Patients with traumatic brain injury: Acute deep venous thrombosis due to low vitamin D.

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Short Communication

Venous Thromboembolism (VTE), which manifests as Deep Venous Thrombosis (DVT) and Pulmonary Embolism (PE), has been found to be more common in Traumatic Brain Injury (TBI) populations. In trauma patients coming to the emergency department with TBI, Virchow's triad (stasis, hypercoagulable condition, and endothelial damage) is prevalent. Bleeding in severe trauma promotes the release of coagulation factors, which can contribute to VTE. Physical and cognitive impairments, when combined, increase the likelihood of developing VTE. TBI patients are frequently immobile, weak, and bedridden, and they may also have additional severe injuries that enhance the risk of venous stasis. According to a 2009 research, TBI patients had a three-to-fourfold higher risk of DVT [1]. In neurosurgical patients, the Well's score and number of days in bed were found to be significant predictors of DVT development. Another study found that major general surgery and significant trauma were both high risk factors for the development of VTE, but extended bed rest was just a minor risk factor.

Chemical DVT prevention may be started later in TBI patients due to the danger of increasing cerebral bleeding. Delaying prophylaxis can raise the risk of VTE morbidity and death. Chemoprophylaxis is not recommended for patients with spontaneous ICH growth within the first 72 hours after injury, according to the existing research. Despite the higher frequency of VTE in this patient population, there is no widely acknowledged guideline for chemoprophylaxis treatment [2]. In an acute care hospital, the choice to start low-molecular-weight heparin or unfractionated heparin is frequently decided by the neurosurgery or trauma team. Bradley et al. investigated the impact of starting chemical DVT prevention in TBI patients who were wearing intraparenchymal pressure monitors. They discovered no new haemorrhages or haemorrhage expansion while using low-molecular-weight or unfractionated heparin.

Vitamin D is a necessary vitamin that aids in the maintenance of many vital physiological functions. Adequate vitamin D levels enhance calcium homeostasis, skeletal integrity, and neurodevelopment. High vitamin D levels have been linked to younger age, better nutrition, and greater physical activity in the general population. Deficiency in vitamin D has been related to dementia, depression, type 2 diabetes, autism, and schizophrenia. Most research on optimal vitamin D levels for health focus on fracture prevention and bone mineral density maintenance. According to the findings of this research, blood 25-hydroxy vitamin D (VitD-25OH) concentrations should be more than 75 nmol/L (30 ng/mL) [3]. Adults find it challenging to maintain appropriate amounts of vitamin D without supplementation, owing to current eating standards and sun avoidance for skin cancer protection.

A trend of vitamin D deficiency in TBI populations has been shown by several research. Pellicane et al. conducted two studies in 2010 and 2011 that found a higher incidence of VitD-25OH deficiency in rehab groups, particularly TBI patients. According to a 2010 research, 66 percent of outpatient rehabilitation patients lacked VitD-25OH. Lower vitamin D levels were linked to non-white race, a history of spinal cord injury, TBI, and genetic musculoskeletal disease. According to a comparable research published in 2011, 77% of patients to acute rehabilitation were VitD-25OH inadequate or deficient. Intiso et al. discovered that severe TBI impairment severity was associated to vitamin D deficiency [4]. Low VitD-25OH levels may be caused by poor diet and insufficient sun exposure. However, a secondary phenomenon may develop as a result of TBI's inflammatory processes.

There has been research into the relationship between vitamin D levels and the development of VTE. Low levels of VitD-25OH were linked to idiopathic lower extremity DVT in a 2014 case-control study of 82 individuals. A cross-sectional research published in 2019 compared vitamin D levels in 42 patients with lower extremity DVT or PE to 42 controls. They observed that individuals with VTE had decreased vitamin D concentrations. Previous research has looked into the link between low vitamin D levels and VTE in different groups, such as those in acute rehabilitation. A 2019 study looked into vitamin D insufficiency, supplementation, and the development of VTE in individuals with spinal cord injuries [5]. While there was no significant difference in the development of VTE in VitD-25OH deficient individuals, those who received vitamin D supplements were less likely to develop VTE. In 2018, Wu et al. presented a research that found vitamin D insufficiency to be independently related with the development of DVT in ischemic stroke patients. The goal of this study was to look at the link between vitamin D levels and the development of DVT in TBI patients in acute rehabilitation.

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