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Risk factors for in-hospital venous thromboembolism in patients with bladder cancer: A retrospective single-center study

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Abstract

Objective In this study, we retrospectively analyzed the incidence of and risk factors for in-hospital venous thromboembolism (VTE) in patients with bladder cancer and explored measures to reduce the incidence of and/or prevent in-hospital VTE and mortality.

Methods The clinical data of 5744 patients with bladder cancer who were admitted to the Second Hospital of Tianjin Medical University during a 5-year period were summarized and then analyzed to determine the risk factors for in-hospital VTE in patients with bladder cancer with the aim of identifying preventive measures.

Results Univariate analysis revealed significant differences between the VTE group and the non-VTE group in terms of age, sex, Caprini score, surgical treatment status, tumor stage, D-dimer level, PT, APTT, and Hb ($P < 0.05$). Multiple factor analysis further confirmed that the Caprini score, surgical treatment status, D-dimer level, PT, and APTT were independent risk factors for VTE.

Conclusion Analyses revealed that a high Caprini score, surgical treatment, an elevated D-dimer level, a short PT, and a short APTT were independent risk factors for in-hospital VTE in patients with bladder cancer and that active preventive measures should be implemented to reduce the incidence of in-hospital VTE as well as mortality.

Keywords Venous thromboembolism, Risk factors, Bladder cancer, Preventive measures

Introduction

Venous thromboembolism (VTE), including deep vein thrombosis (DVT) and pulmonary embolism (PE), is an important health problem that affects approximately 1 in 1000 to 2 in 1000 individuals in the general population annually [1]. The risk of VTE is estimated to be approximately 4–6.5 times greater in cancer patients than in noncancer patients [2, 3]. Moreover, VTE is one of the leading causes of death in cancer patients [4]. Bladder cancer is one of the most common urological malignancies [5]. Patients with bladder cancer have a high risk of VTE [6, 7]. The overall rate of VTE in patients with bladder cancer has been estimated to be 1.9–4.7%, ranging

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from 3 to 24.4% in patients undergoing cystectomy and 3.1–7.9% in metastatic-stage patients, with variations depending on the type of treatment [8–10]. In the literature, multifactorial risk factors for VTE in patients with bladder cancer have been reported, including major surgery, an advanced disease stage, an increased number of comorbidities, and a history of VTE [11–14]. Additionally, the cross-talk between bladder cancer cells and the coagulation system has been shown to increase the risk of VTE in patients with bladder cancer [15–18]. However, the prediction of VTE occurrence based on relevant laboratory indicators and the Caprini score in bladder cancer patients remains to be further explored. This retrospective study analyzed clinical data on in-hospital bladder cancer patients with VTE admitted to the Second Hospital of Tianjin Medical University between October 2018 and October 2023, aiming to identify potential risk factors for in-hospital VTE. The findings are expected to benefit the management of in-hospital VTE in bladder cancer patients.

Materials and methods

Study population

In this study, the matched case-control method was used; 97 patients who were admitted to the Second Hospital of Tianjin Medical University between October 2018 and October 2023 ($n = 5744$) and met the inclusion criteria were included in the VTE group, and 97 patients were randomly assigned to the non-VTE group at a ratio of 1:1, with age and sex used as the matching indices (Fig. 1).

Inclusion and exclusion criteria

The inclusion criteria were as follows: (1) a diagnosis of bladder cancer confirmed by pathology; (2) an imaging examination (ultrasound color doppler and computed tomography angiography) revealing the presence of VTE; (3) no relevant diseases affecting coagulation function; (4) no anticoagulant therapy prior to admission; and (5) complete imaging and clinical data. The exclusion criteria included the following: (1) the use of drugs affecting coagulation within the past 3 months; (2) venous thrombosis associated with other cancers; (3) a diagnosis of VTE at the time of admission; (4) a poor general condition with a life expectancy of less than 6 months; (5) the presence of active bleeding; and (6) a history of VTE.

Data collection methods

The detailed clinical data of all the enrolled patients were collected from the hospital's electronic medical record system, including but not limited to patient age; sex; history of smoking and alcohol consumption; history of hypertension and diabetes mellitus; body mass index (BMI); D-dimer level; thrombin time (TT); activated partial thromboplastin time (APTT); fibrinogen (Fbg) and prothrombin time (PT); hemoglobin(Hb); platelet count; white blood cell count; and Caprini score.

