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Comparative review of hepatic and renal toxicities in Hodgkin and non-Hodgkin lymphoma chemotherapy

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Abstract
Objective: This review aimed to comparatively analyse the existing literature on the impact of standard chemotherapy regimens on hepatic and renal function parameters in patients with Hodgkin Lymphoma (HL) versus Non-Hodgkin Lymphoma (NHL).
Methods: A comprehensive literature search was conducted across PubMed, ScienceDirect, and Google Scholar from inception to March 2024. The review included original research, reviews, meta-analyses, and case reports published in English that focused on hepatic and/or renal function in adult HL or NHL patients receiving standard chemotherapy. Studies solely on paediatric populations, those focusing on other toxicities without a significant hepatic/renal focus, and non-peer-reviewed works were excluded. Data on regimens, organ function abnormalities, toxicity incidence, risk factors, and mechanisms were synthesised.
Results: The analysis revealed distinct toxicity profiles. HL regimens (ABVD, BEACOPP) are associated with direct, though often transient, hepatotoxicity and a lower risk of renal impairment. In contrast, NHL regimens (R-CHOP, R-CVP, BR) carry a significant risk of Hepatitis B Virus reactivation, leading to severe hepatotoxicity. Furthermore, aggressive NHL subtypes are highly susceptible to acute kidney injury driven by Tumour Lysis Syndrome. Key risk modifiers include baseline viral status, pre-existing organ dysfunction, and cumulative drug doses.
Conclusion: The patterns of chemotherapy-induced hepatic and renal toxicity differ markedly between HL and NHL. Recognising these differences is crucial for implementing tailored prophylactic strategies, vigilant monitoring, and timely interventions to mitigate adverse outcomes and improve patient safety and quality of life.

Keywords: Hodgkin Lymphoma, Non-Hodgkin Lymphoma, Chemotherapy, Hepatotoxicity, Nephrotoxicity, Hepatitis B Reactivation, Tumour Lysis Syndrome

Plain English Summary

This paper is a review that looks at the side effects of chemotherapy on the liver and kidneys in patients with the two main types of lymphoma: Hodgkin Lymphoma and Non-Hodgkin Lymphoma. We compared the risks that come with the most common treatment regimens for each disease. The main finding is that the dangers to these organs are quite different between the two types. For Hodgkin Lymphoma, the chemotherapy drugs themselves can sometimes cause direct, though often temporary, damage to the liver, while serious kidney issues are less common. For Non-Hodgkin Lymphoma, the biggest concern for the liver is different; a key drug called rituximab can cause a past hepatitis B infection to flare up, which can lead to sudden and severe liver damage. For the kidneys, patients with aggressive Non-Hodgkin Lymphoma face a significant risk of a complication called Tumour Lysis Syndrome, where the rapid death of cancer cells can overwhelm the kidneys and cause them to fail suddenly. The review concludes that understanding these different risks is vital for doctors. It means they can tailor their approach for each patient, implementing crucial preventative steps like screening for hepatitis B and closely monitoring kidney function in high-risk situations. This proactive management is key to preventing serious complications and ensuring chemotherapy is as safe and effective as possible.

Introduction

Lymphomas represent a diverse group of haematological malignancies originating from lymphocytes, broadly categorised into Hodgkin Lymphoma (HL) and Non-Hodgkin Lymphoma (NHL). While both are cancers of the lymphatic system, they differ significantly in their pathological characteristics, clinical behaviour, and treatment approaches (1).

Chemotherapy remains the cornerstone of treatment for most lymphoma subtypes. Standard regimens for HL, such as ABVD (Doxorubicin, Bleomycin, Vinblastine, Dacarbazine) and escalated BEACOPP (Bleomycin, Etoposide, Doxorubicin, Cyclophosphamide, Vincristine, Procarbazine, Prednisone), have dramatically improved survival rates (2). Similarly, for NHL, regimens like R-CHOP (Rituximab,

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Cyclophosphamide, Doxorubicin, Vincristine, Prednisone) have revolutionised outcomes (3).
Despite their therapeutic efficacy, these potent chemotherapeutic agents are associated with a spectrum of adverse effects. Among the most critical are toxicities affecting the liver and kidneys, which can lead to significant morbidity, necessitate dose modifications, and result in life-threatening complications. This paper aims to provide a comprehensive comparative review of hepatic and renal function parameters in HL and NHL patients undergoing chemotherapy, delineating specific toxicities, exploring underlying mechanisms, identifying risk factors, and discussing implications for clinical management.

