

High rates of venous thromboembolism in endometrial cancer patients requiring systemic therapy: an opportunity for risk assessment and intervention

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HIGHLIGHTS

- 26.7% of endometrial cancer patients requiring upfront chemotherapy developed VTE.
- Cancer stage was the only clinicopathologic factor associated with subsequent VTE.
- Khorana score was not associated with VTE development.
- Risk assessment tools to evaluate VTE prophylaxis in endometrial cancer are needed.

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ABSTRACT

Objectives. We aim to describe the venous thromboembolism (VTE) rate among patients who received chemotherapy for initial endometrial cancer treatment, and identify factors associated with VTE diagnosis in this population.

Methods. This is a retrospective cohort study of patients who received chemotherapy during initial treatment for endometrial cancer from January 2015 to December 2023 within a single academic medical system. The primary outcome is overall VTE incidence. Sociodemographic and biomedical factors were collected. Descriptive statistics were used for analysis.

Results. Two hundred ninety-two patients were identified who received chemotherapy during initial treatment for endometrial cancer; 78 (26.7%) developed a VTE after cancer diagnosis. Cancer stage was associated with VTE development ($p < 0.001$); 50.0% of patients who developed VTE had Stage IV at diagnosis compared to 20.4% of those who did not. Twenty-four patients (30.8%) had carcinomatosis at the time of VTE diagnosis and 14 (17.9%) had ascites. A high-risk Khorana score (≥ 3) at initiation of initial chemotherapy, histology, grade, age, ECOG functional status, tobacco use, body mass index, and receipt of external beam pelvic radiation, immunotherapy, or bevacizumab during initial treatment were not associated with VTE development.

Discussion. More than 1 in 4 patients who received chemotherapy for initial endometrial cancer developed a VTE during their treatment course. Stage at cancer diagnosis was the only factor associated with subsequent VTE; factors including histology, grade, BMI and Khorana score were not associated. As endometrial cancer incidence rises, continued efforts to improve the accuracy of risk assessment tools and evaluate prophylactic anticoagulation in this high-risk population are warranted.

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1. Introduction

Thromboembolic events, including deep vein thrombosis (DVT) and pulmonary embolism (PE), are among the most serious complications affecting individuals with cancer; venous thromboembolism (VTE) contributes to treatment delays and increased healthcare burden, and

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represents the second leading cause of mortality among cancer patients [1]. Several studies have isolated chemotherapy itself as a factor associated with thrombotic events [1,2].

Reported incidence estimates of VTE in cancer patients vary widely, and in some studies reach as high as 40%. These rates are driven by cancer site, heterogeneity in treatment modalities, data abstraction methods and follow-up window [3–8]. Among the gynecologic malignancies, VTE risk in patients receiving chemotherapy has been most extensively documented in ovarian cancer. This risk is most pronounced in patients with high tumor burden and those receiving neoadjuvant chemotherapy, with recent studies consistently confirming elevated VTE rates in this population [9–11]. In response to reported rates of 10.8–42%, the evidence base supporting VTE prophylaxis in ovarian cancer has grown substantially [12–15]. A comprehensive systematic review and meta-analysis confirmed that ovarian cancer patients receiving chemotherapy benefit significantly from VTE prophylaxis, with relative risk reductions of 30–50% observed across multiple studies [14,15].

Despite the well-documented risk of VTE and benefit of prophylactic anticoagulation in ovarian cancer patients, a significant knowledge gap exists in the risk of VTE attributable to chemotherapy treatment in endometrial cancer patients, despite this being the most common gynecologic malignancy and one of few cancers with rising mortality rates [16]. Emerging data suggest that the risk of thrombotic events associated with advanced endometrial cancer may be comparable to that of ovarian cancer [6]. Published reports describe VTE rates in endometrial cancer patients ranging from 0.8% to 27%, with the highest rates observed among patients with high-grade tumors or advanced-stage disease, or in those receiving chemotherapy [6,7]. Most of the existing endometrial cancer-specific literature is anchored in the perioperative setting, lacks uniform treatment exposures, or originates from population-based database cohorts without sufficient granularity to characterize patient risk factors, chemotherapy regimen or timing of treatment relative to VTE [7,17,18]. A recent single-institution retrospective study reported VTE rates as high as 27% among patients with advanced endometrial cancer receiving chemotherapy, particularly during early treatment phases [19]. In this and other studies, the Khorana score, which is used in multiple clinical risk assessment guidelines, has failed to show utility in predicting VTE [20].

The epidemiology of endometrial cancer reveals important demographic considerations that may influence VTE risk. Black women are disproportionately likely to present with later-stage disease and more aggressive histologic subtypes, often requiring neoadjuvant chemotherapy, all of which are established risk factors for highly morbid thrombotic events [21,22]. Importantly, the Khorana risk scores dramatically underestimated VTE incidence in this population [23]. This overall mortality and population-specific risk profile underscores the need for tailored approaches to VTE prevention in endometrial cancer care.

