Sequential fulminant hepatitis and toxic epidermal necrolysis induced by tislelizumab: A case report

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Abstract. Immune checkpoint inhibitors (ICIs)-monoclonal antibodies that block inhibitory pathways such as programmed cell death protein 1 (PD-1)/PD-1 ligand to restore T-cell anti-tumor activity-are increasingly used in advanced non-small cell lung cancer. These agents are associated with severe multisystem immune-related adverse events (irAEs). The present study reported the first case of sequential grade 4 hepatotoxicity and toxic epidermal necrolysis (TEN) induced by the anti-PD-1 antibody tislelizumab. A 70-year-old female patient with advanced lung squamous cell carcinoma developed acute severe mixed liver injury (alanine aminotransferase, 1,741.6 U/l) followed by extensive TEN shortly after initiation of tislelizumab combined with chemotherapy. The toxicities were refractory to conventional hepatoprotective and antihistamine therapies but responded to high-dose corticosteroids combined with intravenous immunoglobulin (IVIG), leading to full recovery. This case highlights that PD-1 inhibitors can induce rare yet life-threatening overlapping irAEs. Clinicians should maintain heightened vigilance for such severe toxicities, particularly in elderly patients receiving combination immunotherapy, and promptly initiate multidisciplinary management including corticosteroids and IVIG.

Introduction

Lung cancer ranks among the most common malignancies worldwide in terms of both incidence and mortality, with non-small cell lung cancer (NSCLC) accounting for the majority of cases. In recent years, immune checkpoint inhibitors (ICIs), particularly monoclonal antibodies targeting programmed cell death protein 1 (PD-1) and its ligand (PD-L1), have revolutionized the treatment landscape for advanced NSCLC. By blocking the PD-1/PD-L1 signaling pathway, these agents release inhibitory effects on T cells and enhance antitumor immune responses, leading to significant and durable survival benefits for patients (1). Tislelizumab is a humanized IgG4 anti-PD-1 monoclonal antibody. Its Fc segment has been engineered to minimize binding to FcγR on macrophages, thereby reducing antibody-dependent cellular phagocytosis and potentially mitigating effector T-cell exhaustion, which may enhance antitumor activity (2,3). It has demonstrated promising efficacy and a manageable safety profile across various malignancies, including advanced squamous and non-squamous NSCLC. However, like other ICIs, tislelizumab can also induce a spectrum of immune-related adverse events (irAEs) (4). Although management guidelines for irAEs are continually being refined, clinical practice still faces substantial challenges (5). This article reports the case of a 70-year-old female with advanced lung squamous cell carcinoma who developed severe dual irAEs involving the liver and skin following treatment with nab-paclitaxel, carboplatin and tislelizumab. The case aims to provide an in-depth analysis of the clinical features, potential mechanisms and management strategies of this rare dual grade 4 toxicity, hoping to alert and inform clinicians in the recognition and management of similar complex cases.

Case presentation

A 70-year-old non-smoking female patient was diagnosed at Liaohua Hospital (Liaoyang, China) with non-keratinizing squamous cell carcinoma of the right upper lobe with multiple intracranial metastases in November 2023. After completing radiotherapy in December 2023, first-line chemotherapy was initiated in January 2024: Nab-paclitaxel (100 mg on day 1, 200 mg on day 8) plus carboplatin (450 mg on day 1), every 3 weeks. The first cycle was well tolerated with no adverse events. At 21 days after the first-line chemotherapy, the patient started the second cycle of the same regimen with the addition of intravenous tislelizumab 200 mg (Fig. 1); this day was designated as day 0. Baseline laboratory tests before treatment showed normal liver function (Table I) and no skin rash. On day 19, the patient developed a sharp elevation in liver enzymes: Alanine transaminase (ALT), 1,741.6 U/l (normal range: 7-40 U/l); aspartate transaminase (AST), 1,310 U/l (normal range: 13-35 U/l). Despite the initiation of hepatoprotective

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Table I. Laboratory values of the patient over the clinical course.

