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## Pigmentation in Leishmaniasis: Common or Different

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The term Leishmaniasis refers collectively to various clinical symptoms and types (Cutaneous, Mucocutaneous and Visceral) that are caused by obligate intracellular protozoan parasite of genus Leishmania [1]. Parasite is transmitted through the bite of an infected female phlebotomine sand fly. Leishmaniasis currently threatens 350 million people in 88 countries around the world. The WHO estimated that there are 12 million cases of all forms of leishmaniasis worldwide, with over 500,000 new cases of visceral disease occurring each year. Cutaneous Leishmaniasis (CL) is characterized by skin lesions i.e open or closed sores, which typically develop within several weeks or months after exposure. The sores first appear months or years later, in the context of trauma such as skin wounds or surgery in some people. Inital symptoms of Mucocutaneous Leishmaniasis (ML) are similar to that of CL in which extensive disfiguring of the nasal septum, lips, and palate occur later on. Visceral leishmaniasis (VL) also known as kala azar (KA) is characterized by irregular bouts of fever, substantial weight loss, swelling of the spleen and liver. If left untreated, the fatality rate of VL in developing countries can be as high as 100% [2]. Postkala-azar dermal leishmaniasis (PKDL) is a complication of VL which it is characterised by a macular, maculopapular, and nodular rash in a patient who has recovered from VL [3]. PKDL develops months to years after the patient's recovery from VL, with cutaneous lesions. The variant occurs in patients 1-2 years and as long as 20 years after recovery from VL.

Pigmentation is commonly observed in the patient of CL, VL and PKDL. Various pigmentation patterns are seen in the person suffering from Leishmaniasis. Pigmentation is the colouring of individual skin. When a person is healthy, his or her skin will appear normal in colour. In the case of Leishmania infection, the person's skin may change colour, becoming darker (hyperpigmentation) or lighter (hypopigmentation) or sometimes total loss in skin colour (depigmented). Hyperpigmentation in skin is caused by an increase in the melanin pigment (responsible for colour of skin). Melanin pigment is found in a special type of cell named as melanocytes. Hypo pigmentation is decrease in the pigment of skin or lightening of the skin. Hypopigmentation in skin is the result of a reduction in melanin production.

Hyperpigmented depressed scars are seen in case of CL and VL/ KA. After CL, healing may occur spontaneously over 2-12 months and is followed by scarring and changes in pigmentation. KA means the black disease, which refers to hyperpigmentation of the skin and it is frequently found in Indian KA patients. Stimulation of melanocyte can occur, causing characteristic skin hyperpigmentation in KA. Some cases of KA do not show great pigmentation of the skin. This hyperpigmentation is not found in Sudanese KA patients [4]. Hypo pigmentation occurs in the patient suffering or recovered from PKDL. It causes have a loss of pigmentation in the affected area. The polymorphic form showing hypopigmented or erythematous macules with papules and/or nodules is the commonest in PKDL and nodules on the face; macules on the rest of the body with virtually total hypopigmentation sparing axillae and inguinal areas [3]. The lesions may be numerous and persist for decades. But sometimes skin returns to normal, repigmentation may take time and cannot be taken as a parameter for cure. Macules take longer time to repigment after completion of therapy.

Pigmentation could be helpful in morphological determination of disease but the pattern is not common or similar in all form of Leishmaniasis. It varies among different forms of leishmaniasis. Different pigmentation pattern has been seen even is same form of leishmaniasis in person to person. The degree of pigment loss may be considerable, mimicking, and sometimes complete, as in vitiligo. The cause of pigmentation in Leishmaniasis has not been well studied. According to histopathology, the pigment loss has been observed to be proportional to the density of the dermal infiltrate, suggesting that the granuloma somehow interferes with local pigment metabolism but clinical correlation is not clear [5]. It is anticipated that increased production of adrenocorticotropic hormone is responsible for skin hyper pigmentation and reduction of cortisol level may cause disappearance of skin hyper-pigmentation but the relationship between Leishmaniasis and skin pigmentation has not been investigated so far. Therefore future research must be done on this particular topic for the development of better understanding of pigmentation pattern in Leishmaniasis.

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