**RESEARCH ARTICLE**
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# Validation and Modeling of Flow Cytometric CD36 Coefficient of Variation (CV) Analysis in the Diagnosis of Lower-Risk Myelodysplastic Syndromes

# Düşük Risk Myelodisplastik Sendrom Tanısında Akım Sitometrik CD36 Varyans Katsayısı Analizinin Validasyonu ve Modellenmesi



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## Abstract

**Objective:** Myelodysplastic syndromes (MDS) are clonal hematopoietic disorders in which flow cytometry plays a limited yet evolving role in diagnosis. Recent studies have identified CD36 coefficient of variation (CV) as a potential marker of dyserythropoiesis. This study aimed to validate the diagnostic utility of CD36 CV in a local cohort, establish control-based cutoff values, and assess the added value of CD36 CV when integrated into the Ogata score for improved detection of lower-risk MDS.

**Materials and Methods:** In this retrospective study, 82 patients who underwent bone marrow aspiration for unexplained cytopenia were analyzed using multiparametric flow cytometry, cytogenetics, and morphological assessment. CD36 CV was measured from erythroid precursors, and diagnostic thresholds were determined based on the distribution of values in the control group. Diagnostic models included CD36 CV alone, a combined binary model with Ogata score, and an expanded five-point scoring system.

**Results:** CD36 CV values were numerically higher in MDS patients (mean 75.81) compared to controls (mean 65.84), though not statistically significant (p=0.099). For low-risk MDS, the 75th percentage cutoff yielded 60% sensitivity and 80% specificity. Integration of CD36 CV into the Ogata score improved specificity from 33.3% to 80% at a ≥3-point threshold, with an AUC of 0.754 (p=0.003). Models using higher cutoffs demonstrated lower sensitivity.

**Conclusion:** Incorporating CD36 CV into flow cytometric evaluation enhances diagnostic specificity for lowerrisk MDS without requiring additional antibody panels. This locally validated marker may improve diagnostic accuracy when combined with myelomonocytic immunophenotyping. Standardization across institutions remains necessary for broader applicability.

**Keywords:** myelodysplastic syndromes, flow cytometry, diagnosis, CD36, erythroid dysplasia

## Özet

**Amaç:** Myelodisplastik sendrom (MDS) klinik, morfolojik ve sitogenetik özelliklerin bir arada değerlendirilmesi ile tanı konulan klonal hematopoetik bir hastalıktır. Kemik iliğinin akım sitometrik immunfenotiplemesi ve bu açıdan geliştirilen skorlama sistemleri tanıyı destekleyici bir

rol üstlenmektedir. Son çalışmalar, CD36 varyans katsayısını (coefficient of variation-CV) diseritropoez için potansiyel bir biyobelirteç olarak tanımlamıştır. Bu çalışmanın amacı, CD36 CV'nin tanısal değerini yerel bir kohortta doğrulamak, kontrol grubuna dayalı eşik değerler belirlemek ve CD36 CV'nin Ogata skoruna entegre edilmesiyle düşük riskli MDS'nin tanısal performansına olan katkısını değerlendirmektir.
**Gereç ve Yöntem:** Bu retrospektif çalışmaya, açıklanamayan sitopeni nedeniyle kemik iliği aspirasyonu yapılan 82 hasta dahil edildi. Hastalar multiparametrik akım sitometrisi, sitogenetik ve morfolojik inceleme ile değerlendirildi. CD36 CV değerleri eritroid öncüller üzerinde ölçülerek eşik değerler kontrol grubundaki dağılıma göre belirlendi. Tek başına CD36 CV, CD36 CV ile Ogata skorunun ikili kombinasyonu ve genişletilmiş beş puanlı bir skorlama sistemi şeklinde oluşturulan tanısal modeller çeşitli eşik değerler üzerinden test edildi.
**Bulgular:** CD36 CV değerleri, MDS hastalarında (ortalama 75.81) kontrol grubuna (ortalama 65.84) kıyasla sayısal olarak daha yüksek olmasına rağmen, istatistiksel olarak anlamlı bulunmamıştır (p=0.099). Düşük riskli MDS için 75. persentil eşik değeri %60 duyarlılık ve %80 özgüllük sağlamıştır. CD36 CV'nin Ogata skoruna entegrasyonu, ≥3 puanlık eşik değerinde özgüllüğü %33.3'ten %80'e yükseltmiş ve AUC değeri 0.754 (p=0.003) olarak bulunmuştur. Daha yüksek eşik değerleriyle oluşturulan modellerde duyarlılık daha düşük olmuştur.
**Sonuç:** CD36 CV'nin akım sitometrik değerlendirmeye dahil edilmesi, ek antikor panellerine ihtiyaç duymadan düşük riskli MDS tanısına katkı sağlamaktadır. Lokal validasyonu sağlanan bu belirteç, miyelomonositik immünfenotipleme ile birlikte kullanıldığında tanısal doğruluğu iyileştirebilir. Ancak bu yöntemin daha geniş ölçekte uygulanabilmesi için kurumlar arası standardizasyon gereklidir.

