Cardiovascular Comorbidities and Thrombotic Risk in Essential Thrombocythemia: A 152-Patient Cohort

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Introduction

Background: Thrombotic complications are the leading cause of morbidity and mortality in Essential Thrombocythemia (ET). While the IPSET-thrombosis score incorporates age, prior thrombosis, cardiovascular (CV) risk factors, and JAK2V617F status, the individual contribution of specific CV comorbidities — particularly hyperlipidemia and hypertension — to thrombotic risk remains incompletely defined in real-world populations (1).

Aim

To comprehensively compare the clinical profiles of ET patients with and without thrombosis, with particular emphasis on cardiovascular risk burden and its contribution beyond conventional IPSET-thrombosis scoring.

Method

Study Design: Retrospective, single-center cohort study Population: 152 WHO-defined ET patients; median follow-up 8.6 years (range 0.0–30.0)
Groups: Thrombotic (n=30) vs. Non-thrombotic (n=122)
Compared parameters: Demographics · Laboratory values · Driver mutation status (JAK2/CALR/MPL) · CV comorbidities · IPSET-thrombosis risk category · Cytoreductive treatment · Disease outcomes
Statistics: Chi-square test (categorical); Mann-Whitney U test (continuous, non-normal); $p<0.05$ significant, Kaplan–Meier Survival Analysis test (log-rank p=0.001).

Conclusions

In this real-world cohort of 152 patients with Essential Thrombocythemia (ET), thrombosis was associated with older age, JAK2V617F positivity, a higher burden of cardiovascular comorbidities, and significantly inferior overall survival. Cerebrovascular events were the most common thrombotic manifestation, with vital organ involvement occurring in half of all events. These findings are consistent with the IPSET-thrombosis model, which recognizes age, JAK2V617F mutation, previous thrombosis, and cardiovascular risk factors as major determinants of thrombotic risk (1). Similarly, Carobbio et al. demonstrated that conventional cardiovascular risk factors significantly contribute to arterial thrombosis in ET (2). Our results further emphasize that optimizing modifiable cardiovascular risk factors should complement disease-specific risk stratification to reduce thrombotic complications and improve long-term outcomes.

Results

Thrombosis occurred in 30/152 patients (19.7%) 73.3% had a single event; 26.7% had ≥2 events. Cerebrovascular events were the most common manifestation (36.7%); vital organ involvement occurred in 50.0%. 30.0% of events occurred despite cytoreductive therapy. Thrombotic patients were older (68.1 vs. 59.4 years; p=0.008) and more often JAK2V617F-positive (63.3% vs. 42.2%; p=0.058). CV comorbidity burden was significantly higher in the thrombotic group — hyperlipidemia (36.7% vs. 18.9%; p=0.048) and a strong hypertension trend (63.3% vs. 44.4%; p=0.090). IPSET-thrombosis high-risk category: 90.0% (thrombotic) vs. 16.7% (non-thrombotic); p<0.001. All-cause mortality was over 4-fold higher with thrombosis (40.0% vs. 8.9%; p<0.001).

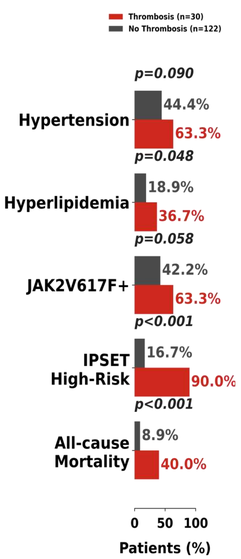
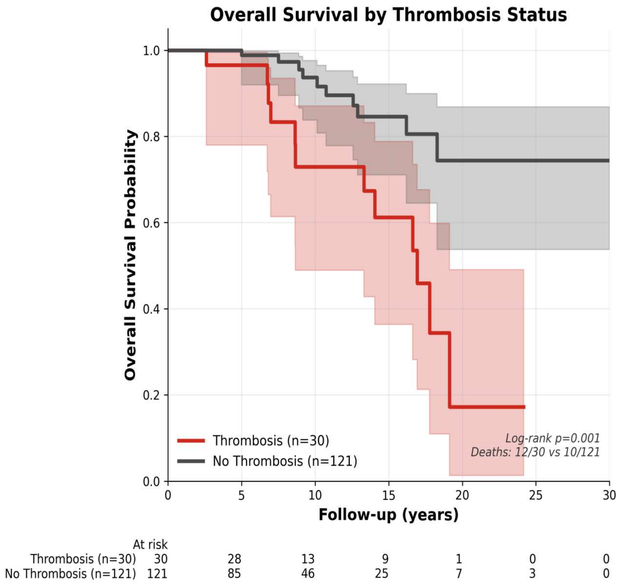


Figure 1. Key clinical variables by thrombosis status (%).



Computed from patient-level data (ET_1.sav), n=151 (1 record excluded: impossible dates).
For 1 patient with recorded death but missing death date, last-known-contact date was used as an approximation.

Variable	Thrombosis (n=30)	No Thrombosis (n=122)	p-value
Age, mean ± SD, years	68.1 ± 14.8	59.4 ± 15.1	0.008
Male sex, n (%)	12 (40.0%)	31 (25.4%)	0.118
JAK2V617F+, n (%)	19 (63.3%)	38 (42.2%)	0.058
Hypertension, n (%)	19 (63.3%)	40 (44.4%)	0.090
Hyperlipidemia, n (%)	11 (36.7%)	17 (18.9%)	0.048
IPSET high-risk, n (%)	27 (90.0%)	15 (16.7%)	<0.001
Antiplatelet/anticoag. use, n (%)	11 (36.7%)	65 (72.2%)	<0.001
All-cause mortality, n (%)	12 (40.0%)	8 (8.9%)	<0.001
Cerebrovascular event, n (%) — thrombotic grp	11 (36.7%)	—	—

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Contact

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