Laboratory value	Normal range	Day 0	Day 19	Day 24	Day 29	Day 32	Day 39	Day 42	Day 50	Day 57
TBiL, μmol/l	0-21	5.7	27.8	73.4	70.1	61.8	83	30.4	28.7	24.8
DBiL, μmol/l	0-6.84	1.5	15.8	48.5	47.5	42.5	54.9	21.9	18.8	15.2
TBA, μmol/l	0-12	3.3	9.8	30.9	77.6	122	8.2	6	5	9
ALT, U/l	7-40	26.9	1741.6	641.1	204.5	122.9	66.4	53	46	57
AST, U/l	13-35	18	1310	1303	752	337	108	81	38	34
ALP, U/l	50-135	67	133	144	151	130	159	77	68	83
GGT, U/l	7-45	22.5	211.3	316	425.1	365.1	343.5	250	124	97
IL-6, pg/ml	<5.9	-	-	-	4.04	-	-	25.8	-	5.2
CRP, mg/l	0-6	-	-	-	9.89	-	56.2	19.29	18.8	3.7

ALT, alanine transaminase; AST, aspartate transaminase; TBA, total bile acid; ALP, alkaline phosphatase; CRP, C-reactive protein; TBil, total bilirubin; DBil, direct bilirubin; GGT, gamma-glutamyl transferase.

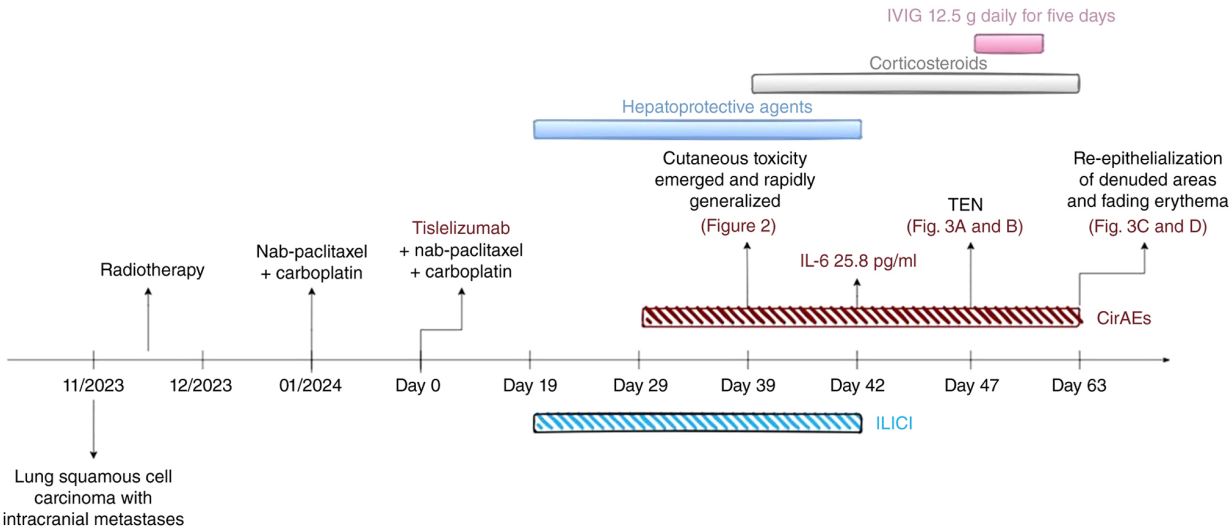

Figure 1. A timeline illustrating the disease course and interventions. ILICI, immune-mediated liver injury caused by immune checkpoint inhibitors; CirAEs, cutaneous immune-related adverse events; TEN, toxic epidermal necrolysis; IL-6, interleukin-6; IVIG, intravenous immunoglobulin.

