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PIGMENTATION

STUDY OF THE EXPRESSION OF LC3-I, LC3-II & BECLIN 1 IN VITILIGO AND HEALTHY CONTROLS

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Introduction: Autophagy is an evolutionarily conserved, highly regulated lysosomal degradative pathway for cellular components, which is essential for cellular maintenance and cell viability. Autophagy functions to eliminate unnecessary macromolecules, damaged organelles, and intracellular pathogens through the fusion of the lysosome with a double-membrane bound autophagosome, which can also sequester cargo. It was found that dysregulated autophagy increased melanocytic sensitivity to H2O2 induced oxidative stress in vitiligo. The exact mechanism of impairment of autophagy in vitiligo has not yet been determined.

Objective: This study's objective is to assess the possible role of autophagy in vitiligo pathogenesis through measuring LC3-I, LC3-II & Beclin 1 in vitiligo.

Materials and Methods: Twenty patients with non-segmental vitiligo as well as 20 age and sex matched healthy volunteers served as controls were recruited from the dermatology outpatient clinic, Kasr Al Ainy hospital, Cairo University. Skin biopsies were taken from patients and controls to be assessed for LC3-I, LC3-II & Beclin 1 expression by western blot analysis. Malondialdehyde DNA (MDA) (oxidative marker) and superoxide dismutase (SOD) (antioxidant marker) will be detected and measured by colorimetry method in tissue homogenate.

Results: The levels of LC3-I, LC3-II, Beclin 1 and SOD were statistically significantly lower in lesional skin than non-lesional skin of patients (P<0.001). On the other hand, the level of MDA was statistically significantly higher in lesional skin than non-lesional skin of patients (P<0.001). The levels of LC3-I, LC3-II, Beclin 1 and SOD were statistically significantly lower in lesional skin of patients than controls (P<0.001). On the other hand, the level of MDA was statistically significantly higher in lesional skin of patients than controls (P<0.001). On the other hand, the level of MDA was statistically significantly higher in lesional skin of patients than controls (P<0.001).

Conclusions: This study revealed lower LC3-I, LC3-II & Beclin 1 tissue levels in vitiliginous











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skin than in normal skin suggesting an imminent role of autophagy in the pathogenesis of vitiligo.



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