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CASE REPORT

Hepatotoxicity Induced by Adalimumab in Chronic Plaque Psoriasis Patient: A Case Report

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Introduction: Adalimumab is a TNF-α antagonist used to treat psoriasis and rheumatologic conditions; it can rarely cause hepatotoxicity manifesting as elevated liver enzymes.

Case Presentation: Here, we present a case of a 24-year-old female with chronic plaque psoriasis with no history of liver disease or any risk factor for liver disease who developed a drug-induced liver injury upon treatment initiation with Adalimumab. This case report highlights the importance of monitoring liver function tests in patients receiving Adalimumab, early recognition, and prompt medication dosage adjustment or discontinuation. Early diagnosis and treatment of chronic plaque psoriasis is critical for improving prognosis, especially in the case of increased risk factors of liver disease due to the administration of Adalimumab.

Conclusion: Our case presented a patient with no history of liver disease; however, she developed a drug-induced liver injury. Therefore, this case report is the first to emphasize the need for early recognition of LFTs upon Adalimumab administration, and prompt medication dosage adjustment or discontinuation is essential in patients receiving Adalimumab.

Keywords: Adalimumab, psoriasis, liver enzyme elevation, drug-induced liver injury, case report

Introduction

Adalimumab is a fully human recombinant immunoglobulin G (IgG) monoclonal antibody with specificity for tumor necrosis factor-alpha (TNF-α). Adalimumab binds to soluble and membrane-bound TNF-α, leading to a blockade of its activity, resulting in apoptosis of cells with membrane-bound TNF.[1] The most common adverse reaction to Adalimumab is injection site reaction, occurring in more than 10% of treated patients; other adverse reactions include the reactivation of tuberculosis, which is the most serious.[2] Drug-induced systemic lupus erythematosus, lymphoma, and demyelinating illness are other serious adverse effects, but these occur infrequently.[3] Hepatotoxicity also affects less than 5% of patients on treatment, which commonly manifests as an asymptomatic increase in liver enzymes.[4]

There are a few case reports on hepatotoxicity and Adalimumab; however, these are primarily on patients diagnosed with other autoimmune diseases, such as rheumatoid arthritis and inflammatory bowel disease. Literature on hepatotoxicity associated with Adalimumab among patients with psoriasis is scarce.[5] However, there is a case report of a patient with psoriasis who developed liver enzyme elevation. The liver failure prognosis was not attributed to Adalimumab because it was discounted, but the liver enzymes were elevated, possibly due to methotrexate.[6] Currently, our case is the first to present a rare occurrence of liver enzyme elevation in a patient with psoriasis treated with Adalimumab. Our patient was a healthy young female who had never been treated previously with any medications aside from Adalimumab and topical treatments.

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Case Presentation

A 24-year-old female with no known co-morbidities and no history of taking any medications was diagnosed with chronic plaque psoriasis in 2017. She first presented with erythematous plaques with silvery fine scaling over the knees as shown in Figure 1, which progressed to involve the elbows as shown in Figure 2, other areas of the lower limbs, and scalp. She has no family history of a similar disease.

Prior to receiving therapy, the patient underwent viral hepatitis, viral load tests, and serologic testing for the hepatitis B and C viruses, all of which came out negative. She had never used drugs or alcohol, received blood transfusions, or had any other history of viral hepatitis risk factors. Initial treatment was done with calcipotriene-betamethasone 4% topical ointment twice a day and tacrolimus 0.1% topical twice daily without any noticeable improvement in the lesions. Adalimumab was subcutaneously administered in December 2020, and the dosage was 40 milligrams every 2 weeks. Clobetasol and calcipotriene solutions were simultaneously administered for the scalp lesions. At 5 months post-treatment, her symptoms resolved. However, her liver function tests (LFTs) revealed an alanine aminotransferase (ALT) level of 152 µ/L (2.3 times elevated the upper limit of normal [ULN]), aspartate aminotransferase (AST) level