Given the substantial risk of VTE in advanced endometrial cancer and the demonstrated benefit of prophylactic anticoagulation in other gynecologic malignancies, there is a pressing need for data to guide decision-making in this understudied population. This study aims to address the current knowledge gap by characterizing VTE rates among endometrial cancer patients receiving chemotherapy and identifying timing and clinical factors associated with VTE development to improve risk stratification and inform future studies on prophylactic anticoagulation in this high-risk population.

2. Methods

2.1. Study design

This retrospective cohort study was conducted at a multi-hospital urban academic healthcare system in the southeastern United States. It was approved by the system's Institutional Review Board. The requirement for informed consent was waived due to the retrospective

nature of the study. The primary outcome of the study was the incidence of venous thromboembolism (VTE), at any point in their cancer care, in patients who received chemotherapy as a component of their initial treatment for endometrial cancer. Secondary outcomes included the incidence of VTE in the neoadjuvant and adjuvant settings, in patients with advanced stage disease, and in patients receiving whole pelvic external beam radiation therapy (EBRT), immunotherapy and bevacizumab in addition to cytotoxic chemotherapy as initial treatment. Additionally, associations with clinicodemographic factors including body mass index (BMI), tobacco use, ECOG functional status, tumor histology and grade, and Khorana score at chemotherapy initiation were evaluated. The treatment course was divided into time periods: pre-treatment (date of cancer diagnosis to initiation of cancer treatment, either surgery or first chemotherapy infusion), neoadjuvant chemotherapy (first chemotherapy infusion to date of surgery), post-operative (date of surgery to 30 days post-surgery), adjuvant chemotherapy (>30 days post-surgery or from first post-operative chemotherapy infusion, whichever was later, through 21 days after final chemotherapy infusion), surveillance (>21 days after final chemotherapy infusion through date of diagnosis of recurrence or last documented encounter if no recurrence), at or during treatment for recurrence (date of diagnosis of recurrence through last documented encounter).

2.2. Study population

Patients over age 18 with endometrial cancer who received chemotherapy as a component of their initial endometrial cancer treatment were included in this study. Patients began endometrial cancer treatment between January 1, 2015 and December 31, 2023 at our institution; data were collected from the entire course of cancer treatment. Initial chemotherapy could have been neoadjuvant or adjuvant; receipt of subsequent lines of chemotherapy for recurrence or progression was noted but not considered part of the inclusion criteria. Patients who did not receive chemotherapy as part of their initial treatment, regardless of subsequent receipt of chemotherapy for recurrence, were excluded. Additionally, patients treated for non-endometrial cancer or multiple cancers simultaneously, or at an outside institution, were excluded.

2.3. Data collection

Patients were identified via review of institutional pharmacy records. All patients receiving any combination of carboplatin, cisplatin, docetaxel, liposomal doxorubicin, paclitaxel or nab-paclitaxel associated with C54 (malignant neoplasm of corpus uteri) or C55 (malignant neoplasm of uterus, part unspecified) ICD-10 diagnostic codes were identified. Electronic medical records were examined for each identified patient; those not meeting inclusion criteria were excluded. Relevant data, including treatment course, sociodemographic characteristics and oncologic factors, were abstracted for those meeting inclusion criteria.

2.4. Statistical analyses

Statistical analyses were completed using SPSS (Version 29.0.2.0, IBM Corporation, Armonk, NY). A univariate analysis was completed. Chi-square and Fisher's Exact tests were used as appropriate to compare categorical variables while Mann-Whitney *U* tests were used to compare continuous variables. A *p*-value less than 0.05 was used for statistical significance.

3. Results

3.1. Patients

Six hundred fifty-six patients were initially identified as described above; 292 ultimately met inclusion criteria and were included in

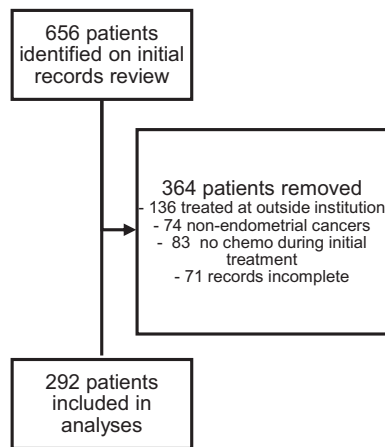


Fig. 1. Patients meeting inclusion criteria and included in analyses.

analyses (Fig. 1). The median age at cancer diagnosis of patients included was 66.1 (IQR 61.2–70.9) years (Table 1). The majority of patients were Black (65.4%); white patients made up 27%, Asian patients made up 4.4% and other/mixed racial groups made up 3.1% of the population. Median BMI was 31.9 (IQR 27.1–38.2) kg/m². Median follow up time was 23.9 (IQR 13.9–41.9) months.

