

Oxaliplatin-induced autoimmune-like hepatitis: A case report highlighting the role of clinical pharmacists

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Abstract. The present study reported the case of a 48-year-old female who developed drug-induced autoimmune-like hepatitis (DI-ALH) during adjuvant oxaliplatin, 5-fluorouracil and leucovorin calcium (FOLFOX) therapy for rectal cancer. The initial liver injury was attributed to non-specific chemotherapy-associated hepatotoxicity, resulting in a delay in the correct diagnosis. Recurrent transaminitis following oxaliplatin rechallenge subsequently suggested an immune-mediated mechanism. A clinical pharmacist played a central role in the multidisciplinary team by leading the causality assessment and developing an individualized corticosteroid management strategy, which enabled completion of all 12 planned cycles of FOLFOX without severe hepatic flares. Liver enzyme levels normalized following completion of chemotherapy and remained stable after glucocorticoid discontinuation during 12 months of follow-up. The present case report highlighted that DI-ALH may closely resemble idiopathic autoimmune hepatitis but typically resolves after withdrawal without the need for long-term immunosuppressive therapy. It also underscored the important contribution of clinical pharmacists in the recognition, assessment and management of complex chemotherapy-associated toxicities.

Introduction

Drug-induced autoimmune-like hepatitis (DI-ALH) is a rare but clinically significant phenotype of the idiosyncratic form of drug-induced liver injury (DILI), and this closely resembles idiopathic autoimmune hepatitis (AIH) in its serological, biochemical and histological features (1). Typically, AIH is characterized by elevated serum transaminase levels, increased immunoglobulin G (IgG) concentrations and detectable autoantibodies, such as anti-nuclear antibody (ANA) or anti-smooth muscle antibody (2). Histologically, AIH is characterized by interface hepatitis, plasma cell infiltration and hepatocyte rosette formation (3). The differential diagnosis of DI-ALH primarily relies on exclusion of AIH. A diagnosis of AIH can generally be excluded when liver injury resolves following withdrawal of the suspected medication and, if glucocorticoids are administered, does not recur after their discontinuation (1). This distinction is clinically important as the majority of patients with AIH require long-term immunosuppressive therapy (4).

Oxaliplatin (OXA), a third-generation platinum-based chemotherapeutic agent, is a key component of the OXA, 5-fluorouracil, leucovorin calcium (FOLFOX) regimen, which remains the standard first-line adjuvant treatment for Stage II and III colorectal cancer (5). The FOLFOX regimen consists of OXA (85 mg/m²) and leucovorin (400 mg/m²), followed by a bolus injection of 5-fluorouracil (400 mg/m²) and a 46-h continuous infusion of 5-fluorouracil (2,400 mg/m²), administered every 2 weeks (6,7). The most commonly reported adverse effects of OXA are neurotoxicity and myelosuppression (7,8). More recently, increasing attention has been directed toward its hepatotoxic potential (9,10). However, OXA-induced immune-mediated liver injury remains poorly recognized.

When hepatitis develops during chemotherapy, distinguishing DI-ALH from idiopathic AIH can be challenging but is essential, given the markedly different therapeutic and prognostic implications. In the present case, recurrent transaminitis following OXA rechallenge, together with the subsequent clinical course, supported a diagnosis of OXA-induced ALH. The present case report highlighted the importance of careful longitudinal assessment, recognition of characteristic clinical patterns and multidisciplinary collaboration, including clinical

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Abbreviations: FOLFOX, oxaliplatin, 5-fluorouracil, leucovorin calcium; ULN, upper limit of normal; DI-ALH, drug-induced autoimmune-like hepatitis; DILI, drug-induced liver injury; OXA, oxaliplatin; ALT, alanine aminotransferase; AST, aspartate aminotransferase; GGT, gamma-glutamyl transpeptidase

Key words: drug-induced liver injury, autoimmune-like hepatitis, oxaliplatin, corticosteroids, clinical pharmacist

pharmacist involvement, in the diagnosis and management of complex chemotherapy-associated hepatotoxicity.

