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To study of thrombocytopenia, with an emphasis on platelet concentrations

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Abstract
Background: Thrombocytopenia can happen when the spleen gets sucked up, when platelets are destroyed more quickly, or when the bone marrow doesn't grow enough. A bone marrow test is the only way to be sure of what is causing thrombocytopenia, but it is expensive and invasive.
Materials and Methods: A prospective observational and case control study was carried out at Department of Pathology, Sree Balaji Medical College and Hospital, Chennai, India. The researchers were part of the Department of Pathology's haematology branch. This study looked at the association between age and thrombocytopenia, as well as the therapeutic relevance of platelet volume characteristics measured by an automated blood cell counter.
Results: This study assessed the utility of platelet parameters from bone marrow examinations, compared to automated blood cell counter results, in differentiating between hyperdestructive and hypoproductive thrombocytopenia etiology. The patients were adults, aged between 11 and 70 years. Thirty percent of patients are found within the age range of 41 to 50, with eighteen cases documented.
Conclusion: The platelet indices in the hyper-destructive group in this study are elevated, which may aid in differentiating between hyper-destructive and hypo-productive causes of thrombocytopenia.

Keywords: Thrombocytopenia, clinical, platelets, total blood count

Introduction

The endothelium is the main defense against bleeding caused by damage to small and large blood vessels. Platelets stick together and gather to keep it strong. If the number of platelets in your blood is less than 150 × 10^9/L, you have thrombocytopenia. Because of this, low platelet numbers often lead to bleeding, since platelets are needed for initial hemostasis and may increase the risk of death in thrombocytopenic patients [1-3].

Splenic sequestration, increased platelet destruction, and bone marrow hypoplasia are among the many potential causes of thrombocytopenia. It occurs when the bone marrow fails to produce an adequate number of red blood cells. Acute myeloid leukemia (AML), megaloblastic anemia, myelodysplastic syndrome, amegakaryocytic thrombocytopenic purpura, and treatment-related adverse effects are among the primary and secondary bone marrow illnesses that can cause this. Concurrent with normal or increased bone marrow production, extramedullary platelet degradation occurs [3-5].

The result can be immune thrombocytopenic purpura, subsequent intravascular coagulopathy, and hyperdestructive thrombocytopenia. Congestive splenomegaly is the most common cause of splenic sequestration. This condition can be brought on by a number of different illnesses, including long-term infections, myeloproliferative disorders, lymphomas, hemoglobin C disease, Gaucher's disease, thalassemia major, homozygous sickle cell disease, hemoglobin C, or any number of others. Thrombocytopenia can only be definitively diagnosed by bone marrow testing, which is intrusive, costly, and directly associated with the risk of bleeding diathesis. I would advise against relying on it only for diagnosing purposes [4-6].

As part of routine complete blood counts, machine-derived parameters called platelet indices, such as average platelet volume (MPV), platelet distribution width (PDW), and platelet criticality (PCT), have lately made it easier to determine the reason of thrombocytopenia. These numbers come from a Beckmann Coulter LH 780 automated blood cell analysis. For a first, non-invasive look at the cause of thrombocytopenia, these measures are useful [5-7].

The aim and objectives of this study was to study of thrombocytopenia, with an emphasis on platelet concentrations.

Materials and Methods

This study was performed at Department of Pathology, Sree Balaji Medical College and Hospital, Chennai, India, inside the hematology division of the Department of Pathology, as a prospective observational and case-control study from August 2016 to July 2017. This study assessed the clinical significance of platelet volume characteristics measured by an automated blood cell counter, together with the age-related causes of thrombocytopenia in the subjects.

Inclusion criteria

1. Patient with low WBC
2. Patients with platelet levels

Exclusion criteria

1. Patients with pseudo thrombocytopenia
2. Patients with coagulation problems

Results

The study utilized 100 people. This research investigated individuals with thrombocytopenia. An automated blood cell counter was employed to gather platelet data and evaluate the age-related progression of the illness. This study aimed to ascertain the utility of platelet measures in differentiating between hypoproductive and hyperdestructive thrombocytopenia.

Table 1: Age Distribution

Sr. No.	Age (Years)	Cases	%
1.	11 to 20	20	20
2.	21 to 30	16	16
3.	31 to 40	28	28
4.	41 to 50	22	22
5.	51 to 60	10	10
	61 to 70	4	4
	Total	100	100

The majority of persons are categorized into age groups ranging from 11 to 70 years. The age group of 31 to 40 years has the highest frequency, succeeded by the age group of 41 to 50 years, as indicated by the data in Table 1 and Figure 1 above.