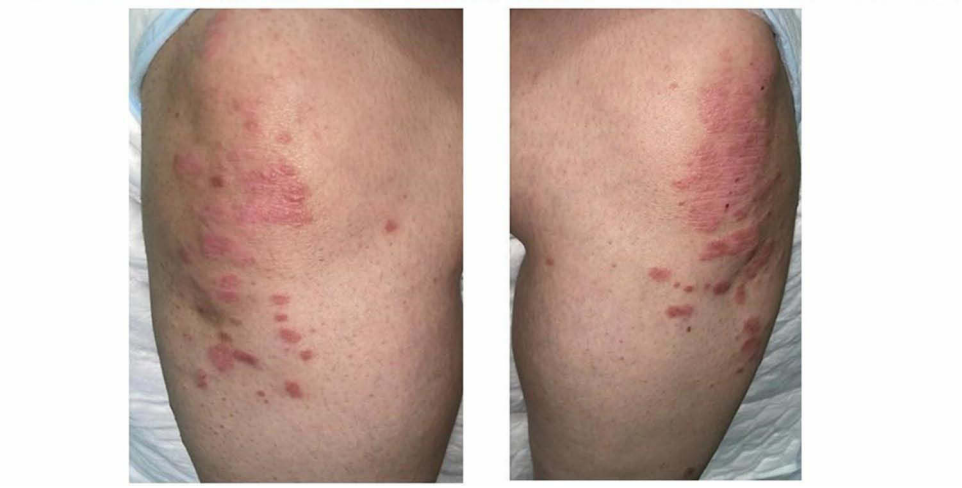

Figure 1 Erythematous plaques with silvery fine scaling over the knees consistent with chronic plaque psoriasis.

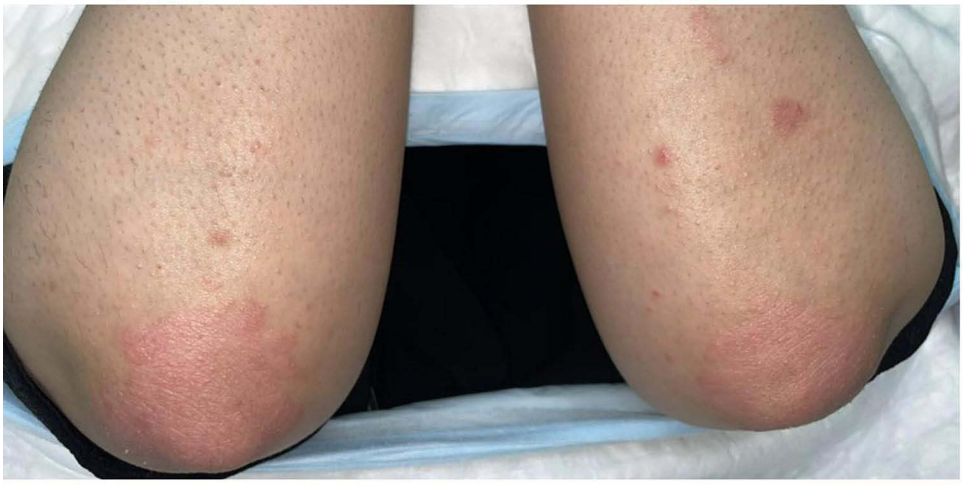

Figure 2 Progression of the erythematous plaques to involve the bilateral elbows.

of 79 μ/L (2.5 times elevated the ULN) and total bilirubin level of 19.83 μmol/L (1.2 times elevated the ULN). Hepatology then assessed her to have developed a drug-induced liver injury.

Repeat viral hepatitis screening (including HBV, HCV, and HAV serologies and PCR when relevant) was required to rule out infectious reasons after liver enzyme increased in April 2021, but all results came back negative. Additionally unremarkable were signs of autoimmune liver disease and abdominal ultrasonography, which supported the diagnosis of drug-induced liver impairment. Following the test results, we discontinued Adalimumab immediately. The patient was then closely monitored with serial liver function tests over the following 4–6 weeks of Adalimumab discontinuation; her liver enzymes returned to baseline levels (LFTs returned to normal: ALT of 30 μ/L, AST of 20 μ/L, and total bilirubin of 10.45 μmol/L) without the need for hospitalization or further treatment. Upon involving the patient in the management plan, we decided to start her on subcutaneous Risankizumab at a dosage of 150mg. At a 6-month follow-up, there was no elevation in the liver enzymes.

Discussion

In the literature, Adalimumab has been linked to a low incidence of increased aminotransferases during treatment. However, such incidences are transient, mild, asymptomatic, and rarely necessitate dosage modification. The rise in levels resolved in 82% of patients upon follow-up.[5] Liver enzyme abnormality was characterized as a twofold or more significant rise above the ULN in two or more of the following components: AST, ALT, total bilirubin, lactate dehydrogenase (LDH), or alkaline phosphatase (ALP).[7] Our patient exhibited significantly increased AST and ALT levels. Although liver injury associated with TNF-α inhibitors such as Adalimumab is generally idiosyncratic, it presents a clinically relevant risk due to its potential cellular damage and variability.[8] The precise mechanism remains unclear; however, the literature sheds some light on its possible involvement with immune-mediated harm or direct hepatocellular toxicity.[9] In the literature, an example of the damage was shown in a case report that described a patient with rheumatoid arthritis treated with Adalimumab. Despite the patient having hepatitis C, their LFTs remained stable before commencing treatment. The patient's gamma-glutamyl transpeptidase (GGT) level surged to 23 times the ULN three months post adalimumab administration, while the ALP level rose to 2.5 times the ULN. Interestingly, the patient did not display elevated AST and ALT levels; Adalimumab was discontinued six months after treatment.[10] Similarly, in an analysis of thirty-four cases, hepatotoxicity linked to TNF-α antagonists varied from asymptomatic enzyme increases to severe liver damage, with most instances resolving upon discontinuation of the drug.[11,12]