agents-bicyclol (50 mg three times daily), polyenylphosphatidylcholine (300 mg three times daily), and ursodeoxycholic acid (250 mg three times daily)-the biochemical abnormalities persisted, accompanied by elevated total bile acids, indicating mixed hepatocellular and cholestatic liver injury. On day 29, the patient experienced unexplained low-grade fever (37.8˚C); IL-6 was normal (normal range: <5.9 pg/ml by chemiluminescence method). After spontaneous defervescence, scattered purpuric macules and papules with pruritus appeared on the occipital region, face and abdomen. Oral cetirizine was ineffective and the rash gradually spread to the trunk, limbs, neck and scalp, with worsening pruritus. On day 39, yellowish vesicles developed, which ruptured upon scratching (Fig. 2). Intravenous methylprednisolone (120 mg; 2 mg/kg/day) was given for 3 days, without improvement. On day 42, the patient was transferred to the dermatology department, where the Severity-of-Illness Score for Toxic Epidermal Necrolysis (SCORTEN) score was determined to be 4 (age ≥40 years, malignancy, heart rate ≥120 bpm, involved body surface area ≥10% on day 1), indicating a high risk of death from toxic epidermal necrolysis (TEN). At that time, liver function had returned to near normal (ALT, 53 U/l; AST, 81 U/l), but IL-6 was markedly elevated to 25.8 pg/ml. Comprehensive treatment was initiated: Methylprednisolone was adjusted to 60 mg (1 mg/kg)/day, combined with antihistamines, gastric mucosal protectants, topical emollients and antiseptic mouthwash. Because the TEN-like skin lesions continued to progress (widespread epidermal detachment involving ~50% of body surface area, positive Nikolsky sign) (Fig. 3A and B), intravenous immunoglobulin (IVIG) was added on day 47 at a dose of 12.5 g/day (0.25 g/kg/day) for 5 days. After treatment, IL-6 gradually decreased, reaching 5.2 pg/ml on day 57, and liver function remained normal. The glucocorticoid was tapered to 40 mg/day orally. On day 63, the skin lesions had markedly improved, with complete re-epithelialization of the eroded areas and resolution of erythema (Fig. 3C and D). The patient was discharged in a stable condition. After discharge, the patient was followed up monthly. No further laboratory or

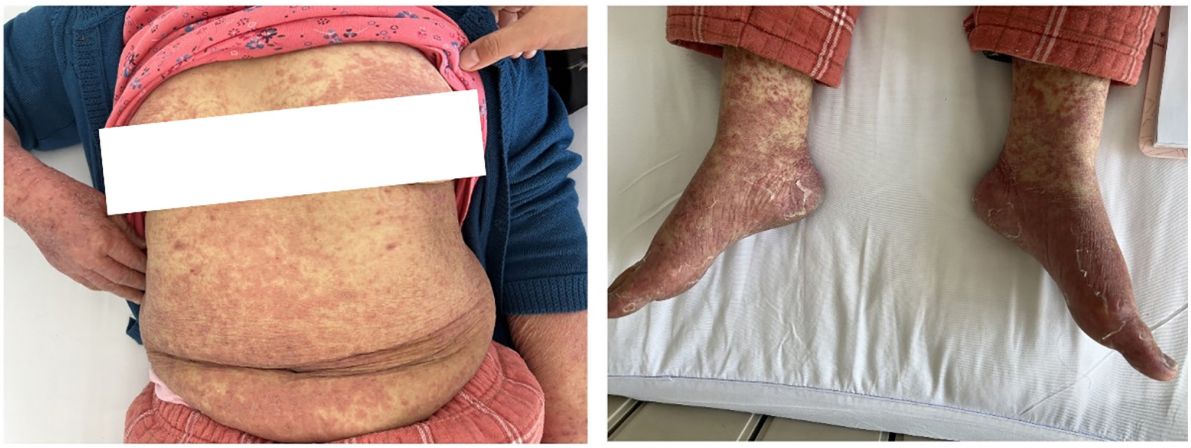

Figure 2. Generalized, extensive dense erythema on the trunk (chest and abdomen), both lower limbs, and both feet. Large areas of epidermal detachment with scaling and desquamation were present on both feet, the lower calves, and the ankles. The trunk showed no large areas of epidermal loss, with predominantly diffuse maculopapular lesions. The total area of epidermal detachment was less than 5% of body surface area, mainly confined to the distal lower limbs.

