Original Article

Disseminated Intravascular Coagulation in Acute Myeloid Leukemia at the time of Presentation

Nikesh Bajracharya[1], Mukesh Kumar Shah[2], Bikram Karki[1], Devendra Acharya[1], Suravi Pandey[1], Neeraj Sharma[1], Nripesh Man Shrestha[1], Sanjay Gelal[1], Jasmine Gurung[3], Sachita Baniya[1], and Bishesh Sharma Poudyal[1]

[1] Department of Hematology and Bone Marrow Transplant Unit, Civil Service Hospital, Kathmandu, Nepal
[2] Department of Emergency Medicine and Pre-Hospital Care, Birat Medical College and Teaching Hospital, Koshi Province, Nepal
[3] Department of Clinical Oncology, Bir Hospital, Kathmandu, Nepal

Article History
Received on: Sep 10, 2025
Acceptance on: Oct 27, 2025

Online Access

DOI: https://doi.org/10.59338/cmj.89

Correspondence

Nikesh Bajracharya
DM Resident (Clinical Hematology)
Department of Hematology and Bone Marrow Transplant Unit
Civil Service Hospital
Kathmandu, Nepal
Email: nikesh.bajracharya@gmail.com

Abstract

Introduction: Disseminated Intravascular Coagulation (DIC) is a serious coagulopathy that can complicate Acute Myeloid Leukemia (AML). Although it is most commonly associated with Acute Promyelocytic Leukemia (APL), DIC may also occur in non-APL AML subtypes. The ISTH-DIC scoring system provides a standardized method to classify patients into overt or non-overt DIC, enabling early detection and management.

Methods: This cross-sectional observational study included 55 newly diagnosed non-APL AML patients treated at Civil Service Hospital, Kathmandu, Nepal, between May 2024 and April 2025. The ISTH-DIC score was calculated using platelet count, PT, fibrinogen, and D-dimer levels. Patients were categorized as overt or non-overt DIC, and clinical/laboratory characteristics were compared using SPSS version 26.

Results: Overt DIC was observed in 17 patients (30.9%). Patients with overt DIC showed significantly prolonged PT ($p < 0.001$) and elevated D-dimer levels ($p = 0.031$). Bleeding manifestations were present in both groups without significant difference, with nasal bleeding being the most common site. Thrombosis was rare, occurring in only one patient. No significant association was found with immunophenotypic markers.

Conclusion: Overt DIC is a common and often asymptomatic complication in non-APL AML. Routine screening using the ISTH-DIC scoring system at diagnosis is recommended. Abnormal PT and D-dimer are useful markers, and further studies are needed to explore additional risk factors.

Keywords: Acute Myeloid Leukemia, Disseminated Intravascular Coagulation, ISTH-DIC score, thrombocytopenia, D-dimer, prothrombin time, fibrinogen.

Introduction

Disseminated Intravascular Coagulation (DIC) is a life-threatening condition caused by systemic activation of the coagulation cascade, leading to widespread thrombus formation in micro- and macro-vasculature, impaired tissue perfusion, multiple organ dysfunction (MODS), and concurrent severe bleeding due to consumption of clotting factors and platelets.[1] Leukemia are one of the important causes of DIC.[2] Patients with leukemia often exhibit abnormal coagulation parameters even in the absence of clinical signs of thrombosis or bleeding, reflecting varying degrees of coagulation activation and indicating an underlying hypercoagulable state.[3,4]

Although DIC is most frequently associated with acute promyelocytic leukemia (APL), it can occur in any morphological subtype of acute myeloid leukemia (AML). The prevalence of DIC in APL has been reported to range from 17% to 100%, while in acute lymphoblastic leukemia

© **The Author(s) 2025.** This work is licensed under a Creative Commons Attribution-Non Commercial 4.0 International (CC BY-NC)

(ALL) and non-APL AML, it varies between 8.5% and 25% across studies.[5] AML, the most common type of leukemia in adults, is characterized by the clonal proliferation of immature blast cells in the peripheral blood and bone marrow, leading to ineffective hematopoiesis and bone marrow failure.[6] The exact mechanism of DIC in AML remains unclear; however, it is hypothesized that leukemic cell granules may release enzymes that degrade coagulation factors or express tissue factor-like substances that activate the coagulation cascade, with additional contributions from inflammatory cytokines, chemotherapy, and infections for DIC.[7]

