliferation can also result to the differences in immune cells infiltrations (Jenkins et al., 2011, Taguchi et al., 2013). Immunocytes migration which belongs to immunocyte homing is essential for immune response and homeostasis, and changes of microenvironments, external stimulus (Byrne et al., 2008, Moser and Loetscher, 2001) and decrease of intrinsic chemotaxis property (Gruber-Wackernagel et al., 2011b) could eventually trigger aberrant immunocyte migration resulting in immune response of inflammation diseases. As to proliferation, it is an important feature of some immune cells. Immunocytes proliferation which proceeded in a split way contributes to strengthening of immune response by releasing cytokines, interacting with other immunocytes and so on(Badr et al., 2012, Jenkins et al., 2011).

Many studies have focus on differential immune cell infiltration

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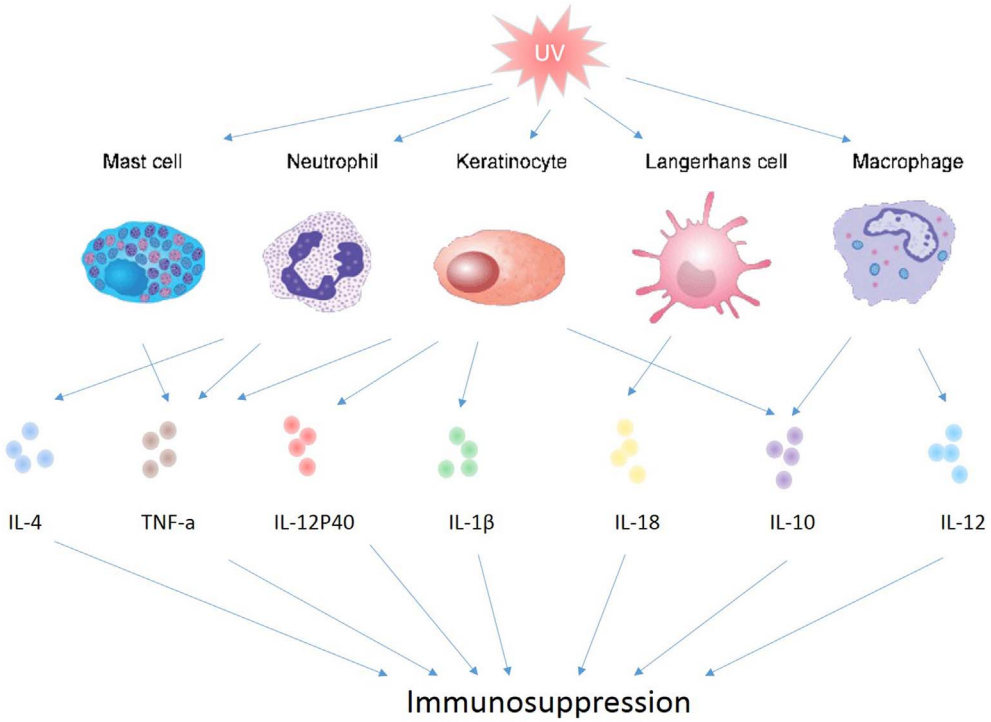

Fig. 1. Interaction network of immune cells and cytokine in PLE development.

Table 1
Differences in infiltration of immune cells and possible inducible factors to the differences.

Immune cells	Under UV		Inducible Factors
	In PLE patients	In healthy individuals	
LC	Persist in the epidermal	Migration from the epidermal to lymph node	Reduction of IL-1,TNF-α
PMN	Significantly decreased infiltration	Migrate from the blood vessels to skin tissue	Reduced chemotaxis ability E-selectin?
Mø	Decreased infiltration	Increased infiltration in epidermis and dermis	Reduced proliferation of local macrophages?
Mastocyte	Decreased infiltration	Migration from skin to draining lymph node	Not shown
Treg	Decreased infiltration	Proliferation occur in situ and in thymus	Reduced low level of vitamin D
KC	Express more IL-10, ICAM-1	Express moreIL-1,IL-6,IL-7, IL-10,IL-12,IL15and TNF	–
pDC	No pDCs expression	Accumulation and increase of chemoattractant	Not shown

LC, langerhans cell; PMC, neutrophil; Mø, macrophages; MC, mast cell; Treg, regulatory T cell; KC, Keratinocyte; pDC, plasmacytoid dendritic cell.

degree in the skin of PLE patients compared with the skin of healthy individuals, which plays a critical role in generating the immune response and contributing to the pathogenesis of PLE, thereby it is necessary to make a summary to the different infiltration degrees of immunocytes in PLE including LC, neutrophil, Mø, mastocyte, T regulatory cell (Treg) and KC (Table 1).

3. Immunocytes

3.1. Langerhans cell

LC affiliated with dendritic cell (DC) is derived from the bone marrow, and can also be differentiated into the peripheral blood mononuclear cells. They account for 3%–8% of the total epidermal cells, and distribute in the basal layer of epidermis and upper appendages. As antigen-presenting cell in skin, LC possesses the function of incepting, disposing and presenting antigens (Sparber, 2014). LC was shown to play a critical immunoregulatory role in UV-induced cutaneous immunosuppression (Fukunaga et al., 2010, Halliday et al., 1998, Kolgen et al., 2003, Schwarz et al., 2010, Taguchi et al., 2013). Following UV radiation, migration of LC contributed to local immunosuppression in healthy skin.

When exposed to UV the number of LC in the epidermis of healthy skin was brought up to be reduced (Ullrich, 1995). Consistent with the result, a further study found after the use of sunscreen the expressions of CD207, CD80 and CD86 on LCs were increased, indirectly demonstrating the decrease of dermal LCs (Chen et al., 2016). UV-induced reduction of LC in healthy skin was mainly caused by the migration of LC rather than apoptosis (Kolgen et al., 2002). Residence of DCs could be maintained by KCs expressed in non-overlapping mode in epidermis by activating integrins αvβ6 and αvβ8 of transforming growth factor (TGF)-β, but UV irradiation could reduce the integrin expression on KCs and reduce the activation of TGF-β which led to the migration of LC (Mohammed and Beura, 2016). The migration of LC from skin could also be modulated by the release of IL-1β, TNF-α and IL-18 (Cumberbatch et al., 1997, Cumberbatch et al., 1999, Tominaga et al., 2000). Moreover, UVB irradiation could inhibit the expression of important molecules on LC surface, including intercellular adhesion molecule (ICAM)-1, B7-2 (CD86) and major histocompatibility complex class II molecules (MHC-II), which resulted in the decrease of LC antigen presenting ability (Lappin et al., 1996). Furthermore, LC migration from the epidermis is compensated by recruitment of DCs and various other leukoyctes (Achachi et al., 2015). On the contrary, research also indicates that UV-induced immune suppression is dependent on Langerin$^+$ cells rather than LC. Wang et al. (2009) observed immunosuppression for both CD8$^+$ T cell expansion and CHS in the absence of LC. They observed that dermal langerin$^+$ cells were essential for this phenomenon. However, the migration model of LC in PLE