Among the entire study population, Stage I disease was the most common (32.2%, $n = 94$), although over half had advanced disease at diagnosis (Stage III 26.0%, $n = 76$ and IV 28.1%, $n = 82$). Serous histology was the most common (38.4%, $n = 112$) followed by endometrioid (22.6%, $n = 66$) and carcinosarcoma (18.8%, $n = 55$) (Table 1). In addition to cytotoxic chemotherapy, initial treatment included EBRT for 28.4% of patients, immunotherapy for 9.2% and bevacizumab for 1.4% (Table 1). At the time of chemotherapy initiation for initial treatment, 65.1% of patients had a Khorana score less than 3 (Table 1).

3.2. VTE incidence, timing and associated clinical factors

Of the 292 patients included in our cohort, 78 (26.7%) were diagnosed with VTE at the time of their cancer diagnosis or during subsequent care (Table 1). Median time from cancer diagnosis to VTE diagnosis was 7.1 (IQR 3.7–15.5) months. Ten percent of VTE diagnoses were made concurrent with the cancer diagnosis or between diagnosis and treatment initiation; 21% while on neoadjuvant chemotherapy; 4% ≤ 30 days after surgery, 27% on adjuvant chemotherapy, 14% in surveillance, and 24% at or during treatment for a recurrence (Fig. 2). For those diagnosed with VTE in recurrence, median time from recurrence to VTE was 4.8 months (IQR 2.9–12.1 months).

Cancer stage (FIGO 2009 staging, [24]) at diagnosis was significantly associated with VTE diagnosis: 50.0% of those who developed VTE had Stage IV disease, compared to 20.4% of those who did not develop VTE ($p < 0.001$, Table 1). Earlier stages followed the opposite pattern: Stage I made up 16.7% of those with VTE versus 38.4% of those without, Stage II 7.7% versus 9.0% and Stage III 17.9% versus 29.4% ($p < 0.001$, Table 1).

Clinicodemographic factors including median age ($p = 0.69$), median BMI ($p = 0.47$), tobacco use ($p = 0.93$), ECOG functional status ($p = 0.96$), tumor histology ($p = 0.96$), non-endometrioid histology ($p = 0.51$), and tumor grade ($p = 0.28$) were not associated with VTE diagnosis in this cohort (Table 1). Receipt of EBRT ($p = 0.96$), immunotherapy ($p = 0.20$), or bevacizumab ($p = 1.00$) as a component of initial treatment was not associated with increased VTE rates (Table 1). Rates of high-risk Khorana score (≥ 3) at chemotherapy initiation were similar between those who developed VTE (37.2%) and those who did not (30.4%) ($p = 0.37$, Table 1).

3.3. Cancer distribution at VTE diagnosis

The distribution of disease at the time of VTE diagnosis is shown in Table 2. The most common sites of disease were uterus (38.5%), carcinomatosis (30.8%) and other distant sites (34.6%); 17.9% had ascites (Table 2). Notably, 28.2% had no evidence of disease at the time of VTE diagnosis. Half of the patients without evidence of disease at the time of VTE diagnosis were receiving adjuvant chemotherapy, and the remainder were in surveillance.

4. Discussion

In our study, more than one out of every four patients (26.7%) diagnosed with endometrial cancer who received chemotherapy as part of their initial treatment were diagnosed with a VTE at some point during their cancer treatment course. This is higher than prior studies with reported VTE incidence rates of 0.8–22%, although these studies were not limited to those receiving chemotherapy [4,6,7,15]. Notably, however, Llanora et al. recently reported a VTE rate of 27.4% (95% CI, 18.8–38.0%) in patients who received either neoadjuvant or adjuvant chemotherapy, similar to our results [19].

It is well known that cancer induces a prothrombotic state, in part due to inflammatory factors such as tissue factor, interleukins, and other inflammatory cytokines, however chemotherapy itself is a significant risk factor for VTE development through direct drug-induced damage to endothelium and activation of procoagulant factors [7,25]. Khorana et al. reported a 47% increase in the rate of VTE in admitted patients receiving chemotherapy for their cancer treatment (3.9 vs 5.7%, $p < 0.0001$), with others noting that the highest incidence occurs just after treatment initiation [1,26]. When evaluated as an independent risk factor, chemotherapy increases a patient's risk of VTE with an odds ratio of 6.5 (95% CI, 2.1–20.2) compared to 4.1 (95% CI, 1.9–8.5) in those with cancer but not receiving chemotherapy [2]. Although we did not evaluate VTE rates in patients who did not receive chemotherapy as part of their initial treatment course, the relatively high rates of VTE in our patients and in the cohort reported by Llanora et al., combined with prior data suggesting that chemotherapy itself is a risk factor for VTE, emphasize the importance of maintaining high suspicion for VTE and potential standardization of prophylaxis in endometrial cancer patients receiving chemotherapy [19].

