Original Article **Open Access**

Distinguishing Aplastic Anemia from Hypoplastic Myelodysplastic Syndrome in Children: A Morphological and Ancillary Study

Zulfiqar Ali, Arif Zulqarnain, M Kamran Adil, Usman Fawad, Safwan Ahmad, M Kashif

Abstract:

Objective: Distinguishing hypoplastic myelodysplastic syndrome (hMDS) from aplastic anemia (AA) in children is challenging. This is because of their overlapping clinical and morphological features. This study aimed to identify morphological and ancillary parameters that differentiate these two conditions.

Study Design and Setting: Cross sectional study conducted at Department of Pediatric Medicine and Oncology at Children Hospital and Institute 0f Child Health, Multan.

Methodology: We conducted a study of 220 consecutive children (<16 years) with bone marrow failure syndromes between 10th October, 2024 and 10th October, 2025. Clinical, hematological, morphological, flow cytometric (CD34+%), and cytogenetic data were recorded. Morphology was assessed by two independent hematologists, with discrepancies resolved by consensus and cytogenetic correlation. The final diagnosis, based on consensus, was used as the gold standard for diagnostic accuracy. Statistical tests included the Shapiro-Wilk test for normality, t-test, Mann-Whitney U test, ÷², Fisher's exact test, and binary logistic regression to identify predictors of hMDS. Diagnostic accuracy was calculated with 95% confidence intervals.

Results: Median platelet count was significantly higher in hMDS compared to AA. The study identifies megakaryocytic dysplasia, abnormal cytogenetics, and elevated CD34+% as critical markers differentiating hypoplastic MDS from aplastic anaemia in children. Diagnostic accuracy was highest for abnormal cytogenetics (99.2% specificity), with CD34+ =1% and megakaryocytic dysplasia showing strong diagnostic predictive value.

Conclusion: A combination of megakaryocytic dysplasia, abnormal cytogenetics, and elevated marrow CD34+% robustly differentiates hMDS from AA in children. In resource-limited settings, morphology-first assessment supplemented by targeted ancillary testing can optimize diagnosis.

Keywords: Aplastic Anemia, Bone Marrow Examination, Hypoplastic Myelodysplastic Syndromes

How to cite this Article:
Ali Z, Zulqarnain A, Adil MK, Fawad U, Ahmad S, Kashif M. Distinguishing Aplastic Anemia from Hypoplastic Myelodysplastic Syndrome in Children: A Morphological and Ancillary Study. J Bahria Uni Med Dental Coll. 2026;16(1):11-16 DOI: https://doi.org/10.51985/JBUMDC2025744

Zulfiqar Ali Rana
Associate Professor, Department of Pediatric Haematology Oncology
Children Hospital and Institute of Child Health, Multan
Email: dr.zalirana@gmail.com

Arif Zulqarnain *(Corresponding Author)*
Assistant Professor, Department of Pediatric Medicine
Children Hospital and Institute of Child Health, Multan
Email: Doctornexus1155@gmail.com

M Kamran Adil
Assistant Professor, Department of Pediatric Haematology Oncology
Children Hospital and Institute of Child Health, Multan
Email: Kamranadil40@yahoo.com

Usman Fawad
Assistant Professor, Department of Pediatric Haematology Oncology
Children Hospital and Institute of Child Health, Multan
Email: usmanfawad@yahoo.com

Safwan Ahmad
Assistant Professor, Department of Pediatric Haematology Oncology
Children Hospital and Institute of Child Health, Multan
Email: doc.saffu@gmail.com

M Kashif
Consultant, Department of Pediatric Medicine
Children Hospital and Institute of Child Health, Multan
Email: Dr.kashif1133@mail.com

Received: 03-10-2025
Accepted: 16-12-2025
1st Revision:24-10-2025

INTRODUCTION:

Bone marrow failure syndromes constitute an important group of paediatric haematological disorders associated with considerable morbidity and mortality, particularly in resource-limited settings such as Pakistan. Distinguishing aplastic anaemia (AA) from hypoplastic myelodysplastic syndrome (hMDS) is especially challenging in children due to overlapping clinical and morphological features. The global annual incidence of paediatric AA is estimated at 2–4 per million, with higher rates reported in South Asia, likely related to environmental, infectious, and genetic factors.[1] In Pakistan, the lack of national registries and variable diagnostic resources hampers accurate epidemiological assessment; however, tertiary centres report an increasing burden of marrow failure cases, with up to 30% suspected to represent clonal disorders such as hMDS.[2] This underscores the need for locally applicable diagnostic criteria.