The International Society of Thrombosis and Hemostasis-Disseminated Intravascular Coagulation (ISTH-DIC) score (Table 1) is a widely accepted tool developed through expert consensus to assess patients with critical illnesses known to trigger DIC.8 It classifies patients into 'overt' or 'non-overt' DIC based on laboratory parameters including platelet count, D-dimer levels, fibrinogen levels, and prolongation of prothrombin time (PT).[9] A total score of 5 or greater indicates overt DIC, while a score of less than 5 suggests non-overt DIC.[8] Non-overt DIC represents a subtle, compensated form of hemostatic dysfunction where there is ongoing but controlled activation of the coagulation system without clinical bleeding or thrombosis.[10] In contrast, overt DIC is a decompensated state marked by widespread coagulation activation, consumption of clotting factors and platelets, and a breakdown of regulatory mechanisms, leading to clinical manifestations such as bleeding, thrombosis, and organ dysfunction.[10] Early recognition of non-overt DIC is crucial to prevent progression to the overt, life-threatening stage.[10] Demonstrating a sensitivity of 91% and specificity of 97%, the ISTH-DIC score is highly accurate in identifying overt DIC.[11] Multiple studies have shown that the presence of overt DIC, as defined by this algorithm, is a significant predictor of mortality.[12,13]

Overt DIC at AML diagnosis is a strong predictor of thrombosis and early 30 day-mortality, with thrombotic risk exceeding 20% in younger and 40% in older patients.[14,15] Libourel et al. (2016) have reported a thrombosis prevalence of 8.7% in younger adults and 10.4% in older adults with AML over a median follow-up period of 478 days.[15] Their study has further highlighted that patients with overt DIC and elevated D-dimer levels are at a significantly increased risk of thrombosis.[15] Similarly, patients with overt DIC at AML diagnosis are at higher risk for bleeding. Chowdhury et al. (2021) have reported a higher risk of bleeding in overt DIC than in non-overt DIC at AML diagnosis (57.1% vs 36.6%).[16] A retrospective study by Paterno et al. (2022) has shown a higher 30-day mortality rate in AML patients with overt DIC than with non-overt DIC [28% vs 9%, HR: 3.307, 95% CI: 1.818-6.015, $p < 0.001$).[14]

Utilizing the ISTH-DIC scoring system can enable early detection of overt DIC in AML, allowing for timely interventions for the symptomatic patients that can help to improve the outcomes.

This study aimed to determine the frequency of overt and non-overt DIC at the time of AML diagnosis using the ISTH-DIC score, compare the clinical and laboratory profiles of patients with and without overt DIC, and identify factors associated with an increased risk of developing overt DIC.

Methods

This cross-sectional observational study was conducted in the Department of Hematology and Bone Marrow Transplant Unit, Civil Service Hospital, Kathmandu, from May 2024 to April 2025, after obtaining ethical approval from the Institutional Review Board (IRB), National Academy of Medical Sciences (NAMS). This study was conducted in accordance with the principles of the Declaration of Helsinki. A total of 55 newly diagnosed AML patients aged 16 years and above, who provided written informed consent (or assent for those under 18 years) were enrolled. Patients with APL, confirmed by morphology and molecular studies, and those receiving anticoagulant or antiplatelet therapy were excluded.

All patients suspected of Acute leukemia based on medical history and complete blood count (CBC) were assessed for DIC at initial presentation. AML diagnosis was confirmed using peripheral blood smear (PBS), bone marrow examination, and flow cytometry. For CBC analysis, 3 mL of venous blood was collected in EDTA vials and processed using a Beckman Coulter DXH800 Hematology Analyzer, with differential counts and platelet counts manually verified. PT, fibrinogen, and D-dimer levels were measured from 2.7 mL venous blood samples collected in trisodium citrate vials, centrifuged at 4,000 rpm for 15 minutes using a Rotina 380 centrifuge, and analyzed on the ACL Elite Pro Fully Automated Coagulation Analyzer. The ISTH-DIC scoring system was used to calculate the DIC score and a cut off value of > 5 was used for labelling as overt DIC.