Statistical methods

The data were analyzed using SPSS 26.0 statistical software. The quantitative data are presented as the means ± standard deviations, and t tests and ANOVA were used to compare differences between groups. Qualitative data are presented as frequencies and rates, and the chi-square (χ^2) test was used to compare differences between groups.

In this study, the test level was set at 0.05. Risk factors with a two-sided P value < 0.05 in the univariate analysis were included in the multifactorial logistic regression to determine the independent risk factors for concurrent VTE in patients with bladder cancer.

Results

Among the 5744 patients, 97 (1.69%) experienced in-hospital VTE. There were statistically significant differences in age, sex, and Caprini scores between the VTE group and the non-VTE group ($P < 0.05$). There were no statistically significant differences between the two groups in terms of BMI, history of smoking, history of alcohol consumption, and history of diabetes mellitus, hypertension, or VTE ($P > 0.05$) (Table 1).

D-dimer levels were higher in the VTE group than in the non-VTE group, and Hb, PT, and APTT were significantly lower in the VTE group than in the non-VTE group ($P < 0.01$) (Table 2).

Statistically significant differences were observed in surgical treatment status as well as in surgical modality ($P < 0.05$). Statistical differences were also observed among different clinical stages of bladder cancer. However, there was no statistically significant difference among the different chemotherapy regimens ($P > 0.05$) (Tables 3 and 4).

A multifactorial logistic analysis was performed with age, sex, Caprini score, surgical treatment, tumor stage, D-dimer level, PT, APTT, and Hb as the independent variables, with the occurrence of VTE as the dependent variable. The results revealed that a high Caprini score, surgical treatment, an elevated D-dimer level, and a short PT and APTT were independent risk factors for VTE in patients with bladder cancer (Table 5).

Discussion

Patients with malignant tumors have a high incidence of VTE. The annual incidence of VTE is significantly greater in patients with malignant tumors (approximately 0.5%) than in the general population (approximately 0.1%), and patients with active malignant tumors account for approximately 20% of the total number of patients with VTE [19]. The complication of VTE not only affects the

