

A Short Note on Venous Thromboembolism and Sickle Cell Disease

Neeharika Singh*

Department of Microbiology, Delhi University, Delhi, India

DESCRIPTION

Sickle Cell Disease (SCD) is an inherited hemoglobinopathy characterized by intermittent occurrences of hemolysis and vasoocclusion that negatively impact the systemic and pulmonary vasculature. The vasoocclusion in SCD is abnormal relations between erythrocytes, leukocytes, platelets, and the vascular endothelium leading to creation of inflammation, thrombosis, and oxidative stress. As similar, SCD is a hypercoagulable state with abnormalities in platelet and coagulation factors contributing to increased thrombotic threat. Thromboses can do within both the arterial and venous vasculatures. While Vasoocclusive Events (VOE) contributes to increased thrombosis threat, the donation of Venous Thromboembolic Events (VTE) to the overall progression of SCD and threat of mortality is frequently overlooked.

VTE do at increased frequency in cases with SCD compared to the general population, yet the epidemiology and the optimal approach to operation remains unclear. One single-center study plant that 25 of their adult cases with SCD had endured a VTE. Review of the Cooperative Study of Sickle Cell Disease (CSSCD) and California Case Discharge Databases suggest that, by age 40, 11-12 of cases with SCD have endured a VTE and this increases their mortality threat nearly triple. Also, among 877 cases in the California Case Discharge Database with incident VTE, the 1-time and 5- time accretive prevalence of rush was 13.2 (95 CI11.0-15.5) and 24.1 (95 CI21.2-27.1) with a case casualty rate for VTE rush of 3.1 (95 CI,1.0-5.2). For comparison, in the general population of cases with one provoked VTE, the rush rate is 10 at one time and 25 at five times, with 4 of intermittent events performing in death. Taken together, these studies suggest that, in cases with SCD, VTE are common, do at a youngish age than in those without SCD, have a high rush rate, and are

associated with increased mortality, occasionally directly related to this rush. Still, the mileage of these findings is hampered by the lack of specific individual and treatment data; accordingly, our understanding of the correct approach to webbing, opinion, and treatment of VTE in SCD is limited. Bettered understanding of VTE issues in the SCD is important because the American Society of Hematology SCD Cardiopulmonary and Renal clinical guidelines group presently recommends life-long anticoagulation after a first time VTE for individualities with SCD grounded on their overall increased thrombotic threat. While this is supported by the literature, there's concern that this may be associated with an increased hemorrhagic threat. We hypothecated that there's a subset of SCD cases who are at increased threat for VTE and rush and that by relating threat factors for VTE that act in musicale with the known hypercoagulability of SCD in these cases, increased granularity to the understanding of the epidemiology of VTE in SCD could be achieved. A secondary thesis was that a VTE wasn't an insulated event but impacted long-term clinical issues for cases with SCD and that these adverse issues may regard for the observed increased mortality threat.

In conclusion, we've demonstrated a high frequency of VTE and rush in grown-ups with sickle cell complaint, particularly within the first five times of their original event. There appears to be increased VTE threat associated with known VTE threat factors similar as advanced BMI and previous splenectomy and conceivably SCD-specific threat factors similar as leukocytosis which may indicate asub-group of the population for whom lesser clinical alert is warranted. These data may support the need for lifelonganti-coagulation for anon-provoked first time VTE in this population and the need for a lesser understanding of how thrombosis contributes to the pathophysiology of SCD.

Correspondence to: Neeharika Singh, Department of Microbiology, Delhi University, Delhi, India, E-mail: neeharikasingh@gmail.com

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