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Case Report

Case report: Effects of TNF-blockers in lowering Crp, Esr and D-dimer in Psoriatic arthritis

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psoriatic arthritis, TNF-blocker, Etanercept, D-dimer, CRP, ESR, biologic therapy, systemic inflammation

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ABSTRACT

Psoriatic arthritis (PsA) is a chronic inflammatory disease affecting both the joints and skin, often associated with elevated systemic inflammatory markers such as C-reactive protein (CRP), erythrocyte sedimentation rate (ESR), and D-dimer. TNF-α inhibitors have become pivotal in the management of moderate to severe PsA. We report the case of a 32-year-old male with a 9-year history of psoriasis who developed PsA following a COVID-19 infection. The patient presented with severe bilateral knee pain, inflammatory arthritis, and markedly elevated inflammatory markers: CRP 139 ng/L, ESR 86 mm/hr, and D-dimer 3303 ng/ml. Treatment with Etanercept over a 24-month period led to significant clinical improvement and normalization of inflammatory markers (CRP 5 ng/L, ESR 16 mm/hr, D-dimer 110 ng/ml). Pain scores decreased from 10/10 to 1/10, and the patient's psoriatic skin lesions resolved. This case highlights the efficacy of Etanercept in achieving both clinical and biochemical remission in PsA and suggests a potential role for D-dimer as a marker of systemic inflammation and thrombotic risk in PsA patients.

1. Introduction

Psoriatic arthritis (PsA) is a chronic inflammatory condition that not only affects the joints but is also often accompanied by systemic features, leading to significant impairment in a patient's quality of life (1). The disease is characterized by both musculoskeletal inflammation and elevated levels of inflammatory biomarkers such as C-reactive protein (CRP), erythrocyte sedimentation rate (ESR), and, in certain cases, D-dimer—reflecting underlying inflammation and thrombotic risk (2). Managing these inflammatory markers effectively is crucial for controlling disease activity and preventing long-term complications (3).

Tumor necrosis factor (TNF) blockers have emerged as a cornerstone in the treatment of moderate to severe PsA. These biologic agents target key inflammatory pathways and are designed to reduce inflammation, alleviate symptoms, and halt disease progression (3, 4). Beyond their effects on joint and skin manifestations, TNF-blockers have demonstrated the potential to reduce systemic inflammation, as evidenced by declines in laboratory markers such as CRP, ESR, and D-dimer (5).

This case report illustrates the impact of TNF-blocker therapy in a patient with PsA, focusing on its effectiveness in lowering CRP, ESR, and D-dimer levels, while also contributing to a marked improvement in the patient's quality of life. It highlights the broader systemic benefits of targeted biologic therapy and provides insight into the role of TNF-blockers in achieving both clinical and biochemical remission.

2. Case report

The case studied is a 32-year-old patient, with the initials E.D., who presented to our Clinic with bilateral knee pain, inflammatory joint pain, generalized psoriatic squamous plaque-like cutaneous elements, accompanied by pruritus, and a temperature of 38.5 degrees Celsius for two days. He reports that these complaints have started more than 1 year ago (2 months after the patient was diagnosed with Covid-19).

The pain level was assessed 10/10 (The pain level was assessed by the Rheumatologist on a scale of 0-1, where 0 is no pain at all and 10 is very severe pain that affects the quality of life).

The patient has been diagnosed with Psoriasis for 9 years.

The patient had previously been treated with methylprednisolone 40 mg, but no significant improvement in the general condition was observed. On clinical examination, signs of synovitis were evident in the radiocarpal (RC), metacarpophalangeal (MCP), and talocrural (TC) joints, all of which were infiltrated and tender to digital pressure. The bilateral knee joints (genua) were also tender and edematous, with evidence of hypertrophic synovium on palpation. The coxofemoral (CF) joints demonstrated restricted range of motion during maneuvering. Additionally, spinal tenderness was noted during flexion movements.

The patient was admitted to the Rheumatology Clinic for further evaluation and initiation of biologic therapy. Etanercept, a tumor necrosis factor-alpha (TNF-α) inhibitor, was selected as the biological agent for treatment.