While the data are more limited, VTE rates in patients receiving immunotherapy appear to be similar to rates in patients receiving cytotoxic chemotherapy, and the combination may result in a higher VTE rate than cytotoxic therapy alone [27,28]. We did not find any association between receipt of immunotherapy and VTE diagnosis, however only 9.2% of patients included in our study received immunotherapy with their initial cytotoxic chemotherapy as the majority began their treatment prior to the widespread adoption of immunotherapy into the initial treatment paradigm for advanced endometrial cancer. With the increased use of immunotherapy in endometrial cancer treatment, monitoring for increased VTE risk will be critical.

Prior studies have shown patient age, higher stage, elevated CA-125, and non-endometrioid histology as risk factors for VTE in endometrial cancer patients; in our analysis stage was the only factor associated with a diagnosis of a VTE ($p < 0.001$) [6,29]. Ascites at cancer diagnosis has been identified as a risk factor for VTE development in ovarian cancer [30]. Tumor burden and distribution of disease is thought to influence VTE risk in solid tumors, however the precise risk is difficult to elucidate as other factors, including chemotherapy, surgical status, and patient comorbidities, confound this data [31]. Among patients in our cohort with VTE, 17.9% had ascites, 30.8% had carcinomatosis, and 34.6% had other distant disease at the time of VTE diagnosis.

The majority of patients in our study had high grade histology, as expected based on our inclusion criteria, which may limit our ability to evaluate the role tumor grade has in predisposing patients to VTE. However, while serous was the most common histology in our cohort, other

Table 1

Sociodemographic and clinical characteristics of included patients with and without VTE. Percentages may not total 100 due to rounding.

	Overall population, n (%) ^a	VTE, n (%) ^a	no VTE, n (%) ^a	p-value
All patients	292 (100)	78 (26.7)	214 (73.3)	
Age median (IQR), years	66.1 (61.2–70.9)	66.0 (60.9–70.1)	66.3 (61.2–71.2)	0.69
Race				
Black	191 (65.4)	51 (65.3)	140 (65.4)	
White	79 (27.0)	21 (26.9)	58 (27.1)	
Asian	13 (4.5)	2 (2.6)	11 (5.1)	
Other	9 (3.1)	4 (5.1)	5 (2.3)	
BMI median (IQR) kg/m2	31.9 (27.1–38.2)	32.2 (26.8–39.9)	31.9 (27.2–37.5)	0.47
ECOG performance score				0.96
0	113 (38.7)	34 (43.6)	79 (36.9)	
1	17 (5.8)	4 (5.1)	13 (6.1)	
2	7 (2.4)	2 (2.6)	5 (2.3)	
3	2 (0.7)	0 (0)	2 (0.9)	
4	0 (0)	0 (0)	0 (0)	
Not recorded	153 (52.4)	38 (48.7)	115 (53.7)	
Tobacco use				0.93
Never	231 (79.1)	63 (80.8)	168 (78.5)	
Previous	50 (17.1)	12 (15.4)	38 (17.8)	
Current	11 (3.8)	3 (3.8)	8 (3.7)	
Stage (FIGO 2009)				<0.001
I	94 (32.2)	13 (16.7)	83 (38.8)	
II	25 (8.6)	6 (7.7)	19 (8.9)	
III	76 (26.0)	14 (17.9)	63 (29.4)	
IV	82 (28.1)	39 (50.0)	43 (20.0)	
Unstaged	12 (4.1)	6 (7.7)	6 (2.8)	
Histology				0.96
Endometrioid	66 (22.6)	20 (25.6)	47 (22.0)	
Serous	112 (38.4)	28 (35.9)	85 (39.7)	
Clear cell	19 (6.5)	6 (7.7)	13 (6.1)	
Un/dedifferentiated	8 (2.7)	2 (2.6)	6 (2.8)	
Mixed	25 (8.6)	5 (6.4)	20 (9.3)	
Carcinosarcoma	55 (18.8)	16 (20.5)	40 (18.7)	
Other	4 (1.4)	1 (1.3)	3 (1.4)	
Grade				0.28
1–2	41 (14.0)	14 (17.9)	27 (12.6)	
3	251 (86.0)	64 (82.1)	187 (87.4)	
Radiation				0.96
None or VBT alone	209 (71.6)	56 (71.8)	153 (71.5)	
EBRT +/- VBT	83 (28.4)	22 (28.2)	61 (28.5)	
Immunotherapy				0.20
Yes	27 (9.2)	10 (12.8)	17 (7.9)	
No	265 (90.8)	68 (87.2)	197 (92.1)	
Bevacizumab				1.00
Yes	4 (1.4)	1 (1.3)	3 (1.4)	
No	288 (98.6)	77 (98.7)	211 (98.6)	
Khorana score at chemotherapy initiation				0.37
<3	190 (65.1)	49 (62.8)	141 (65.9)	
≥3	94 (32.2)	29 (37.2)	65 (30.4)	
Data missing	8 (2.7)	0 (0)	8 (3.7)	

Vaginal brachytherapy (VBT), external beam radiation therapy (EBRT). ^aExcept age and BMI.

high-grade histologies were well represented and we found no difference in VTE rates between them.