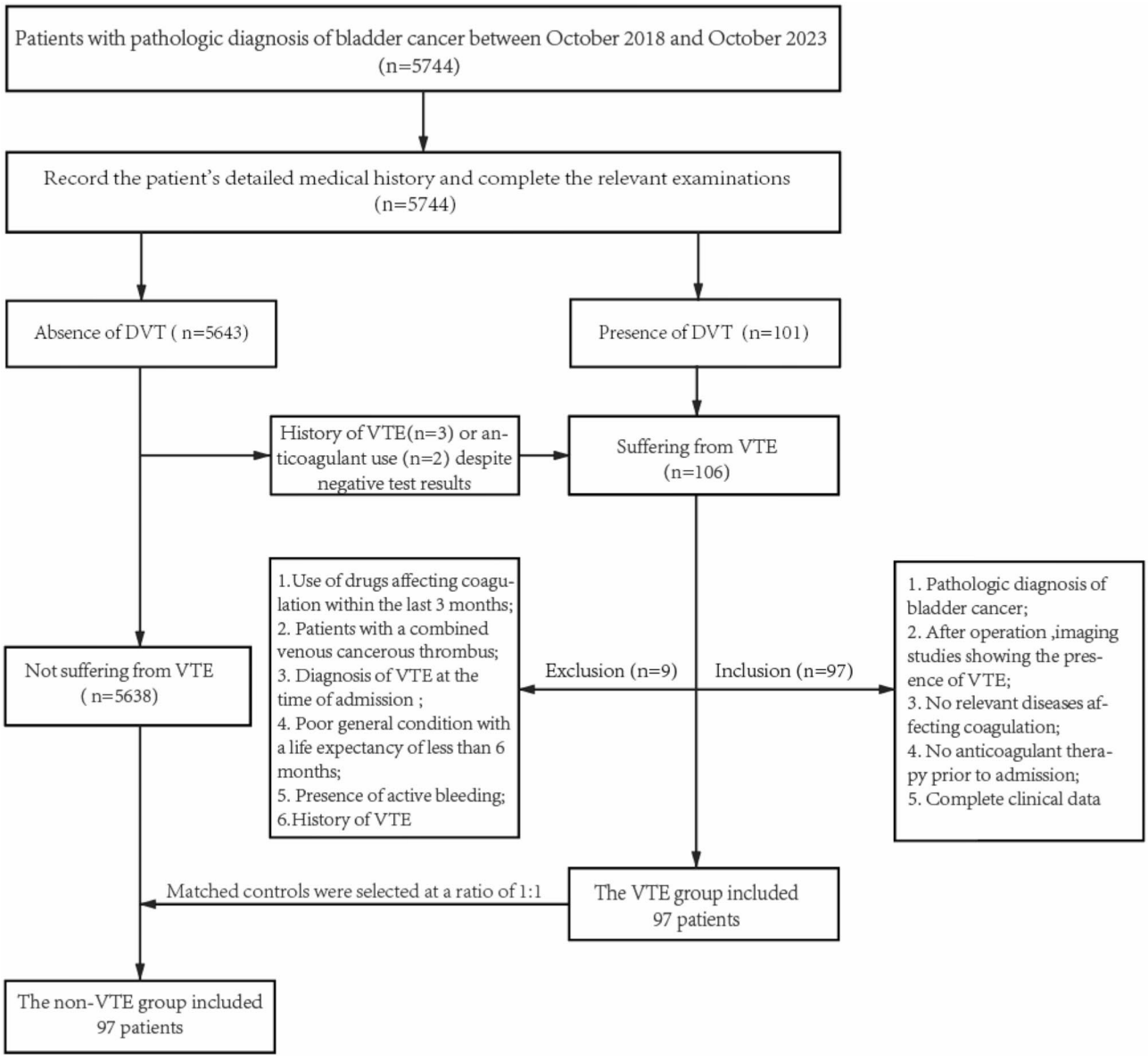

Fig. 1 Flowchart for patient enrollment

quality of life of patients with bladder cancer but is significantly associated with decreased survival time [11]. Previous studies have revealed an association between VTE and bladder cancer, but the extent and mechanism of this association have not yet been clarified [11]. It is possible to diagnose VTE if it is detected early, and VTE can be prevented if the relevant indicators are addressed. Prevention or early diagnosis can improve patient prognosis and quality of life.

D-dimer is a soluble fibrin degradation product that is an important indicator of hypercoagulability and activation of the fibrinolytic system in vivo, and elevated D-dimer levels have been shown to be an independent risk factor for VTE [20]. However, elevated plasma D-dimer levels lack specificity; in addition to venous thrombosis, infection, pregnancy, renal insufficiency, and other conditions can also cause elevated D-dimer levels [21]. The plasma D-dimer assay is simple and well validated, and D-dimer is still an irreplaceable screening indicator for bladder cancer-related VTE [22, 23]. In this study, the D-dimer level was 3.95 ± 10.12 mg/L in the VTE group and 0.47 ± 0.36 mg/L in the non-VTE group, indicating that the D-dimer level was significantly greater in the VTE group. This study revealed that the D-dimer level is not only an independent risk factor for thrombosis but also an important monitoring index during the treatment and follow-up of patients with bladder cancer complicated by VTE.

Table 1 Comparison of differences in general clinical data between the VTE group and the non-VTE group

Variable	VTE group ($n=97$)	Non-VTE group ($n=97$)	t/χ^2	p
Age (y), Mean ± SD	72.37 ± 8.43	68.82 ± 11.97	-2.385	0.018*
Gender, n (%)				
male	74(76.29%)	85(87.63%)	4.218	0.040*
female	23(23.71%)	12(12.37%)		
BMI (kg/m^2), Mean ± SD	24.19 ± 3.17	25.05 ± 4.47	1.541	0.125
Smoking, n (%)	44(45.36%)	40(41.24%)	0.336	0.562
Alcohol consumption, n (%)	27(27.84%)	18(18.56%)	2.344	0.126
Hypertension, n (%)	46(47.42%)	45(46.39%)	0.021	0.886
Diabetes mellitus, n (%)	30(30.93%)	22(22.68%)	1.681	0.195
History of VTE, n (%)	2(2.06%)	2(2.06%)	0.000	0.992
Caprini score, Mean ± SD	5.42 ± 1.57	4.16 ± 1.12	-6.441	< 0.001**