Case report

A 48-year-old female patient (height, 155 cm; weight, 59 kg) presented to Yichang Central People's Hospital (Yichang, China) in August 2024, where she received her diagnosis and all subsequent treatment, and underwent laparoscopic resection for stage IIIB rectal adenocarcinoma (pT3N1M0). This diagnosis was established by macroscopic inspection and histopathological assessment of the resected specimen (data not shown; based on pathology reports) according to the American Joint Committee on Cancer Tumour-Nodes-Metastasis staging criteria (11). Histology demonstrated moderately differentiated adenocarcinoma, non-special type with focal mucinous differentiation. The tumour invaded connective tissue beyond the muscularis propria (pT3), and metastatic carcinoma was identified in one out of 13 perirectal lymph nodes (pN1). No distant metastasis was detected. The first cycle of postoperative adjuvant FOLFOX chemotherapy was administered one month after surgery in September 2024. Baseline liver function tests were within healthy limits and there was no personal or family history of liver disease. Contrast-enhanced (CE) pelvic magnetic resonance imaging (MRI), CE upper and lower abdominal computed tomography (CT) and thoracic CE-CT were acquired in September 2024 prior to the initiation of chemotherapy (Fig. 1). The scans revealed post-surgical changes of rectal carcinoma, with no pelvic lymphadenopathy or distant metastatic lesions, confirming stable disease at this time-point. Asymptomatic elevations in liver enzymes were detected 8 days after the initiation of postoperative adjuvant FOLFOX chemotherapy in September 2024, including alanine aminotransferase (ALT) at 129.9 U/l [3.2 x upper limit of normal (ULN)], aspartate aminotransferase (AST) at 101.8 U/l (2.9 x ULN) and gamma-glutamyl transferase (GGT) at 100 U/l (2.2 x ULN). Both total bilirubin (TBL) and the international normalized ratio (INR) were within the normal range (TBL, 4.6 μ mol/l, reference range: 3.4–17.1 μ mol/l; INR, 1.0, reference range: 0.8–1.2). Hepatoprotective therapy, including glutathione and magnesium isoglycyrrhizinate, resulted in normalization of liver enzyme levels and FOLFOX chemotherapy was temporarily suspended due to acute liver injury. Treatment was subsequently switched to concurrent chemoradiotherapy with capecitabine from September 2024 to November 2024, in accordance with the National Comprehensive Cancer Network Clinical Practice Guidelines in Oncology (NCCN guidelines) for Rectal Cancer, version 3.2024 (12). For this patient with stage IIIB rectal adenocarcinoma (pT3N1M0), the version 3.2024 NCCN guidelines permit two sequential adjuvant strategies: 12 cycles of adjuvant FOLFOX followed by capecitabine-based chemoradiotherapy, or upfront capecitabine chemoradiotherapy followed by 12 cycles of FOLFOX. The initial plan was FOLFOX first, followed by pelvic radiotherapy. However, transaminitis developed after the first FOLFOX cycle. To reduce systemic chemotherapy intensity and allow hepatic recovery, the sequence was reversed to deliver capecitabine-based chemoradiotherapy upfront. This adjustment was consistent with the permissive sequence modifications allowed by the NCCN guidelines.

During this period of concurrent capecitabine chemoradiotherapy, only mild fluctuations in liver enzyme levels (1–2 x ULN) were observed, which responded to hepatoprotective treatment with glutathione (1.2 g intravenously once daily), magnesium isoglycyrrhizinate (100 mg intravenously once daily) and polyene phosphatidylcholine (465 mg intravenously once daily). Post-chemoradiotherapy imaging (pelvic CE-MRI, upper and lower abdominal CE-CT and thoracic CE-CT) was performed following completion of chemoradiotherapy in November 2024 (Fig. 1). No local recurrence at the surgical resection bed or distant metastatic disease was identified, consistent with sustained stable rectal adenocarcinoma at this time-point.

The clinical pharmacist played a central role in multidisciplinary team (MDT) discussions. After careful consideration of the oncological benefits and potential hepatic risks, the MDT elected to proceed with an OXA rechallenge under close monitoring, supported by two key considerations. First, incomplete FOLFOX would significantly increase recurrence risk, as 12 cycles are guideline-mandated (12). Second, the initial liver injury did not meet the criteria for permanent OXA discontinuation: ALT peaked at 3.2 x ULN, without clinical symptoms (fatigue, nausea, vomiting, right upper quadrant pain, fever, rash) or laboratory abnormalities (TBL >2 x ULN, INR >1.5) (13). Given the substantial antitumour benefit of completing adjuvant chemotherapy outweighed the low risk of recurrent hepatotoxicity, the MDT proceeded with rechallenge under rigorous monitoring. The monitoring protocol included liver function tests (ALT, AST, alkaline phosphatase, bilirubin) performed before each cycle, 24 h post-infusion and weekly between cycles, with clinical evaluation for jaundice, fatigue and abdominal discomfort at each visit (13). Written informed consent was obtained and personally signed by the patient for this procedure.

FOLFOX chemotherapy was resumed in November 2024. Mild elevations in transaminases were detected [ALT at 45.1 U/l (1.1 x ULN) and AST at 36.8 U/l (1.1 x ULN)] 3 days after this resumption in December 2024, prompting administration of glutathione (1.2 g intravenously once daily), magnesium isoglycyrrhizinate (100 mg intravenously once daily) and polyene phosphatidylcholine (465 mg intravenously once daily). However, reassessment in December 2024 demonstrated marked worsening of liver injury, with ALT at 303.5 U/l (7.6 x ULN), AST at 275.8 U/l (7.9 x ULN) and GGT at 164 U/l (3.6 x ULN). Viral hepatitis screening was negative except for hepatitis B surface antibody positivity, consistent with prior vaccination. All serological and autoantibody tests were performed using standard clinical laboratory assays. Autoantibody testing revealed positivity for ANA (titre 1:100, negative reference), anti-mitochondrial M2 antibody (AMA-M2; 3.07 AU/ml, reference range: 0–1 AU/ml) and anti-SPI00 antibody (2.88 AU/ml, reference range: 0–1 AU/ml), whereas serum IgG concentrations remained within the healthy range (13.04 g/l, reference range: 7–16 g/l).