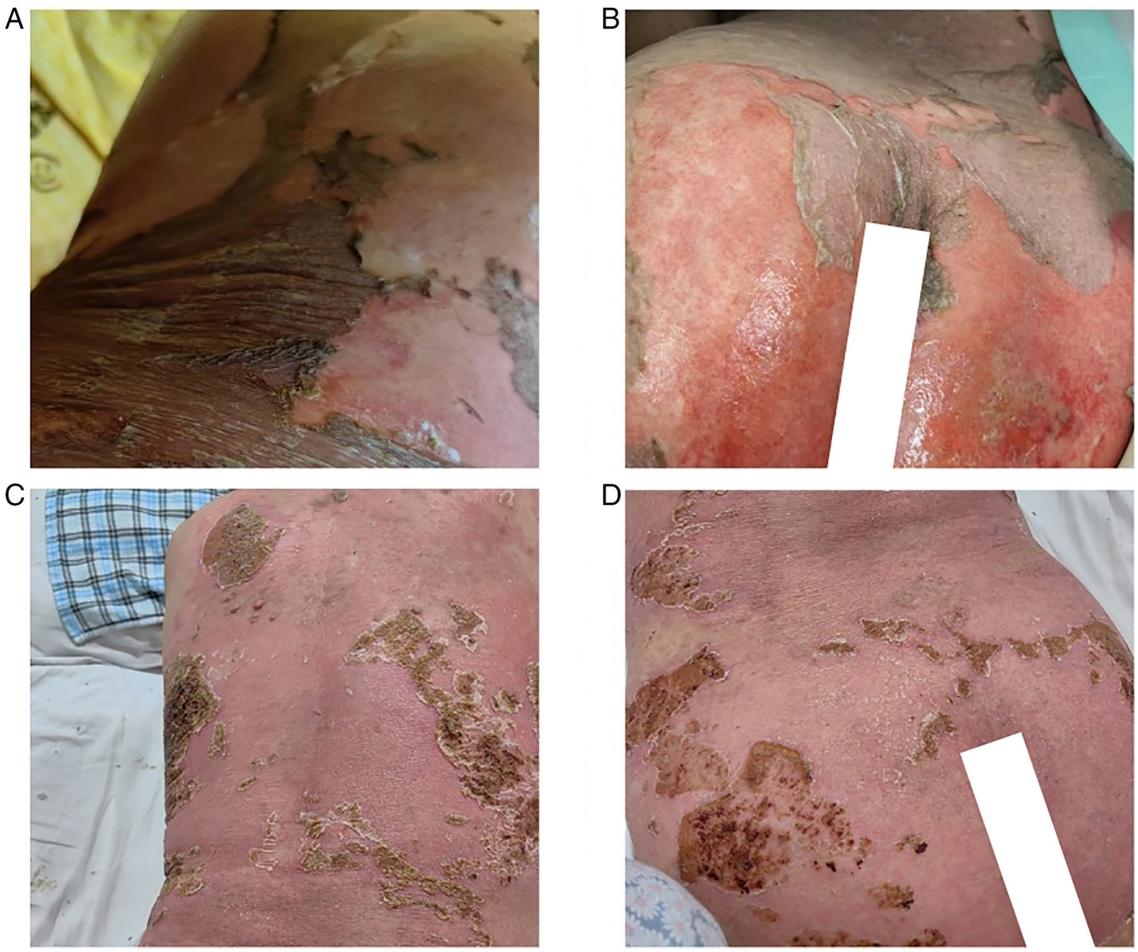

Figure 3. Before treatment, extensive confluent erythema on (A) the back and (B) buttocks, with large areas of flaccid epidermal detachment, exposing the dermis and obvious exudation. The estimated area of epidermal detachment was approximately 50% of body surface area, and Nikolsky sign was positive. After treatment, complete resolution of erythema on (C) the back and (D) buttocks, with full re-epithelialization of the detached surfaces, covered by pinkish healed skin, no residual crusts or erosions, and mild residual hyperpigmentation.

imaging examinations were performed. The patient received no additional anti-tumor therapy and was managed with palliative symptomatic care only. The patient died in July 2024. No autopsy was performed.

