PB1844 THROMBOTIC EVENTS DURING REMISSION INDUCTION OF ACUTE MYELOID LEUKEMIA – FREQUENCY AND RELATED FACTORS

Topic: 04. Acute myeloid leukemia - Clinical

Wellington Silva1, Raphael Melo1, Fernanda Mendes1, Elvira Velloso1, Vanderson Rocha1, Eduardo Rego1

1 Hematology, Instituto do Cancer do Estado de Sao Paulo, Faculdade de Medicina da Universidade de Sao Paulo, Sao Paulo, Brazil

Background: Thrombotic events in newly diagnosed patients with acute myeloid leukemia (AML) have been considered rare and have not received a great deal of attention. These events may occur at the diagnosis, during the remission induction, or later. The incidence of thromboembolism in AML varied markedly among studies, ranging from 2 to 13%, and are usually divided between arterial and venous events. Limited evidence points that using markers of disseminated intravascular coagulation (DIC) might predict thrombosis in AML, and the Khorana score is not useful for this purpose.

Aims: In this work, we evaluated a cohort of intensively treated adults with non-promyelocytic AML in order to assess the thrombosis rate and baseline factors that might be associated with this event.

Methods: This is a retrospective cohort encompassing newly diagnosed AML adult patients (pts) treated at Instituto do Cancer do Estado de Sao Paulo, Brazil, between June 2011 and June 2020. All patients received the “7+3” regimen as the frontline regimen, and all thrombotic events until day 60 from diagnosis were captured. Logistic regression was used to find risk factors for thrombosis. Cutoffs for numeric variables were defined by Akaike’s information criteria.

Results: Overall, 204 pts were included, with a median age of 54 years (range,17-74). The main baseline features of this cohort are summarized in Table 1. Overall, 24 thrombotic events were registered – the thrombotic rate of 11.9% (95% CI 7.9-17.4%). Most events were catheter-related (9/24), followed by upper (10) and lower (3) limb events. Two patients presented spontaneous jugular vein thrombosis. DIC score (Taylor et al., 2001) was calculated for 107 patients with laboratory data fully available and resulted in overt DIC (≥ 5) in 45.2%. By univariate analysis, morphologic subtype, AML genetic classification, fibrinogen, d-dimer, and DIC score were not associated with thrombosis during induction in AML. Excluding those catheter-related cases, we noticed a higher monocyte count in patients who developed thrombosis (15.8 vs. 4.7x10⁹/L, p=0.009). Incidence of thrombosis in patients with monocyte count >8.5x10⁹/L was 16% vs. 6.2%. The overall 60-day mortality was 25.8%, with no death related to thrombosis. DIC score was not associated with early mortality during AML induction (p=0.19). Remaining coagulation parameters when individually assessed were also not linked with thrombosis or mortality during induction.

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Summary/Conclusion: In this study with intensively treated newly diagnosed AML, we noticed a similar incidence of thrombosis to that previously reported. While most events reported in this cohort were related to catheter devices, spontaneous thrombosis was associated with a higher baseline monocyte count. While monocytic AML may present with DIC at the diagnosis, monocytic cells are a major source of blood tissue factor, pointing it as an independent marker for thrombosis in AML. Our data, although limited, highlights that the understanding of the thrombosis landscape in AML is complex, and the sole use of already recognized markers of coagulation activation might not be sufficient for prediction.