Liver biopsy was recommended to establish a definitive diagnosis. However, despite healthy coagulation parameters, the patient declined the procedure because of concerns regarding potential bleeding complications and procedural discomfort. No liver imaging was performed during the

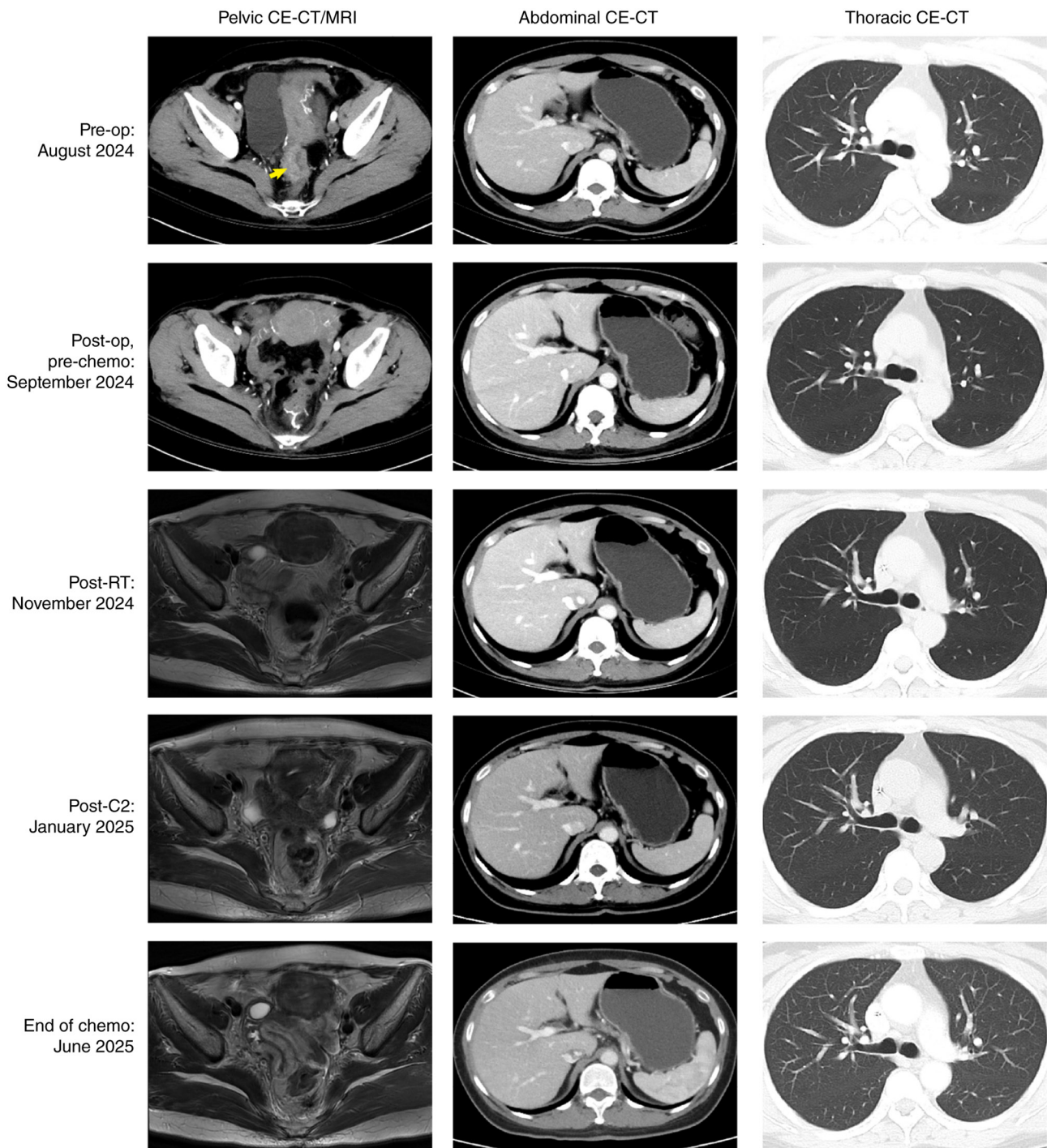


Figure 1. Serial imaging showed no local pelvic recurrence or distant metastases throughout treatment. The five rows represent key time-points: Pre-op staging (August 2024), Post-op and Pre-chemo (September 2024), post- RT (November 2024), Post-C2 (January 2025) and end of chemo (June 2025). The three columns display representative axial slices of the pelvis, upper and lower abdomen and thorax. Pelvic imaging: CE-CT was used at the first two visits (August 2024, September 2024), with T2-weighted MRI adopted for subsequent follow-ups due to superior soft-tissue contrast for detecting subtle recurrence and small lymph nodes. Abdominal and thoracic scans: Only CE-CT phases are shown, as they provide sufficient diagnostic information. For pelvic MRI, only T2-weighted images are presented to better distinguish postoperative scarring from potential recurrence. A coloured arrow marks the primary rectal lesion on the preoperative pelvic CT; no markers are added elsewhere, as no recurrence or metastases were identified at any time-point. CE-CT, contrast-enhanced computed tomography; MRI, magnetic resonance imaging; Pre-op, pre-operative; Post-op, post-operative; Pre-chemo, before chemotherapy; RT, radiotherapy; Post-C2, after 2 cycles of chemotherapy; end of chemo, upon chemotherapy completion.

