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EVALUATION OF MEGAKARYOCYTIC QUANTITATIVE STATUS IN PATIENTS PRESENTING WITH THROMBOCYTOPENIA AND THROMBOCYTOSIS AND CORRELATE THESE FINDINGS WITH PERIPHERAL PLATELET COUNTS

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ABSTRACT

Background: Bone marrow megakaryocytes are the precursors of platelets and their quantitative status reflects underlying thrombopoietic activity. Quantitative megakaryocytic assessment in thrombocytopenic and thrombocytotic states may aid in differentiating mechanisms of platelet derangements and guide diagnosis and management.[1] **Objective:** To evaluate the megakaryocytic quantitative status in patients presenting with thrombocytopenia and thrombocytosis and correlate these findings with peripheral platelet counts. **Methods:** In this prospective observational study, bone marrow aspirates from 100 patients were analyzed. Thrombocytopenia (platelet count <150 × 10⁹/L) was observed in 70 patients, while thrombocytosis (platelet count >450 × 10⁹/L) was seen in 30 patients. Quantitative megakaryocytic status was categorized as increased, normal, or decreased based on established cytological criteria. Megakaryocytic counts were correlated with peripheral counts and analyzed using descriptive statistics. **Results:** In thrombocytopenia (n=70), megakaryocytes were increased in 26 (37.1%), normal in 21 (30.0%), and decreased in 23 (32.9%). In thrombocytosis (n=30), megakaryocytes were increased in 26 (86.7%), normal in 2 (6.7%), and decreased in 2 (6.7%). Increased megakaryocytes were significantly more frequent in thrombocytotic samples ($p < 0.001$). Decreased megakaryocytes were relatively common in thrombocytopenia, suggesting hypoproliferative marrow. Normal megakaryocytes were observed across both groups but were less frequent in thrombocytosis.[1–4] **Conclusion:** Quantitative megakaryocytic assessment demonstrates distinct patterns in thrombocytopenia and thrombocytosis, with increased megakaryocytes predominating in thrombocytosis and variable counts in thrombocytopenia. These patterns can help differentiate mechanisms of platelet disorders and support diagnosis.

KEYWORDS

Megakaryocytes, thrombocytopenia, thrombocytosis, bone marrow, thrombopoiesis.

INTRODUCTION

Megakaryocytes are large bone marrow cells responsible for platelet production through a process known as thrombopoiesis, under the regulatory influence of thrombopoietin and other cytokines.[1–3] Peripheral platelet counts are maintained by a dynamic balance between platelet production by megakaryocytes and peripheral destruction/consumption. In clinical hematology, quantitative and qualitative changes in megakaryocytes reflect underlying pathophysiological processes in both thrombocytopenic and thrombocytotic disorders.[1–5]

Thrombocytopenia, defined as a platelet count <150 × 10⁹/L, may result from decreased production, increased destruction, sequestration, or dilution.[1] Conversely, thrombocytosis >450 × 10⁹/L arises from reactive causes (infection, inflammation, iron deficiency) or primary myeloproliferative neoplasms (e.g., essential thrombocythemia).[2] Quantitative megakaryocytic assessment in bone marrow aspirates may distinguish hypoplastic, hyperplastic, or normal megakaryopoiesis and help elucidate disease mechanisms.[2–4]

Despite its diagnostic utility, quantitative megakaryocytic evaluation remains underutilized in routine practice. Published studies have largely focused on qualitative dysplasia rather than systematic quantitation of megakaryocyte numbers in diverse hematological contexts.[3–4] This study aimed to characterize megakaryocytic counts in patients with thrombocytopenia and thrombocytosis and to explore patterns associated with these conditions.

Materials and Methods

The study was conducted in the Post Graduate Department of Pathology in Acharya Shri Chander College of Medical Sciences and Hospital Sidhra, Jammu after obtaining clearance from the Institutional Ethics Committee.

STUDY DURATION

The study was conducted for a period of twenty two months from 1" June 2024 to 31 March 2026.

STUDY DESIGN:

The study is an observational cross- sectional study.

