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Serum TWEAK: A cutoff between segmental and nonsegmental vitiligo

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Abstract

Background: TWEAK/Fn14 is expressed in many tissues including the skin, playing an important role in many inflammatory, autoimmune, and neoplastic cutaneous disorders.

Aims: To assess the serum levels of tumor necrosis factor-like weak inducer of apoptosis (TWEAK) in vitiligo patients.

Methods: This case-control study included 100 subjects (50 vitiligo patients and 50 control subjects) recruited from Dermatology Outpatient Clinic, Benha University. All patients were subjected to complete cutaneous examination, to evaluate the clinical type, distribution and severity of vitiligo using the Vitiligo Area Scoring Index (VASI).

Results: TWEAK serum levels were significantly higher in patients than in the control subjects (644.76 ± 688.93 vs 282.75 ± 125.67, respectively). Serum levels were significantly elevated in segmental versus nonsegmental vitiligo. Receiver operating characteristic (ROC) analysis revealed that TWEAK shows 80% sensitivity and 56.67% specificity in diagnosing vitiligo and 100% sensitivity and 80.09% specificity in differentiating segmental from nonsegmental vitiligo.

Conclusion: TWEAK may play a role in vitiligo pathogenesis. It may be used in the differentiation between segmental and nonsegmental vitiligo and represent a promising therapeutic target in vitiligo.

KEYWORDS

segmental, TWEAK, Vitiligo

1 | INTRODUCTION

Vitiligo is a common disorder of pigmentation caused by acquired melanocyte destruction. Multiple theories have been proposed to outline the pathogenesis of this depigmentation; however, the exact pathogenesis is not yet clarified. The most accepted theory is considering vitiligo as an autoimmune disorder affecting genetically predisposed individuals.[1] Among the involved cytokines in this complex disorder, interferon (IFN)-$^{\gamma}$ plays an essential role in recruiting the $CD8^{+}$ T cells, which attack self-melanocytes.[2] Other cytokines that participate in the pathogenesis of vitiligo, with less understood role, include TNF-α, IL-6, and IL-17.[3]

Tumor necrosis factor-like weak inducer of apoptosis (TWEAK) is a multifunctional cytokine, which belongs to the tumor necrosis factor receptor family ligands.[4] At first, it was considered to be an apoptosis inducer only, and then, its role in regulating many vital processes such as angiogenesis, inflammation, and cell proliferation has been discovered.[5] TWEAK exerts its role via binding to its receptor, fibroblast growth factor-inducible 14 (Fn14). This binding triggers a cascade of intracellular events ending in activation of nuclear factor-kappa B (NF-κB) with subsequent expression of multiple inflammatory molecules and enhancing the inflammatory effects of other proinflammatory cytokines, for example, TNF-α, interleukin (IL)-1, IL-6, and interferon-γ.[6]