*$p < 0.05$ **$p < 0.01$

Table 2 Comparison of laboratory indicators between the VTE group and the non-VTE group

Variable	VTE group ($n=97$)	Non-VTE group ($n=97$)	t/χ^2	p
D-dimer level (mg/L), Mean ± SD	2.44 ± 3.00	0.47 ± 0.36	-3.384	0.001**
PT (s), Mean ± SD	11.45 ± 1.45	13.12 ± 1.43	8.091	< 0.001**
APTT (s), Mean ± SD	27.62 ± 6.45	35.58 ± 4.22	10.149	< 0.001**
TT (s), Mean ± SD	17.37 ± 1.20	18.11 ± 3.54	1.926	0.056
Fbg (g/L), Mean ± SD	3.50 ± 1.11	3.66 ± 1.93	0.740	0.460
Platelet count (×10^9/L), Mean ± SD	223.09 ± 80.49	228.47 ± 67.90	0.503	0.615
Leukocyte count (×10^9/L), Mean ± SD	6.80 ± 2.38	6.43 ± 1.99	1.51	0.134

*$p < 0.05$ **$p < 0.01$

Table 4 Comparison of tumor clinical stages between the two groups

	VTE group ($n=97$)	Non-VTE group ($n=97$)	t/χ^2	p
Tumor Clinical Stage, n (%)				
I	28(28.87%)	43(46.24%)	13.139	0.004**
II	39(40.21%)	40(43.01%)		
III	23(23.71%)	8(8.60%)		
IV	7(7.22%)	2(2.15%)		

*$p < 0.05$ **$p < 0.01$

Hypercoagulability is an important risk factor for VTE in patients with bladder cancer. In this study, we further analyzed the PT and APTT data of patients in the thrombosis group and nonthrombosis group, and the PT and APTT values in the VTE group were significantly lower than those in the non-VTE group ($P < 0.001$). The decreases in PT and APTT usually suggest that the blood is in a state of hypercoagulability, which may indicate insufficient quantity of or sensitivity to anticoagulant medication, although PT and APTT may be affected by a variety of factors and cannot be used alone to confirm the diagnosis of VTE.

Table 3 Comparison of treatment modalities between the two groups

Variable	VTE group($n=97$)	Non-VTE group($n=97$)	t/χ^2	p
Surgical treatment, n (%)	88(90.72%)	41(42.27%)	51.109	< 0.001**
Surgical procedures, n (%)				
Cystoscopy	54(61.36%)	35(85.37%)	11.239	0.004**
Laparoscopy	31(35.23%)	3(7.32%)		
Open surgery	3(3.41%)	3(7.32%)		
Drug treatment, n (%)	40(41.24%)	16(16.49%)	14.460	< 0.001**
Drug treatment modalities, n (%)				
Immunotherapy	8(17.78%)	1(5.88%)	7.512	0.111
Chemotherapy plus immunotherapy	17(37.78%)	3(17.65%)		
Chemotherapy	11(24.44%)	10(58.82%)		
Targeted therapy	4(8.89%)	2(11.76%)		

*$p < 0.05$ **$p < 0.01$

In major urologic surgery, VTE is among the most serious complications [24]. The incidence of VTE in patients who undergo radical cystectomy is approximately 6-24.4% [10], and the incidence of PE is about 2.5% [25]. If reconstruction of the inferior vena cava is required during surgery, such as in radical nephrectomy, the incidence of VTE is approximately 22% [26]. Although robot-assisted laparoscopic surgery is less invasive, 14% of patients who undergo this type of surgery are readmitted to the hospital due to VTE, which is the most important reason for postoperative readmission [27]. In this study, 88 (90.72%) patients in the VTE group received surgical treatment after admission, including 54 (61.36%) who underwent cystoscopic surgeries, 31 (35.23%) who underwent laparoscopic surgeries, and 3 (3.41%) who underwent open surgeries. Surgical treatment is an independent risk factor for VTE, and appropriate mechanical and pharmacological prophylaxis should be administered to this group of patients according to their situation, as it is highly important for reducing the incidence of VTE and mortality [28–31].