Data analysis was performed using the Statistical Package for Social Sciences (SPSS) version 26. The Shapiro-Wilk test was used to assess data normality, given the sample size (N=55). Categorical variables such as age groups and gender were presented as frequencies and percentages. Continuous variables including age, platelet count, white blood cell (WBC) count, PT, fibrinogen, D-dimer, and peripheral blood blasts were expressed as mean ± standard deviation for normally distributed data, or as median with

interquartile range (IQR) for non-normally distributed data. Associations between categorical variables were analyzed using the Pearson Chi-Square test or Fisher's Exact test, as appropriate. The independent samples t-test was used to compare means between two groups when data were normally distributed, while the Mann-Whitney U test was applied to compare medians between two groups for non-normally distributed variables. A p-value of <0.05 was considered statistically significant for all analyses.

Results

A total of 55 patients with newly diagnosed AML were enrolled in this study. The mean age of the cohort was 43.65 ± 17.32 years, with an age range of 16 to 81 years. Most patients (32.7%) were between 41 and 60 years of age. There was a slight female predominance, with 30 females (54.5%) and 25 males (45.5%). The age and gender distribution of the patients is presented in Table 2. All patients exhibited some degree of abnormality in DIC parameters at the time of diagnosis as shown in Table 3. Specifically, 40 patients (89.1%) had platelet counts below 100×10⁹/L, 25 patients (45.5%) had PT, 44 patients (80%) had elevated D-dimer levels, and only 2 patients (3.6%) had fibrinogen levels below 1 g/L.

Using the ISTH DIC scoring system, 17 patients (30.9%) were diagnosed with overt DIC, while the remaining 38 patients (69.1%) were categorized as having non-overt DIC. The mean age of patients with overt DIC was slightly higher than that of those without overt DIC (48.82 ± 17.45 years vs. 41.34 ± 16.97 years), although the difference was not statistically significant (p = 0.140). There was also no significant difference in gender distribution between the overt and non-overt DIC groups (p = 0.456).

Bleeding manifestations at presentation were observed in 30 patients (54.54%). Among those with overt DIC, 9 of 17 patients (52.94%) experienced bleeding, compared to 21 of 38 patients (55.25%) in the non-overt DIC group. The difference in bleeding frequency between the two groups was not statistically significant (p = 0.873). The most common site of bleeding was nasal (26.7%), followed by gingival and gastrointestinal bleeding (20% each). Other reported sites included per vaginal (13.3%), urinary tract (10%), and intraocular bleeding (10%). Thrombosis at presentation was observed in one patient (1.8%), who had a pulmonary embolism and met the criteria for overt DIC.

Platelet counts were lower in the overt DIC group compared to the non-overt group, with a median platelet count of 20 ×10⁹/L (IQR: 15–45) versus 29.5 ×10⁹/L (IQR: 14–57), though this difference was not statistically significant (p = 0.266). At the time of presentation, 40 out of 55 patients (72.7%) had platelet counts below 50 ×10⁹/L. Among these, 25 patients (62.5%) presented with bleeding, indicating a trend toward increased bleeding risk with lower platelet counts, although the association did not reach statistical significance (Fisher's Exact Test, p = 0.071).

Patients with overt DIC demonstrated significantly prolonged PT values compared to those without overt DIC (p < 0.001), and D-dimer levels were also significantly higher in the overt DIC group (p = 0.031). Fibrinogen levels were slightly lower in the overt DIC group (3.40 ± 1.69 g/L) compared to the non-overt group (3.72 ± 1.40 g/L), but this difference was not statistically significant (p = 0.468). Most patients (96.4%) had fibrinogen levels above 1 g/L.

