

Conference Abstracts

Vol. 6, Issue Supplement 2, 2026

Myeloid Malignancy Risk in Large Vessel Vasculitis: A Propensity Score-Matched Analysis

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Keywords: Clonal Hematopoiesis, Myeloid Neoplasm, Myelodysplastic syndrome, Myeloproliferative neoplasm

Submission received: 2026-08-15 / Accepted: 2026-08-25 / Published: 2026-09-28

<https://doi.org/10.53876/001a.129831>

Abstract

Background

Recent evidence suggests several inflammatory and autoimmune diseases, including large vessel vasculitis (LVV) - giant cell arteritis (GCA) and Takayasu arteritis (TAK) - may be associated with clonal hematopoiesis of indeterminate potential (CHIP). Smaller studies also link inflammatory disorders to myeloid neoplasms (MNs), but the risk of incident hematologic malignancy following LVV diagnosis remains poorly characterized.

Methods

Using the TriNetX US Collaborative Network, we identified adults with LVV (GCA, TAK; 2005–2025) and no prior malignancy, indexed at first LVV diagnosis. Seborrhic keratosis (SK) patients served as controls, given comparable healthcare engagement without inflammatory, autoimmune, or hematologic burden- an established comparator group used in similar real-world studies. Cohorts were propensity-matched 1:1 on age, sex, race/ethnicity, comorbidities, and immunosuppressive medications. Incident outcomes included any hematologic malignancy, lymphoma, leukemia, myelodysplastic syndrome (MDS), and myeloproliferative neoplasm (MPN),

assessed via Kaplan-Meier analysis and hazard ratios (HR).

Results

After matching, 40,355 LVV patients and 40,355 SK controls were analyzed (mean age 68.5 years; 70.8% female; median follow-up 1,277 days). LVV was associated with increased hematologic malignancy risk overall (HR 1.272, 95% CI 1.146-1.412, p=0.004), driven mainly by MDS (HR 1.604, 95% CI 1.234-2.086, p<0.001) and MPN (HR 1.285, 95% CI 1.061-1.556, p=0.006); lymphoma (HR 1.153, p=0.117) and leukemia (HR 1.158, p=0.695) were not significantly elevated.

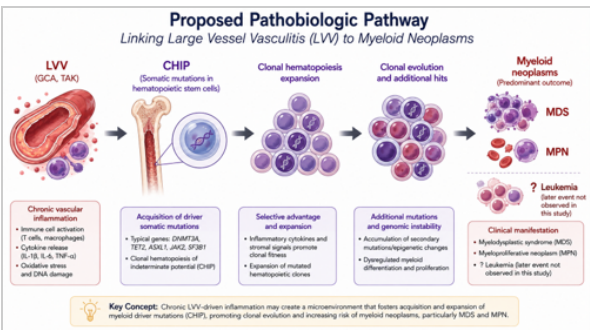


Figure 1. Proposed Pathobiologic Pathway Linking Large Vessel Vasculitis (LVV) to Myeloid Neoplasms

Conclusion

LVV is independently associated with increased hematologic malignancy risk, primarily driven by

chronic myeloid neoplasms. This myeloid predominance suggests a shared origin, with CHIP a plausible link between LVV-driven inflammation and myeloid clonal evolution. The absent leukemia signal may reflect limited follow-up, as transformation occurs over years

and warrants longer-term study. Clinically, findings support early hematology referral, CHIP screening in high-risk rheumatology patients, and structured surveillance at specialized CHIP clinics.

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