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Complications - Other

Individualized Risk Model for Venous Thromboembolism After Total Joint Arthroplasty

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ABSTRACT

Background: Venous thromboembolism (VTE) after total joint arthroplasty (TJA) is a potentially fatal complication. Currently, a standard protocol for postoperative VTE prophylaxis is used that makes little distinction between patients at varying risks of VTE. We sought to develop a simple scoring system identifying patients at higher risk for VTE in whom more potent anticoagulation may need to be administered.

Methods: Utilizing the National Inpatient Sample data, 1,721,806 patients undergoing TJA were identified, among whom 15,775 (0.9%) developed VTE after index arthroplasty. Among the cohort, all known potential risk factors for VTE were assessed. An initial logistic regression model using potential predictors for VTE was performed. Predictors with little contribution or poor predictive power were pruned from the data, and the model was refit.

Results: After pruning of variables that had little to no contribution to VTE risk, using the logistic regression, all independent predictors of VTE after TJA were identified in the data. Relative weights for each factor were determined. Hypercoagulability, metastatic cancer, stroke, sepsis, and chronic obstructive pulmonary disease had some of the highest points. Patients with any of these conditions had risk for postoperative VTE that exceeded the 3% rate. Based on the model, an iOS (iPhone operating system) application was developed (VTEstimator) that could be used to assign patients into low or high risk for VTE after TJA.

Conclusion: We believe individualization of VTE prophylaxis after TJA can improve the efficacy of preventing VTE while minimizing untoward risks associated with the administration of anticoagulation.

Venous thromboembolism (VTE) after total joint arthroplasty (TJA) is a devastating complication, feared by both patients and surgeons [1]. In an effort to prevent VTE, numerous prophylactic modalities have been developed, including the introduction of potent anticoagulation drugs in recent years. Many organizations and regulatory bodies have issued statements or guidelines pertinent to this matter. The American College of Chest Physicians was one of the first organizations to issue guidelines for prevention of VTE after TJA [2]. The American College of Chest Physicians guideline has undergone remarkable changes over time, with their recent guidelines accepting mechanical prophylaxis and aspirin as effective VTE prophylaxes after TJA and arthroplasty for hip fracture [3]. The American Academy of Orthopaedic Surgeons has also issued best practice guides for prevention of VTE after TJA [4].

Despite the presence of these guidelines and the availability of numerous VTE prophylactic measures, including many potent drugs, the incidence of VTE after TJA has remained fairly unchanged over the last few decades [5]. Furthermore, it is known whether the administration of anticoagulation prophylaxis is associated with serious complications such as persistent wound drainage, bleeding, hematoma formation, periprosthetic joint infection, and even all-time mortality [6-9]. The question that then arises is what, if any, strategy is needed to improve the efficacy of prophylactic measures

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Table 1
Predictors for VTE That Were Used in the Final Model, Based on the Logistic Regression.

Predictor	Points	Occurrence in NIS Population (%)
Anemia (either blood loss* or deficiency*)	16	15.4
Congestive heart failure*	22	3.04
Coagulation deficiency*	39	1.78
Lymphoma*	30	0.28
Fluid and electrolyte disorders*	45	8.00
Metastatic cancer*	87	0.16
Peripheral vascular disease*	10	1.94
Nonmetastatic solid tumors*	16	1.00
Weight loss*	46	0.36
Chronic pulmonary heart disease (ICD-9416.x)	61	0.54
Blood transfusion (99.01-05)	32	21.70
History of VTE (V12.51)	30	2.91
Myeloproliferative disorders (238.4, 238.71, 289.89.289.9)	38	0.19
Hypercoagulability state (289.81)	100	0.17
Myocardial infarction (410-412)	13	3.69
Varicose veins (454-456)	28	0.49
Fracture (820-829)	43	1.74
Inflammatory bowel disease (555-556)	17	0.40
Sepsis (038,115.2995.91-92,785.52)	72	0.25
Periprosthetic joint infection (996.66)	27	1.24
Atrial fibrillation (427.31)	30	5.81
Stroke (433.x1,434.x1,436)	78	0.13
Apnea (327.2x,780.51,780.53,780.57)	9	6.79
Bilateral joints	21	
Not primary THA	43	
Age	0.267*y over 40	

These are all factors that have a large and statistically significant effect on the rate of in-hospital VTE, adjusted for year. Comorbidity fields that are part of the existing NIS database are marked with a (*). Otherwise, fields defined by ICD-9 codes are indicated by codes in parentheses.
NIS, Nationwide Inpatient Sample; ICD, International Classification of Diseases; VTE, venous thromboembolism; THA, total hip arthroplasty.

while reducing the aforementioned adverse effects associated with the administration of prophylactic anticoagulation.