Materials and Methods
This comparative analysis is based on a comprehensive review of existing literature focusing on hepatic and renal function parameters in Hodgkin Lymphoma (HL) and Non-Hodgkin Lymphoma (NHL) patients following chemotherapy. A systematic search was conducted across major medical and scientific databases, including PubMed, ScienceDirect, and Google Scholar, for studies published from database inception to March 2024. The search strategy employed a combination of keywords related to lymphoma types ('Hodgkin Lymphoma', 'Non-Hodgkin Lymphoma'), chemotherapy regimens ('ABVD', 'BEACOPP', 'R-CHOP', 'R-CVP', 'BR'), organ function ('hepatic function', 'renal function', 'liver toxicity', 'nephrotoxicity'), and specific complications ('Hepatitis B reactivation', 'Tumour Lysis Syndrome', 'acute kidney injury').

Inclusion Criteria
The review considered original research articles, review articles, meta-analyses, and case reports published in English that reported on hepatic and/or renal function parameters in adult patients with a diagnosis of HL or NHL who were treated with standard chemotherapy regimens. Articles were also included if they discussed the mechanisms of liver or kidney injury, the incidence of toxicity, associated risk factors, or relevant management strategies.

Exclusion Criteria
Studies were excluded if they focused exclusively on paediatric populations. Articles that primarily addressed other chemotherapy-related toxicities—such as cardiotoxicity or pulmonary toxicity—without a significant focus on hepatic or renal aspects were also omitted. Furthermore, non-peer-reviewed publications and conference abstracts for which the full text was not available were not included in the analysis.

The identified literature was critically appraised for relevance and quality. Data extracted included: specific chemotherapy regimens, reported hepatic and renal function abnormalities (e.g., elevated ALT, AST, ALP, GGT, bilirubin, creatinine, BUN, eGFR), incidence rates of hepatotoxicity and nephrotoxicity, identified risk factors (e.g., baseline HBV status, pre-existing CKD, age, cumulative drug doses), and proposed mechanisms of organ injury. The data were synthesised through thematic analysis, comparing findings between HL and NHL regimens to generate the comparative overview presented in the results section.

Results
Hepatic Toxicity
Chemotherapy-induced hepatotoxicity is a significant concern in both HL and NHL patients, though the specific agents, mechanisms, and risk profiles differ. The key hepatic function parameters monitored include Alanine Transaminase (ALT), Aspartate Transaminase (AST), Alkaline Phosphatase (ALP), Gamma-Glutamyl Transferase (GGT), total and conjugated bilirubin, albumin, and prothrombin time (PT)/International Normalised Ratio (INR) (4, 5).

Hodgkin Lymphoma: ABVD and BEACOPP Regimens
ABVD Regimen: The ABVD regimen is generally associated with a relatively low incidence of severe hepatotoxicity. Mild and transient elevations in liver enzymes are common, often resolving spontaneously (6). While rare, severe liver injury, including acute liver failure, has been reported; dacarbazine has been linked to veno-occlusive disease (VOD) (7, 8, 9).
BEACOPP Regimen: This more intensive protocol carries a higher potential for hepatic toxicity. Components like cyclophosphamide and procarbazine are known to possess hepatotoxic potential, and etoposide can contribute to elevated liver enzymes (10, 11). Close monitoring of liver function tests (LFTs) is standard practice.

Non-Hodgkin Lymphoma: R-CHOP, R-CVP, and BR Regimens
R-CHOP Regimen: While liver enzyme elevations are common, the primary hepatic concern is Hepatitis B Virus (HBV) reactivation. Rituximab-induced B-cell depletion compromises immune control over HBV, leading to viral rebound and potentially severe hepatitis or acute liver failure (12, 13, 14). Pre-screening and prophylactic antiviral therapy are mandatory (15). The cytotoxic agents (cyclophosphamide, doxorubicin) can also cause direct hepatotoxicity (16).
R-CVP Regimen: This regimen shares the rituximab-associated risk of HBV reactivation but is generally considered less hepatotoxic than R-CHOP due to the absence of doxorubicin (17).
BR Regimen: Bendamustine can cause liver enzyme elevations, and caution is advised in patients with pre-existing liver impairment. The risk of HBV reactivation remains due to the rituximab component (18).