The fundamental risk factors for liver disease, particularly in metabolic syndrome and fatty liver diseases in patients with psoriasis, enhance the chances of hepatotoxicity at the initial stages.[13] Therefore, it is recommended that liver function tests be performed before the commencement of therapy and periodically during therapy. This predisposing risk is why routine monitoring must be employed to enable early detection. According to the recent literature, Madani and Almuhaideb described a case study of a patient with Down syndrome and psoriasis receiving Adalimumab whose liver enzymes flare up due to hepatitis B virus infection.[14] Björnsson et al stated concern about drug-induced liver injury attributable to TNF-α antagonists.[8] However, the stated overall risk remains relatively low as long as other predisposing factors are considered. In addition, Hagel et al[9] noted that such injuries are highly unique and thus require monitoring to detect any subsequent similar injuries.

More specifically, the precise cause of liver injury related to Adalimumab is still unknown and appears to be quite random.[9,12] Further research is, however, required to identify other factors that may predispose a patient to this side effect. In addition, more extensive research and surveys on a more significant population sample are needed to establish the injury patterns of hepatotoxicity. Our current case focuses on the importance of early identification and risk mitigation of drug-induced liver injury. LFTs should be performed before starting treatment and periodically throughout treatment.[11] In this case, and indeed in managing most ADRs, withdrawal of the offending agent remains the strength of treatment, which we are the first to present. Active comparators like Risankizumab, which were proven to have a better hepatic safety profile in this patient, give hope for patients developing adverse reactions to Adalimumab.

In comparison with previous studies on the subject, our case report is the first to highlight the importance of routine testing of LFTs in patients receiving Adalimumab, early recognition, and prompt medication dosage adjustment or discontinuation (see Table 1). While Adalimumab has shown a favorable safety profile over 15 years of use,[15] clinicians must remain vigilant for rare adverse events, such as hepatotoxicity, particularly in patients with additional risk factors.

Table 1 Comparison of Current Cases with Previously Published Cases of Adalimumab-Induced Hepatotoxicity

Case	Patient Demographics	Underlying Conditions	Adalimumab Regimen	Hepatotoxicity Manifestation	Outcome
Current Case	24-year-old female	Chronic plaque psoriasis	40 mg every 2 weeks	ALT 152 μ/L (2.3x ULN), AST 79 μ/L (2.5x ULN), Total bilirubin 19.83 μmol/L (1.2x ULN)	LFTs normalized after discontinuation; switched to risankizumab without recurrence.
Hagel et al[9]	44-year-old female	Psoriasis, no prior liver disease	Initial dose of 80 mg, followed by 40 mg every 2 weeks	ALT 202.2 μ/L (5.9x ULN), AST 117.4 μ/L (3.8x ULN), hepatomegaly, jaundice	Sub-acute liver failure; LFTs improved after drug discontinuation
French et al[10]	Rheumatoid arthritis patient, Hepatitis C	Rheumatoid arthritis	Not specified	GGT 23x ULN, ALP 2.5x ULN (AST/ALT not elevated)	Discontinued Adalimumab; LFTs stabilized
Frider et al[12]	35-year-old male patient with no underlying liver conditions	Autoimmune condition	Not specified	Cholestatic liver injury	Liver function tests improved after discontinuation
Madani and Almuhaideb[14]	Down syndrome patient with psoriasis, Hepatitis B	Psoriasis and Hepatitis B	Not specified	Elevated liver enzymes during flare-up	Managed with concurrent hepatitis B treatment
Ghabril et al[11]	Various patients from a 34-case study	Autoimmune conditions	TNF-α inhibitors (adalimumab)	Varied: asymptomatic enzyme increases to severe liver damage	Most cases are resolved upon discontinuation of treatment

Conclusions

Early diagnosis and treatment of chronic plaque psoriasis is critical for improving prognosis, especially in the case of increased risk factors of liver disease due to the administration of Adalimumab. Our case presented a patient with no history of liver disease; however, she developed a drug-induced liver injury. Although the mechanism of liver injury could be idiosyncratic, liver injury is an uncommon, underreported adverse effect of this drug. Therefore, this case report emphasizes the need for early recognition of LFTs upon Adalimumab administration, and prompt medication dosage adjustment or discontinuation is essential in patients receiving Adalimumab.

Data Sharing Statement

All the data for this case report will be made available upon reasonable request. Further inquiries can be directed to the corresponding author.

Consent for Publication

All authors gave their consent for publication, and written informed consent was obtained from the patient to publish the details of their medical care and any accompanying images.

Study Approval Statement

Ethical approval is not required for case reports at our institution while not disclosing patient identity.

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Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors have no conflicts of interest to declare in this work.

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