Khorana scores were calculated for all patients at the start of their chemotherapy treatment, and 37.2% of patients ultimately diagnosed with a VTE had a high risk Khorana score (≥ 3) compared with 30.4% of patients who did not develop a VTE. High risk Khorana score was not associated with VTE risk ($p = 0.32$). Similar findings were reported by Piver et al. who found that a higher Khorana score (≥ 2 or ≥ 3) was not sufficient to predict VTE in patients receiving chemotherapy for endometrial cancer, and by Llanora et al. who reported that $>50\%$ of patients who developed VTE had an intermediate risk score and would not have qualified for prophylaxis [19,32]. The Khorana score may have limited use in the endometrial cancer population. Other risk stratification models for VTE in cancer have been published and incorporate a variety of factors including medical comorbidities, serum biomarkers, genetics, surgical approach and specific treatment regimens [27]. Furthering our investigation to include these could identify additional factors associated with VTE development in our cohort of endometrial cancer patients.

Sixty-five percent of the patients in our cohort were Black, making this a unique dataset. Although our results do suggest that higher stage at diagnosis is associated with VTE diagnosis and Black women with endometrial cancer more frequently present with Stage IV disease compared to white women, Black race was not associated with development of VTE in our cohort ($p = 0.995$) [33]. However, Datta et al., in an analysis of a statewide cancer registry demonstrated a higher risk of cancer-associated thrombosis in Black and African American patients across almost all cancer types compared to white patients, with the most significant difference in uterine cancer (HR 2.11; 95% CI, 1.80–2.48) [33]. Race and ethnicity were independent risk factors for cancer-associated thrombosis in their analysis and other studies have demonstrated similar results, even when cancer stage at VTE diagnosis is consistent across racial groups [23,34].

Limitations of this study include its retrospective nature, limited historic sociodemographic and comorbidity data, and a single-institution patient population. We acknowledge the competing risk of death in advanced stage cancer cohort could bias the incidence, yielding lower rates of VTE, while selecting a cohort of cancer patients on

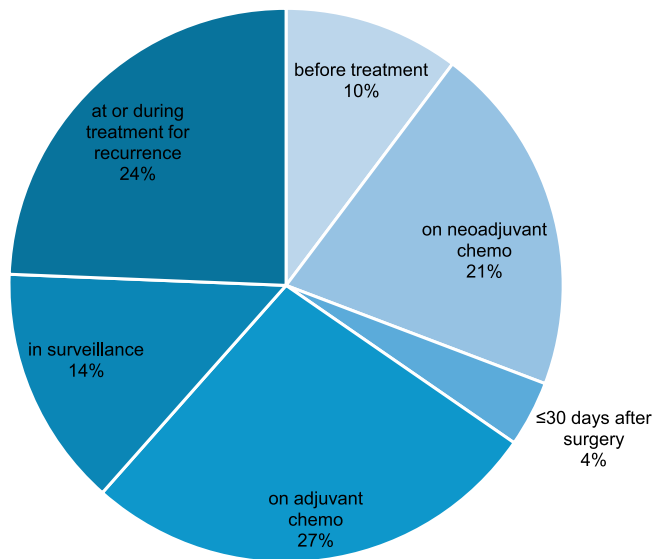


Fig. 2. Timing of VTE diagnosis in the course of cancer treatment.

Table 2
Distribution of disease at the time of VTE diagnosis.

Disease location	n (%)
Uterus	30 (38.5)
Vagina	5 (6.4)
Adnexae or pelvic mass	11 (14.1)
Pelvic LN	19 (24.4)
Para-aortic LN	15 (19.2)
Carcinomatosis	24 (30.8)
Ascites	14 (17.9)
Other distant site	27 (34.6)
No evidence of disease	22 (28.2)

chemotherapy undergoing regular imaging studies likely increases the overall detection rate of asymptomatic VTE. Strengths include the total number of patients evaluated and capture of granular data over the entire cancer course of a chemotherapy-restricted cohort, as well as the racial and histologic diversity among included patients.