hepatic flare; monitoring relied solely on serial liver function tests and clinical examination. Liver imaging was not emergently arranged for the following reasons: i) The patient had no focal hepatic symptoms (severe right upper quadrant pain, hepatomegaly, jaundice or ascites); ii) serial liver

function tests adequately tracked hepatotoxicity severity; and iii) CE-CT for tumour staging performed shortly before showed no hepatic lesions, biliary obstruction or vascular abnormalities. A structured causality assessment was subsequently conducted by the clinical pharmacist using

the temporal association between drug exposure and liver injury, the dechallenge-rechallenge response pattern and the Roussel Uclaf Causality Assessment Method (RUCAM). The RUCAM score was 10, indicating a highly probable causal association between OXA and the liver injury. Based on the strong temporal association, positive rechallenge, exclusion of alternative etiologies and autoimmune features, OXA-induced ALH was considered the most likely diagnosis. The decision to proceed with empirical treatment was made by the oncology team with support from the clinical pharmacist.

Glucocorticoids are considered first-line therapy for DI-ALH. Current guidelines recommend prednisolone at a dose of 0.5-1.0 mg/kg/day (14). Taking into account the patient's body weight and impaired hepatic function, the clinical pharmacist guided the administration of intravenous methylprednisolone 40 mg/day in December 2024, approximately 2-3 weeks after re-initiation of FOLFOX chemotherapy following radiotherapy completion. This dose is roughly equivalent to 50 mg/day of prednisolone and falls within the recommended therapeutic range. Methylprednisolone was selected because it does not require hepatic activation, thereby providing more predictable pharmacological activity in the setting of acute liver injury. Marked improvement in liver enzyme levels was observed within three days, with ALT and AST decreasing to 133.7 and 55.0 U/l, respectively. Oral prednisone 10 mg daily was commenced 3 days after initiation of intravenous methylprednisolone in December 2024, and liver enzyme levels normalized within 1 week. Following the third cycle of FOLFOX chemotherapy, recurrent transaminase elevations were observed later that same month [ALT at 124.0 U/l (3.1 x ULN) and AST at 91.7 U/l (2.6 x ULN)]. Given the immune-mediated nature of the liver injury and the importance of completing 12 cycles of adjuvant chemotherapy to reduce the risk of cancer recurrence, the clinical pharmacist proposed a prophylactic corticosteroid strategy consisting of intravenous methylprednisolone 40 mg daily for 3 days before each FOLFOX cycle, followed by oral prednisone 10 mg daily until the subsequent treatment cycle. Subsequent clinical observations supported this strategy, as each chemotherapy cycle was associated with only mild and transient transaminase elevations. Corresponding written informed consent was also obtained from the patient for this regimen. It was continued until completion of all 12 planned cycles of FOLFOX chemotherapy in May 2025. Imaging comprising pelvic CE-MRI, upper and lower abdominal CE-CT and thoracic CE-CT was completed in January 2025 (Fig. 1). There was no evidence of local relapse at the resection site or distant metastases, verifying stable rectal tumour status at this follow-up visit. Thereafter, serial radiological assessment and serum tumour marker surveillance were scheduled every two cycles of FOLFOX chemotherapy. No local recurrence or distant metastases were detected throughout the entire treatment period, including during the episode of immune-mediated liver injury. A post-treatment CE scan was obtained 1 month after the final cycle of 12-cycle FOLFOX chemotherapy in June 2025 (Fig. 1). The imaging findings revealed stable size and morphology of the primary rectal adenocarcinoma, with no local progression, lymph node recurrence or distant metastasis. The patient's rectal adenocarcinoma remained

consistently stable throughout surveillance. During this entire period, liver enzyme levels remained <3 x ULN, enabling uninterrupted completion of adjuvant treatment. The strategy effectively controlled liver injury while minimizing exposure to prolonged high-dose corticosteroid therapy.

The dynamic changes in liver enzyme levels during chemotherapy and glucocorticoid treatment are presented in Fig. 2. Following completion of chemotherapy in May 2025, glucocorticoids were discontinued. During the subsequent 12-month follow-up period ending in June 2026, monthly laboratory assessments consistently demonstrated healthy liver function, indicating sustained remission without recurrence of liver injury. Quarterly radiological surveillance was routinely performed and serial imaging obtained in September 2025, December 2025, March 2026 and June 2026 demonstrated continuously stable rectal adenocarcinoma, with no local progression, regional recurrence or distant metastasis (Fig. 3). On thoracic CT performed in March 2026, a 3x3 mm micronodule was detected in the lateral segment of the right middle lobe (Fig. 3; yellow arrow) and managed with active surveillance. The nodule had spontaneously resolved on follow-up CT in June 2026, implying an infective aetiology. Given the stable disease status and spontaneous resolution of the pulmonary micronodule, no additional systemic or local intervention was required. The patient continued scheduled quarterly radiological and serum tumour-marker surveillance for colorectal cancer, with ongoing clinical follow-up.

