SCHOLARLY PUBLICATIONS
School of Medical Sciences
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Journal Name: Cancers **IF: 4.8**

Title: Pathogenic Mutations in the Tumor Microenvironment Drive Tumor Progression in Diffuse Large B-Cell Lymphoma Through Tumor–Stroma Cross-Talk

Author: Aggarwal V.; Srinivasan R.; Bal A.; Malhotra P.; Varma S.; Das A.

Details: Volume 18, Issue 11, June 2026

Abstract: Background: Diffuse Large B-cell Lymphoma (DLBCL) is a biologically heterogeneous subtype of non-Hodgkin's lymphoma (NHL), accounting for 30–40% of cases worldwide. Despite the incorporation of rituximab into standard chemo-immunotherapy regimen, approximately one-third of patients present with relapsed or refractory disease, implicating the need for improved prognostic markers and therapeutic targets. Gene expression profiling successfully classified DLBCL into Germinal Center B-cell-like (GCB) and non-GCB subtypes, which differ in genetic alterations, response to therapy, and clinical outcome. Results: A total of 176 DLBCL patients were screened, of which 113 were enrolled based on availability of complete clinical data. The cohort demonstrated male predominance (male:female ratio: 2.1:1), advanced disease stage in 72.6% of patients, and elevated serum lactate dehydrogenase levels in 57.5%. Based on immunohistochemistry, 43.4% cases were classified as GCB-DLBCL and 56.6% as non-GCB DLBCL. Although the International Prognostic Index (IPI) retained prognostic significance for event-free survival (EFS) and overall survival (OS), considerable heterogeneity was observed within similar risk groups. Whole-exome sequencing (WES) uncovered recurrent somatic mutations in key oncogenic and epigenetic regulators, including TNFAIP3, NFIB, NOTCH1, TSC2, EZH2, EP300, KMT2D, and B2M, with subtype-specific distribution. Functional validation through primary cell culture demonstrated significantly elevated Th2 (IL-4, IL-6, IL-10) and Th17 (IL-17) cytokines in co-cultures containing both neoplastic cells and stromal components, underscoring the role of TME in DLBCL progression. Conclusions: Taken together, this study provides novel insights into stromal mutational signatures and cytokine-mediated tumor–stroma interactions, offering potential prognostic biomarkers and therapeutic targets for the improved management of DLBCL.

URL: https://www.mdpi.com/2072-6694/18/11/1697

SCHOLARLY PUBLICATIONS
School of Medical Science
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Journal Name: Journal of Biological Chemistry **IF: 4.1**

Title: Dysregulated HELLS expression alters cellular processes and serves as a potential prognostic marker in acute leukemia

Author: Madhulika, S; Panda, TS; Samal, P; Kumar, SS; Mohanty, S; Ghosh, M; Chakraborty, S; Das, A; Panda, J; Saha, S; Padhy, M; Mallick, TP; Mohapatra, PP; Prasad, P

Details: Volume-302, Issue-7, July 2026

Abstract: Spatiotemporal gene expression is regulated by the SNF2 family of ATPases that remodel chromatin, among which helicase lymphoid-specific (HELLS) play an essential role in DNA-templated processes. Analysis from cancer databases revealed HELLS upregulation in several cancers, including acute myeloid leukemia (AML). Using an in vitro myeloid differentiation model, we found that HELLS deficiency promotes myeloid differentiation, apoptosis, cell-cycle arrest, and chromatin instability. Furthermore, HELLS deficiency enhanced myeloid differentiation and apoptosis in HL-60 cells when treated with chemotherapy drugs, including 5-azacytidine, cytarabine, and doxorubicin. Epigenetically, loss of HELLS reduced active histone mark (H3K4me3) and enhanced repressive promoter (H3K27me3), active enhancer (H3K27ac) marks, and chromatin accessibility. Transcriptomic analysis of BeatAML data and bioinformatics analysis revealed that HELLS upregulation is associated with adverse prognosis, as defined by the ELN-2017 classification, and significantly poorer survival. Classification of AML patients into $HELLS^{High}$ and $HELLS^{Low}$ gene-expression groups showed that lower HELLS expression was associated with a favorable prognosis and improved remission. HELLS upregulation was positively and negatively associated with leukemic stem/progenitor cell (LSPC) markers and myeloid lineage markers, respectively. Ex vivo validation in AML patient-derived LSPCs demonstrated that HELLS deficiency reduces leukemic marker burden (CD123 and TIM-3) and increases apoptosis. Overall, we show HELLS upregulation drives leukemogenesis with poor outcomes, highlighting its potential as a prognostic marker and therapeutic target in AML.