Discussion

Immunotherapy has revolutionized the cancer treatment landscape over the past few decades. With the increasingly

widespread application of ICIs in oncology, there has been a corresponding significant rise in reports of irAEs (5). Most irAEs occur in barrier organs, including the skin, gastrointestinal tract, liver and lungs (6). Unlike adverse reactions induced by chemotherapy or targeted therapy, irAEs are challenging to predict in terms of both severity and timing. These toxicities may lead to treatment delays or discontinuation and can even be life-threatening (7). Notably, the occurrence of irAEs is closely associated with the efficacy of ICIs. Certain studies suggest that patients who develop irAEs are more likely to mount a potent antitumor response. For instance, a retrospective study of cancer patients treated with ICIs indicated that the development of cutaneous irAEs (cirAEs) served as a significant protective factor for treatment response and overall survival (8). Similar findings have been observed in multisystem irAEs. An analysis of patients with NSCLC treated with ICIs targeting both PD-1 and programmed death-ligand 1 (PD-L1) suggested that multisystem irAEs were associated with improved survival following immunotherapy (9). Another retrospective study in patients with stage IV SCLC receiving PD-(L)1 inhibitors demonstrated that those who developed multisystem irAEs had longer survival compared to those with single-system irAEs or no irAEs-an association that persisted even after the administration of systemic corticosteroids for irAE management (10). Therefore, early recognition, timely intervention and multidisciplinary collaboration are crucial in the management of irAEs to improve patient outcomes.

Immune-mediated liver injury caused by ICIs (ILICI) is one of the commonly reported irAEs (11-13). The mechanisms driving ILICI are not yet fully elucidated. The liver possesses a unique immune profile, typically maintained in a state of immune tolerance (14-17), achieved through anti-inflammatory responses by both non-parenchymal and parenchymal cells under homeostatic conditions, along with checkpoint molecule expression across various cell subsets. After ICI blockade, this hepatic immune tolerance may be compromised, rendering the organ more susceptible to tissue inflammation. Data indicate that immune-related acute hepatitis occurs in ~18% of patients receiving combined anti-PD-1 and anti-cytotoxic T-lymphocyte-associated protein 4 monoclonal antibodies, whereas the incidence drops to 1-4% with PD-1 inhibitors alone; severe fatal hepatotoxicity is relatively rare in clinical practice (17,18). Liver injury induced by anti-PD-1/PD-L1 monoclonal antibodies primarily manifests as lobular hepatitis, characterized by inflammatory cell infiltration within the lobules. The most common clinical presentations are elevated ALT and AST levels, occasionally accompanied by abnormal bilirubin (19). Management of PD-1/PD-L1 inhibitor-induced liver injury primarily follows guidelines from authoritative oncology and hepatology societies (20-23). For grade 1-2 liver injury, the emphasis is on identifying and excluding other potential causative factors, closely monitoring liver function and temporarily withholding ICIs. For grade 3-4 hepatotoxicity, ICIs should be discontinued immediately, hospitalization considered and corticosteroid therapy initiated. In the present case, the patient developed sharply elevated ALT and AST along with significantly increased total bile acids after the second cycle of combined PD-1 inhibitor therapy, suggesting mixed liver injury. Despite prompt administration of multiple hepatoprotective agents (such as bicyclol, polyene phosphatidylcholine and ursodeoxycholic acid), liver enzymes continued to fluctuate until corticosteroids were introduced, leading to gradual recovery. This course underscores the potential severity of immune-mediated hepatotoxicity and its poor responsiveness to conventional liver-protective therapies. It is noteworthy that hepatotoxicity is closely associated with T-cell overactivation and cytokine release, making early recognition and steroid intervention critical for controlling disease progression.