Table 1: Scoring system for Overt DIC according to ISTH-DIC scoring system8

A. Risk assessment: Does the patient have an underlying disorder known to be associated with overt DIC? If yes: proceed If no: do not use this algorithm				
B. Order global coagulation tests: Prothrombin time (PT), platelet count, fibrinogen, fibrin related marker				
C. Score the test results:				
Tests	Points			
	0	1	2	3
PT prolongation (sec)	< 3 sec	3-6 sec	> 6 sec	
Fibrinogen (g/l)	> 1	< 1		
D-dimer (ng/ml)	< 400		400-4,000	> 4,000
Platelet count (×10⁹/l)	> 100	50-100	< 50	
D. Calculate score: ≥ 5 = compatible with overt DIC < 5 = suggestive of non-overt DIC				

Table 2: Age and gender distribution of study population

Age Groups (years)	Frequency (n)	Percentage (%)
≤ 20	8	14.5
21-40	17	30.9
41-60	18	32.7
≥ 61	12	21.8
Gender		
Male	25	45.5
Female	30	54.5

Table 3: Characteristic of DIC parameters in the study population at presentation

Parameters	Value	Frequency (%)
Platelets (×10⁹/L)	≥ 100	6 (10.90%)
	50-100	9 (16.40%)
	< 50	40 (72.70%)
Increase in PT (sec)	< 3	30 (54.5%)
	≥ 3	25 (45.5%)
D-dimer (ng/ml)	Normal	11 (20%)
	Increased	44 (80%)
Fibrinogen (g/L)	≥ 1	53 (96.4%)
	< 1	2 (3.6%)
ISTH-DIC Score	< 5	38(69.1%)
	≥ 5	17 (30.9%)

In terms of hematologic parameters, the median white blood cell (WBC) count in the overt DIC group was 18.51 ×10⁹/L (IQR: 6.70–41.06), compared to 10.80 ×10₂/L (IQR: 2.30–45.40) in the non-overt DIC group. The median peripheral blast percentage was also higher in the overt DIC group (51%, IQR: 24–73.5) than in the non-overt group (33.5%, IQR: 9.25–62). However, these differences were not statistically significant (p = 0.729 for WBC count and p = 0.107 for blast percentage). Comparison of clinical and laboratory profiles between the DIC groups are shown in Table 4.

Table 4: Comparison of clinical and laboratory characteristics between overt DIC and non-overt DIC

Parameters	Overt Dic	Non- Overt Dic	p-value
Mean age (years)	48.82 ± 17.45	41.34 ± 16.97	0.140[a]
Gender (%)			
Male	9 (16.36%)	16 (29.10%)	0.456[b]
Female	8 (14.54%)	22 (40.00%)	
Bleeding at presentation			
Present	9 (16.36%)	21 (38.20%)	0.873[b]
Absent	8 (14.54%)	17 (30.90%)	
Median platelet count (×10⁹/L)	20	29.5	0.266[c]
[IQR]	[15-45]	[14-57]	
Median PT (sec)	16.6	13.2	0.001[c]
[IQR]	[15-18.20]	[12.60-14.40]	
Median D-dimer (ng/dl)	1,410	780	0.031[c]
[IQR]	[610-2165]	[337.5-1337.5]	
Mean Fibrinogen (g/L)	3.40 ± 1.69	3.72 ± 1.40	0.468[a]
Median WBC count (×10⁹/L)	18.51	10.80	0.729[c]
[IQR]	[6.70-41.06]	[2.30-45.40]	
Median Peripheral Blast (%)	51	33.5	0.107[c]
IQR	[24-73.50]	[9.25-62]	

a: Independent samples t-test, b: Pearson Chi-square test, c: Mann-Whitney U test

A comprehensive immunophenotypic analysis, including markers such as CD13, CD33, MPO, CD36, CD64, CD15, CD56, CD11c, HLA-DR, CD34, CD117, CD38, and aberrant markers, was conducted to evaluate their association with DIC status. No statistically significant differences were observed between the overt and non-overt DIC groups in relation to the expression of these markers. Comparison of immunophenotypic marker expression between the DIC groups are shown in Table 5.

Table 5: Comparison of Immunophenotypic marker expression between overt DIC and non-overt DIC groups