URL: https://www.sciencedirect.com/science/article/pii/S002192582602082X

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URL: https://www.sciencedirect.com/science/article/pii/S002192582602082X?via%3Dihub

Journal Name: Journal of Clinical and Experimental Hepatology **IF: 3.4**

Title: Overlap Syndromes and Associated Immunoglobulin-G4-Related Liver Diseases

Author: Malakar S.; Kumar S.; Roy A.; Giri S.; Rungta S.; Agarwal M.; Singh A.; Bassi N.; Samanta A.; Ghoshal U.C.; Singh V.

Details: Volume 16, Issue 4, July-August 2026

Abstract: Overlap syndromes (OS) are mixed hepatitic-cholestatic variants of autoimmune liver diseases (AILD), characterized by features of autoimmune hepatitis (AIH), bile duct injury, and circulating autoantibodies related to primary biliary cholangitis (PBC) or primary sclerosing cholangitis (PSC). Few cases of PBC-PSC have also been reported. Following the recognition of immunoglobulin G4-related diseases (IgG4-RD), IgG4-hepatopathy, IgG4-AIH, and PSC-with high serum IgG4-related disorders are being increasingly identified. Patients with this group of disorders often present like OS. High serum IgG4 levels in patients with PSC have been associated with worse overall survival. Hence, treatment and prognosis may differ between patients with IgG4-AIH and those with IgG4-hepatopathy. Early recognition and management of OS and related cholestatic diseases may improve outcomes and extend transplant-free survival. This review aims to formulate strategies for the diagnosis and management of OS based on current evidence. It also delves into the intricate relationship between the IgG4-related disease and AILD.

URL: https://www.sciencedirect.com/science/article/pii/S0973688326000964?via%3Dihub

SCHOLARLY PUBLICATIONS
School of Medical Sciences
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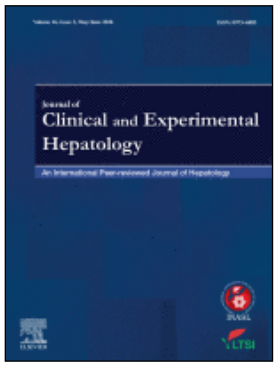
Journal Name: Journal of Clinical and Experimental Hepatology **IF: 3.4**

Title: Clip-Assisted Cyanoacrylate Injection for the Management of a Proximal Colonic Varix

Author: Giri S.; Maheswari S.; Praharaj D.L.; Das S.; Sahu M.K.

Details: Volume 16, Issue 4, July-August 2026

Abstract: Colonic varices are a rare manifestation of portal hypertension and an uncommon cause of lower gastrointestinal bleeding, particularly when involving the proximal colon. We report a case of bleeding hepatic flexure varices in a 23-year-old male with extrahepatic portal vein obstruction (EHPVO) and prior splenectomy with lienorenal shunt. The patient presented with recurrent hematochezia and severe anemia. Upper gastrointestinal endoscopy was negative for a bleeding source, while colonoscopy revealed large tortuous varices at the hepatic flexure with stigmata of recent hemorrhage. Contrast-enhanced CT confirmed cavernomatous transformation of the portal vein with extensive mesenteric collaterals, thrombosis of the prior shunt, and afferents to the varix arising from the superior mesenteric vein. Endoscopic therapy was performed using clip-assisted flow modulation of the afferent and efferent venous channels, followed by N-butyl-2-cyanoacrylate injection into the main variceal trunk. The procedure resulted in successful obliteration of the varices without complications, with no further bleeding on follow-up. This case highlights hepatic flexure varices as a rare cause of lower gastrointestinal bleeding in EHPVO and demonstrates the feasibility of clip-assisted cyanoacrylate injection as an effective endoscopic treatment strategy for high-flow colonic varices.



URL: https://www.sciencedirect.com/science/article/pii/S0973688326000927?via%3Dihub