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A commentary on 'Risk factors for incident venous thromboembolism in patients with renal tumor and inferior vena cava tumor thrombus: A retrospective case–control study'

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Dear Editor,

Despite a recent trend toward earlier diagnosis of renal cell carcinoma (RCC), 4–10% of patients are still diagnosed with advanced disease and tumor invasion into the inferior vena cava[1]. Patients with kidney cancers, specifically RCCs, are at increased risk of venous thromboembolism (VTE), with rates varying between 1.2 and 3.5%[2,3]. To identify preoperative thrombotic risk factors in patients with renal tumor and inferior vena cava tumor thrombus, Wang *et al*.[4] performed a retrospective case–control study and concluded that in addition to the risk factors contributing to mechanical obstruction, a higher level of tumor thrombus, inferior vena cava blockage status, and inferior vena cava wall invasion included, renal sinus fat invasion, higher neutrophils, and serum albumin levels are significant risk factors for preoperative VTE development in patients with renal tumor and inferior vena cava tumor thrombus.

Although hemogram results, such as the platelet count and hemoglobin, were not associated with an increased incidence of VTE, several studies have reported that older age and increased C-reaction protein (CRP) were identified as risk factors for VTE[5]. The CRP is a representative inflammatory marker; an inflammatory state could contribute to VTE through the activation of vascular endothelial cells and adhesion molecules, which could activate the coagulation cascade. Older age is an established risk factor for VTE, which may be associated with underlying illnesses or reduced blood flow and stasis that increase the potential for coagulation. The tumor thrombus itself may induce a procoagulant response mediated by the presence of a foreign body within the vessel lumen, and the coagulation cascade can be further aggravated by the effect of turbulent flow due to venous bloodstream obstruction. Therefore, obviously 'the risk of preoperative VTE' is increased in RCC patients when major retroperitoneal venous structures are involved, and it is not uncommon to detect the coexistence of a bland thrombus and a tumor thrombus at the time of diagnosis.

It is important to note the limitations of the study. First, the misdiagnosis of tumor thrombus is a potential limitation. Tumor thrombus cases were diagnosed on the basis of computed tomographic imaging. Although contrast-enhanced computed tomography and magnetic resonance imaging have equal sensitivities in detecting venous involvement in the renal vein and the inferior vena cava, non-contrast magnetic resonance imaging has a higher sensitivity and specificity in detecting tumor thrombus than non-enhanced computed tomography. Under these circumstances, presumably, many cases of bland thrombus (i.e. VTE-positive cases) may have been misinterpreted as negative for VTE in this study. Moreover, we believe that it is also difficult to establish the exact moment at which the embolic event occurred, especially if these episodes were not associated with overt symptoms (as most are purportedly asymptomatic). Second, propensity score matching to reduce potential bias was not performed owing to the relatively small sample size. Finally, the total population and event rates were low, which could have resulted in an overestimation of the risks.

Ethical approval

Not applicable.

Consent

Not applicable.

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J.F.: wrote the paper; Y.C.: study design; J.W.: data analysis.

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