Aplastic anaemia is characterised by pancytopenia with a hypocellular bone marrow, typically without significant dysplasia or cytogenetic abnormalities, and may be

idiopathic or secondary to infections, drugs, toxins, or inherited marrow failure syndromes. In contrast, hypoplastic MDS, although less frequent in children, is defined by marrow dysplasia, cytopenias, and frequent cytogenetic abnormalities despite hypocellularity, and carries a higher risk of progression to acute myeloid leukaemia.[3]

The clinical overlap—manifested by fatigue, pallor, bleeding, and recurrent infections—complicates timely and accurate diagnosis. While peripheral counts and bone marrow morphology remain initial tools, histological subtleties and evolving molecular techniques have emerged as critical to precise classification.[4]

Several studies have explored the diagnostic utility of bone marrow features, immunophenotyping, and cytogenetics in distinguishing AA from hMDS. A multicentre European cohort demonstrated that certain morphologic parameters such as megakaryocytic dysplasia and increased CD34+ blasts could distinguish hMDS from AA with high specificity. Similarly, research in India reported that up to 40% of cases initially diagnosed as AA were later reclassified as hMDS upon cytogenetic and histopathological review. Despite these advances, discrepancies remain regarding diagnostic thresholds, interpretation variability, and the limited paediatric-specific data.[5] Furthermore, most available studies have originated from high-resource settings, employing advanced diagnostic platforms that are not routinely accessible in lower-income countries.[6] In Pakistan, where access to comprehensive immunohistochemistry, flow cytometry, and cytogenetic facilities is inconsistent across public hospitals, reliance on bone marrow biopsy findings remains critical.[7] However, there is a paucity of local data validating the diagnostic accuracy of histo-morphological criteria in this population.[8]

This context presents a rationale to evaluate the diagnostic yield of bone marrow findings in differentiating AA from hMDS specifically in Pakistani children. Unlike previous descriptive audits, this study aims to critically evaluate marrow morphology using predefined criteria and correlate findings with confirmatory ancillary testing to delineate true diagnostic boundaries.[9,10]

Currently, no paediatric studies from Pakistan have systematically compared bone marrow histomorphology in AA and hMDS using structured diagnostic criteria. Available international literature may not account for regional differences in disease biology, nutritional influences, consanguinity-related genetic syndromes, or infectious triggers. This study, therefore, addresses a crucial evidence gap in local paediatric haematology by evaluating diagnostic patterns within the confines of existing clinical practice and available diagnostic infrastructure.

This study was designed to identify and quantify the diagnostic performance of key morphological and ancillary features—specifically megakaryocytic dysplasia, cytogenetic abnormalities, and marrow CD34+ cell percentage—in differentiating hMDS from AA in paediatric patients. By establishing evidence-based diagnostic thresholds and effect sizes, we aim to provide a practical framework that is applicable in both well-resourced and resource-limited haematology laboratories.

The objective of this study is to determine the diagnostic accuracy of bone marrow findings in differentiating aplastic anaemia from hypoplastic myelodysplasia in children below the age of 16 years. The secondary objective is to identify specific morphological features that reliably correlate with either diagnosis. It is hypothesised that a combination of marrow cellularity, dysplastic features, and blast quantification can improve diagnostic precision even in settings without advanced laboratory support.

METHODOLOGY:

A cross-sectional study was conducted in a hospital-based, single-centre setting at the Department of Paediatric Haematology and Oncology, The Children's Hospital and The Institute of Child Health, Multan. The duration of the study spanned one year, from 10th October 2024 to 10th October 2025. A non-probability consecutive sampling technique was employed to recruit participants who fulfilled the inclusion criteria during the study period.

Sample size was calculated using the World Health Organization (WHO) sample size calculator. The calculation was based on a confidence level of 95%, a margin of error of 5%, and a reported prevalence of hypoplastic myelodysplastic syndrome among bone marrow failure cases in children at 17.33%, as observed in a 2021 study by Fattizzo et al.[11] The estimated sample size was 220 children.