Discussion

Among the components of the FOLFOX regimen, OXA and 5-fluorouracil were the principal agents with potential hepatotoxicity. Leucovorin functions solely as a pharmacological modulator and was therefore not considered a suspect drug in the causality assessment. The temporal course of liver enzyme changes supports OXA as the primary cause of liver injury: Transaminases rose after initial FOLFOX (5-fluorouracil plus OXA), subsided during capecitabine monotherapy and rose again upon FOLFOX rechallenge. Given that capecitabine is metabolized to 5-fluorouracil *in vivo*, this pattern of liver enzyme fluctuation suggests a stronger causal association with OXA than with 5-fluorouracil. Formal causality evaluation using the RUCAM scale further specifically ruled out other FOLFOX constituents as primary triggers. Points corresponding to the co-administered suspect agent 5-fluorouracil were deducted during scoring; even after this deduction, the patient's overall RUCAM score remained 10, consistent with a highly probable causal link between OXA and observed DILI.

Developed by an international group of hepatologists, regulatory scientists and the Roussel Uclaf Drug Safety Department in the early 1990s (15), RUCAM remains the most widely used diagnostic tool for assessing DILI (16). The clinical pharmacist derived the score as follows: Time to onset (+3; reaction occurring within 5-90 days of drug exposure), course after drug withdrawal (+3; ALT decreased by ≥50% from a peak of 3.2 x ULN within 8 days), risk factors (0; no alcohol consumption and age <55 years), concomitant drugs (-3; potential contribution of 5-fluorouracil), exclusion of non-drug causes (+2), previous evidence of hepatotoxicity (+2;

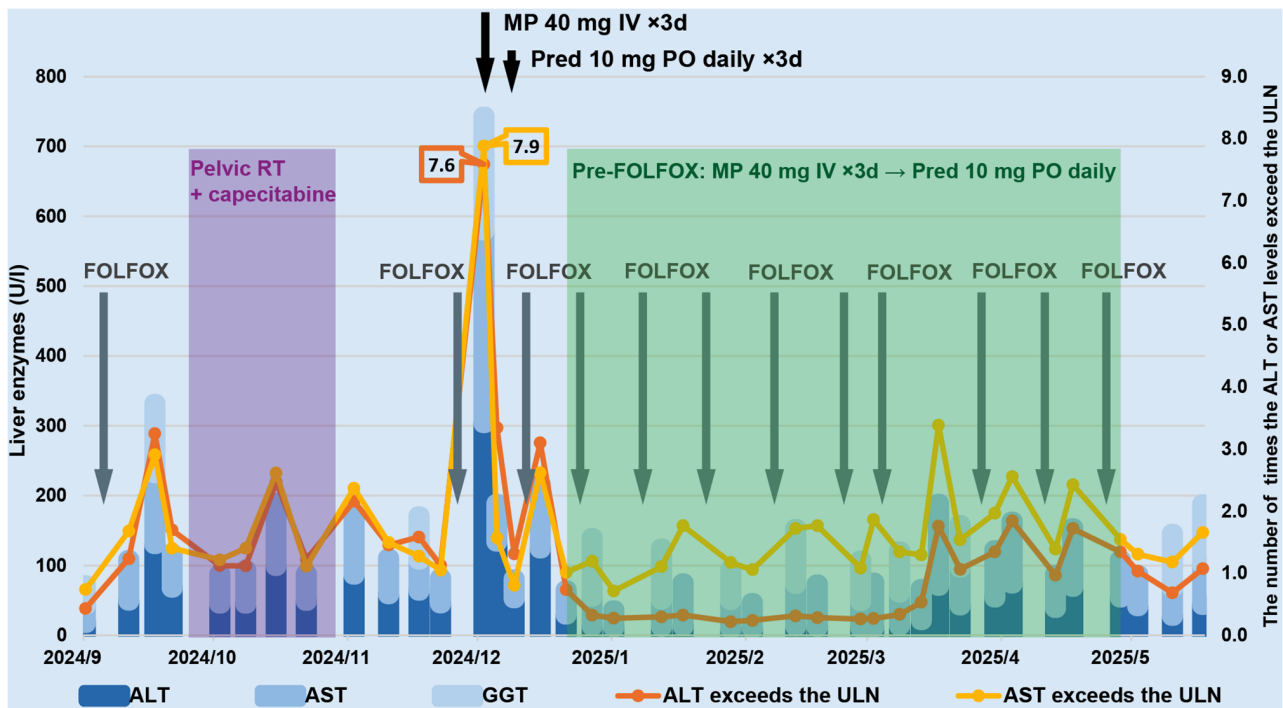


Figure 2. Trends in transaminase levels during chemotherapy and glucocorticoid treatment from September 2024 to May 2025. The timeline extends from initiation of FOLFOX chemotherapy in September 2024, to completion of all 12 treatment cycles in May 2025, and illustrates temporal changes in liver enzyme levels. The x-axis represents the date. The left y-axis indicates serum transaminase concentrations, whereas the right y-axis shows the fold increase above the ULN. Blue bars represent transaminase levels, with palest blue indicating GGT, light blue indicating AST and dark blue indicating ALT. Yellow line graphs represent the fold increase above the ULN over time, with light yellow indicating AST and orange indicating ALT; fold-ULN values were not calculated for GGT, and therefore no corresponding line is presented for this parameter. A total of 12 grey arrows indicate the timing of the 12 FOLFOX chemotherapy cycles. The purple shaded area denotes the period of concurrent chemoradiotherapy (pelvic RT plus capecitabine). Green shaded areas indicate administration of prophylactic IV methylprednisolone (40 mg once daily) for three consecutive days after the start of each FOLFOX cycle, followed by maintenance oral prednisone (10 mg once daily) until the subsequent treatment cycle. A marked increase in transaminase levels was observed following the second FOLFOX cycle, followed by a rapid decline after initiation of glucocorticoid therapy (black arrow). ALT, alanine aminotransferase; AST, aspartate aminotransferase; FOLFOX, oxaliplatin, 5-fluorouracil, leucovorin; GGT, gamma-glutamyl transferase; ULN, upper limit of normal; RT, radiotherapy; MP, methylprednisolone; Pred, prednisone; IV, intravenous; PO, per os (oral); d, days.

