Dermatology Practical & Conceptual

Hepatotoxicity Associated with Adalimumab in Hidradenitis Suppurativa: A Report of Two Cases of Drug-Induced Liver Injury

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Introduction

Adalimumab, a TNF-α inhibitor, is widely used in hidradenitis suppurativa (HS). While generally considered safe, hepatotoxicity has been documented as a potential adverse effect. We present two cases of drug-induced liver injury (DILI) associated with adalimumab in patients with HS, highlighting the variability in onset and severity of hepatic damage.

Case Presentation

The first case involves a 47-year-old female diagnosed with HS Hurley III in 2019, with a medical history of endometriosis managed with oral contraceptives, no known drug allergies, no hypertension, diabetes, or dyslipidemia, and no personal or family history of liver disease. She was initially treated with rifampicin and clindamycin for two months while undergoing pre-biological screening. Subsequently, she started adalimumab 80 mg every two weeks. In 2021, her therapy was switched to a biosimilar at the same dosage. In July 2024, after more than two years of treatment, she developed right upper quadrant discomfort with transaminases elevated fivefold. Liver elastography, previously normal, revealed severe steatosis; antinuclear and liver-specific antibodies were negative. Adalimumab was discontinued, and she received a tapering course of oral corticosteroids, with progressive improvement in liver enzymes.

The second case describes a 56-year-old female, smoker (eight cigarettes/day), with a history of anxiety managed intermittenly with lorazepam, no drug allergies or chronic comorbidities, and no personal or family history of liver disease. Diagnosed with HS Hurley III in February 2022, she initially received clindamycin for one month during her pre-biological screening. Then, adalimumab was initiated in April 2022

with an induction dose of 160 mg. Within just three days of this first and only dose, she developed severe epigastric pain. Laboratory tests revealed elevated ALT (1595 U/L), AST (587 U/L), and GGT (556 U/L). Liver elastography was normal; antinuclear and liver-related antibodies were negative. Adalimumab was immediately discontinued, and liver enzymes gradually normalized during follow-up. The final diagnosis was DILI, attributed to recent adalimumab initiation.

Hepatotoxicity occurred at different stages- delayed in the first case and acute in the second- highlighting the unpredictable latency of DILI. As noted by Frider et al. [1], adalimumab-related liver injury is often idiosyncratic, likely driven by an abnormal immune response rather than a direct dose-dependent toxicity. Elevated transaminases were the main laboratory finding in both cases, reflecting the degree of hepatocellular injury. In the second case, the rapid onset with transaminase elevation within days of starting treatment suggests a more severe inflammatory response, indicating acute liver damage.

The underlying mechanism of adalimumab-induced hepatotoxicity is not entirely understood. It may involve aberrant immune activation due to TNF-α blockade, potentially resulting in eosinophilic infiltration and hepatic inflammation, as seen in liver biopsies from affected patients [2]. The resolution of liver enzyme abnormalities following drug discontinuation, as observed in our patients, supports the idea that prompt cessation of therapy is crucial to preventing severe liver damage [3].

Conclusion

We present two cases of DILI associated with adalimumab in patients with HS, an occurrence not previously described in the literature. While adalimumab remains a cornerstone of HS treatment, these cases underscore the need for routine hepatic monitoring, especially at early stages of therapy. Clinicians should be aware of the possibility of severe hepatotoxicity and act promptly when clinical or biochemical abnormalities appear. Further research is needed to better understand the risk factors and mechanisms of adalimumab-induced liver injury, ensuring safer use of this treatment.

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