STUDY UNIT

The study subjects included patients with platelet count less than 1,50,000 per microliter or more than 400000 per microliter of blood referred to the department of Pathology in Acharya Shri Chander College of Medical Sciences & Hospital, Jammu.

SAMPLING METHOD:

Simple Random Sampling Technique was used for sample collection for patients visiting the Department of Pathology and fulfilling the inclusion and exclusion criteria.

SELECTION CRITERIA OF PATIENTS:

INCLUSION CRITERIA:

Patients of all genders with a platelet count of less than 1,50,000 per microliter or more than 400000 per microliter of blood.

EXCLUSION CRITERIA

Patients on antiplatelet drugs and other medications causing thrombocytopenia will be excluded. > Patients who have been transfused with platelet concentrate or whole blood in the previous 9 days. > Inadequate bone marrow aspirate.

Each patient's clinical details including history and physical findings were documented. Complete blood count, peripheral smears and relevant laboratory investigations were performed.

COLLECTION OF BLOOD SAMPLE

Under all aseptic precautions, venous blood was collected for the study with Ethylene Diamine Tetra Acetic Acid (EDTA) used as an anticoagulant.

Bone marrow aspirates were obtained from the posterior iliac crest using standard techniques, air-dried, and Leishman-stained. Megakaryocytes were quantified by trained hematopathologists. Megakaryocytic status was categorized as increased, normal, or decreased relative to marrow cellularity and reference counts established in departmental validation studies. Increased megakaryocytes signified hyperplasia suggestive of heightened thrombopoiesis; decreased counts implied hypoplastic marrow production; normal counts indicated baseline megakaryopoiesis.

Statistical Analysis

Descriptive statistics were used. Comparisons between groups (thrombocytopenia vs thrombocytosis) were analyzed using chi-square tests with a significance level set at $p < 0.05$.

Observation and Results

Of the 100 patients studied, 70 presented with thrombocytopenia and 30 with thrombocytosis.

Quantitative Megakaryocytic Findings are summarized in table 1.

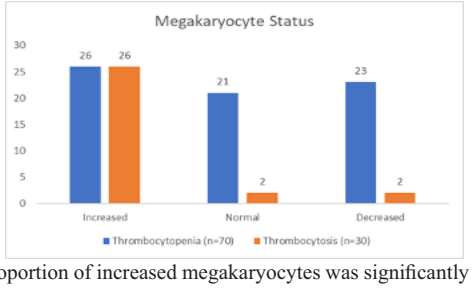
1. Quantitative Megakaryocyte Status in Thrombocytopenia and Thrombocytosis

Megakaryocyte Status	Thrombocytopenia (n=70)	Thrombocytosis (n=30)	p-value
Increased	26 (37.14)	26 (86.67)	0.000003
Normal	21 (30)	2 (6.67)	
Decreased	23 (32.86)	2 (6.67)	

In thrombocytopenia (n=70) , increased megakaryocytes were observed in 37.14% , normal in 30% and decreased in 32.86% of cases. In thrombocytosis (n=30) , increased megakaryocytes predominated with 86.67% , while normal and decreased counts were each 6.67%.

Increased megakaryocytes were 49.53% points higher in Thrombocytosis than Thrombocytopenia. The association between platelet disorder and megakaryocyte status was statistically significant. (p=0.000003)

FIG 1: HISTOGRAM SHOWING QUANTITATIVE MEGAKARYOCUTE STATUS IN THROMBOCYTOPENIA AND THROMBOCYTOSIS



The proportion of increased megakaryocytes was significantly higher in thrombocytosis compared with thrombocytopenia ($p < 0.001$). Decreased megakaryocytes were mainly observed in thrombocytopenia. Increased megakaryocytes in thrombocytopenia may reflect compensatory thrombopoietic response in disorders involving peripheral platelet destruction.