## 1. Introduction

Myelodysplastic syndromes (MDS) are clonal hematopoietic disorders diagnosed through an integrated assessment of clinical, morphological, and cytogenetic features. Risk stratification is based on the percentage of bone marrow blasts and the presence of specific molecular and cytogenetic abnormalities. (1)

Due to the lack of global standardization in flow cytometric analysis, immunophenotyping currently plays only a supportive role in MDS diagnostics. Most flow cytometric scoring systems are based on evaluating the myelomonocytic lineage, and standardized protocols for assessing dyserythropoiesis remain under development. (2)

The Ogata score is one of the most widely used flow cytometric tools for the diagnosis of myelodysplastic syndromes (MDS). It evaluates four parameters, each assigned one point if the abnormal threshold is met: the proportion of $CD34^+$ myeloid progenitors among total nucleated cells (≥ 2%), the percentage of B-cell precursors among $CD34^+$ cells (≤ 5%), the mean fluorescence intensity (MFI) ratio of CD45 in lymphocytes to myeloblasts (≤ 4 or ≥ 7.5), and the side scatter (SSC) ratio of granulocytes to lymphocytes (≤ 6). A total score of 2–4 points is considered supportive of MDS, with reported sensitivity of approximately 69% and specificity of 92%. (3,4) The enriched or extended Ogata score incorporates additional aberrant immunophenotypic features, by adding 1 point when CD5 or CD7 was expressed on myeloid progenitors or CD56 on monocytes with a threshold of 30% positive cells, thereby improving sensitivity in MDS(5). More recent studies have also confirmed the prognostic relevance of the Ogata score and validated its diagnostic performance in different patient cohorts.(6,7)
During normal erythropoiesis, early erythroid progenitors express high levels of transferrin receptor (CD71) and stem cell factor receptor (CD117), which progressively decrease as cells mature, while CD36 expression becomes more homogeneous. In parallel, the mean fluorescence intensity (MFI) of CD71 declines and the proportion of CD117-positive cells is reduced as differentiation proceeds, reflecting the transition from immature to mature erythroid stages. (8) CD36 (thrombospondin receptor) is a transmembrane glycoprotein expressed on various hematopoietic and non-hematopoietic cells. It is involved in angiogenesis, lipid metabolism, apoptosis, and thrombosis, depending on its ligands. (9) During erythropoiesis, CD36 is expressed from the proerythroblast stage, progressively downregulated during maturation, and absent in reticulocytes. Increased flow cytometric coefficient of variation (CV) of CD36, indicating surface expression heterogeneity, has been identified as a potential marker of dyserythropoiesis. (10)
In patients with myelodysplastic syndromes, these physiological patterns are altered. An increased coefficient of variation (CV) of CD36 or CD71 indicates a broader heterogeneity of antigen expression among erythroid precursors, consistent with dysplastic maturation. A decreased CD71 MFI reflects abnormally low transferrin receptor density, suggesting impaired proliferative activity or defective iron uptake. Similarly, an increased proportion of CD117-positive cells denotes a relative accumulation of immature erythroid progenitors, a hallmark of ineffective erythropoiesis (2). A

multicenter study identified CD36-CV, CD71-CV, CD71 mean fluorescence intensity (MFI), and CD117 positivity as key markers associated with erythroid dysplasia. Incorporating these into myelomonocytic flow cytometric evaluation improved the diagnostic sensitivity of the Ogata score from 76% to 84%. (2,11)

In this study, we aimed to validate the diagnostic relevance of elevated CD36 CV in MDS patients in a local cohort, establish a locally applicable cutoff value by comparison with anemic controls, and evaluate the performance of a revised scoring model integrating CD36 CV into the Ogata system.