		Overt Dic	Non-Overt Dic	P-value
CD13	Positive Negative	16 1	33 5	0.654[a]
CD33	Positive Negative	12 5	32 6	0.286[a]
MPO	Positive Negative	10 7	22 16	0.949[b]
CD36	Positive Negative	0 17	4 34	0.299[a]
CD64	Positive Negative	2 15	10 28	0.304[a]
CD15	Positive Negative	1 2	16 36	1[a]
CD56	Positive Negative	1 2	16 36	1[a]
CD11c	Positive Negative	0 17	2 36	1[a]
HLA-DR	Positive Negative	16 1	33 5	0.654[a]
CD34	Positive Negative	11 6	26 12	0.786[b]
CD38	Positive Negative	11 6	28 10	0.498[b]
CD117	Positive Negative	15 2	27 11	0.303[a]
Aberrant	Positive Negative	4 13	7 31	0.722[a]

a: 2-sided Fisher's Exact test, b: Pearson Chi-square test

Discussion

This study examined the prevalence and clinical characteristics of overt and non-overt DIC among patients newly diagnosed with non-APL AML, using the ISTH-DIC scoring system. A key finding was the relatively high frequency of overt DIC (30.9%) in this cohort, which is significantly higher than figures reported in earlier studies by Chowdhury et al. (2021) (14.6%) and Libourel et al. (2016) (8.5%).[15,16] This notable variation could reflect regional differences in disease biology, genetic predispositions, or disparities in healthcare infrastructure and early diagnostic capabilities. The high rate of overt DIC in our study underscores the importance of routinely assessing coagulation profiles at the time of AML diagnosis, especially in resource-limited settings where patients often present at more advanced stages of disease.

The demographic profile of our patients contrasts with Western epidemiological data, such as those from the American Cancer Society, which typically report a median age of AML diagnosis at around 69 years and a male predominance.[17] In contrast, the mean age in our study was substantially lower (43.65 years), with a slight female predominance. This pattern was consistent with findings from similar South Asian studies, including those conducted in Bangladesh by Chowdhury et al. (2021) and Nepal by Chaurasiya et al. (2023).[16,18] Such differences may be influenced by regional factors including environmental exposures (e.g., industrial toxins, pesticides), socioeconomic status, healthcare-seeking behaviors, and genetic or epigenetic susceptibilities unique to the population. These observations highlight the necessity of region-specific data in tailoring diagnostic and management strategies for AML.

Nearly all patients in this study exhibited at least one abnormality in DIC-related parameters, reaffirming that coagulopathy is a common and early feature of AML. Elevated D-dimer and prolonged PT were especially prevalent and were found to be significantly higher in patients with overt DIC. These findings support the use of these parameters as practical and sensitive indicators of more advanced coagulopathy. D-dimer reflects ongoing fibrin degradation and fibrinolysis, while prolonged PT indicates consumption of clotting factors—both of which are central features of overt DIC. Their significant elevation in overt DIC cases underscores their diagnostic and prognostic utility.

Interestingly, while fibrinogen levels were slightly lower in overt DIC patients, the difference was not statistically significant, and most patients (96.4%) had levels above 1 g/L. This finding aligns with previous studies by Dixit et al. (2007) and Choudhury et al. (2021), which also reported preserved fibrinogen levels in most AML patients at diagnosis.[16,19] Several explanations have been proposed for this phenomenon, including compensatory hepatic fibrinogen synthesis or early-phase DIC where consumptive coagulopathy is not fully established. It also raises questions about the sensitivity of hypofibrinogenemia as a marker for DIC in AML and supports a more nuanced interpretation of fibrinogen values within the clinical context.

Another important observation in our study was the frequency and pattern of bleeding manifestations. Bleeding occurred in over half of the patients (54.5%), with no significant difference between the overt and non-overt DIC groups. Notably, bleeding was slightly more frequent in the non-overt DIC group, which contradicts the expected trend and previous reports, including that by Chowdhury et al. (2021), who observed a higher bleeding

rate among patients with overt DIC.[16] This paradoxical finding in our study may reflect the variability in clinical presentation of DIC. However, given the small sample size and lack of statistical significance, this result should be interpreted with caution. It does, however, highlight the clinical variability in DIC presentation and underlines the importance of comprehensive coagulation monitoring regardless of DIC categorization.

Thrombosis was relatively rare in our cohort, with only one case (1.8%) presenting with a pulmonary embolism at diagnosis. This is in line with Libourel et al. (2016), who reported a similar incidence of thrombotic events (1.5%) in a larger AML cohort.[15] Nonetheless, the presence of thrombosis in a patient with overt DIC reiterates the dual nature of DIC as both a hemorrhagic and thrombotic disorder. While bleeding may be the more visible clinical challenge, clinicians must remain aware of the thrombotic risks, especially in patients with elevated D-dimer and other procoagulant profiles.