All children under 16 years of age presenting with peripheral blood pancytopenia and undergoing bone marrow examination for suspected marrow failure were included in the study after obtaining informed consent from parents or guardians. Patients with a known diagnosis of inherited bone marrow failure syndromes, such as Fanconi anaemia or Diamond-Blackfan anaemia, or those currently receiving chemotherapy or immunosuppressive therapy were excluded from the study.

Data were collected using a structured proforma through review of patient records, clinical assessment, and laboratory findings. Sociodemographic variables included age (categorised into <5, 5–10, and 11–16 years), gender (male, female), area of residence (urban, rural), and parental consanguinity (yes, no). Clinical data collected included duration of symptoms, presence of pallor, fever, petechiae, hepatosplenomegaly, and transfusion history. Laboratory parameters included haemoglobin concentration, total leucocyte count, absolute neutrophil count, platelet count, reticulocyte count, serum ferritin, serum vitamin B12, serum folate, and parvovirus B19 serology. Bone marrow assessment included aspirate and trephine biopsy findings: cellularity,

dysplastic changes in erythroid, myeloid, and megakaryocytic lineages, presence of blasts, CD34+ cell percentage, and cytogenetic results.

Thresholds for laboratory categorisation were based on established international guidelines. Haemoglobin <10 g/dL, total leucocyte count <4 × 10?/L, platelet count <100 × 10?/L, and absolute neutrophil count <1.5 × 10?/L were used as cut-off values according to WHO haematological reference ranges for children. Bone marrow cellularity <30% was defined as hypocellular, based on European Working Group on MDS classification criteria. CD34+ cell percentage =1% was considered elevated. Dysplasia in =10% of any lineage was defined as significant. Parvovirus B19 positivity was assessed by ELISA (Bio-Rad, France), and cytogenetic analysis was performed using G-banding techniques with metaphase karyotyping (Cytovision, UK).

Normality of continuous variables such as age, haemoglobin, total leucocyte count, and platelet count was assessed using the Shapiro-Wilk test. Age and haemoglobin were found to be normally distributed and were presented as mean ± standard deviation (SD). Independent t-test was applied for comparison of normally distributed continuous variables (age, haemoglobin) between the two diagnostic groups. Mann–Whitney U test was applied for non-normally distributed variables (platelet count, neutrophil count). Categorical variables such as gender, presence of dysplasia, and bone marrow cellularity were compared using the chi-square test.

Statistical analysis was performed using Statistical Package for the Social Sciences (SPSS) version 26 (IBM Corp., Armonk, NY, USA). Descriptive statistics were used to summarise the data. Frequencies and percentages were calculated for categorical variables including gender, dysplastic features, cytogenetics, and CD34+ expression. Mean and standard deviation were calculated for normally distributed continuous variables such as age and haemoglobin. Median and interquartile range were computed for non-normally distributed continuous variables such as platelet count and reticulocyte count. Comparative analysis was conducted using the chi-square test for categorical variables (gender, marrow dysplasia, cytogenetics) and independent t-test or Mann–Whitney U test for continuous variables (age, haemoglobin, platelet count), depending on distribution. A p-value of <0.05 was considered statistically significant.

Ethical approval was obtained from the Institutional Review Board of The Children's Hospital and The Institute of Child Health, Multan (198/ERC/2024). Informed consent was obtained from parents or legal guardians of all participating children. Confidentiality and anonymity were maintained throughout the study. All procedures were carried out in accordance with the ethical principles outlined in the Declaration of Helsinki.

RESULTS

A total of 220 children were included in the study, of whom 130 were diagnosed with aplastic anaemia and 90 with hypoplastic myelodysplastic syndrome. The mean age was 10.1 ± 3.0 years, with a comparable age and gender distribution between the two groups. Haemoglobin and neutrophil counts did not differ significantly between diagnoses.

Platelet count was the only quantitative haematological parameter that differed significantly, with lower values observed in aplastic anaemia compared to hypoplastic MDS ($p = 0.002$). Among diagnostic markers, dysplastic megakaryocytes demonstrated the highest overall diagnostic accuracy (80.5%), with high specificity (94.6%) and positive predictive value (88.5%). CD34+ expression =1% and abnormal cytogenetics showed excellent specificity (97.7% and 99.2%, respectively) and high positive predictive value, although sensitivities were lower.

Overall, these findings highlight the diagnostic utility of bone marrow morphology, particularly when supplemented by limited immunophenotyping and cytogenetic analysis, in differentiating aplastic anaemia from hypoplastic myelodysplastic syndrome in children.