## 2. Materials and Methods

### 2.1. Patients

This retrospective study included patients who underwent bone marrow aspiration between January 2019 and June 2024 due to unexplained cytopenias and were evaluated by multiparametric flow cytometry. Cytogenetic and morphological analyses were also performed as part of the diagnostic workup. Following the diagnostic workup, according to the WHO 2016 classification, a diagnosis of MDS was established if morphological evaluation of the bone marrow aspirate revealed ≥10% dysplasia in at least one myeloid lineage, or, in cases of equivocal dysplasia, if one of the defining cytogenetic/molecular genetic markers described in the same WHO criteria was present. (12) Clinical, demographic, and laboratory data were extracted from electronic medical records. Patients who received a diagnosis of MDS formed the study group, while those in whom MDS was excluded—based on alternative, non-hematological causes of anemia—constituted the control group. The control group consisted of patients with cytopenias and pathological findings suggestive of a preliminary diagnosis of MDS, while a healthy control group was not included in the study. Upon retrospective evaluation, 15 patients with flow cytometry data deemed sufficient for analysis were allocated to the control group, whereas 67 patients were included in the MDS cohort. Risk stratification was performed using the Revised International Prognostic Scoring System (IPSS-R). (13)

Tekirdağ Namık Kemal University Ethics Committee approval was obtained. (No: 2025.16.01.16)

### 2.2. Flow Cytometry

Bone marrow aspirates were processed within 24 hours (most within 1 hour) and analyzed using the Navios EX Flow Cytometer (Beckman Coulter, CA, USA), acquiring a minimum of 20,000 events per sample.A two-tubes, 10-color antibody panel was used. ( Tube 1: CD11b-FITC, CD117-PE, CD10-ECD, CD33-PC5.5, CD34-PC7, CD13-APC, CD123-A700, CD38-A750, HLA-DR-PB, CD45-KO; Tube-2: CD15-FITC, CD16-PE, CD19-ECD, CD56-PC7, CD36-APC, CD7-A700, CD64-A750, CD14-PB, CD45-KO) Antibodies were prepared in sequence according to the manufacturer's instructions (FITC/PE/ECD/PC5/PC7/APC/A750/A700/PB/KO). Following antibody preparation, 100 µl of sample was added to the antibody tube, vortexed, and incubated in the dark at room temperature for 15 minutes. After addition of 500 µl OptiLyse C (Beckman Coulter, Inc.; California, USA) and a 10-minute incubation for red blood cell lysis, 500 µl PBS was added and incubated for another 10 minutes. Cells were then washed twice with PBS (300 g, 5 min) and finally resuspended in 500 µl PBS for flow cytometric analysis. Quality control procedures were implemented at each step according to the manufacturer's calibration protocols, and results were systematically recorded and reported.

Using Kaluza analysis software (Beckman Coulter, CA, USA), CD36-positive events were gated from SSClowCD45dim populations to calculate CD36 CV (Figure 1). Ogata scores were calculated and recorded for each patient (Figure 2).

### 2.3. Statistical Analysis and Modeling

Statistical analyses were performed using SPSS version 23 (IBM, USA). Depending on the variable type, comparisons utilized the chi-square test, Fisher’s exact test, independent samples t-test, Mann–Whitney U test, and ROC curve analysis.

Three cutoff values for CD36 CV were derived from the control group: (i) the 75th percentile (P75), (ii) the 90th percentile (P90), and (iii) the 90th percentile calculated using the method described by Westers et al. (WP90).(11)

Modeling approaches included: CD36 CV alone; combination of CD36 CV positivity with Ogata score positivity; and

an expanded 5-point scoring system adding one point for CD36 CV positivity to the original Ogata score. Sensitivity, specificity, and ROC analyses were performed for all models and cutoff thresholds.

3. Results

A total of 82 patients were included: 67 diagnosed with MDS and 15 assigned to the anemic control group. Among the MDS group, 12 (17.9%) were high-risk, while 55 (82.1%) were classified as low- or intermediate-risk by IPSS-R (Table 1).

In the control group, after exclusion of MDS by morphology and cytogenetics, the etiologies of anemia were: renal failure (26.6%), rheumatologic disease (20%), drug-induced anemia (20%), chronic liver disease (13.3%), iron deficiency due to gastrointestinal bleeding (6.6%), CMV infection secondary to prolonged steroid use (6.6%), and chronic inflammation (6.6%).