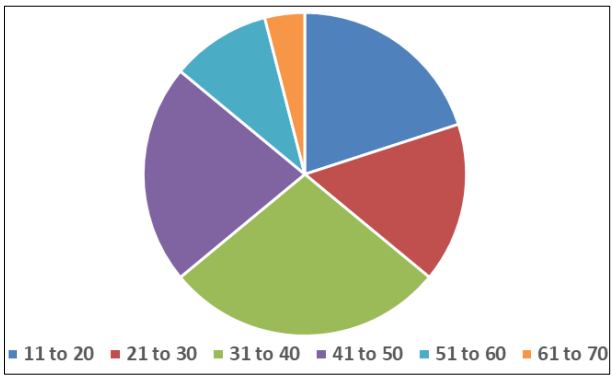

Fig 1: Age Distribution

Mean Age

The age distribution of people with hyperdestructive thrombocytopenia was 13.92 years, with a mean age of 45.33 years. The average age of the individuals with hypoproductive thrombocytopenia was 40.51 years, accompanied by a standard deviation of 14.91 years. The average age of all patients was 42.2 years, with a standard deviation of 14.63 years (See Table 2 and Figure 2).

Table 2: Distribution of hypoproductive thrombocytopenia patients

Sr. No.	Diagnosis	Patients	%
1.	Megaloblastic anaemia	50	50
2.	Post chemotherapy	20	20
3.	Lymphoma	15	15
4.	CML	15	15
5.	Acute leukemia	10	10
	Total	100	100

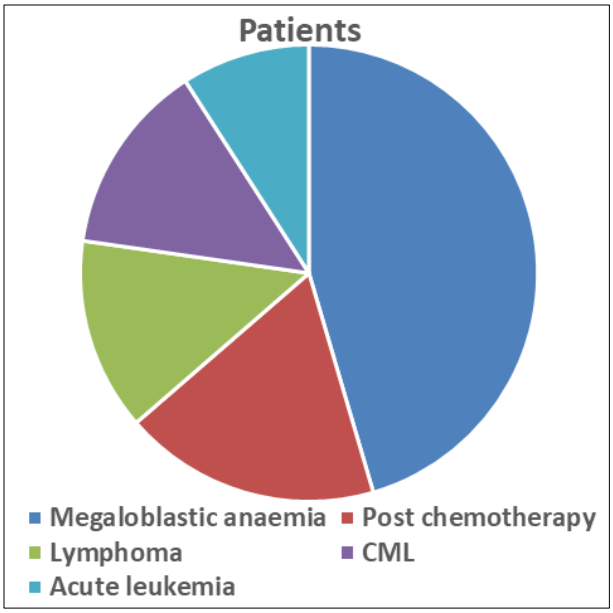

Fig 2: Distribution of hypo productive thrombocytopenia patients

The allocation of occurrences predicated on hyper destructive origins. Immune-mediated thrombocytopenia was identified as the cause in all forty cases, constituting 100% of the cases. Hypodestructive thrombocytopenia exhibited lower mean values for MPV, PDW, and PCT compared to the other type of thrombocytopenia when analyzing platelet properties in both groups.

Discussion

Platelets are made in the bone marrow by megakaryocytes, which are complex anucleate cells. When activated properly, platelets change into amazing cells that can contract and make chemicals that are useful for the body. A carefully made peripheral blood film shows a lot of details about the number, size, location, and shape of platelets. If there is an artifact, a wrong diagnosis could happen. Automated cell counters are being used more and more in both emerging and developed countries because they are reliable [7-9].

Here, we evaluated the use of platelet parameters obtained from bone marrow assays with those obtained from an automated blood cell counter in order to determine the etiology of thrombocytopenia and to distinguish between hyperdestructive and hypoproductive forms of the condition. The ages of the patients treated varied from eleven years old to seventy. Thirty percent of the cases included individuals

aged 41–50, with 18 cases falling within this age bracket [10–12]. People in the age bracket of 31–40 account for 14.3% of all cases, making them the second largest age group statistically. Individuals with hyperdestructive thrombocytopenia were included in this research. The average age of patients with hyperdestructive thrombocytopenia was 49.1 years, whereas the average age of patients with hypoproductive thrombocytopenia was 72 years. There's been a difference of 30 years. We observed a different gender ratio than the 1.5:1 previously observed in previous groups of patients with hyperdestructive thrombocytopenia [11–13].