The Caprini score is a widely used VTE risk assessment tool [32]. The mean Caprini score in the VTE group in this study was 5.42 ± 1.57, which was significantly higher than that in the non-VTE group (4.16 ± 1.1), and the occurrence of VTE in patients with bladder cancer

Table 5 Binary logistic regression analysis of independent risk factors for VTE in patients with bladder cancer

Variable	Beta.	SE	z	Wald χ2	*p*	OR	95% CI
Age	-0.021	0.045	-0.476	0.226	0.634	0.979	0.897 ~ 1.069
Gender	1.267	0.944	1.342	1.801	0.180	3.549	0.558 ~ 22.568
Tumor stage	-0.319	0.478	-0.668	0.446	0.504	0.727	0.285 ~ 1.854
Surgical treatment	3.654	1.028	3.555	12.639	< 0.001**	38.628	5.152 ~ 289.586
D-dimer	4.941	1.137	4.346	18.886	< 0.001**	139.877	15.066 ~ 1298.646
PT	-0.825	0.377	-2.190	4.795	0.029*	0.438	0.209 ~ 0.917
APTT	-0.415	0.116	-3.572	12.756	< 0.001**	0.661	0.526 ~ 0.829
TT	0.192	0.146	1.311	1.718	0.190	1.212	0.909 ~ 1.614
Hb	-0.005	0.021	-0.257	0.066	0.797	0.995	0.954 ~ 1.036
Caprini score	1.149	0.451	2.549	6.497	0.011*	3.154	1.304 ~ 7.629

*$p < 0.05$**$p < 0.01$

Table 6 Risk of VTE in the included patients [34]

Risk level	Total score (Caprini)	VTE group ($n = 97$)	Non-VTE group ($n = 97$)
Low risk	0–1	0	0
Medium risk	2	0	8
High risk	3–4	1	58
Very high risk	≥ 5	96	31

was not limited to the postoperative stage. The biological properties of the tumor itself, the implementation of multiple interventions during cancer treatment, and a patient's own coagulation status may promote the occurrence of VTE [33]. Therefore, the role of the Caprini score in the assessment of VTE risk in patients with bladder cancer should not be limited to a single point in time but should be used as a dynamic monitoring tool to continuously guide clinical decision-making (Table 6).

According to the recommendations in the current guidelines, prophylactic anticoagulants should be initiated for hospitalized patients with a confirmed diagnosis or suspected case of bladder cancer if there are no contraindications [35, 36]. In patients with urogenital pelvic tumors who do not undergo prophylaxis, the incidence of postoperative VTE is greater than 20%, and most cases occur after discharge from the hospital [37], which is why drug prophylaxis for VTE should be continued for 4 weeks after major abdominal or pelvic surgery [37]. Pariser et al. reported that continuous administration of enoxaparin for 28 days after cystectomy led to a decrease in the incidence of VTE from 12–5% [38], suggesting that all patients should receive prophylactic anticoagulants for at least 28 days after radical cystectomy and that although anticoagulation increases the risk of bleeding somewhat, most patients still benefit from extended prophylactic anticoagulation after surgery [39]. Studies have shown that Vitamin K antagonist (VKA) for anticoagulation increases the risk of bleeding by 2–6 times in cancer patients and therefore is not recommended [40].

Low-molecular-weight heparin (LMWH), owing to its long half-life, high bioavailability, and lack of need for frequent coagulation monitoring, has become the first-line drug for VTE prophylaxis recommended in several guidelines [41, 42]. Non-vitamin K antagonist oral anticoagulants (NOACs) have a wide therapeutic window, can be administered orally, and do not require frequent coagulation testing, making them more convenient than LMWH and VKA for prophylactic anticoagulation in patients with malignant tumors, especially outpatients [43]. However, owing to the small number of studies on the efficacy and safety of NOACs for VTE prevention after cancer surgery, there is no direct evidence supporting the replacement of LMWH with NOACs for the prevention of postoperative VTE in patients with cancer, and the highest dose of LMWH is still recommended for prophylaxis in patients with postoperative VTE according to current guidelines [42].

This study has a few limitations. It was a retrospective study from a single center, which might cause selection bias and limit the generalizability of the study results. So, the clinical applicability and reproducibility of risk factors for in-hospital VTE in patients with bladder cancer still need to be confirmed. Further prospective, multicenter studies may be beneficial to further validate our findings.