The mean CD36 CV value in the control group was 65.84 (range: 57.99–92.19), while it was 75.81 (range: 46.23–139.57) among all patients diagnosed with MDS. When stratified by risk group, the mean CD36 CV was 73.16 (range: 46.23–122.56) in the low- to intermediate-risk subgroup and 80.25 (range: 47.67–139.57) in the high-risk group. Although the mean CD36 CV values were numerically higher in all MDS subgroups compared to the control group, these differences did not reach statistical significance ($p=0.099$ for all MDS vs. control; $p=0.146$ for low-risk vs. control; and $p=0.083$ for high-risk vs. control), as shown in Table 1.

To assess diagnostic thresholds, upper cutoff values for CD36 CV were calculated based on the control group distribution. These were 70.9 for the 75th percentile, 87.16 for the 90th percentile, and 95.47 using the 90th percentile as defined by the method described by Westers et al. (WP90).
For low-risk MDS diagnosis, corresponding sensitivities were 60%, 18.2%, and 3.6%; specificities were 80%, 93.3%, and 100%, respectively (Table 2). CD36 CV alone was not statistically significant in ROC analysis (AUC: 0.623, 95% CI: 0.475–0.771, $p=0.146$).

Using an Ogata score threshold of ≥2 yielded 85.5% sensitivity and 33.3% specificity in lower-risk MDS (ROC AUC: 0.720, 95% CI: 0.584–0.856, $p=0.009$) (Table 2 and 3).

A revised 5-point scoring model was developed by adding one point for CD36 CV above the specified cutoff. ROC analysis showed statistical significance for all three cutoffs. The model using the 75th percentile cutoff achieved the best performance, with 72.7% sensitivity and 80% specificity at a ≥3-point threshold (detailed data in Supplementary Table).

**4. Discussion**

MDS diagnosis relies on morphological and cytogenetic analysis, along with exclusion of secondary causes of cytopenia. Unlike acute leukemias or chronic lymphocytic leukemia, flow cytometry lacks definitive diagnostic power in MDS. (14) Most current flow cytometry-based strategies focus on the myelomonocytic lineage, despite erythroid dysplasia being the most common dysplastic feature.

Aberrant CD36 expression is a recognized dysplastic marker in both granulocytic and erythroid cells. The European LeukemiaNet Working Group considers CD36 upregulation on granulocytes as a dysplastic feature. (15) Interpretation, however, is complicated by factors such as eosinophil or apoptotic cell contamination, and CD36 variability on monocytes. On erythroid cells, expression variability of CD36 and CD71 is diagnostically valuable. The "Red Score," which combines CVs of CD36 and CD71 with hemoglobin levels, achieved 77.5% sensitivity and 90% specificity for erythroid dysplasia. (16)The study by Lu et al investigates the expression of CD36 on $CD105^{pos}$ nucleated erythroid cells using multiparameter flow cytometry to differentiate MDS from megaloblastic anemia. Key findings indicate that the relative mean fluorescence intensity of CD36 is significantly decreased in MDS compared to megaloblastic anemia and anemia controls. Additionally, CD36 CV is increased in MDS versus controls. (17)
Westers et al. conducted a comprehensive analysis of erythroid markers—CD36, CD71, CD105, CD117, CD235a—evaluating CV, MFI, and antigen positivity. (11) Among these, CD36 CV, CD71 CV, CD71 MFI, and CD117 positivity were considered feasible for routine use. Later, Cremers et al. demonstrated that adding erythroid parameters to myeloid scoring significantly enhanced sensitivity from 74% to 86% without reducing specificity. (2) In a more comprehensive and recent study comparing five flow cytometric diagnostic criteria, the sensitivity of the Ogata score—focused on the

myeloid lineage—was reported as 57%, while the Red score—focused on the erythroid lineage—demonstrated a sensitivity of 43%. (18) The integrated iFS score proposed by Cremers et al., which incorporates both lineages, achieved a sensitivity of 79%. (2) However, the combined and extensive iFS scoring system is time-consuming and highly antibody-intensive for daily routine practice (requiring either 13 tubes with 4-color or 7 tubes with 8-color panels). Therefore, there is a need to develop diagnostic algorithms that rely on fewer parameters.