Comparative Summary of Hepatic Toxicity
Table 1 provides a comparative overview of hepatic toxicity profiles between common HL and NHL chemotherapy regimens.

Table 1: Comparative Hepatic Toxicity Profiles in Hodgkin Lymphoma vs. Non-Hodgkin Lymphoma Chemotherapy

Feature	Hodgkin Lymphoma (ABVD/BEACOPP)	Non-Hodgkin Lymphoma (R-CHOP/R-CVP/BR)
Primary Concern	Direct drug-induced liver injury	HBV reactivation (major concern with rituximab)
Key Agents	Dacarbazine, Cyclophosphamide, Procarbazine	Rituximab, Cyclophosphamide, Doxorubicin, Bendamustine

Severity	Generally mild and transient; severe cases are rare	Can be severe, especially with HBV reactivation
Monitoring	Routine LFTs	Routine LFTs, mandatory HBV screening and monitoring
Prophylaxis	Not routinely indicated	Antiviral prophylaxis for HBV-positive patients is critical

Renal Toxicity

Renal function parameters, including serum creatinine, blood urea nitrogen (BUN), estimated glomerular filtration rate (eGFR), and electrolytes, are crucial indicators of kidney health (4, 5). Chemotherapy can impact renal function through various mechanisms.

Hodgkin Lymphoma: ABVD and BEACOPP Regimens

ABVD Regimen: Renal toxicity is generally low. Mild, transient elevations in creatinine can occur, but severe acute kidney injury (AKI) is rare. Bleomycin may rarely contribute to renal dysfunction, and Tumour Lysis Syndrome (TLS) can occur in patients with high tumour burden (6, 19, 20, 21).

BEACOPP Regimen: This regimen carries a higher potential for renal toxicity. Etoposide can cause renal toxicity at higher doses, and cyclophosphamide can cause haemorrhagic cystitis or SIADH, affecting fluid balance (22, 23). If platinum agents are used in salvage therapy, they are highly nephrotoxic (24).

Non-Hodgkin Lymphoma: R-CHOP, R-CVP, and BR Regimens

R-CHOP Regimen: Renal complications are more frequent, often due to the disease itself or TLS. TLS is a major concern in aggressive NHLs (e.g., DLBCL, Burkitt Lymphoma) due to rapid tumour cell breakdown, leading to metabolic derangements and AKI (25, 26). Direct renal infiltration by NHL is also more common than in HL (27).

R-CVP Regimen: TLS remains a significant risk, particularly in indolent lymphomas with high tumour burden. It is generally considered less nephrotoxic than R-CHOP due to the absence of doxorubicin.

BR Regimen: Bendamustine can cause renal toxicity and requires dose adjustments in patients with pre-existing renal impairment. The risk of TLS is present due to rituximab (28).

Comparative Summary of Renal Toxicity

Table 2 provides a comparative overview of renal toxicity profiles.

Table 2: Comparative Renal Toxicity Profiles in Hodgkin Lymphoma vs. Non-Hodgkin Lymphoma Chemotherapy

Feature	Hodgkin Lymphoma (ABVD/BEACOPP)	Non-Hodgkin Lymphoma (R-CHOP/R-CVP/BR)
Primary Concern	Direct renal infiltration (rare), TLS (less common)	Tumour Lysis Syndrome (TLS), direct renal involvement
Key Agents	Bleomycin, Etoposide, Doxorubicin (indirect)	Chemosensitivity leading to TLS, Bendamustine
Severity	Generally low; severe AKI is rare	Can be severe, often requiring aggressive management
Monitoring	Routine renal function tests	Aggressive TLS monitoring (electrolytes, uric acid)
Prophylaxis	Allopurinol/hydration for TLS risk	Allopurinol/Rasburicase and aggressive hydration

Discussion

This comparative review synthesises evidence on the distinct patterns of hepatic and renal toxicity associated with chemotherapy for HL and NHL. The differences are driven by variations in disease biology, specific chemotherapeutic agents, and patient-related risk factors. Our analysis reveals that while direct organ injury is a feature of both lymphoma types, the predominant concerns are different: direct hepatotoxicity in HL versus HBV reactivation in NHL, and a generally lower risk of renal complications in HL versus a high risk of TLS-driven AKI in aggressive NHL.

