

A Case of Recalcitrant Psoriasis Treated with Secukinumab

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ABSTRACT

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Psoriasis is a chronic, inflammatory papulosquamous disorder characterized by erythematous, scaly plaques. Treatment for recalcitrant cases can be challenging. This case report discusses the successful management of a 55 year old male with recalcitrant psoriasis, treated with Secukinumab, an IL-17A monoclonal antibody. After failing multiple therapies, including methotrexate and cyclosporine, the patient showed significant improvement following Secukinumab administration, emphasizing the effectiveness of this biologic agent in managing moderate to severe psoriasis.

Keywords: Recalcitrant Psoriasis, Secukinumab

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CASE DESCRIPTION

55 years old male known case of psoriasis since 18 years, hypertension and type 2 diabetes mellitus since 15 years and dyslipidemia since 5 years presented with asymptomatic scaly lesions over the trunk, buttocks, bilateral upper limbs and lower limbs. He had history of exacerbation of lesions since 2 months following steroid injection. He had taken multiple treatments in the past including methotrexate and cyclosporine. Systemic examination was within normal limits. On cutaneous examination there were multiple erythematous and hyperpigmented scaly plaques present over the trunk, buttocks, bilateral upperlimbs and lower limbs. Scaly plaques were present over scalp. Longitudinal ridges and trachyonychia were present on all finger nails and toe nails. Auspitz sign was positive. A clinical diagnosis of psoriasis was made.

INVESTIGATIONS AND TREATMENT

Skin biopsy was done and histopathological examination showed focal parakeratosis, acanthosis, regular elongation of rete ridges, focal hypogranulosis, focal supra papillary thinning, neutrophils were noted in the mounds of parakeratosis. Papillary dermis showed mild

edema, numerous congested blood vessels and mild perivascular lymphohistiocytic infiltrate. Deep dermis and dermal appendages were uninvolved. Histopathological findings confirmed the diagnosis of psoriasis.

Complete blood count, Erythrocyte sedimentation rate, liver function test, renal function test, urine routine examination, HBsAg antigen, Anti HCV IgM, ELISA, chest Xray and Quantiferon TB Gold test were done as pretreatment work up for secukinumab injection and all were within normal limits. Patient was administered inj. Secukinumab 300mg (150mg on each arm) subcutaneously at 0,1,2,3 and 4 weeks interval followed by maintenance dose of 300mg subcutaneous injection at 4 weeks interval. Patient showed drastic change after each dose. After 6 doses of secukinumab injection all the lesions healed and the nails showed significant improvement. Patient was monitored with complete blood count monthly for 3 months and 3 monthly thereafter.

DISCUSSION

Psoriasis is a chronic inflammatory and proliferative condition of the skin associated with systemic manifestations. The most characteristic lesions consist of

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Figure 1. Before treatment



Figure 2. After 1st dose of secukinumab

bilaterally symmetrical well defined erythematous scaly plaques present particularly over the extensor surfaces and scalp though other morphological variants are also described. The disease is believed to be multifactorial with both genetic and environmental factors playing a role in its development. Psoriasis is a hyperproliferative disorder, the proliferation is driven by a complex cascade of inflammatory mediators. T-cells and cytokines play pivotal roles in the pathophysiology of psoriasis. The pathogenesis of psoriasis is influenced

by the interleukin (IL)-23/IL-17 pathway, which is involved in the inflammatory processes underlying this chronic skin condition. IL-23 is a cytokine that helps the production of IL-17.

There are six cytokines in the IL-17 cytokine family. IL-17 A-F and five receptors IL-17 A-E. IL-17A, -C and -F mRNA levels are elevated in psoriasis plaques and increased number of IL-17A-positive cells are found in involved psoriasis skin compared to controls.¹ The cellular source of IL-17 in psoriasis includes lympho-



Figure 3. After 3rd dose of secukinumab



Figure 4. After 5th dose of secukinumab

cytes but also innate immune cells such as neutrophils, mast cells, and innate lymphoid cells. The mechanism of action of several antipsoriatic treatments currently in use and under development is aimed at blocking IL-17 and its mediated downstream immunological cascade.

Secukinumab is a novel biologic agent specifically targeting interleukin-17A. It is a fully human monoclonal antibody. Many clinical trials have demonstrated its efficacy in managing plaque psoriasis and psoriatic

arthritis.² FDA approved indications include moderate to severe psoriasis, hypertrophic palmoplantar psoriasis, generalised pustular psoriasis and psoriatic arthritis. Secukinumab is available as lyophilized powder of 150mg in a vial that require storage at 2-8 degree celsius. Before administration vial has to be kept aside for 20-30 minutes until it reaches room temperature. Reconstitution is performed with 1 ml sterile distilled water followed by gentle stirring to dissolve the powder and kept aside for 10minutes. Adult dosage is 300mg

subcutaneous injection at 0,1,2,3 and 4 weeks followed by maintenance dose of 300mg subcutaneous injection every four weeks.³ Each 300mg dose is given as two subcutaneous injections of 150mg. Presence of active infections, latent or active tuberculosis, hepatitis B&C, HIV are contraindications for secukinumab. Adverse effects include nasopharyngitis, upper respiratory tract infection, neutropenia, candidiasis and flare of inflammatory bowel disease. No significant drug interactions have been reported. Live vaccines should not be given concurrently. Patient has to be monitored by doing complete blood count monthly for 3 months and 3 monthly thereafter. Annual screening for hepatitis and tuberculosis has to be done.

CONCLUSION

Secukinumab provides a promising treatment option for patients with severe, recalcitrant psoriasis. This case demonstrates that Secukinumab can achieve substantial improvement, even in patients who have failed conventional therapies. Its ability to target the IL-17 pathway represents a significant advancement in the management of this chronic inflammatory skin condition.

END NOTE

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