A combined immunophenotypic analysis of CD36 with other erythroid markers such as CD71 may allow a clearer distinction. Nevertheless, due to the retrospective nature of this study, only routinely processed antibody panels were available, and therefore the validation in this study focused specifically on CD36. In our cohort, integrating CD36 CV into the Ogata score improved specificity for low-risk MDS from 33.3% to 80%, though sensitivity decreased from 85.5% to 72.7%.

Despite promising results, technical variability in flow cytometry limits global standardization of CV and MFI values. Local standardization using institutional control groups is essential. (19)

Assessing erythroid dysplasia by flow cytometry has additional challenges. RBC lysis during sample preparation may variably affect nucleated erythroid precursors and alter antigen expression, potentially leading to misinterpretation. (20) As in this study, immunophenotypic assessment from lysed samples is limited to erythroid precursors and nucleated red blood cells, whereas mature erythrocytes cannot be analyzed. Similarly, while sample preparation and scatter-based gating strategies minimize platelet contamination, a complete distinction between erythroid precursors and residual platelets cannot be fully ensured with the currently available antibody panels.

Our findings suggest that incorporating CD36 CV into flow cytometric evaluation can enhance diagnostic performance in lower-risk MDS, particularly when used alongside myelomonocytic immunophenotyping, without incurring extra costs or needing additional antibody panels.

Limitations of this study include its retrospective design, small sample size, and absence of a healthy control group. Future research will focus on standardization and validation in larger, prospective cohorts.

Conflict-of-Interest Statement: The authors declare that they have no conflicts of interest and no financial disclosures.

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Tables:
Table-1: Patients characteristics
Table-2: Sensitivity and specificity of Ogata score, CD36 coefficient of variation (CV) cut-offs, and the revised 5-point model for the diagnosis of all MDS and lower-risk MDS.
Table-3: ROC analyses results of selected diagnostic models

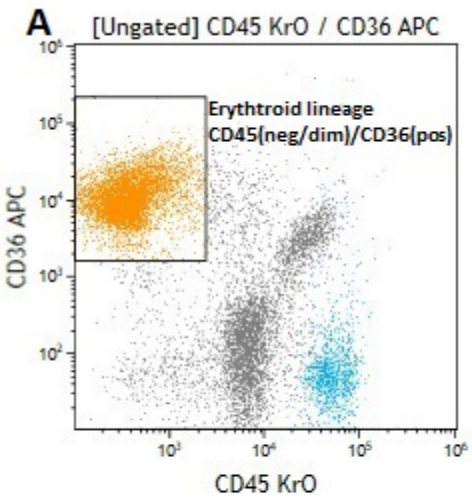
Figures:

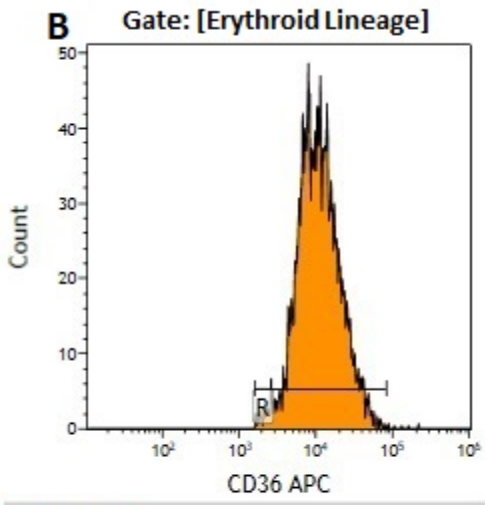
Figure-1: Flow cytometric gating strategy for CD36 coefficient of variation (CV) analysis.
(A) Erythroid lineage cells were identified as CD45^negative/dim/CD36^positive events (orange) within the CD45 KrO vs. CD36 APC dot plot.
(B) Histogram representation of CD36 APC expression in the gated erythroid population, where a region (R) was defined to calculate the coefficient of variation (X-CV) of CD36 expression.

Figure-2: Representative example of Ogata score calculation.
(A) CD34 vs. CD10 plot showing 2.24% myeloblasts and 0.17% B-progenitor cluster size.
(B) Side scatter histogram displaying granulocyte (red) and lymphocyte (blue) peaks, used to calculate a granulocyte-to-lymphocyte SSC ratio of 5.43.
(C) Histogram of lymphocyte CD45 expression with a mean fluorescence intensity (MFI) of 53,147.
(D) Histogram of myeloblast CD45 expression with an MFI of 8,004. The resulting lymphocyte-to-myeloblast CD45 ratio was 6.64.