In conclusion, over a quarter of our patients with endometrial cancer who received chemotherapy as part of their initial treatment, representing a high risk population, developed VTE either during their initial treatment, surveillance, or during recurrence therapy. The only significant risk factor identified was stage at diagnosis, with patients with Stage IV disease significantly more likely to develop VTE compared to others. Ongoing research to evaluate a broader patient population and range of potential risk factors, including medical comorbidities, serum biomarkers, and specific treatment regimens, to identify risk factors predictive of VTE is critical to optimizing our ability to risk-stratify patients and potentially implement standardized prophylaxis protocols to reduce the burden of VTE in endometrial cancer patients.

CRediT authorship contribution statement

Elizabeth G. Thayer: Writing – review & editing, Writing – original draft, Methodology, Investigation, Formal analysis, Conceptualization. **Zoe A. Roecker:** Writing – original draft, Investigation, Formal analysis, Conceptualization. **Kelsey A. Roof:** Writing – original draft, Investigation, Formal analysis, Conceptualization. **Hannah B. Gold:** Writing – original draft, Investigation. **Kristen D. Starbuck:** Writing – review & editing, Supervision, Methodology, Conceptualization.

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Consent statement

This study was a retrospective chart review and was deemed exempt from the consent requirement by the institutional IRB.

Declaration of competing interest

The authors declare no relevant or financial interests in the research described in this paper.

References

- [1] A.A. Khorana, C.W. Francis, E. Culakova, N.M. Kuderer, G.H. Lyman, Frequency, risk factors, and trends for venous thromboembolism among hospitalized cancer patients, *Cancer* 110 (2007) 2339–2346, <https://doi.org/10.1002/cncr.23062>.
- [2] J.A. Heit, M.D. Silverstein, D.N. Mohr, T.M. Petterson, W.M. O'Fallon, L.J. Melton 3rd, Risk factors for deep vein thrombosis and pulmonary embolism: a population-based case-control study, *Arch. Intern. Med.* 160 (2000) 809–815, <https://doi.org/10.1001/archinte.160.6.809>.
- [3] A.O. Rodriguez, A.M. Gonik, H. Zhou, G.S. Leiserowitz, R.H. White, Venous thromboembolism in uterine cancer, *Int. J. Gynecol. Cancer* 21 (2011) 870–876, <https://doi.org/10.1097/JGCO.0b013e31821a367e>.
- [4] T. Satoh, K. Matsumoto, K. Uno, M. Sakurai, S. Okada, M. Onuki, T. Minaguchi, Y.O. Tanaka, S. Homma, A. Oki, H. Yoshikawa, Silent venous thromboembolism before treatment in endometrial cancer and the risk factors, *Br. J. Cancer* 99 (2008) 1053–1057, <https://doi.org/10.1038/sj.bjc.6604658>.
- [5] A.T. Cohen, A. Katholing, S. Rietbrock, L. Bamber, C. Martinez, Epidemiology of first and recurrent venous thromboembolism in patients with active cancer: a population-based cohort study, *Thromb. Haemost.* 117 (2017) 57–65, <https://doi.org/10.1160/TH15-08-0686>.
- [6] S. Pin, J. Mateshaytis, S. Ghosh, E. Batuyong, J.C. Easaw, Risk factors for venous thromboembolism in endometrial cancer, *Curr. Oncol.* 27 (2020) 198–203, <https://doi.org/10.3747/co.27.5981>.