Conclusion

This study revealed that a high Caprini score, surgical treatment, an elevated D-dimer level, and a short PT and APTT are independent risk factors for in-hospital VTE in patients with bladder cancer, and preventive measures should be initiated for patients with bladder cancer to reduce the incidence of in-hospital VTE, morbidity, and mortality.

Abbreviations

VTE	Venous thromboembolism
Hb	Hemoglobin
DVT	Deep vein thrombosis
Fbg	Fibrinogen
PE	Pulmonary embolism
BMI	Body mass index
PT	Prothrombin time

LMWH	Low-molecular-weight heparin
APTT	Activated partial thromboplastin time
VKA	Vitamin K antagonist
TT	Thrombin time
NOACs	Non-vitamin K antagonist oral anticoagulants

Author contributions
Conceptualization, H.Y. and Z.L.; Data curation, T.Z. and H.Y.; Formal analysis, T.Z. and K.W.; Funding acquisition, Y.L.; Investigation, B.C. and K.W.; Methodology, Y.C. and Z.L.; Project administration, H.H. and Y.C.; Resources, B.C. and H.Z.; Software, G.F. and Z.J.; Supervision, H.H. and Y.L.; Visualization, H.Z. and H.Y.; Validation, G.F. and Z.J.; Writing– original draft, H.Y., T.Z. and Z.L.; Writing– review & editing, H.H. and Y.L. All the authors have read and agreed to the submission of the manuscript for consideration for publication.

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Data availability
Data is provided within the manuscript.

Declarations

Ethics approval and consent to participate
This study was conducted in accordance with the ethical principles outlined in the Declaration of Helsinki. Ethical approval was obtained from the Ethics Committee of the Second Hospital of Tianjin Medical University (approval number KY2024K211 from 15, April 2024), which also granted a waiver of informed consent for this retrospective study. The research utilized anonymized medical records collected during routine clinical consultations, and the Ethics Committee confirmed that the waiver of informed consent would not adversely affect the rights, welfare, or health of the study participants.

Consent for publication
Not apply.

Competing interests
The authors declare no competing interests.