The trend toward increased bleeding with lower platelet counts suggests a clinically relevant relationship that is consistent with findings from Chowdhury et al. (2021).[16] In their study, patients with platelet counts <50 ×10⁹/L had a significantly higher bleeding risk ($p < 0.001$). Our findings support this trend but also highlight the limitations of small sample size in achieving statistical significance. Nonetheless, the data emphasize the critical need for aggressive platelet monitoring and transfusion support in thrombocytopenic AML patients to prevent bleeding complications.

Hematologic parameters, such as WBC count and peripheral blast percentage, were higher in the overt DIC group, but the differences did not reach statistical significance. This contrasts with Guo et al. (2020), who reported a significant association between elevated leukocyte count and overt DIC.[20] The lack of significance in our study could reflect the relatively small sample size, heterogeneity in leukemia subtypes, or differences in leukemic burden and organ involvement. Despite this, elevated leukocyte counts, and blast percentages should still be considered in risk stratification, as they may contribute to increased tissue factor expression and endothelial damage, both of which promote DIC.

The study also explored the association between leukemic immunophenotypes and DIC status. Contrary to prior reports by Uchiumi et al. (2007) and Chowdhury et al. (2021), which linked certain markers (e.g., negative CD13 and HLA-DR expression) with overt DIC, we did not observe any statistically significant differences in marker expression between the overt and non-overt DIC groups.[16,21] This could be due to population-based genetic variability, the complexity of leukemic clone biology, or sample size constraints. Nevertheless, it suggests that while immunophenotyping remains essential for disease classification and prognostication, its role in predicting DIC risk may be limited or population-specific.

In summary, our findings highlight the high prevalence of both overt and non-overt DIC in newly diagnosed AML patients and affirm the value of the ISTH-DIC scoring system in identifying patients at risk. Parameters such as PT and D-dimer offer reliable insights into coagulopathy severity, while bleeding and thrombotic complications remain critical concerns across the DIC spectrum. Though limitations such as small sample size and single-center design must be acknowledged, this study adds to the growing body of literature advocating for routine coagulation monitoring at AML diagnosis. Future multicenter studies with larger cohorts are needed to confirm these findings, explore predictive biomarkers, and develop tailored management strategies to mitigate DIC-related morbidity and mortality in AML patients.

Conclusion

This study found that overt DIC was present in 30.9% of newly diagnosed non-APL AML patients, with no significant associations with age, gender, or bleeding manifestations. Although overt DIC was associated with significantly prolonged PT and elevated D-dimer levels, fibrinogen levels remained largely preserved. Thrombocytopenia and higher WBC counts, and blast percentages were more common in overt DIC but did not reach statistical significance. Additionally, immunophenotypic markers showed no significant association with DIC status, contrasting with earlier reports. Given that many AML patients with DIC may be asymptomatic and bleeding is not always present, routine DIC screening using the ISTH-DIC score at presentation is crucial for timely diagnosis and intervention. A thorough evaluation of both clinical and laboratory parameters is recommended to better understand the risk factors for DIC and guide effective management strategies in AML.

Limitations

This study has a few important limitations. First, the number of patients enrolled was small (only 55) and all were from a single hospital, so the results may not apply to patients in other regions or countries. Second, since this was a cross-sectional study, it only looked at patients at one point in time. This means we couldn't study how DIC develops or changes over time. Third, some factors that can affect blood clotting, like infections or liver problems, were not fully studied. We also excluded patients with

APL, so we couldn't compare DIC across all types of AML. In addition, we only checked for DIC at the time of diagnosis and not later, which might have missed changes in clotting status. Fourth, while we studied surface markers on leukemia cells, we couldn't include genetic or molecular tests due to limited resources, which might have provided more detailed risk information. Lastly, we didn't compare the mortality between the two groups in this study.

Acknowledgements

The authors express their sincere gratitude to the Department of Hematology and Bone Marrow Transplant, Civil Service Hospital, Kathmandu, and to all the participants for their valuable support and cooperation.

Ethical approval

This study was conducted after obtaining ethical approval from Institutional Review Board (IRB), National Academy of Medical Sciences (NAMS) (Committee's Ref. No.: 1481/2080/81).