reaction described in OXA product information) and response to re-administration (+3; positive rechallenge with ALT increasing to 7.6 x ULN). To further support the assessment, the Refined Electronic Causality Assessment Method (17) was also applied and yielded a score of 3, indicating a 'possible' association. In a multicentre post-marketing safety assessment involving 3,678 patients, OXA was found to exert a potentially specific effect on hepatic function, the occurrence of which appeared to be independent of the dose (9). On the basis of the above evidence, the patient in the present case report received the full course of the standard-dose FOLFOX regimen as described in the Introduction, and no dose reduction was made even after DILI had developed.

Although liver biopsy was recommended, the patient declined the procedure. Histological confirmation is not mandatory for the diagnosis of DI-ALH, particularly when strong clinical evidence is available (2). In the present case, the characteristic dechallenge-rechallenge pattern, exclusion of competing etiologies and highly probable RUCAM score provided compelling evidence supporting OXA-induced liver injury.

Importantly, the progression of liver injury after initial drug withdrawal does not exclude DI-ALH, as immune-mediated hepatic injury may continue to evolve despite cessation of the triggering agent. This phenomenon was reflected by

the marked increase in transaminase levels following rechallenge with FOLFOX after completion of chemoradiotherapy. The serological profile observed in the present case, characterized by positive ANA, AMA-M2 and SP100 antibodies in the presence of normal IgG concentrations, is atypical for classical AIH. Elevated IgG levels are a hallmark feature of idiopathic AIH (4). The expected IgG concentration, absence of a prior history of autoimmune disease and negative viral hepatitis results collectively supported a drug-induced immune-mediated process rather than unmasking of underlying AIH. This profile is consistent with previous reports of DI-ALH (13,18).

Following the second cycle of FOLFOX chemotherapy, transaminase levels continued to rise despite treatment with anti-inflammatory and antioxidant hepatoprotective agents. By contrast, administration of glucocorticoids resulted in rapid normalization of liver enzymes. Furthermore, after completion of all 12 cycles of adjuvant FOLFOX chemotherapy, corticosteroids were successfully discontinued without biochemical relapse. Collectively, these findings strongly support classification of the liver injury as OXA-induced ALH rather than conventional DILI.

To date, reports associating OXA to DI-ALH with autoimmune serological features remain limited. LiverTox, the National Institutes of Health-sponsored database of DILI,