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References

1. Goldhaber SZ. Venous thromboembolism: epidemiology and magnitude of the problem. Best Pract Res Clin Haematol. 2012;25(3):235–42.
2. Walker AJ, Card TR, West J, Crooks C, Grainge MJ. Incidence of venous thromboembolism in patients with cancer - a cohort study using linked united Kingdom databases. Eur J Cancer. 2013;49(6):1404–13.
3. Heit JA, Silverstein MD, Mohr DN, Petterson TM, O'Fallon WM, Melton LJ 3. Risk factors for deep vein thrombosis and pulmonary embolism: a population-based case-control study. Arch Intern Med. 2000;160(6):809–15.
4. Khorana AA, Francis CW, Culakova E, Kuderer NM, Lyman GH. Thromboembolism is a leading cause of death in cancer patients receiving outpatient chemotherapy. J Thromb Haemost. 2007;5(3):632–4.
5. Lenis AT, Lec PM, Chamie K, Mshs MD. Bladder cancer: A review. JAMA. 2020;324(19):1980–91.
6. Pastori D, Cormaci VM, Marucci S, Franchino G, Del Sole F, Capozza A, Fallarino A, Corso C, Valeriani E, Menichelli D et al. A comprehensive review of risk factors for venous thromboembolism: from epidemiology to pathophysiology. Int J Mol Sci 2023, 24(4).
7. Khorana AA, Connolly GC. Assessing risk of venous thromboembolism in the patient with cancer. J Clin Oncol. 2009;27(29):4839–47.
8. Zareba P, Duivenvoorden WCM, Pinthus JH. Thromboembolism in patients with bladder cancer: incidence, risk factors and prevention. Bladder Cancer. 2018;4(2):139–47.
9. Abdullah O, Parashar D, Mustafa IJ, Young AM. Venous thromboembolism rate in patients with bladder cancer according to the type of treatment: A systematic review. Cureus. 2022;14(3):e22945.
10. Saluja M, Gilling P. Venous thromboembolism prophylaxis in urology: A review. Int J Urol. 2017;24(8):589–93.
11. Sandhu R, Pan CX, Wun T, Harvey D, Zhou H, White RH, Chew HK. The incidence of venous thromboembolism and its effect on survival among patients with primary bladder cancer. Cancer. 2010;116(11):2596–603.
12. Del Giudice F, Tresh A, Li S, Basran S, Prendiville SG, Belladelli F, Asero EDEB, Scornajenghi V, Carino CM. The impact of venous thromboembolism before open or minimally-invasive radical cystectomy in the USA: insurance claims data on perioperative outcomes and healthcare costs. Minerva Urol Nephrol. 2024;76(3):320–30.
13. Fantony JJ, Gopalakrishna A, Noord MV, Inman BA. Reporting bias leading to discordant venous thromboembolism rates in the united States versus Non-US countries following radical cystectomy: A systematic review and Meta-analysis. Eur Urol Focus. 2016;2(2):189–96.
14. Fantony JJ, Inman BA. Thromboembolism and bleeding in bladder cancer. Oncol (Williston Park). 2014;28(10):847–54.
15. John A, Robador JR, Vidal YSS, Houdek P, Wladykowski E, Günes C, Bolenz C, Schneider SW, Bauer AT, Gorzelanny C. Urothelial carcinoma of the bladder induces endothelial cell activation and hypercoagulation. Mol Cancer Res. 2020;18(7):1099–109.
16. Lima LG, Monteiro RQ. Activation of blood coagulation in cancer: implications for tumour progression. Biosci Rep 2013, 33(5).
17. Förster Y, Meye A, Albrecht S, Kotzsch M, Füssel S, Wirth MP, Schwenzer B. Tissue specific expression and serum levels of human tissue factor in patients with urological cancer. Cancer Lett. 2003;193(1):65–73.
18. Robinson CM, Poon BPK, Kano Y, Pluthero FG, Kahr WHA, Ohh M. A Hypoxia-Inducible HIF1-GAL3ST1-Sulfatide axis enhances CcRCC immune evasion via increased tumor Cell-Platelet binding. Mol Cancer Res. 2019;17(11):2306–17.
19. Fernandes CJ, Morinaga LTK, Alves JL Jr., Castro MA, Calderaro D, Jardim CVP, Souza R. Cancer-associated thrombosis: the when, how and why. Eur Respir Rev 2019, 28(151).
20. Ay C, Vormittag R, Dunkler D, Simanek R, Chiriac AL, Drach J, Quehenberger P, Wagner O, Zielinski C, Pabinger I. D-dimer and prothrombin fragment 1 + 2 predict venous thromboembolism in patients with cancer: results from the Vienna cancer and thrombosis study. J Clin Oncol. 2009;27(25):4124–9.
21. Goodacre S, Sampson FC, Sutton AJ, Mason S, Morris F. Variation in the diagnostic performance of D-dimer for suspected deep vein thrombosis. QJM. 2005;98(7):513–27.
22. Crawford F, Andras A, Welch K, Sheares K, Keeling D, Chappell FM. D-dimer test for excluding the diagnosis of pulmonary embolism. Cochrane Database Syst Rev. 2016;2016(8):Cd010864.
23. van Es N, Di Nisio M, Cesarman G, Kleinjan A, Otten HM, Mahé I, Wilts IT, Twint DC, Porreca E, Arrieta O, et al. Comparison of risk prediction scores for venous thromboembolism in cancer patients: a prospective cohort study. Haematologica. 2017;102(9):1494–501.
24. Scarpa RM, Carrieri G, Gussoni G, Tubaro A, Conti G, Pagliarulo V, Mirone V, De Lisa A, Fiaccavento G, Cormio L, et al. Clinically overt venous thromboembolism after urologic cancer surgery: results from the @RISTOS study. Eur Urol. 2007;51(1):130–5. discussion 136.
25. McAlpine K, Breau RH, Mallick R, Cnossen S, Cagiannos I, Morash C, Carrier M, Lavallée LT. Current guidelines do not sufficiently discriminate venous thromboembolism risk in urology. Urol Oncol. 2017;35(7):e457451–8.
26. Hicks CW, Glebova NO, Piazza KM, Orion K, Pierorazio PM, Lum YW, Abularrage CJ, Black JH 3. Risk of venous thromboembolic events following inferior Vena Cava resection and reconstruction. J Vasc Surg. 2016;63(4):1004–10.
27. Schmid M, Chiang HA, Sood A, Campbell L, Chun FK, Dalela D, Okwara J, Sammon JD, Kibel AS, Menon M, et al. Causes of hospital readmissions after urologic cancer surgery. Urol Oncol. 2016;34(5):e236231–211.
28. Cerruto MA, D'Elia C, Piccoli M, Cacciamani G, Corsi DDEM, Cavalleri PVDEM, Artibani S. Association between postoperative thromboembolism prophylaxis and complications following urological surgery. Exp Ther Med. 2016;11(1):157–63.
29. Behranwala KA, Williamson RC. Cancer-associated venous thrombosis in the surgical setting. Ann Surg. 2009;249(3):366–75.
30. Tikkinen KAO, Cartwright R, Jensen AG, Violette PD. European guidelines on peri-operative venous thromboembolism prophylaxis: first update.: chap. 12: urology. Eur J Anaesthesiol. 2024;41(8):618–21.
31. Holzheimer RG. Laparoscopic procedures as a risk factor of deep venous thrombosis, superficial ascending thrombophlebitis and pulmonary