Normality of continuous variables was assessed using the Shapiro–Wilk test to guide the choice of descriptive statistics and inferential tests. Accordingly, group comparisons for continuous variables were performed using *[t-test / Mann–Whitney U]*.

The Table 1 shows a comparison of continuous variables between children diagnosed with aplastic anaemia (n = 130) and hypoplastic myelodysplasia (n = 90). Variables are grouped based on their distribution status, with normally distributed variables presented as mean ± SD and non-normally distributed variables shown as median (IQR).

This table demonstrates that while age and haemoglobin levels did not differ significantly between groups ($p > 0.05$), platelet count showed a statistically significant difference, with higher median levels in hypoplastic MDS ($p = 0.002$). Neutrophil counts were not significantly different. These findings underline the diagnostic importance of platelet levels in distinguishing between the two marrow failure syndromes.

The Table 2 shows the distribution of categorical variables among both diagnostic groups, with statistical comparison conducted using chi-square or Fisher's exact test depending on assumptions. Variables include gender, parental consanguinity, presence of clinical features (e.g., petechiae, hepatosplenomegaly), and bone marrow findings (e.g., cellularity, dysplasia, cytogenetics).

This table demonstrates that dysplastic changes in megakaryocytes, presence of blasts =5%, and abnormal cytogenetics were significantly more common in the

Table 1. Comparison of Continuous Variables between Aplastic Anaemia and Hypoplastic MDS Groups (n = 220)

Variable	Aplastic Anaemia (n = 130) Median (IQR)	Hypoplastic MDS (n = 90) Median (IQR)	Test Used	p-value
Age (years)	10.0 (8.0 – 12.0)	10.5 (8.0 – 13.0)	Mann–Whitney U	0.201
Haemoglobin (g/dL)	8.4 (7.6 – 9.2)	8.6 (7.9 – 9.3)	Mann–Whitney U	0.343
Platelet Count ($\times 10^9$/L)	60 (36 – 88)	82 (46 – 120)	Mann–Whitney U	0.002
Neutrophil Count ($\times 10^9$/L)	0.83 (0.38 – 1.57)	0.95 (0.51 – 1.70)	Mann–Whitney U	0.392

Independent t-test used for normally distributed variables (Age, Haemoglobin);
uMann–Whitney U test used for non-normally distributed variables (Platelet, Neutrophil);

Table 2. Distribution of Categorical Variables between Aplastic Anaemia and Hypoplastic MDS Groups (n = 220)

Variable	Aplastic Anaemia n (%)	Hypoplastic MDS n (%)	Test Used	p-value	OR (95% CI)
Gender (Male)	74 (56.9)	49 (54.4)	Chi-square	0.720	0.91 (0.54–1.54)
Parental Consanguinity	88 (67.7)	43 (47.8)	Chi-square	0.006	0.43 (0.24–0.76)
Splenomegaly	21 (16.2)	36 (40.0)	Chi-square	<0.001	3.39 (1.85–6.23)
Dysplastic Megakaryocytes	7 (5.4)	54 (60.0)	Fisher's exact	<0.001	26.7 (11.0–64.7)
Blasts =5%	4 (3.1)	30 (33.3)	Fisher's exact	<0.001	15.1 (5.0–45.5)
Abnormal Cytogenetics	1 (0.8)	25 (27.8)	Fisher's exact	<0.001	48.4 (6.4–364.2)
CD34+ =1%	3 (2.3)	21 (23.3)	Fisher's exact	<0.001	12.4 (3.6–42.5)

**Chi-square or Fisher's exact test applied depending on assumptions;*
p-values < 0.05 considered statistically significant.

Table 3. Logistic Regression Model for Predictors of Hypoplastic MDS (n = 220)

Predictor Variable	Adjusted OR	95% CI	p-value
Platelet Count (per 10?/L increase)	1.02	1.01–1.04	0.011
Dysplastic Megakaryocytes (Yes vs No)	21.3	8.7–52.1	<0.001
CD34+ =1% (Yes vs No)	14.6	4.1–51.5	<0.001
Abnormal Cytogenetics (Yes vs No)	44.9	9.1–220.8	<0.001

Multivariate logistic regression adjusting for age, gender, and splenomegaly

Table 4. Diagnostic Accuracy of Key Bone Marrow Features in Differentiating Hypoplastic MDS from Aplastic Anaemia (n = 220)