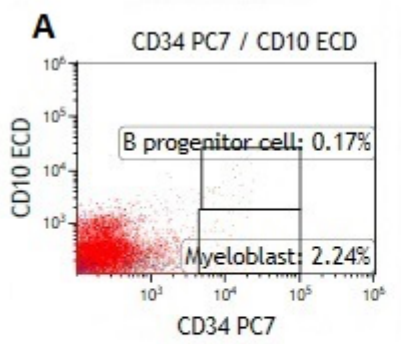
According to these parameters, the Ogata score for this case is 3 (myeloblasts >2%, score 1; B-progenitor cluster size <5%, score 1; granulocyte-to-lymphocyte SSC ratio <6, score 1; lymphocyte-to-myeloblast CD45 ratio between 4 and 7.5, score 0).

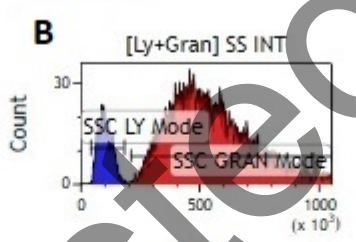
Abbreviations: MFI, mean fluorescence intensity; SSC, side scatter.



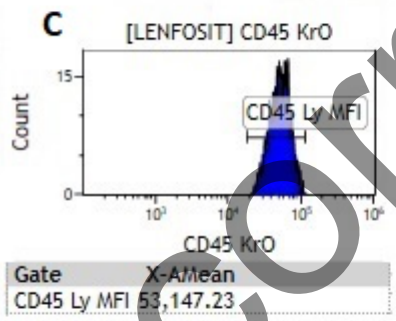




| Gate | X-CV |
|---|---|
| All | 78.79 |
| R | 69.91 |

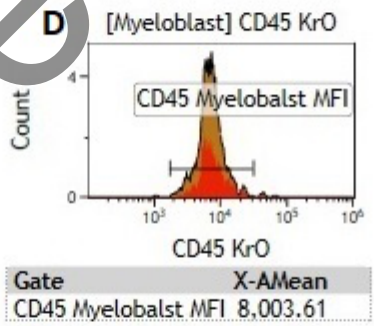



| Gate | X-AMean |
|---|---|
| SSC GRAN Mode | 544,612.63 |
| SSC LY Mode | 100,226.30 |




| Gate | X-AMean |
|---|---|
| CD45 Ly MFI | 53,147.23 |




| Gate | X-AMean |
|---|---|
| CD45 Myelobalst MFI | 8,003.61 |

| Parameter | Cut-off values | Score |
|---|---|---|
| Myeloblast(%of CD45+cells) | >2% | 1 |
| B-Progenitor-related cluster size(%ofCD34+) | <5% | 1 |
| Lymphocyte to myeloblast CD45 ratio | <4 or>7.5 | 1 |
| Granulocyte to lymphocyte SSC ratio | <6 | 1 |

Table-1: Patients characteristics

| | MDS (n=67) | LR-MDS (n=55) | Control (n=15) | P (MDS-control, LR-MDS-control) |
|---|---|---|---|---|
| Sex | | | | |
| Male | 42 (62.7%) | 35 (63.6%) | 5 (33.3%) | P=0.047, p=0.036 |
| Female | 25 (37.3%) | 20 (36.4%) | 10 (66.7%) | |
| Age | 70.42 | 71.11 | 67.93 | P<0.01, P<0.01 |
| Hgb (gr/dl) | 8.67 | 8.73 | 10.52 | P<0.01, P<0.01 |
| Neu(/uL) | 2368 | 2575 | 4469 | P<0.01, P<0.01 |
| Plt (*10^3/uL) | 177 | 201 | 226 | P<0.01, P<0.01 |
| CD36 CV | 75.81 | 73.16 | 65.84 | p=0.099, p=0.146 |
| Ogata Score | | | | |
| 1 | 10 (14.9%) | 8 (14.5%) | 5 (33.3%) | p=0.014 , p=0.04 |
| 2 | 17 (25.4%) | 17 (30.9%) | 8 (53.3%) | |
| 3 | 34 (50.7) | 29 (52.7%) | 2 (13.3%) | |
| 4 | 6 (9%) | 1 (1.8%) | 0 (0) | |
| Dysplasia | | | | |
| SLD | 20 (29.9%) | 20 (36.4%) | NA | NA |
| MLD | 47 (70.1%) | 35 (63.6%) | NA | NA |
| IPSS-R | | | | |
| Very-low | 8 (11.9 %) | NA | NA | NA |
| Low | 27(40.3 %) | NA | NA | NA |
| Intermediate | 20 (29.9%) | NA | NA | NA |
| High | 5 (7.5 % | NA | NA | NA |
| Very-high | 7 (10.4 %) | NA | NA | NA |