- [7] J.S. Yuk, B. Lee, K. Kim, M.H. Kim, Y.S. Seo, S.O. Hwang, Y.K. Cho, Y.B. Kim, Incidence and risk of venous thromboembolism according to primary treatment type in women with endometrial cancer: a population-based study, *BMC Cancer* 21 (2021) 1166, <https://doi.org/10.1186/s12885-021-08853-x>.
- [8] V.M. Wagner, R.N. Piver, M.D. Levine, F.J. Backes, L.J. Chambers, D.E. Cohn, L.J. Copeland, C.M. Cosgrove, C.I. Nagel, D.M. O'Malley, K.L. Bixel, Postoperative venous thromboembolism risk stratification in patients with uterine cancer, *Am. J. Obstet. Gynecol.* 228 (2023) 555.e1–555.e8, <https://doi.org/10.1016/j.ajog.2022.12.310>.
- [9] D. Basaran, T. Boerner, J. Suhner, D. Sassine, Y. Liu, R.N. Grisham, W.P. Tew, G.J. Gardner, O. Zivanovic, Y. Sonoda, K. Long Roche, D.S. Chi, N.R. Abu-Rustum, G.A. Soff, E.L. Jewell, Risk of venous thromboembolism in ovarian cancer patients receiving neoadjuvant chemotherapy, *Gynecol. Oncol.* 163 (2021) 36–40, <https://doi.org/10.1016/j.ygyno.2021.07.030>.
- [10] L. Ye, L. Cai, Y. Fu, D. Zhuang, X. Hu, Y. Jie, The prevalence, risk factors, and prognostic value of venous thromboembolism in ovarian cancer patients receiving chemotherapy: a systematic review and meta-analysis, *World J. Surg. Oncol.* 19 (2021) 12, <https://doi.org/10.1186/s12957-020-02101-5>.
- [11] P.S. Greco, A.A. Bazzi, K. McLean, R.K. Reynolds, R.J. Spencer, C.M. Johnston, J.R. Liu, S. Uppal, Incidence and timing of thromboembolic events in patients with ovarian cancer undergoing neoadjuvant chemotherapy, *Obstet. Gynecol.* 129 (2017) 979–985, <https://doi.org/10.1097/AOG.0000000000001980>.
- [12] F. Abu Saadeh, L. Norris, S. O'Toole, N. Gleeson, Venous thromboembolism in ovarian cancer: incidence, risk factors and impact on survival, *Eur. J. Obstet. Gynecol. Reprod. Biol.* 170 (2013) 214–218, <https://doi.org/10.1016/j.ejogrb.2013.06.004>.
- [13] Y. Matsuura, G. Robertson, D.E. Marsden, S.N. Kim, V. GebSKI, N.F. Hacker, Thromboembolic complications in patients with clear cell carcinoma of the ovary, *Gynecol. Oncol.* 104 (2007) 406–410, <https://doi.org/10.1016/j.ygyno.2006.08.026>.
- [14] H.D. McLaughlin, P. Greco, A.M. Straubhar, A. Rolston, K. McCool, M. Brackmann, J.H. Siedel, K. McLean, R.K. Reynolds, S. Uppal, Implementation of routine venous thromboembolism prophylaxis during neoadjuvant chemotherapy for patients with ovarian cancer, *Gynecol. Oncol.* 178 (2023) 89–95, <https://doi.org/10.1016/j.ygyno.2023.10.001>.
- [15] J.R. Salinaro, K. McQuillen, M. Stemple, R. Boccaccio, J. Ehrisman, A.M. Lorenzo, L. Havrilesky, A.A. Secord, V.G. Turner, K.N. Moore, B. Davidson, Incidence of venous thromboembolism among patients receiving neoadjuvant chemotherapy for advanced epithelial ovarian cancer, *Int. J. Gynecol. Cancer* 30 (2020) 491–497, <https://doi.org/10.1136/ijgc-2019-000980>.
- [16] W.D. Hazelton, M. Prest, L. Chen, K. Rouse, E.B. Elkin, J.S. Ferris, X. Xu, N.A. Bickell, C.Y. Kong, S. Blank, E.J. Feuer, G. Samimi, B.M. Heckman-Stoddard, T.M. Layne, J.D. Wright, E.R. Myers, L.J. Havrilesky, Trends in uterine cancer incidence and mortality: insights from a natural history model, *J. Natl. Cancer Inst.* 117 (2025) 1891–1903, <https://doi.org/10.1093/jnci/djaf135>.