Consent for publication

Written informed consent was obtained from the respondent for data collection and for inclusion in a scientific publication.

Availability of data and materials

The data used in this study are not publicly available due to privacy or ethical reasons but can be provided by the corresponding author upon reasonable request.

Conflicts of Interest

The authors declare that there are no conflicts of interest related to the publication of this study.

Funding: None

Contributions

Nikesh Bajracharya conceived, conceptualized, and designed the study. Literature review was conducted by Nikesh Bajracharya, Bikram Karki, Sachita Baniya, and Bishesh Poudyal. Data collection was carried out by Nikesh Bajracharya, Bikram Karki, Devendra Acharya, Suravi Pandey, Nripesh Man Shrestha, Sanjay Gelal, and Neeraj Sharma. Data analysis was performed by Nikesh Bajracharya, Devendra Acharya, and Jasmine Gurung. The manuscript was drafted by Nikesh Bajracharya and Mukesh Kumar Sah, and edited by Bishesh Poudyal, Bikram Karki, and Nripesh Man Shrestha. All authors critically reviewed the manuscript and approved its final version for submission and publication.

References

1. Costello RA, Nehring SM. Disseminated Intravascular Coagulation. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024 [cited 2024 Feb 20]. Available from: http://www.ncbi.nlm.nih.gov/books/NBK441834/
2. Nur S, Anwar M, Saleem M, Ahmad PA. Disseminated intravascular coagulation in acute leukaemias at first diagnosis. European Journal of Haematology [Internet]. 1995 [cited 2024 Feb 20];55(2):78-82. Available from: DOI: 10.1111/j.1600-0609.1995.tb01813.x PMID: 7628593
3. Rickles FR, Levine M, Edwards RL. Hemostatic alterations in cancer patients. Cancer Metast Rev [Internet]. 1992 Nov 1 [cited 2025 May 6];11(3):237-48. Available from: DOI: 10.1007/BF01307180 PMID: 1423816
4. Falanga A, Barbui T, Rickles FR, Levine MN. Guidelines for Clotting Studies in Cancer Patients. Thrombosis and Haemostasis [Internet]. 2018 Jul 5 [cited 2025 May 6];70:540-2. DOI: 10.1055/s-0038-1649619 PMID: 8259561
5. Cate H ten, Leader A. Management of Disseminated Intravascular Coagulation in Acute Leukemias. Hamostaseologie [Internet]. 2021 Apr [cited 2024 Feb 19];41(2):120-6. DOI: 10.1055/a-1393-8302 PMID: 33860520
6. Vakiti A, Mewawalla P. Acute Myeloid Leukemia. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024 [cited 2024 Feb 19]. DOI: 10.1007/978-981-99-9283-6_42
7. Bick RL. Disseminated Intravascular Coagulation and Related Syndromes: A Clinical Review. Semin Thromb Hemost [Internet]. 1988 Oct [cited 2024 Feb 20];14(04):299-338. DOI: 10.1055/s-2007-1002793 PMID: 3057630
8. Toh CH, Hoots WK. The scoring system of the Scientific and Standardisation Committee on Disseminated Intravascular Coagulation of the International Society on Thrombosis and Haemostasis: a 5-year overview1. Journal of Thrombosis and Haemostasis [Internet]. 2007 Mar 1 [cited 2025 May 8];5(3):604-6. Available from: DOI: 10.1111/j.1538-7836.2007.02313.x PMID: 17096704

9. DIC Score [Internet]. [cited 2024 Apr 15]. Available from: https://reference.medscape.com/calculator/649/dic-score

10. Papageorgiou C, Jourdi G, Adjambri E, Walborn A, Patel P, Fareed J, et al. Disseminated Intravascular Coagulation: An Update on Pathogenesis, Diagnosis, and Therapeutic Strategies. Clin Appl Thromb Hemost [Internet]. 2018 Dec [cited 2025 Jun 8];24(9 Suppl):8S-28S. DOI: 10.1177/1076029618806424
PMID: 30296833 PMCID: PMC6710154