embolism–case report and review of the literature. Eur J Med Res. 2004;9(9):417–22.
32. Lobastov K, Urbanek T, Stepanov E, Lal BK, Marangoni J, Krauss ES, Cronin M, Dengler N, Segal A, Welch HJ, et al. The thresholds of Caprini score associated with increased risk of venous thromboembolism across different specialties: A systematic review. Ann Surg. 2023;277(6):929–37.
33. Lee AY, Levine MN. Venous thromboembolism and cancer: risks and outcomes. Circulation. 2003;107(23 Suppl 1):I17–21.
34. Gould MK, Garcia DA, Wren SM, Karanicolas PJ, Arcelus JI, Heit JA, Samama CM. Prevention of VTE in nonorthopedic surgical patients: antithrombotic therapy and prevention of thrombosis, 9th ed: American college of chest physicians Evidence-Based clinical practice guidelines. Chest. 2012;141(2 Suppl):eS227–77.
35. Falanga A, Ay C, Di Nisio M, Gerotziafas G, Jara-Palomares L, Langer F, Lecumberri R, Mandala M, Maraveyas A, Pabinger I, et al. Venous thromboembolism in cancer patients: ESMO clinical practice guideline. Ann Oncol. 2023;34(5):452–67.
36. Streiff MB, Holmstrom B, Angelini D, Ashrani A, Elshoury A, Fanikos J, Fertrin KY, Fogerty AE, Gao S, Goldhaber SZ, et al. Cancer-Associated venous thromboembolic disease, version 2.2021, NCCN clinical practice guidelines in oncology. J Natl Compr Canc Netw. 2021;19(10):1181–201.
37. Sagalovich D, Say R, Kaouk J, Mehrazin R. The role of extended venous thromboembolism prophylaxis following urologic pelvic surgery. Urol Oncol. 2018;36(3):83–7.
38. Kukreja JEB. Perioperative venous thromboembolism in urologic oncology procedures, risk factors, and prevention. Curr Opin Urol. 2018;28(3):227–32.
39. Klaassen Z, Arora K, Goldberg H, Chandrasekar T, Wallis CJD, Sayyid RK, Fleshner NE, Finelli A, Kutikov A, Violette PD, et al. Extended venous thromboembolism prophylaxis after radical cystectomy: A call for adherence to current guidelines. J Urol. 2018;199(4):906–14.
40. Ay C, Pabinger I, Cohen AT. Cancer-associated venous thromboembolism: burden, mechanisms, and management. Thromb Haemost. 2017;117(2):219–30.
41. Samama CM, Afshari A. European guidelines on perioperative venous thromboembolism prophylaxis. Eur J Anaesthesiol. 2018;35(2):73–6.
42. Farge D, Frere C, Connors JM, Khorana AA, Kakkar A, Ay C, Muñoz A, Brenner B, Prata PH, Brilhante D, et al. 2022 International clinical practice guidelines for the treatment and prophylaxis of venous thromboembolism in patients with cancer, including patients with COVID-19. Lancet Oncol. 2022;23(7):e334–47.
43. Ramcharitar RK, Man L, Khaja MS, Barnett ME, Sharma A. A review of the past, present and future of Cancer-associated thrombosis management. Heart Int. 2022;16(2):117–23.

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