Hypoproductive thrombocytopenia affected 1.20 times more men than women. Hyperdestructive thrombocytopenia is characterized by platelet counts below 80,000/microliter, with 60,000/microliter being the median. In the hyperdestructive age group, the average platelet count was 43.5±20.9, whereas in the control group, it was 228 ± 10^3 55.2. Significant statistical analysis revealed this disparity [14–16]. Hyperdestructive thrombocytopenia, in comparison to the standard type, is significantly worse. The hyperdestructive group had a higher concentration of low-platelet cells compared to the hypoproductive group. Our current understanding is that immune thrombocytopenic purpura was the underlying cause of every case in the hyperdestructive group. Between the two variants, the hypoproductive one was more prevalent in the sixty cases with thrombocytopenia that were examined. Multiple employees conducted observations simultaneously [17–19]. Because they aid in the early identification and categorization of thrombocytopenia and in determining the efficiency of the bone marrow in individuals with platelet problems, platelet indices including mean platelet volume, platelet distribution width, and platelet count have been the subject of increased research. Once other potential causes of low platelets have been eliminated, ITP is typically diagnosed [18–20]. While white blood cell and red blood cell counts were within normal ranges, platelet counts were lower than typical, according to the peripheral blood smear. A dramatic decrease in platelet count and an increase in megakaryocyte count were revealed by the bone marrow aspirate. A large number of tiny megakaryocytes with hypolobes can be found in the medullary cavity of long bones [19-21].

Vacuolations can be formed by megakaryocytes. Megakaryocytes in this study tended to be younger, less developed, more spherical, and less polypoid in the majority of ITP cases. Just a few number of mature megakaryocytes were able to produce platelets. It is possible that the age of the hematological analyzer employed in the research is to blame for the various MPV threshold values observed in the different investigations [20-22]. Old automatic analyzers may have been utilized in the study, which could explain why platelets didn't stand out from other similar-sized particles such as immune complexes, damaged red or white blood cells, cell debris, and so on. Additionally, only standard-sized platelets are listed because they cannot be distinguished from red blood cells. According to research, there are several factors that can affect MPV. These include the time that has passed since the venipuncture, the anticoagulant type, the storage temperature of the specimen, and the testing procedure. Another explanation could be a fundamental disparity in platelet indices among the categories [21–23]. It has been examined platelet indices in healthy Chinese individuals from various regions using the Sysmex XT 2100, and they found regional variances. The platelet crit value, which is the number of platelets in a certain amount, is not affected by how bad the hyperdestructive or hypoproductive thrombocytopenia. Because most of our patients were in the normal range, we weren't able to tell the difference between hyperdestructive and hypoproductive thrombocytopenia. The P-LCR index values of patients with myeloid failure were much lower than those in the control group, but those with ITP had values that were much higher [24-26].

Conclusion

Extremely low platelet counts are often associated with hyperdestructive thrombocytopenia, which is also associated with an increase in platelet indices such as MPV and PDW. Neither this elevation nor severe thrombocytopenia are hallmarks of hypoproductive thrombocytopenia. Neither of the thrombocytopenia' platelet levels have improved. Prior to the publication of bone marrow data, platelet indices may provide vital early information on the type of thrombocytopenia. Because they may be finished while counting blood cells, they don't necessitate an extra blood sample, additional time, or extra money.

Conflict of Interest: None

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References

1. Vinholt PJ, Hvas AM, Nybo M. An overview of platelet indices and methods for evaluating platelet function in thrombocytopenic patients. European journal of haematology. 2014 May;92(5):367-376.
2. Sekhon SS, Roy V. Thrombocytopenia in adults: A practical approach to evaluation and management. Southern medical Journal-Birmingham Alabama. 2006 May 1;99(5):491.
3. Frelinger III AL, Grace RF, Gerrits AJ, Berny-Lang MA, Brown T, Carmichael SL, *et al*. Platelet function tests, independent of platelet count, are associated with bleeding severity in ITP. Blood, The Journal of the American Society of Hematology. 2015 Aug 13;126(7):873-879.
4. Drachman JG. Inherited thrombocytopenia: when a low platelet count does not mean ITP. Blood. 2004 Jan 15;103(2):390-398.
5. Greinacher A, Selleng S. How I evaluate and treat thrombocytopenia in the intensive care unit patient. Blood, the Journal of the American Society of Hematology. 2016 Dec 29;128(26):3032-3042.
6. Smock KJ, Perkins SL. Thrombocytopenia: an update. International journal of laboratory hematology. 2014 Jun;36(3):269-278.
7. Murray NA, Howarth LJ, McCloy MP, Letsky EA, Roberts IA. Platelet transfusion in the management of severe thrombocytopenia in neonatal intensive care unit patients. Transfusion medicine. 2002 Feb;12(1):35-41.
8. Warkentin TE. Platelet count monitoring and laboratory testing for heparin-induced thrombocytopenia: recommendations of the College of American Pathologists. Archives of pathology & laboratory medicine. 2002 Nov 1;126(11):1415-1423.