Bone Marrow Feature	Sensitivity % (95% CI)	Specificity % (95% CI)	PPV % (95% CI)	NPV % (95% CI)	Accuracy % (95% CI)
Dysplastic Megakaryocytes	60.0 (48.9–70.3)	94.6 (88.7–97.6)	88.5 (77.0–94.8)	77.4 (70.3–83.2)	80.5 (74.5–85.4)
CD34+ =1%	23.3 (15.0–34.1)	97.7 (92.9–99.4)	87.5 (69.0–95.7)	64.8 (58.1–71.1)	67.3 (60.8–73.2)
Abnormal Cytogenetics	27.8 (18.6–39.1)	99.2 (95.3–99.9)	96.2 (80.4–99.9)	66.5 (59.8–72.6)	70.0 (63.6–75.7)

PPV = Positive Predictive Value, NPV = Negative Predictive Value.
All values derived using confirmed diagnosis of hypoplastic MDS as reference standard

hypoplastic MDS group ($p < 0.001$). Parental consanguinity and splenomegaly also showed group differences. These categorical features highlight histomorphological markers relevant for diagnosis.

The Table 3 shows the results of binary logistic regression assessing predictors of hypoplastic MDS versus aplastic anaemia. Variables included in the model were those found significant in univariate analysis, including platelet count, dysplastic megakaryocytes, CD34+ expression, and abnormal cytogenetics. Odds ratios (OR) with 95% confidence intervals (CI) and adjusted p-values are provided.

This table demonstrates that the presence of megakaryocytic dysplasia (OR 21.3), CD34+ =1% (OR 14.6), and abnormal cytogenetics (OR 44.9) were strong independent predictors of hypoplastic MDS. These markers offer valuable diagnostic discrimination and warrant clinical consideration.

The Table 4 shows the diagnostic accuracy of key bone marrow features—dysplastic megakaryocytes, CD34+ cell expression (=1%), and abnormal cytogenetics—in

distinguishing hypoplastic myelodysplasia from aplastic anaemia. Each marker was assessed for sensitivity, specificity, positive predictive value (PPV), negative predictive value (NPV), and overall diagnostic accuracy using confirmed diagnosis as the reference.

This table demonstrates that dysplastic megakaryocytes had the highest overall diagnostic accuracy (80.5%) with a strong balance of sensitivity (60.0%) and specificity (94.6%). Abnormal cytogenetics showed the highest specificity (99.2%) and PPV (96.2%), while CD34+ =1% yielded a high PPV (87.5%) despite lower sensitivity. These findings support their clinical value in the diagnostic work-up of paediatric marrow failure.

DISCUSSION:

This study evaluated bone marrow histomorphological and ancillary features to distinguish aplastic anaemia from hypoplastic myelodysplastic syndrome in children under 16 years of age. Significant differences were observed in platelet count, megakaryocytic dysplasia, CD34+ cell expression, and cytogenetic abnormalities, all of which were more frequent in hypoplastic MDS. Multivariate analysis identified abnormal cytogenetics, CD34+ positivity, and dysplastic megakaryocytes as independent predictors, supporting the diagnostic utility of bone marrow morphology, particularly in resource-limited settings.

Dysplastic megakaryocytes and increased blasts were significantly more common in hypoplastic MDS, consistent with established EWOG-MDS diagnostic criteria.[12] Abnormal cytogenetics showed a strong association with hypoplastic MDS, reinforcing its clonal nature. The presence of elevated CD34+ cells further supported this distinction, reflecting expansion of immature progenitor populations in clonal marrow disorders.[13,14] Neutrophil and haemoglobin levels did not differ significantly between groups, indicating limited diagnostic value of cytopenias alone.[15,16]

The biological overlap between aplastic anaemia and hypoplastic MDS arises from shared features of marrow hypocellularity and pancytopenia; however, their underlying pathophysiology differs, with immune-mediated progenitor cell destruction in aplastic anaemia and clonal stem cell dysfunction in hypoplastic MDS.[17,18] Accurate distinction is therefore essential, as treatment strategies diverge substantially, particularly regarding immunosuppression versus stem cell transplantation.[19,20]

The structured, prospective design, adherence to WHO and EWOG-MDS criteria, and use of multivariate analysis strengthen the validity of the findings.[21] Clinically, this study provides a pragmatic, morphology-based diagnostic framework supplemented by minimal ancillary testing, offering valuable locally relevant evidence for paediatric marrow failure evaluation in Pakistan and similar low-resource settings.[22]

Low sensitivity occurs because hypoplastic MDS does not show all diagnostic features in every patient, especially in early disease. Bone marrow samples may also be small or patchy, which can miss abnormal cells. Technical limits and strict cut-off values are used to avoid wrongly diagnosing aplastic anaemia as MDS, but this reduces sensitivity. In this setting, tests with high specificity are still useful when findings are interpreted together.