The key mechanistic difference lies in the role of immunosuppression. The efficacy of rituximab in NHL comes with the cost of HBV reactivation, a potentially fatal complication that is not a typical feature of HL regimens (13, 15). This underscores a non-negotiable requirement for pre-emptive viral screening and prophylaxis in all NHL patients scheduled for anti-CD20 therapy. Conversely, the intensive multi-drug nature of BEACOPP explains its higher potential for direct organ injury compared to ABVD (10, 11).

Regarding renal toxicity, the biology of aggressive NHL itself is a major risk factor. The high proliferative rate and chemosensitivity of subtypes like DLBCL create a perfect storm for TLS, a phenomenon less commonly observed in the typically more structured spread of HL (25, 27). This necessitates a risk-stratified approach at diagnosis, where patients with aggressive NHL and high tumour burden receive aggressive hydration and prophylactic uric acid-lowering therapy from the outset.

Several risk modifiers cut across both toxicity types. Pre-existing organ dysfunction (CKD, liver disease) significantly increases vulnerability (29). Furthermore, the cumulative dose of nephrotoxic or hepatotoxic agents can lead to progressive damage over time, highlighting the need for long-term monitoring even after successful treatment.

Clinical Implications and Recommendations

For clinicians, this review supports tailored monitoring strategies:

Hepatic Monitoring: Mandatory HBV screening (HBsAg, anti-HBc) is essential before starting NHL regimens containing rituximab. Prophylactic antiviral therapy should be initiated in at-risk patients and continued post-treatment. Regular LFT monitoring is crucial for all patients.

Renal Monitoring: Assessment of TLS risk should be part of initial staging for NHL patients. High-risk patients require intensive monitoring of electrolytes, uric acid, and renal function, especially during the first cycles. Prophylaxis with

allopurinol or rasburicase and aggressive hydration is paramount.
Dose adjustments for renally excreted drugs like bendamustine are necessary in patients with baseline renal impairment (28).

Limitations and Future Directions
This review is limited by the heterogeneity of the included studies in terms of design, definitions of toxicity, and patient populations. Many studies focus on acute toxicities, while the long-term hepatic and renal consequences of chemotherapy in lymphoma survivors represent an important area for future research. Prospective studies with standardised toxicity criteria are needed to allow for more robust comparative meta-analyses. Future research should also focus on validating predictive biomarkers for organ injury and exploring the toxicity profiles of novel agents and immunotherapies.

Conclusion
Chemotherapy for HL and NHL, while highly effective, presents distinct challenges regarding hepatic and renal toxicities. HL regimens can cause direct liver and kidney injury, though severe cases are less common. In contrast, NHL regimens, particularly those containing rituximab, carry a significant risk of HBV reactivation and a higher susceptibility to TLS-induced AKI. A thorough understanding of these comparative toxicity profiles, coupled with vigilant, risk-adapted monitoring and proactive management strategies, is paramount for minimising treatment-related morbidity and improving overall outcomes for lymphoma patients.

List of Abbreviations
AKI: Acute Kidney Injury
ALP: Alkaline Phosphatase
ALT: Alanine Transaminase
AST: Aspartate Transaminase
BR: Bendamustine Rituximab
BUN: Blood Urea Nitrogen
CKD: Chronic Kidney Disease
eGFR: estimated Glomerular Filtration Rate
GGT: Gamma-Glutamyl Transferase
HBV: Hepatitis B Virus
HL: Hodgkin Lymphoma
INR: International Normalised Ratio
LFTs: Liver Function Tests
NHL: Non-Hodgkin Lymphoma
PT: Prothrombin Time
R-CVP: Rituximab, Cyclophosphamide, Vincristine, Prednisone
R-CHOP: Rituximab, Cyclophosphamide, Doxorubicin, Vincristine, Prednisone
TLS: Tumour Lysis Syndrome
VOD: Veno-occlusive Disease.

Declarations
Ethics Approval and Consent to Participate
Not applicable. This article is a literature review and does not report on original patient data.

Consent for Publication
All authors have reviewed the final manuscript and provided consent for its publication under the Creative Commons Attribution Non-Commercial 4.0 International License.

Availability of Data and Materials
All data and materials analysed in this review are available from the corresponding author upon reasonable request.

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Authors' contributions
ASHK: Conceptualisation, Writing - Original Draft, Writing - Review & Editing.
HAU: Writing - Original Draft, Methodology.
AAMA: Writing - Original Draft, Data Curation. All authors reviewed and approved the final manuscript.

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