9. Budak YU, Polat M, Huysal K. The use of platelet indices, Plateletcrit, mean platelet volume and platelet distribution width in emergency non-traumatic abdominal surgery: A systematic review. Biochemia medica. 2016 Jun 15;26(2):178-193.
10. Rodeghiero F, Michel M, Gernsheimer T, Ruggeri M, Blanchette V, Bussel JB, *et al*. Standardization of bleeding assessment in immune thrombocytopenia: report from the International Working Group. Blood, The Journal of the American Society of Hematology. 2013 Apr 4;121(14):2596-2606.
11. Slichter SJ. Relationship between platelet count and bleeding risk in thrombocytopenic patients. Transfusion medicine reviews. 2004 Jul 1;18(3):153-167.
12. Barsam SJ, Psaila B, Forestier M, Page LK, Sloane PA, Geyer JT, *et al*. Platelet production and platelet destruction: assessing mechanisms of treatment effect in immune thrombocytopenia. Blood, The Journal of the American Society of Hematology. 2011 May 26;117(21):5723-5732.
13. Thiery-Antier N, Binquet C, Vinault S, Meziani F, Boisramé-Helms J, Quenot JP. Is thrombocytopenia an early prognostic marker in septic shock?. Critical care medicine. 2016 Apr 1;44(4):764-772.
14. Thiele T, Selleng K, Selleng S, Greinacher A, Bakchoul T. Thrombocytopenia in the intensive care unit—diagnostic approach and management. InSeminars in hematology. WB Saunders. 2013;50(3)239-250.
15. Nahirniak S, Slichter SJ, Tanael S, Rebulla P, Pavenski K, Vassallo R, *et al*. Guidance on platelet transfusion for patients with hypoproliferative thrombocytopenia. Transfusion medicine reviews. 2015 Jan 1;29(1):3-13.
16. Cremer M, Sallmon H, Kling PJ, Bührer C, Dame C. Thrombocytopenia and platelet transfusion in the neonate. InSeminars in Fetal and Neonatal Medicine 2016;21(1):10-18. WB Saunders.
17. Wazny LD, Ariano RE. Evaluation and management of drug-induced thrombocytopenia in the acutely ill patient. Pharmacotherapy: The Journal of Human Pharmacology and Drug Therapy. 2000 Mar;20(3):292-307.
18. McCrae KR, Bussel JB, Mannucci PM, Remuzzi G, Cines DB. Platelets: An update on diagnosis and management of thrombocytopenic disorders. ASH Education Program Book. 2001 Jan 1;2001(1):282-305.
19. Khellaf M, Michel M, Schaeffer A, Bierling P, Godeau B. Assessment of a therapeutic strategy for adults with severe autoimmune thrombocytopenic purpura based on a bleeding score rather than platelet count. Haematologica. 2005 Jan 1;90(6):829-832.
20. Arnold DM, Nazi I, Warkentin TE, Smith JW, Toltl LJ, George JN, Kelton JG. Approach to the diagnosis and management of drug-induced immune thrombocytopenia. Transfusion medicine reviews. 2013 Jul 1;27(3):137-145.
21. Bowles KM, Cooke LJ, Richards EM, Baglin TP. Platelet size has diagnostic predictive value in patients with thrombocytopenia. Clinical & Laboratory Haematology. 2005 Dec;27(6):370-373.
22. Afdhal N, McHutchison J, Brown R, Jacobson I, Manns M, Poordad F, *et al*. Thrombocytopenia associated with chronic liver disease. Journal of hepatology. 2008 Jun 1;48(6):1000-1007.
23. Skipper MT, Rubak P, Larsen OH, Hvas AM. Thrombocytopenia model with minimal manipulation of blood cells allowing whole blood assessment of platelet function. Platelets. 2016 May 18;27(4):295-300.
24. Lippi G, Pavesi F, Pipitone S. Evaluation of mean platelet volume with four hematological analyzers: harmonization is still an unresolved issue. Blood Coagulation & Fibrinolysis. 2015 Mar 1;26(2):235-237.
25. Hoffmann JJ. Reticulated platelets: analytical aspects and clinical utility. Clinical Chemistry and Laboratory Medicine (CCLM). 2014 Aug 1;52(8):1107-1117.
26. Greinacher A, Selleng K. Thrombocytopenia in the intensive care unit patient. Hematology 2010, the American Society of Hematology Education Program Book. 2010 Dec 4;2010(1):135-143.