

THE PORTAL HYPERTENSIVE PHENOTYPE OF SPLANCHNIC VEIN THROMBOSIS IN MYELOPROLIFERATIVE NEOPLASMS: A NATIONAL INPATIENT SAMPLE ANALYSIS, 2022–2023

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INTRODUCTION

Myeloproliferative neoplasms (MPNs) are strongly associated with unusual-site venous thrombosis, including splanchnic vein thrombosis (SVT). SVT encompasses portal vein thrombosis, Budd-Chiari syndrome, and mesenteric or splenic vein thrombosis.

MPN-associated SVT is clinically important because morbidity may extend beyond thrombosis itself to include portal hypertension, ascites, varices, variceal bleeding, hepatic encephalopathy, splenomegaly, and anticoagulation–bleeding complexity.

Knowledge gap: National inpatient data characterizing the portal hypertensive phenotype and outcomes of MPN-associated SVT remain limited.

AIM

To compare portal hypertensive complications, anticoagulation documentation, and discharge disposition between hospitalized adults with MPN-associated SVT and MPN hospitalizations without SVT, with exploratory pooled analysis of in-hospital mortality.

METHODS

- Design: Retrospective NIS analysis; primary cohort = coding-consistent 2022–2023 era
- Validation: A sharp D47.3 discontinuity at the FY2022 boundary coincided with introduction of D75.838/D75.839
- Population/exposure: Adults with MPN; primary SVT = I81 or I82.0
- Analysis: Survey-weighted logistic regression adjusted for demographics, hospital factors, MPN subtype, and cirrhosis; pooled and broad-definition analyses assessed robustness

CONCLUSIONS

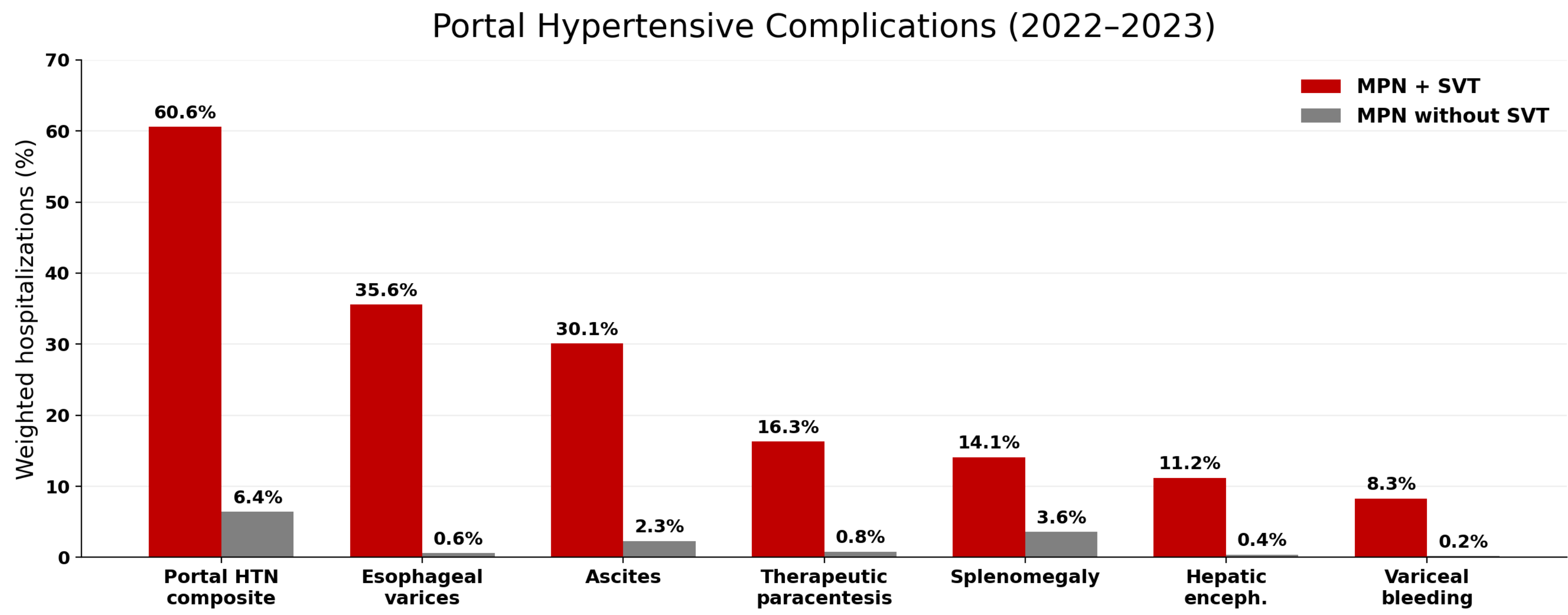
Concurrent SVT identified a portal hypertension–dominant inpatient phenotype: the portal hypertension composite occurred in 60.6% versus 6.4% and remained strongly associated with SVT after adjustment (aOR 10.35, 95% CI 7.59–14.12). Esophageal varices showed the strongest individual association (aOR 28.37).

The SVT association was stable across coding eras (interaction P = 0.70), persisted after excluding cirrhosis/chronic liver disease (aOR 13.60), and was robust across exposure definitions. Severe sepsis, thrombocytopenia, and discharge to home/home health were not independently associated with SVT.