- [17] H.S. Kahr, O.B. Christiansen, C. Hogdall, A. Grove, R.N. Mortensen, C. Torp-Pedersen, A. Knudsen, O. Thorlacius-Ussing, Endometrial cancer does not increase the 30-day risk of venous thromboembolism following hysterectomy compared to benign disease: a Danish national cohort study, *Gynecol. Oncol.* 155 (2019) 112–118, <https://doi.org/10.1016/j.ygyno.2019.07.022>.
- [18] K.G. Dinkins, G.M. Lee, P.W. Ketch, M.D. Toboni, C.A. Leath III, J.M. Straughn Jr., T.K.L. Boitano, Venous thromboembolism risk for patients with BMI ≥ 40 kg/m² undergoing robotic surgery for endometrial cancer, *Gynecol. Oncol. Rep.* 59 (2025), 101779, <https://doi.org/10.1016/j.gore.2025.101779>.
- [19] J.D. Llanora, S. Uppal, H.D. McLaughlin, Incidence and timing of venous thromboembolism in patients with advanced endometrial cancer, *Obstet. Gynecol.* 146 (2025) 293–296, <https://doi.org/10.1097/AOG.0000000000005978>.
- [20] A. Kretz, L. Prichett, S. Olson, Y. Wang, A. Beavis, Evaluation of the Khorana score and association with venous thromboembolism incidence among patients with uterine cancer: implications for patient care, *Gynecol. Oncol.* 190 (2024) S408, <https://doi.org/10.1016/j.ygyno.2024.07.601>.
- [21] B. Mukerji, C. Baptiste, L. Chen, A.I. Tergas, J.Y. Hou, C.V. Ananth, A.I. Neugut, D.L. Hershman, J.D. Wright, Racial disparities in young women with endometrial cancer, *Gynecol. Oncol.* 148 (2018) 527–534, <https://doi.org/10.1016/j.ygyno.2017.12.032>.
- [22] T.R. Sponholtz, J.R. Palmer, L. Rosenberg, C. Chen, Y. Chen, M.A. Clarke, T. Clendenen, M. Du, L. Johnson, L.M. Liao, K.A. Michels, K. O'Connell, S.H. Olson, S. Petruzella, T.R. Rebbeck, V.W. Setiawan, B. Trabert, N.S. Weiss, N. Wentzensen, L. Wilkens, L.A. Wise, Risk factors for endometrial cancer in Black women, *Cancer Causes Control* 34 (2023) 421–430, <https://doi.org/10.1007/s10552-022-01653-z>.
- [23] N.O. Addo-Tabiri, R. Chudasama, R. Vasudeva, O. Leiva, B. Garcia, J.D. Ravid, T. Bunze, L. Rosen, M. Belghasem, J. Francis, M. Brophy, B. Johnson, R. Ferguson, J. Weinberg, V.C. Chitalia, Black patients experience highest rates of cancer-associated venous thromboembolism, *Am. J. Clin. Oncol.* 43 (2020) 94–100, <https://doi.org/10.1097/COC.0000000000000639>.
- [24] FIGO Committee on Gynecologic Oncology, Revised FIGO staging for carcinoma of the vulva, cervix, and endometrium, *Int. J. Gynaecol. Obstet.* 105 (2009) 103–104, <https://doi.org/10.1016/j.ijgo.2009.02.012>.
- [25] M.Y. Sun, S.M.M. Bhaskar, Venous thromboembolism in cancer patients undergoing chemotherapy: a systematic review and meta-analysis, *Diagnostics* 12 (2022) 2954, <https://doi.org/10.3390/diagnostics12122954>.
- [26] J. Harry, R. Bucciol, D. Finnigan, H. Hashem, A. Araki, M. Othman, The incidence of venous thromboembolism by type of solid cancer worldwide: a systematic review, *Cancer Epidemiol.* 95 (2025), 102764, <https://doi.org/10.1016/j.canep.2025.102764>.
- [27] C. Fowler, S.M. Pastores, Venous thromboembolic events in cancer immunotherapy: a narrative review, *J. Clin. Med.* 14 (2025) 4926, <https://doi.org/10.3390/jcm14144926>.
- [28] C. Iwai, T. Jo, T. Konishi, A. Fujita, N. Michihata, H. Matsui, K. Fushimi, H. Yasunaga, Thrombotic risk of platinum combination chemotherapy with and without immune checkpoint inhibitors for advanced non-small cell lung cancer: a nationwide inpatient database study, *Cancer Immunol. Immunother.* 72 (2023) 3581–3591, <https://doi.org/10.1007/s00262-023-03508-1>.
- [29] K. Matsuo, A.A. Yessaian, Y.G. Lin, H.Q. Pham, L.I. Muderbach, H.A. Liebman, C.P. Morrow, L.D. Roman, Predictive model of venous thromboembolism in endometrial cancer, *Gynecol. Oncol.* 128 (2013) 544–551, <https://doi.org/10.1016/j.ygyno.2012.12.014>.
- [30] K.S. Weeks, E. Herbach, M. McDonald, M. Charlton, M.L. Schweizer, Meta-analysis of VTE risk: ovarian cancer patients by stage, histology, cytoreduction and ascites at diagnosis, *Obstet. Gynecol. Int.* 2020 (2020), 2374716, <https://doi.org/10.1155/2020/2374716>.
- [31] A.A. Khorana, N. Mackman, A. Falanga, I. Pabinger, S. Noble, W. Ageno, F. Moik, A.Y.Y. Lee, Cancer-associated venous thromboembolism, *Nat. Rev. Dis. Primers* 8 (2022) 11, <https://doi.org/10.1038/s41572-022-00336-y>.
- [32] R.N. Piver, V.M. Wagner, M.D. Levine, F.J. Backes, L.J. Chambers, D.E. Cohn, L.J. Copeland, C.M. Cosgrove, C.I. Nagel, D.M. O'Malley, K.L. Bixel, Use of the Khorana score to predict venous thromboembolism in patients undergoing chemotherapy for uterine cancer, *Gynecol. Oncol. Rep.* 46 (2023), 101156, <https://doi.org/10.1016/j.gore.2023.101156>.
- [33] M.L. Hicks, M.M. Hicks, R.P. Mathews, D. Khabele, C.A. Clare, O. Balogun, Y.R. Lawson, R.H. Tillman, R. Butler, C.O. Spann, G.P. Parham, Racial disparities in endometrial cancer: where are we after 26 years? *Gynecol. Oncol.* 184 (2024) 236–242, <https://doi.org/10.1016/j.ygyno.2024.01.054>.
- [34] T. Datta, A. Brunson, A. Mahajan, T. Keegan, T. Wun, Racial disparities in cancer-associated thrombosis, *Blood Adv.* 6 (2022) 3167–3177, <https://doi.org/10.1182/bloodadvances.2021006209>.