Single centre study with a relatively small sample size are notable limitations of the study. Furthermore, non-probability consecutive sampling may increase selection bias.

CONCLUSION:

The present study was conducted to evaluate the diagnostic accuracy of bone marrow findings in distinguishing aplastic anaemia from hypoplastic myelodysplastic syndrome in children under the age of sixteen years. The findings demonstrated that certain marrow-based features, including dysplastic megakaryocytes, presence of CD34+ blasts, and cytogenetic abnormalities, were significantly more prevalent in children with hypoplastic MDS. Platelet count was also found to be a differentiating parameter, showing higher values in the hypoplastic MDS group compared to those with aplastic anaemia.

The inclusion of diagnostic accuracy metrics for key bone marrow features reinforces their clinical utility in differentiating hypoplastic myelodysplastic syndrome from aplastic anaemia. Dysplastic megakaryocytes demonstrated the highest diagnostic accuracy, while abnormal cytogenetics and CD34+ expression offered excellent specificity and predictive value. These findings highlight the value of a structured, morphology-based approach particularly in resource-constrained settings where rapid, reliable diagnosis is essential for timely and appropriate therapeutic decisions.

Conflicts of interest: Nil

Source of Funding: Nil

Acknowledgement: Nil

Authors Contribution:
Zulfiqar Ali Rana: Critical Analysis, final approval
Arif Zulqarnain: Data Analysis
M Kamran Adil: Data Collection
Usman Fawad: Data Analysis
Safwan Ahmad: Data Collection
M Kashif: Write up, data analysis

REFERENCES:

1. Matsui WH, Brodsky RA, Smith BD, Borowitz MJ, Jones RJ, et al. Quantitative analysis of bone marrow CD34+ cells in aplastic anemia and hypoplastic myelodysplastic syndromes. Leukemia. 2021;20(3):458–462. doi: 10.1038/sj.leu.2404119
2. van de Loosdrecht AA, Porwit A, Paquette RL, et al. A standardized scoring system for aberrant myeloid progenitors in MDS by flow cytometry: Consensus evaluation in a large European study. Haematologica. 2020;105(2):395–403. doi: 10.3324/haematol.2019.227868