DISCUSSION

Quantitative assessment of megakaryocytes in bone marrow is a key diagnostic parameter that reflects underlying thrombopoietic activity and can help distinguish mechanisms contributing to abnormal platelet counts. Megakaryocytes are the large marrow cells that give rise to platelets through a complex process of proliferation, maturation, polyploidization, and proplatelet shedding, orchestrated primarily by thrombopoietin signaling and other cytokines. Alterations in their counts often correlate with clinical conditions of thrombocytopenia and thrombocytosis, and may serve as a surrogate for marrow thrombopoietic response.[1–4]

In this study, increased megakaryocytes were present in 86.7 % of patients with thrombocytosis, while 37.1 % of thrombocytopenic patients also showed increased megakaryocytes. The high frequency of increased megakaryocytes in thrombocytosis aligns with expected marrow responses in reactive and clonal conditions, where elevated thrombopoietic drive leads to megakaryocytic hyperplasia. This finding parallels observations in thrombocytotic disorders, including essential thrombocythemia and reactive thrombocytosis, where megakaryocytic hyperplasia is a characteristic morphological hallmark.[5–7]

Conversely, the heterogeneous megakaryocytic patterns seen in thrombocytopenia reflect the diverse etiologies underlying low platelet counts. Increased megakaryocytes in thrombocytopenia, seen in over one-third of cases, likely represent a compensatory marrow response to peripheral platelet destruction or immune-mediated clearance such as in immune thrombocytopenic purpura (ITP). Elevated megakaryocyte counts in ITP have been reported in multiple studies, reflecting a marrow effort to offset peripheral platelet loss.[8–10] On the other hand, decreased megakaryocytes (32.9 %) in thrombocytenic patients suggest marrow hypoplasia or impaired thrombopoiesis, often encountered in bone marrow failure, aplastic anemia, or marrow infiltrative disorders. These conditions deprive the marrow of its capacity to produce sufficient megakaryocyte lineage cells, resulting in low platelet output.[11,12] Normal megakaryocyte counts, observed in both groups, may occur in early or mild disease states where marrow compensation has not fully developed or in conditions with balanced production and destruction.[13]

Morphological alterations complement quantitative assessments. Dysplastic megakaryocytes, micromegakaryocytes, hypolobated nuclei, and bare nuclei are frequently reported in thrombocytopenic conditions, particularly in myelodysplastic syndromes (MDS), acute leukemia, megaloblastic anemia, and ITP. Although dysplasia alone cannot confirm MDS, its presence alongside quantitative data enhances diagnostic accuracy when correlated with clinical and hematological parameters.[14–16] Studies have demonstrated that dysplastic features, including nuclear abnormalities, are more specific for MDS but may also appear in non-MDS thrombocytopenic disorders, emphasizing the need for comprehensive interpretation.[14,17] The findings of this study suggest that quantitative megakaryocyte evaluation provides useful insights into the marrow's thrombopoietic activity and may help differentiate between production- vs destruction-dominant causes of thrombocytopenia as well as confirm hyperproliferative states in thrombocytosis. Integrating this parameter with peripheral blood indices, reticulated platelet counts, and clinical parameters enhances diagnostic precision and may inform therapeutic decisions.[8,18]

Several prior studies underscore the importance of megakaryocyte morphology and counts in diagnostic hematology. Muhury et al. demonstrated that detailed marrow examination can differentiate dysplastic patterns in leukemia vs non-myelodysplastic thrombocytopenias.[3] Similarly, cytological alterations in ITP and other causes of thrombocytopenia have been described and may have diagnostic and prognostic significance.[4]

Quantitative megakaryocytic assessment should be integrated with clinical and laboratory findings, including reticulated platelet counts, thrombopoietin levels, and cytogenetics, to optimize diagnostic accuracy.[5] Limitations of this study include its single-center design and lack of correlation with specific etiologies; future studies should expand on disease-specific megakaryocytic signatures.

CONCLUSION

Quantitative megakaryocytic assessment in bone marrow aspirates reveals distinct patterns in thrombocytopenia and thrombocytosis. Increased megakaryocytes predominate in thrombocytosis, reflecting enhanced thrombopoiesis, while thrombocytopenia shows variable megakaryocytic status consistent with diverse pathophysiological mechanisms. Combined with clinical data, megakaryocytic quantitation can enhance diagnostic precision in platelet disorders.

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