maior risco tromboembólico ao paciente. Neste momento, é importante aplicar a dose completa de antigoagulante naqueles pacientes cuja contagem de plaquetas seja superior a 50.000 μL. O grupo de pacientes com nível de plaquetas entre 25.000 μL e 50.000 μL apresenta uma maior variabilidade em relação as possibilidades de tratamento e em relação aos resultados obtidos. De maneira geral, em eventos trombóticos de maior risco, como o tromboembolismo pulmonar ou a trombose venosa profunda proximal, é recomendável utilizar a dose plena de HBPM associada à utilização de transfusão de plaquetas para manter a contagem acima de 50.000 μL. Já nos eventos trombóticos de menor risco, como aqueles em extremidade distal inferior ou relacionados ao uso de cateter, é aconselhável haver uma redução da dose terapêutica do anticoagulante. Naqueles pacientes com nível plaquetário inferior à 25.000 μL, é aconselhável a suspenção da anticoagulação até ser obtido e mantido um nível satisfatório de contagem plaquetária. Por outro lado, para os pacientes com alto risco de sangramento (aqueles com contagem plaquetária inferior à 10.000 μL), mesmo sem evidências de sangramento ativo, a melhor alternativa parece ser a observação clínica sem o emprego de anticoagulantes. **Conclusão:** Não existe conduta unânime em relação ao uso de anticoagulantes para os pacientes em tratamento para câncer que estejam apresentando evento trombótico agudo em vigência de plaquetopenia. Portanto, há necessidade de realização de estudos mais rígidos para consolidar o manejo terapêutico frente a cada situação clínica, considerando os riscos e benefícios.

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CHALLENGES IN PREDICTING THROMBOEMBOLIC RISK IN ACUTE LEUKEMIA: CLINICAL PROFILING AND LIMITATIONS OF CURRENT SCORING SYSTEMS

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Introduction: Patients with newly diagnosed Acute Leukemia (AL) are prone to an increased risk of thrombosis through mechanisms not completely understood. The described incidence of leukemia-associated thrombosis ranges from 2% to 25%, and prior studies addressing its traditional risk factors are scarce and heterogeneous. In other cancers, the Khorana score and the ISTH disseminated intravascular coagulation (DIC) score have been used, with limited applicability in AL. In this study, we aim to identify clinical and laboratory factors that can predict thrombosis in AL patients, as well as validate prior scores and report their clinical profiles and associated findings related to these events. **Methods:** This is a retrospective analysis of a cohort of patients with newly diagnosed AL in a reference cancer center in Brazil, between 2009 and 2022. All AL subtypes were included. Data on epidemiological and laboratory parameters at diagnosis and at the time of a thrombotic event were collected. The primary endpoint was the cumulative incidence of symptomatic venous or arterial thrombosis in the first 60 days from diagnosis, with death as a competing event. Correlation between baseline parameters and thrombotic events was assessed through logistic regression. **Results:** 421 patients were included, 49% diagnosed with AML, 23% with APL, and 28% with ALL. The 60-day cumulative incidence of thrombosis was 15% (95% Confidence Interval [95% CI] 12%–18%), with incidence rates of 21%, 11%, and 20% in ALL, AML, and APL, respectively. The original phenotype was associated with VTE incidence ($p = 0.031$). Most events occurred within the first 30 days (72%), and the remaining cases were detected at disease presentation (24%) or between 30–60 days from diagnosis (4%). Arterial thrombosis represented 8 out of 69 events, all cerebrovascular and more frequent in APL (32%). Regarding venous thrombosis, the most common sites were the upper limbs (47%), catheter-related events (20%) and lower limbs (13%). Prophylactic anticoagulation was administered in 15% of patients with thrombosis. In the ALL subset, 44% of thrombosis occurred after the use of asparaginase. Cerebral Venous Thrombosis (CVT) represented 25% of this subgroup's events. Overall, thrombosis recurred in 5 patients. At the date of thrombosis, 33% of subjects still displayed circulating blasts, and 25% presented less than 30×10^9/L platelets. In univariate analysis, there was no association between baseline clinical and laboratory (biochemical, hematologic, or coagulation) parameters and the risk of thrombosis. Patients presented low, intermediate, and high Khorana risk scores in 3%, 95%, and 2% of cases, respectively, with no statistical association with thrombosis ($p = 0.19$). The ISTH DIC score was "positive"(≥ 5) in 86%, and it was also not associated with thrombotic risk in our cohort ($p = 0.806$). The SiAML-thrombosis score (Owattanapanich et al. Thrombosis Journal, 2023), which includes different cut-offs for WBC, platelets, and D-dimer, was also not predictive of thrombosis ($p = 0.926$). **Conclusion:** In this real-life clinical cohort, we could not identify laboratory parameters associated with an increased risk of thrombosis, despite its high overall incidence. Existing scores showed limited utility in predicting thrombosis. These events occur even in the presence of low platelet counts, prolonged prothrombin time or hypofibrinogenemia, suggesting a complex coagulopathy leading to hypercoagulability in AL. Identifying novel biomarkers of coagulation, particularly related to global coagulation or thromboinflammation, may contribute to a better characterization of thrombotic and bleeding risks in AL. New prospective studies exploring different coagulation parameters are warranted.

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