11. Bakhtiari K, Meijers JCM, de Jonge E, Levi M. Prospective validation of the International Society of Thrombosis and Haemostasis scoring system for disseminated intravascular coagulation*. Critical Care Medicine [Internet]. 2004 Dec [cited 2024 Feb 20];32(12):2416. DOI: 10.1097/01.CCM.0000147769.07699.E3
PMID: 15599145

12. Gando S, Wada H, Asakura H, Iba T, Eguchi Y, Okamoto K, et al. Evaluation of New Japanese Diagnostic Criteria for Disseminated Intravascular Coagulation in Critically Ill Patients. Clin Appl Thromb Hemost [Internet]. 2005 Jan 1 [cited 2024 Feb 20];11(1):71-6. DOI: 10.1177/107602960501100108
PMID: 15678275

13. Sivula M, Tallgren M, Pettilä V. Modified score for disseminated intravascular coagulation in the critically ill. Intensive Care Med [Internet]. 2005 Sep 1 [cited 2024 Feb 20];31(9):1209-14. DOI: 10.1007/s00134-005-2685-2
PMID: 15959760

14. Paterno G, Palmieri R, Forte V, Bonanni F, Guarnera L, Mallegni F, et al. PB1835: THE ISTH-DIC SCORE PREDICTS 30-DAYS OUTCOME IN NON-M3 ACUTE MYELOID LEUKEMIA PATIENTS. Hemasphere [Internet]. 2022 Jun 23 [cited 2024 Feb 18];6(Suppl):1715-6. DOI: 10.1097/01.HS9.0000850192.01228.bc
PMCID: PMC9431317

15. Libourel EJ, Klerk CPW, van Norden Y, de Maat MPM, Kruip MJ, Sonneveld P, et al. Disseminated intravascular coagulation at diagnosis is a strong predictor for thrombosis in acute myeloid leukemia. Blood [Internet]. 2016 Oct 6 [cited 2024 Feb 18];128(14):1854-61. DOI: 10.1182/blood-2016-02-701094
PMID: 27354723

16. Chowdhury MU, Begum M, Khan MR, Kabir AL, Islam S, Layla KN, et al. Disseminated Intravascular Coagulation at Diagnosis in Acute Myeloblastic Leukaemia. Journal of Biosciences and Medicines [Internet]. 2021 Sep 30 [cited 2024 Feb 17];9(10):124-34. DOI: 10.4236/jbm.2021.910011

17. Key Statistics for Acute Myeloid Leukemia (AML) [Internet]. [cited 2025 May 7]. Available from: https://www.cancer.org/cancer/types/acute-myeloid-leukemia/about/key-statistics.html

18. Chaurasiya PS, Lamsal DK, Khatri A, Parajuli A, Shrestha PR, Aryal L, et al. Prevalence of acute myeloid leukemia and its associated risk factors at a tertiary care center: a retrospective cross-sectional study. Ann Med Surg (Lond) [Internet]. 2023 Aug 14 [cited 2024 Feb 18];85(10):4794-8. DOI: 10.1097/MS9.0000000000001189
PMID: 37811085 PMCID: PMC10553139

19. Dixit A, Chatterjee T, Mishra P, Kannan M, Choudhry DR, Mahapatra M, et al. Disseminated Intravascular Coagulation in Acute Leukemia at Presentation and During Induction Therapy. Clin Appl Thromb Hemost [Internet]. 2007 Jul 1 [cited 2024 Feb 17];13(3):292-8. DOI: 10.1177/1076029607302435
PMID: 17636191

20. Guo Z, Chen X, Tan Y, Xu Z, Xu L. Coagulopathy in cytogenetically and molecularly distinct acute leukemias at diagnosis: Comprehensive study. Blood Cells, Molecules, and Diseases [Internet]. 2020 Mar 1 [cited 2024 Apr 18];81:102393. DOI: 10.1016/j.bcmd.2019.102393
PMID: 31918382

21. Uchiumi H, Matsushima T, Yamane A, Doki N, Irisawa H, Saitoh T, et al. Prevalence and Clinical Characteristics of Acute Myeloid Leukemia Associated with Disseminated Intravascular Coagulation. Int J Hematol [Internet]. 2007 Aug 1 [cited 2024 Apr 17];86(2):137-42. DOI: 10.1532/IJH97.06173
PMID: 17875527