*Age, Hgb, Neu, plt and CD36 CV were given in mean values. MDS: myelodysplastic syndromes, LR-MD: low-intermediate risk MDS, Hgb: hemoglobulin, Neu:neutrophile count, plt: platelet count, CV: coefficient of variation, SLD: single lineage dysplasia, MLD: multi-lineage dysplasia IPSS-R: **Revised International Prognostic Scoring System NA: Not applicable**

Table-2: Sensitivity and specificity of Ogata score, CD36 coefficient of variation (CV) cut-offs, and the revised 5-point model for the diagnosis of all MDS and lower-risk MDS.

| | | **All- MDS** | | **Lower-Risk MDS** | |
|---|---|---|---|---|---|
| | **Threshold value** | **Sensitivity (%)** | **Specificity (%)** | **Sensitivity (%)** | **Specificity (%)** |
| Ogata Score | | | | | |
| ≥2/4 | | 85.1 | 33.3 | 85.5 | 33.3 |

| | | | | | |
|---|---|---|---|---|---|
| ≥3/4 | | 59.7 | 86.7 | 54.5 | 86.7 |
| CD36 Coefficient Of Variation (CV) | | | | | |
| CV>P75 | | 61.2 | 80 | 60 | 80 |
| CV>P90 | | 22.4 | 93.3 | 18.2 | 93.3 |
| CV>WP90 | | 9 | 100 | 3.6 | 100 |
| 5-point Model | | | | | |
| CD36CV Cut-off: P75 | ≥3/5 | 74.6 | 80 | 72.7 | 80 |
| | ≥4/5 | 38.8 | 93.3 | 32.7 | 93.3 |
| CD36CV Cut-off: P90 | ≥3/5 | 61.2 | 86.7 | 56.4 | 86.7 |
| | ≥4/5 | 20.9 | 100 | 12.7 | 100 |
| CD36CV Cut-off: WP90 | ≥3/5 | 59.7 | 86.7 | 54.5 | 86.7 |
| | ≥4/5 | 14.9 | 100 | 5.5 | 100 |
| | | | | | |
| CD 36CV: CD36 Coefficient of Variation | | | | | |
| P75: **the75th percentile of control group CD36 CV, P90: the 75th percentile of control group CD36 CV, WP90: the 90th percentile of control group CD36 CV calculated using the method described by Westers et al (2017)** | | | | | |
| **5-point Model: 5-point scoring system adding one point for CD36 CV positivity (with respective cut-off values; P75, P90, WP90) to the Ogata score** | | | | | |

Table-3: ROC analyses results of selected diagnostic models

| Model | All- MDS | | | LR-MDS | | |
|---|---|---|---|---|---|---|
| | AUC | %95 CI | p | AUC | %95 CI | p |
| Ogata score | 0.740 | 0.617-0.864 | 0.004 | 0.720 | 0.584-0.856 | 0.009 |
| CD36 CV | 0.637 | 0.500-0.774 | 0.099 | 0.623 | 0.475-0.771 | 0.146 |
| 5p-WP90 | 0.744 | 0.624-0.864 | 0.003 | 0.722 | 0.589-0.856 | 0.009 |
| 5p-P90 | 0.760 | 0.644-0.875 | 0.002 | 0.742 | 0.614-0.871 | 0.004 |
| 5p-P75 | 0.800 | 0.680-0.919 | <0.001 | 0.794 | 0.663-0.924 | 0.001 |
| | | | | | | |
| CD 36CV: CD36 Coefficient of Variation | | | | | | |
| P75: **the75th percentile of control group CD36 CV, P90: the 75th percentile of control group CD36 CV, WP90: the 90th percentile of control group CD36 CV calculated using the method described by Westers et al (2017)** | | | | | | |
| **5p: 5-point scoring system adding one point for CD36 CV positivity (with respective cut-off values; P75, P90, WP90) to the Ogata score** | | | | | | |