We believe that VTE prevention needs to take into account the differences in the risk profile of patients undergoing TJA, and prophylaxis needs to be individualized. Although the American Academy of Orthopaedic Surgeon, in their first guidelines, attempted to stratify patients into low- and high-risk groups for VTE, the absence of reliable data prevented the organization from completing this task [10]. Caprini et al [11] have also developed a scoring system that can identify patients at higher risk of VTE in the general surgical population. The Caprini scoring system has taken into account only a handful of risk factors and is not "validated" for the orthopedic patient population. In fact, in this model, all patients undergoing orthopedic procedures are considered to be at a very high risk for development of VTE and are deemed to require a potent anticoagulation.

The propensity to develop VTE after a surgical procedure has also been demonstrated to have a clear genetic element [12]. Although genetic conditions such as Factor V Leiden deficiency, protein C and S deficiency, and a few other conditions have been described, most patients developing VTE following after procedures in general, and TJA in particular, do not seem to have a predetermined genetic risk factor for developing VTE. It appears that VTE can occur in patients without recognized genetic risk factors. Although future research is likely to unravel further genetic contributions to the risk of VTE, at the present time no clear risk stratification for VTE in TJA patients exists. Several studies, however, have attempted to stratify patients undergoing TJA for various complications including VTE [13,14].

The intention of our study was to use national registry and institutional data to further refine the risk model for VTE and provide a simple algorithmic approach for VTE prophylaxis. Numerous risk factors for VTE were evaluated, and using a logistic regression and nomogram model, a relative weight to each risk factor was assigned. Analysis of the available data has allowed the study to design a simple iOS application that can be used to assign each patient into the appropriate risk category for VTE and allow administration of the appropriate prophylactic measure.

Materials and Methods

Using the Nationwide Inpatient Sample (NIS) database, we extracted data for all patients undergoing TJA (International Classification of Diseases, Ninth Revision [ICD-9] codes 81.51, 81.53, 81.54, 81.55, 00.70-00.73, 00.80-00.84) between 2002 and 2011. All patients with pulmonary embolism ([PE]: ICD-9 codes 415.11 or 415.19) or deep vein thrombosis ([DVT] ICD-9 codes 455.1, 451.2, 451.8, 451.9, 453.2, 453.4, 453.8, or 453.9) were identified. To ensure that the predictive model was based on a typical range of patients, we excluded patients younger than 40 years and older than 100 years and those listed as receiving more than one type of

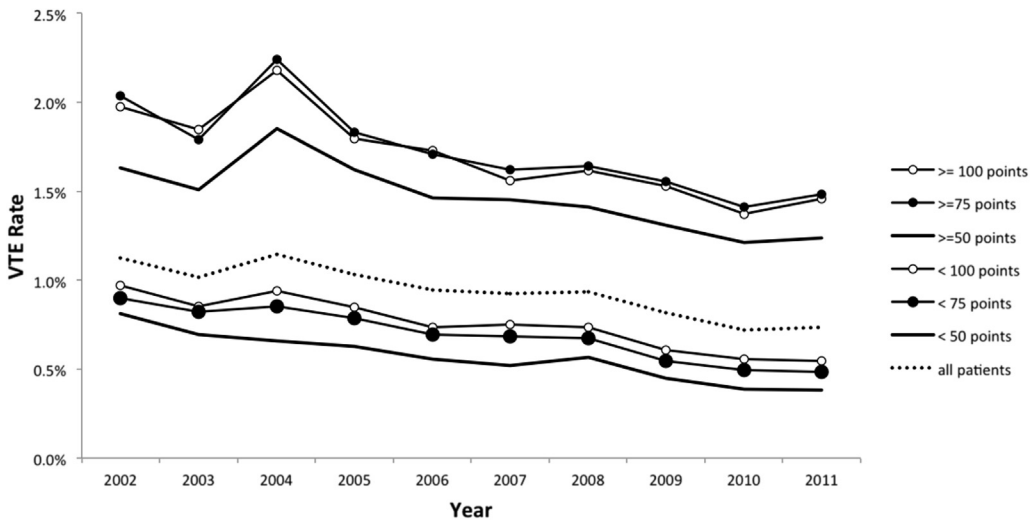

Fig. 1. In-hospital VTE rates per year, overall (dotted line in middle) and for different scoring thresholds. VTE, venous thromboembolism.