Journal of Hematology & Thromboembolic Diseases
ISSN: 2329-8790
OPEN ACCESS Freely available online
Commentary

Brief Note on Epidemiology and Treatment of Venous Thromboembolism

Adel Gouri*

Department of Medicine, Badji Mokhtar University, Annaba, Algeria

DESCRIPTION

The multifactorial condition known as Venous Thromboembolism (VTE) is brought on by inherited or acquired thrombosis predispositions as well as clinical risk factors. It is viewed as a continuum that includes acute Pulmonary Embolism (PE), Deep Vein Thrombosis (DVT), thrombus in transit, and chronic sequel. Post thrombotic Syndrome (PTS) and persistent thromboembolic pulmonary hypertension are two thrombosis sequelae that are often neglected in current therapeutic techniques, which primarily concentrate on the prevention and treatment of acute occurrences Chronic Thromboembolic Pulmonary Hypertension (CTEPH). Following initial VTE, a proportion of patients experience lifetime morbidity, requiring expensive medical treatment. The therapy options for CTEPH have increased as a result of recent research, however PTS prevention continues to be the primary management method. Comprehensive understanding of risk factors, pathobiology, and tailored treatments are the cornerstones of effective chronic complication prevention and treatment, as well as disease recurrence prevention.

Epidemiology

Incidences of VTE in Caucasian patients range from 104 to 183 per 100,000 person-years. PE and Deep Vein Thrombosis (DVT) incidence rates per 100,000 person-years vary from 29 to 78 and 45 to 117, respectively. According to epidemiological estimates, VTE is responsible for about 300,000 deaths annually in the European Union. Observational studies have discovered a number of genetic and environmental risk factors for VTE, many of which are linked to blood flow reduction, vascular damage, or hypercoagulability.

Age, gender, obesity, malignant disease, surgery, trauma, immobility during pregnancy or puerperium, and usage of contraceptives or hormone intake are some of the significant clinical risk factors for VTE.

Up to 25% of people die or have another thromboembolic event within 10 years after their original thromboembolic event as a result of VTE, which frequently recurs. The presence or absence of prothrombotic risk factors at the time of the index VTE has a significant impact on the likelihood of recurrence. Patients who first develop symptomatic VTE in connection with a temporary risk factor (such as surgery) typically have a substantially lower chance of recurrence than patients who get VTE without any apparent cause.

Treatment with anticoagulant for VTE

The three phases of anticoagulant treatment for VTE are initial, chronic, and prolonged. Rapid parenteral anticoagulation (intravenous unfractionated heparin, low-molecular-weight heparin, fondaparinux) initiation characterises the initial phase of treatment. This helps to minimise propagation, recurrence, and death. Vitamin K Antagonists (VKA) are the preferred treatment for the chronic and protracted phase of the condition (except cancer patients). Apixaban, dabigatran, edoxaban, and rivaroxaban are currently all licenced nonvitamin K-dependent oral anticoagulant medicines (NOACs) that are effective and secure therapeutic options for VKA. According to compiled data, these medications are superior in terms of serious bleeding problems and noninferior in terms of VTE recurrence compared to VKA-based therapy regimens. In appropriate patients, NOACs are preferred over traditional VKA for the treatment of DVT or PE. Low-molecular-weight heparins are chosen as a long-term anticoagulant therapy in patients with cancer-associated VTE. Unfortunately, 20% of the time on average, VKA-treated patients are below therapeutic range, which may promote the growth of chronic VTE-related problems. Given that PTS development is linked to time spent outside of the therapeutic range, it has been demonstrated that the therapeutic intensity of VKA treatment is a critical determinant for PTS development. Due to their more consistent pharmacological profiles, NOACs may be able to compensate for VKAs' drawbacks.

Unfortunately, the effects of NOACs on chronic sequelae of VTE have not been covered by the majority of published Phase III studies of NOACs. From the EINSTEIN study, only one post hoc analysis produced results that were neutral with regard to the emergence of PTS. Other modest studies had encouraging findings but were methodologically constrained. Acute PE has a low incidence of CTEPH, and CTEPH may either develop as

Correspondence to: Adel Gouri, Department of Medicine, Badji Mokhtar University, Annaba, Algeria, E-mail: Gouriadel@gmail.com

Received: 22-Jul-2022, Manuscript No. JHTD-22-18524; **Editor assigned:** 27- Jul-2022, Pre Qc No. JHTD-22-18524(PQ); **Reviewed:** 10-Aug-2022, Qc No. JHTD-22-18524; **Revised:** 16-Aug-2022, Manuscript No. JHTD-22-18524 (R); **Published:** 23-Aug-2022, DOI: 10.35248/2329-8790.22.10.487.

Citation: Gouri A (2022) Brief Note on Epidemiology and Treatment of Venous Thromboembolism. J Hematol Thrombo Dis.10.487.

acute PE or without ever exhibiting acute PE symptoms. Further comprehensive assessments of CTEPH as a study endpoint have been discouraged by data that CTEPH may not be avoided by thrombolysis of the acute PE episode. However, neither trials nor registries have addressed the issue of whether NOACs represent an alternate treatment for persistent anticoagulation in CTEPH. NOACs may be linked to post-procedural fresh thrombus at the interventional site, according to observations of patients having Balloon Pulmonary Angioplasty (BPA), suggesting that NOACs may not be the best anticoagulants for BPA patients. The use of NOACs for chronic anticoagulation is encouraged by their advantages, such as higher patient compliance with long-term use, but because of their limited mechanism of action (i.e., excluding the contact activation phase of clotting), it is unclear whether NOACs are a suitable alternative to VKAs in preventing and treating CTEPH.