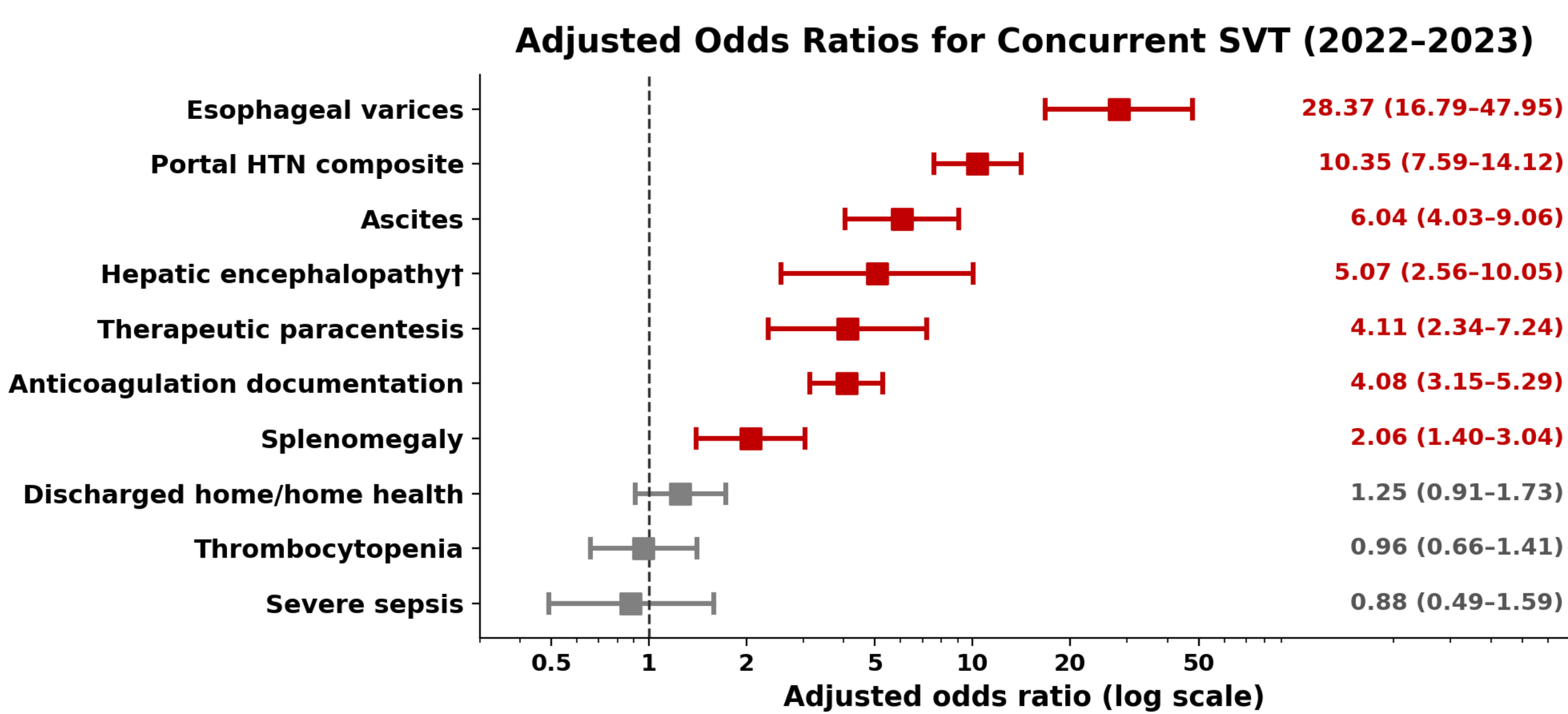
These findings support focused evaluation for portal hypertensive complications and bleeding–anticoagulation complexity, with hematology–hepatology co-management when clinically indicated.

RESULTS

18,595 unweighted hospitalizations represented 92,975 weighted MPN hospitalizations; 1,560 weighted hospitalizations (1.68%) had SVT.



* P < 0.001 for all comparisons; primary coding-consistent cohort, 2022–2023 NIS



Adjusted for age, sex, payer, income quartile, year, hospital teaching status/bed size, MPN subtype, and cirrhosis/chronic liver disease. †Sparse events; estimate stable in parsimonious model (aOR 5.09). Differential ascertainment (SVT-triggered imaging/endoscopy) may contribute to effect magnitude.

Table 1. Portal hypertension composite — robustness across specifications

Analysis	aOR	95% CI	P
Primary: coding-consistent 2022–2023 cohort	10.35	7.59–14.12	<0.001
Excluding cirrhosis/chronic liver disease	13.60	9.75–18.96	<0.001
Excluding D47.1/chronic MPN NOS	11.24	7.91–15.95	<0.001
Broad SVT definition (+I82.890/I82.891)	9.58	7.12–12.88	<0.001
Pooled 2020–2023 (sensitivity)	10.63	8.69–13.00	<0.001

Table 2. Secondary outcomes (2022–2023)

Outcome	MPN + SVT (%)	MPN without SVT (%)	aOR (95% CI)	P
Long-term anticoagulation documentation	43.9	21.7	4.08 (3.15–5.29)	<0.001
Discharged home or home health	82.4	70.3	1.25 (0.91–1.73)	0.175
Thrombocytopenia	12.5	8.8	0.96 (0.66–1.41)	0.844
Severe sepsis	4.8	6.2	0.88 (0.49–1.59)	0.674

Pooled exploratory mortality: aOR 0.91 (95% CI 0.61–1.35); no significant difference, imprecise. Z79.01 reflects documentation of long-term anticoagulant use, not actual inpatient treatment.

CONTACT INFORMATION

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