CirAEs are among the earliest and most frequently observed irAEs (6). Mild to moderate skin irAEs most commonly include pruritus, nonspecific maculopapular rash, lichenoid/lichen planus-like eruptions, psoriasiform dermatitis and eczematous rash, followed by vitiligo and alopecia. Severe cutaneous irAEs encompass bullous pemphigoid, Stevens-Johnson syndrome and TEN (24). Notably, significant associations exist between specific cirAE morphological features and types of irAEs (25); for instance, mucositis has been linked with overall irAE risk, gastrointestinal irAEs and gastroenteritis, while psoriasis is associated with endocrine irAEs. TEN is a rare yet life-threatening T-cell-mediated severe cutaneous adverse reaction with substantial mortality (26). TEN induced by PD-1 inhibitors exhibits sustained immune hyperactivation distinct from conventional drug-induced TEN, rendering monotherapy with glucocorticoids often inadequate to halt progression (27). For severe cases, IVIG combined with adjunctive immunosuppressants such as infliximab or cyclosporine should be considered (28). In the present case, the patient presented with generalized purpuric macules, ecchymoses and blisters that progressed to extensive epidermal detachment, consistent with TEN-like manifestations. Markedly elevated IL-6 levels suggested involvement of a cytokine storm in the pathogenesis. Treatment with methylprednisolone combined with IVIG played a key role in gradually controlling the condition and promoting re-epithelialization. This case illustrates that multimodal treatment strategies, including immunosuppression and supportive care, are indispensable for managing severe cutaneous irAEs.

Unfortunately, liver and skin biopsies were not performed due to the patient's financial constraints. Histopathological examination would have provided definitive evidence of immune-mediated injury: In the liver, interface hepatitis with lymphocytic infiltration, lobular necroinflammation and exclusion of other etiologies (e.g., biliary obstruction or viral inclusions) (11); in the skin, full-thickness epidermal necrosis, sparse lymphocytic infiltrate (typical of TEN) and distinction from other severe cutaneous reactions such as acute generalized exanthematous pustulosis or linear IgA disease (26). The absence of tissue confirmation modestly reduces diagnostic certainty for both hepatotoxicity (grade 4) and TEN, as rare confounding pathologies [e.g., drug-induced autoimmune-like hepatitis (29) or paraneoplastic pemphigus (30)] cannot be formally ruled out. Nevertheless, the triad of a robust temporal association with tislelizumab, systematic exclusion of common alternative etiologies (viral, autoimmune, chemotherapy-alone, drug reaction with eosinophilia and systemic symptoms) (31), and a marked clinical response to corticosteroids combined with IVIG supports the clinical diagnoses with a high degree of confidence. Non-invasive alternatives, while not equivalent to histology, include serial serum biomarkers (elevated IL-6 and

CRP) and the SCORTEN score (32) (score 4 in this case), which collectively strengthen the clinical reasoning. Future studies should validate non-invasive panels as surrogates for biopsy in similar resource-limited settings. Although older patients are traditionally considered less tolerant to chemotherapy, the mechanism of action of ICIs differs fundamentally from that of chemotherapeutic agents, relying on T-cell-mediated antitumor immune activation (33). Multiple studies have shown that age itself does not significantly influence the incidence of irAEs or overall survival (24-37); however, combination with chemotherapy may substantially increase the severity and diversity of irAEs (38). Therefore, when using combined immunotherapy in elderly populations, heightened vigilance for multisystem toxicity and individualized risk management are essential.

In conclusion, this case appears to be the first to document the sequential development of grade 4 drug-induced liver injury and TEN induced by tislelizumab. The patient was successfully managed with glucocorticoids combined with IVIG. Further prospective studies are warranted to optimize immunotherapy strategies and improve the management of irAEs.

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Availability of data and materials

The data generated in the present study are included in the figures and/or tables of this article.

Authors' contributions

ZW was involved in conceptualization, data curation, writing-original draft and writing-review & editing. CL performed data curation and investigation. LY contributed to conceptualization and writing-review & editing. ZW and CL confirm the authenticity of all the raw data. All authors have read and approved the final manuscript.

Ethics approval and consent to participate

Not applicable.

Patient consent for publication

Written informed consent was obtained from the patient for publication of this case report and accompanying images.

Competing interests

The authors declare that they have no competing interests.

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