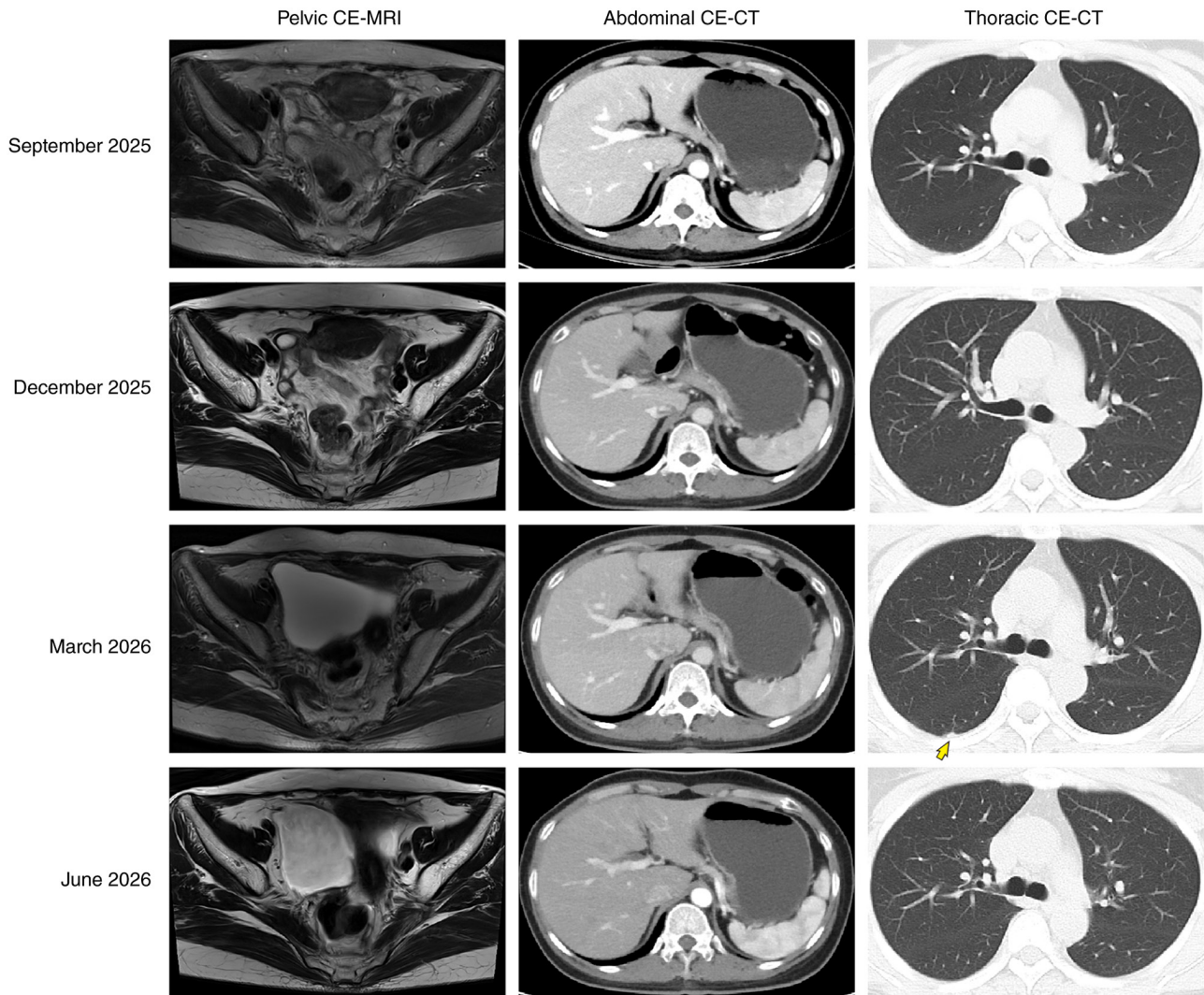


Figure 3. Serial surveillance imaging during post-chemotherapy follow-up. A total of four sequential quarterly imaging assessments were performed in September 2025, December 2025, March 2026 and June 2026. Each time-point comprises pelvic CE-MRI (first column), abdominal CE-CT (second column) and thoracic CE-CT (third column). Abdominal and thoracic scans: Only CT phases are shown, as they provide sufficient diagnostic information. For pelvic MRI, only T2-weighted images are presented to better distinguish postoperative scarring from potential recurrence. A yellow arrow indicates a 3x3 mm right middle lobe lateral segment micronodule on the March 2026 thoracic CE-CT for surveillance. The lesion resolved at the same location later that same month, possibly caused by infection. All serial scans demonstrate stable rectal adenocarcinoma without evidence of local progression, regional recurrence or distant metastasis. CE-MRI, contrast-enhanced magnetic resonance imaging; CE-CT, contrast-enhanced computed tomography.

contains detailed descriptions of >1,200 agents associated with hepatotoxicity (19). The majority of antineoplastic agents possess varying degrees of hepatotoxic potential, typically presenting as transient elevations in transaminases and bilirubin levels during treatment (19). In the majority of patients, liver function recovers rapidly following dose adjustment or drug discontinuation (19). However, the clinical course observed in the present case differed substantially from these typical patterns.

After the second cycle of FOLFOX, the patient developed a marked increase in transaminase levels that progressed despite hepatoprotective therapy. Review of the LiverTox database indicated that liver injury associated with either 5-fluorouracil or OXA is generally not accompanied by autoimmune serological abnormalities. Hepatotoxicity associated with 5-fluorouracil is typically characterized by elevations in transaminases and bilirubin and, in severe cases, may progress to hyperammonaemic encephalopathy. In addition,

5-fluorouracil has been implicated as a risk factor for hepatic steatosis (13). Although OXA-induced hepatotoxicity is relatively uncommon, it is often associated with more severe hepatic injury, predominantly manifesting as isolated transaminase elevations. Sinusoidal obstruction syndrome and nodular regenerative hyperplasia are among the most frequently reported OXA-associated liver lesions (20). Concurrent elevations in both transaminases and bilirubin are uncommon. Published data indicate that the FOLFOX regimen is associated with a 16% incidence of transaminase elevation, with only approximately 2% of patients developing liver enzyme abnormalities exceeding 5 x ULN (21). Existing literature of FOLFOX-related hepatic injury predominantly describes parenchymal vascular damage manifesting as sinusoidal obstruction syndrome (SOS) (22). Oxidative stress constitutes the core pathogenic pathway behind this form of FOLFOX-induced liver toxicity, and concurrent administration of antioxidants has been shown to exert protective