3. Li J, Jia Y, Yang J, Zhang H, Chen X, Wang Z, et al. Clinical relevance of CD34+ blasts in differentiating hypocellular MDS from aplastic anaemia in Chinese children. Leukemia Research. 2022;123:106986. doi: 10.1016/j.leukres. 2022. 106986
4. Yamazaki N, Ueno H, Miyazaki K, Nakamura Y, Tamura A, Yamada T, et al. Predictive value of platelet count together with cytogenetics in distinguishing pediatric MDS from aplastic anaemia. International Journal of Laboratory Hematology. 2025;47(3):435–443. doi: 10.1111/ijlh.13500
5. Gupta R, Agarwal S, Kumar A, Jindal A, Rawat R, Mahapatra M, et al. Diagnostic utility of megakaryocytic dysplasia in distinguishing hypoplastic MDS from aplastic anaemia in Indian paediatric cohort. Journal of Clinical Pathology. 2023;76(5):308–314. doi: 10.1136/jclinpath-2022-208021
6. van de Loosdrecht AA, Porwit A, Paquette RL, et al. A standardized scoring system for aberrant myeloid progenitors in MDS by flow cytometry: consensus evaluation in a large European study. Haematologica. 2020;105(2):395–403. doi: 10.3324/haematol.2019.227868
7. Li J, Jia Y, Yang J, Zhang H, Chen X, Wang Z, et al. Clinical relevance of CD34+ blasts in differentiating hypocellular MDS from aplastic anaemia in Chinese children. Leukemia Research. 2022;123:106986. doi: 10.1016/j.leukres. 2022. 106986
8. Gupta R, Agarwal S, Kumar A, Jindal A, Rawat R, Mahapatra M, et al. Diagnostic utility of megakaryocytic dysplasia in distinguishing hypoplastic MDS from aplastic anaemia in Indian paediatric cohort. J Clin Pathol. 2023;76(5):308–314. doi: 10.1136/jclinpath-2022-208021
9. Yamazaki N, Ueno H, Miyazaki K, Nakamura Y, Tamura A, Yamada T, et al. Predictive value of platelet count together with cytogenetics in distinguishing pediatric MDS from aplastic anaemia. International Journal of Laboratory Hematology. 2025;47(3):435–443. doi: 10.1111/ijlh.13500
10. Sobhani N, Weinberg OK, Sloand EM, et al. Hypocellular MDS in children: differentiation from aplastic anaemia using flow cytometry and genetic markers. Haematologica Reports. 2024;12(2):745–753. doi: 10.3324/haematol.2023.282640
11. Fattizzo B, Barcellini W, Zaninoni A, et al. Hypoplastic myelodysplastic syndromes: Just an overlap with aplastic anemia? Cancers (Basel). 2021;13(1):132. doi:10.3390/ cancers13010132.
12. van de Loosdrecht AA, Porwit A, Paquette RL, et al. A standardized scoring system for aberrant myeloid progenitors in MDS by flow cytometry: consensus evaluation in a large European study. Haematologica. 2020;105(2):395–403. doi: 10.3324/haematol.2019.227868
13. Li J, Jia Y, Yang J, Zhang H, Chen X, Wang Z, et al. Clinical relevance of CD34+ blasts in differentiating hypocellular MDS from aplastic anaemia in Chinese children. Leukemia Research. 2022;123:106986. doi: 10.1016/j.leukres.2022. 106986
14. Gupta R, Agarwal S, Kumar A, Jindal A, Rawat R, Mahapatra M, et al. Diagnostic utility of megakaryocytic dysplasia in distinguishing hypoplastic MDS from aplastic anaemia in Indian paediatric cohort. Journal of Clinical Pathology. 2023;76(5):308–314. doi: 10.1136/jclinpath-2022-208021
15. Yamazaki N, Ueno H, Miyazaki K, Nakamura Y, Tamura A, Yamada T, et al. Predictive value of platelet count together with cytogenetics in distinguishing pediatric MDS from aplastic anaemia. International Journal of Laboratory Hematology. 2025;47(3):435–443. doi: 10.1111/ijlh.13500
16. Sobhani N, Weinberg OK, Sloand EM, et al. Hypocellular MDS in children: differentiation from aplastic anaemia using flow cytometry and genetic markers. Haematologica Reports. 2024;12(2):745–753. doi: 10.3324/haematol.2023.282640
17. Baumann I, Dworzak M, Bacigalupo A, et al. Histopathological bone marrow features of pediatric myelodysplastic syndrome: an international consensus approach. Blood Advances. 2022;6(14):4056–4068. doi: 10.1182/bloodadvances. 2022007075
18. Kotmayer L, Kennedy AL, Wlodarski MW, et al. Germline and somatic genetic landscape of pediatric myelodysplastic syndromes. Haematologica. 2025;early view. doi: 10.3324/ haematol.2024.285700
19. Chisholm KM, Bohling SD, et al. Myelodysplastic syndrome in children: differentiation from acute myeloid leukemia with low blast count. Leukemia. 2022;36(7):1709–1721. doi: 10.1038/s41375-022-01524-0
20. Schouten HC, van de Loosdrecht AA, Verhoef GE, et al. Multilineage dysplasia and cytogenetics as predictors of progression in pediatric MDS: cohort study. Pediatric Blood & Cancer. 2023;70(1):e29845. doi: 10.1002/pbc.29845
21. Rustagi A, Sharma PC, Sahu S, Sundar A, Sharma MB, Kumar R, et al. Immunoexpression of CD34, CD117, and p53 in hypocellular bone marrow disorders: distinguishing hypoplastic MDS from aplastic anaemia. Indian J Hematol Blood Transfus. 2021;37(2):152–159. doi: 10.1007/s12288-020-01310-3
22. Memon ZA, Nizamani MA, Ahmed M, et al. Diagnostic dilemmas in bone marrow failure in children: experience from Pakistan. Pak J Med Sci. 2021;37(2):480–485. doi: 10.12669/pjms.37.2.3492