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HISTORY OF CANCER IN PATIENTS WITH SEVERE HEREDITARY THROMBOPHILIA VERSUS NO THROMBOPHILIA: A SINGLE-CENTER COHORT OF PATIENTS WITH VENOUS THROMBOSIS

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Introduction. Hypercoagulability is a hallmark of cancer and may contribute to tumor progression. Severe hereditary thrombophilia represents a lifelong prothrombotic condition, but its association with cancer remains unclear.

Aim. To compare the prevalence of cancer in patients with severe hereditary thrombophilia versus patients without thrombophilia.

Methods. We conducted a single-center observational study including consecutive patients with venous thrombosis. Patients were classified as having severe hereditary thrombophilia or no thrombophilia based on standardized testing. The primary outcome was history of cancer (ever cancer), defined as any solid or hematological malignancy diagnosed at any time. Age at thrombosis was used as age at study inclusion. Cancer prevalence was compared using Poisson regression models with log link and robust standard errors to estimate prevalence ratios (PR), adjusted for age and sex.

Results. A total of 400 patients were included (200 with severe hereditary thrombophilia and 200 without) (Tab 1). Cancer prevalence was similar between groups (11.0% vs 12.5%). In multivariable regression adjusted for age and sex, severe hereditary thrombophilia was not associated with a higher prevalence of cancer (PR 1.33, 95%CI 0.67–2.58, p=0.403). Increasing age was independently associated with cancer prevalence (PR per year 1.05, 95%CI 1.03–1.07, p<0.001), while male sex was associated with a lower prevalence of cancer (PR 0.51, 95%CI 0.26–0.94, p=0.039). Thrombophilia-by-sex interaction was not significant (p=0.712). Among patients with cancer, those with severe thrombophilia were diagnosed at a younger age (58 [IQR 46–67] y) vs. patients without (64.5 [IQR 54–73] y) in univariable analysis. However, in exploratory multivariable linear regression adjusting for sex and age at thrombosis, severe thrombophilia was not independently associated with age at cancer diagnosis (β −3.9 years, p=0.31). In sensitivity analyses, thrombophilia subtypes were grouped into defects of natural anticoagulants and other thrombophilias. No independent association with cancer prevalence was observed.

Conclusions. Severe hereditary thrombophilia was not associated with an increased prevalence of cancer. Age was the main determinant of cancer burden, and the apparent younger age at cancer diagnosis among thrombophilic patients was largely explained by baseline age differences. These findings do not support intensified cancer screening based solely on thrombophilia status.

Table 1 Characteristics of the study population

Characteristic	Overall (n = 400)	No thrombophilia (n = 200)	Severe thrombophilia (n = 200)
Age, median (IQR), years	53.0 (39.0–67.0)	58.0 (46.0–73.0)	47.0 (33.0–60.0)
Male sex, n (%)	217 (54.2%)	116 (58.0%)	103 (51.5%)
History of cancer, n (%)	47 (11.8%)	25 (12.5%)	22 (11.0%)
Age at cancer diagnosis, median (IQR), years	61.0 (50.5–71.0)	64.5 (54.0–73.0)	58.0 (47.0–66.8)
Type of thrombophilia, n (%)	—	—	—
Natural anticoagulant deficiencies (PC, PS, Antithrombin)	—	—	150 (75.0%)
Factor V Leiden/PT20210A homozygous	—	—	18 (9.0%)
Combined thrombophilia	—	—	32 (16.0%)

PC: protein C; PS: protein S; PT: prothrombin variant