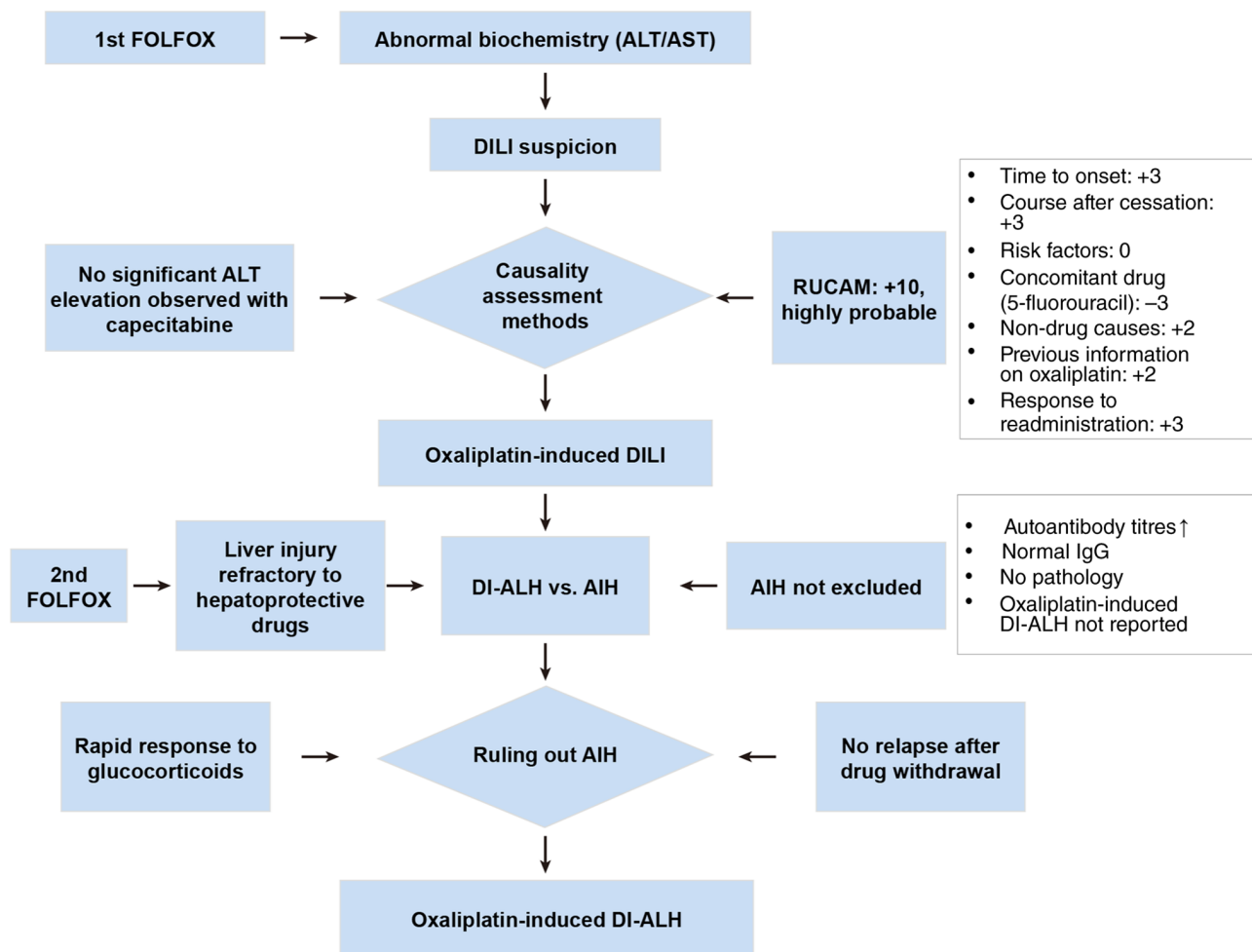


Figure 4. Diagnostic pathway for oxaliplatin-induced ALH. The flowchart summarizes the clinical assessment process leading to the diagnosis of oxaliplatin-induced ALH, including evaluation of liver enzyme abnormalities, exclusion of alternative causes, causality assessment and differentiation from idiopathic autoimmune hepatitis. ALH, autoimmune-like hepatitis; ALT, alanine aminotransferase; AST, aspartate aminotransferase; DILI, drug-induced liver injury; FOLFOX, oxaliplatin, 5-fluorouracil, leucovorin calcium; AIH, autoimmune hepatitis.

effects against SOS development (23). These previously documented vascular hepatic toxicities exhibit markedly different biochemical, clinical and therapeutic profiles compared with the immune-mediated DI-ALH observed in the present patient.

Overall, the available evidence supports a diagnosis of OXA-induced ALH. The diagnostic pathway is summarized in Fig. 4. Given the apparent rarity of this presentation, greater awareness among clinicians is warranted.

Importantly, the present case highlights the critical role of the clinical pharmacist in the management of high-risk DILI. Recognition of the characteristic dechallenge-rechallenge pattern facilitated timely identification of an immune-mediated mechanism and supported prompt initiation of corticosteroid therapy, enabling safe continuation of potentially curative adjuvant chemotherapy. The prophylactic corticosteroid strategy successfully prevented severe hepatic flares during subsequent treatment cycles and provided a practical approach to balancing oncological benefit against hepatic risk. This case illustrates how pharmacist-led medication review and causality assessment can contribute substantially to integrated, patient-centred cancer care.

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Availability of data and materials

The data generated in the present study may be requested from the corresponding author.

Authors' contributions

XZ performed the case review and wrote and revised the draft. TL reviewed imaging data and participated in the writing and revision of draft. GJ provided a case review and contributed to the writing and revision of the draft. XZ and TL checked and confirmed the authenticity of all the raw data. All authors have read and approved the final manuscript.

Ethics approval and consent to participate

The present study was approved by the Ethics Committee of Yichang Central People's Hospital (approval no. 2025-231-01). Informed consent was obtained from the patient for participating in this case report in accordance with the principles of the Declaration of Helsinki.

Patient consent for publication

Written informed consent for the publication of this case report and any accompanying medical images was obtained from the patient included in the present study.

Competing interests

The authors declare that they have no competing interests.

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