Etanercept treatment on dosagee Etanercept 50mg/ml lasted a total of 4 sessions/month (one session/ week) over a period of 24 months (accorded to EULAR, 2019, EULAR recommendations for the management of psoriatic arthritis with pharmacological therapies).

The patient was taken for assessment in periods every 6 months.

After treatment with Etanercept 50mg/ml, an improvement in pain was observed, synovial inflammation was reduced, and psoriatic squamous plaques were reduced. The joints appeared without inflammation, were not edematous, and were not tender to finger pressure and to range of motion.

The pain level after treatment was rated 1/10. (Pain was assessed by the rheumatologist using a scale from 0 to 10, where 0 indicates no pain and 10 indicates severe pain that significantly affects quality of life.)

The examinations performed and included in this case report are summarized in Table I:

Table I. *Laboratory values before and after treatment.*

Laboratory test	Values before treatment	Values after treatment	Normal values
CRP	139	5	0-5ng/l
ERS	86	16	0-20mm/hr
D-Dimer	3303	110ng/ml	0-500ng/ml
ALP	376	267	0-270U/L

3. Discussion

This case highlights the effectiveness of TNF-α inhibitor therapy in the management of severe psoriatic arthritis (PsA), with significant clinical improvement and normalization of inflammatory markers. Of particular interest was the marked reduction of D-dimer levels, which dropped from 3303 ng/mL to 110 ng/mL following treatment.

While C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR) are routinely used as indicators of disease activity in PsA, the elevation of D-dimer is notable. D-dimer, commonly used in the diagnosis of venous thromboembolism (VTE), has also emerged as a non-specific biomarker of systemic inflammation (6). In this case, thromboembolic disease was clinically excluded, and the elevation was attributed to systemic inflammation associated with active PsA.

There is growing evidence linking chronic inflammatory states such as PsA to a prothrombotic profile, suggesting that D-dimer may serve as a surrogate marker for cardiovascular and thrombotic risk in these patients (7, 8). Elevated D-dimer levels have been documented in patients with PsA, correlating with disease activity and systemic inflammatory load (9). The normalization of D-dimer after anti-TNF therapy in this patient reinforces the hypothesis that biologic treatment may reduce thromboinflammatory risk.

The temporal association with COVID-19 infection—which occurred two months prior to the onset of PsA symptoms—raises the possibility of a viral trigger in the pathogenesis of autoimmune disease. Several mechanisms, including molecular mimicry, bystander activation, and immune dysregulation, have been proposed as ways through which SARS-CoV-2 may precipitate autoimmunity in genetically predisposed individuals (10, 11). Emerging case reports have also described new-onset PsA and psoriasis exacerbations following COVID-19 infection (12, 13), supporting the need for vigilance in post-viral autoimmune manifestations.

This case illustrates the importance of comprehensive disease monitoring in PsA, not only evaluating joint and skin symptoms but also systemic markers like D-dimer, which may provide insights into inflammatory activity and cardiovascular risk.

4. Conclusions

This case underscores the effectiveness of etanercept, a TNF-α inhibitor, in inducing clinical remission in a patient with severe PsA. Treatment resulted in:

- Complete resolution of joint inflammation and skin lesions
- Substantial reduction in pain
- Normalization of inflammatory biomarkers including CRP, ESR, and D-dimer

The sharp decline in D-dimer levels may reflect attenuation of systemic inflammation and thrombotic risk, reinforcing the thromboinflammatory connection in PsA. Moreover, the temporal relationship with COVID-19 infection raises considerations about viral triggers in autoimmune pathogenesis.

This case advocates for the early initiation of biologic therapy in PsA and suggests potential value in monitoring D-dimer as a supplementary biomarker for both disease activity and systemic risk. Further research is needed to validate the prothrombotic role of D-dimer in PsA and the post-viral autoimmu-

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DISCLOSURE

All authors report no conflict of interest.

DATA AVAILABILITY STATEMENT

Data openly available in a public repository that issues datasets with DOIs.

ETHICS STATEMENT

The authors obtained written consent from patients for their photographs and medical information to be published in print and online and with the understan-ding that this information may be publicly available. Patient consent forms were not provided to the journal but are retained by the authors.

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