**Title of the manuscript: Relationship between Reticular Fibrosis With Platelet Surface Marker (CD 41A, CD 42A, CD 42B, CD 61) and Prognostic markers (WBC,PLT) in Acute Promyelocytic Leukemia**

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**Conflict of interest:**The authors declare that there is no conflict of interest.

**Ethıcal approval:**Our study was approved by the Inonu University Ethics Committee with the approval number 2022/4216

**AUTHOR CONTRIBUTIONS**

Ahmet Kaya; Conducting the study and article writing process, Mehmet Ali Erkurt; Supervision of the study and article writing process, İrfan Kuku; department approval and Providing support for the study and article writing process, Emin Kaya; Providing support for the study and article writing process, İlhami Berber; Providing support for the study and article writing process, Zehra Bozdağ; Re-examination of pathology preparations and providing pathological support during the writing phase, Soykan Biçim; Providing support for the study and article writing process, Süleyman Arslan; Providing support for the study and article writing process, Fatma Hilal Yagın; providing biostatistical support for the study and article.

**The Novelty Statements**

In this study, it was hypothesized that elevated platelet surface markers on the surface of blasts in acute promyelocytic leukemia trigger reticular fibrosis in the bone marrow, but our study did not support this.

As a result of this study, it was observed that there was no relationship between platelet surface markers on the surface of blasts in acute premyocytic leukemia and WBC and platelet values, which are prognostic indicators, and bone marrow reticular fibrosis. After treatment, there was a regression in the level of reticular fibrosis in some patients, no change in some patients, and progress in some patients. Although it is stated in the literature that bone marrow fibrosis in acute leukemias is the result of cytokine release by blasts, our study did not support this information. In patients who were put into remission by reducing the blast rate after treatment, there were patients whose bone marrow fibrosis did not regress.

Since bone marrow fibrosis is not an expected situation in acute promyelocytic leukemia, this study provides positive data to the literature that bone marrow fibrosis may be present in acute promyelocytic leukemia. It is not parallel to the literature in terms of conflicting results in post-treatment reticular fibrosis and provides new information. The fact that bone marrow fibrosis did not regress in patients who went into remission by reducing the blast rate after treatment has led to the idea that other possible mechanisms other than the release of cytokines from blasts are also effective on bone marrow fibrosis..

In acute promyelocytic leukemia, bone marrow fibrosis is an important factor that prolongs the bone marrow recovery process after treatment and reduces the bone marrow reserve. Elucidating the possible mechanisms of this existing condition during diagnosis may affect the treatment process. Just as bone marrow fibrosis affects the treatment process in acute leukemia, its effect on transplant complications with lower reserves in bone marrow transplantation are issues that need to be clarified.