Original Article

DOI: 10.7860/JCDR/2023/62697.18242

Pathology Section

Distribution and Aetiology of Thrombocytosis in Inpatients Setting of a Tertiary Care Hospital: A Cross-section Study from Western Maharashtra, India

SUDITI WASNIK[1], ASHWINI REGE GUNDAWAR[2], MANGALA NAGARE[3], VARUN DAKE[4], HARSHA DANGARE[5], SMITA BHIDE[6]

ABSTRACT

Introduction: Thrombocytosis is characterised by an increased platelet count in the blood, defined as a count greater than 450,000 cells/μL. The incidental discovery of thrombocytosis often leads to unnecessary investigations and referrals, causing anxiety among physicians.

Aim: This study aims to examine the presence, frequency, and etiological distribution of thrombocytosis in various disease conditions.

Materials and Methods: This cross-sectional observational study was conducted at the Central Clinical Laboratory of MIMER Medical College and Dr. BSTR tertiary care hospital in Talegaon Dabhade, Pune, Maharashtra, India, from June 1st, 2021, to August 31st, 2021. Clinical and laboratory data were collected from adult patients with a platelet count greater than 450,000 cells/μL and entered into a Microsoft Excel sheet. The parameters studied included age, sex, clinical diagnosis, platelet count, Total Leucocyte Count (TLC), absolute neutrophil count, Neutrophil Lymphocyte Ratio (NLR), haemoglobin levels, and C-reactive protein (CRP). Pearson's correlation coefficient was calculated using Statistical Package for the Social Sciences (SPSS) software version 26.0.

Results: A total of 194 patients with a platelet count greater than 450,000 cells/μL were included in the study. The frequency of thrombocytosis was 8.50% (194 patients), with 113 cases in the Medicine Inpatient Department (IPD) and 81 cases in the Surgery IPD. The lowest platelet count observed was 454,000 cells/μL, while the highest was 855,000 cells/μL. Primary thrombocytosis was found in 2 (1.03%) patients, while secondary thrombocytosis was found in 192 (98.96%) patients. A statistically significant association was observed between thrombocytosis and ferritin (p-value=0.032). Additionally, significant associations were found between thrombocytosis and absolute neutrophil count (p-value=0.023) and NLR (p-value=0.047).

Conclusion: Elevated platelet counts, discovered during routine blood examinations, carry diagnostic, prognostic, and therapeutic implications as they can be indicative of various clinical situations with diverse underlying aetiologies. It is essential to rule out secondary thrombocytosis before further investigating for primary thrombocytosis. Thrombocytosis warrants thorough investigations and careful clinical correlation.

Keywords: Blood platelet counts, Complete blood count, Polycythaemia vera, Thrombocythaemia

INTRODUCTION

Thrombocytosis is defined as an increase in the number of platelets in the blood, with a platelet count greater than 450,000 cells/μL [1]. Platelets, also known as thrombocytes, play a crucial role in hemostasis and inflammation [2]. However, an excessive number of platelets can have detrimental effects, leading to thrombotic episodes or, rarely, bleeding episodes [3]. These unexpected events, such as cerebrovascular accidents or myocardial infarction, can occur in patients who were previously asymptomatic [4,5]. Additionally, it is important to note that normal platelet levels can vary based on age, sex, and geographic location [6-8].

With the increasing use of automated haematology analysers, platelet counts are routinely obtained as part of the Complete Blood Count (CBC) work-up [9-11]. Consequently, thrombocytosis is often detected as an unexpected finding, posing a diagnostic challenge, particularly for primary caregivers. Thrombocytosis can have various aetiologies, including spurious, primary, and secondary/reactive causes [12-14]. Primary thrombocytosis is typically associated with Myeloproliferative Neoplasms (MPNs) and is often asymptomatic until severe thrombotic or bleeding events occur [15-18]. Primary thrombocytosis is more commonly linked to thrombotic or bleeding events compared to secondary thrombocytosis. However, primary thrombocytosis should be diagnosed by excluding secondary causes, as secondary thrombocytosis is usually transient [19,20]. Secondary thrombocytosis is known to be associated with thromboembolic events in the presence of co-morbidities, and it can serve as a marker for hidden diseases, such as unresolved infections or other occult conditions [20,21]. Thrombocytosis has also been shown to predict poor outcomes in various clinical conditions [22]. Moreover, existing scientific literature suggests that thrombocytosis can independently predict outcomes, including death, in different clinical scenarios [23,24]. The presence of thrombocytosis alerts physicians to the need for a thorough evaluation of the patient and the possibility of more intensive treatment protocols. It is also associated with complications in various medical conditions, such as retinopathy in type 2 diabetes mellitus, invasiveness of fungal infections, intraventricular haemorrhage, myocardial infarctions, and postsplenectomy complications [25-27].

The unexpected discovery of thrombocytosis can lead to anxiety among physicians ordering blood work-ups and may result in unnecessary referrals and investigations. In many cases, primary care physicians are unaware of the significance of this finding, potentially leading to a lack of further evaluation when necessary. While thrombocytosis has been extensively studied in other countries, there is a lack of research on this topic in India [28,29]. Therefore, there is a need to bridge this knowledge gap and understand the frequency and associated comorbidities of thrombocytosis in routine

examinations in the Indian setting. The objective of this study was to determine the frequency and etiological distribution of thrombocytosis in non pregnant adult patients admitted to a tertiary care rural teaching hospital in Western Maharashtra, India, and to correlate the presence of thrombocytosis with various blood parameters.

MATERIALS AND METHODS

This was a cross-sectional observational study conducted on adult patients admitted to MIMER Medical College and Dr. BSTR tertiary care hospital at the Central Clinical Laboratory, Talegaon Dabhade, Pune, Maharashtra, India, from June 1st, 2021, to August 31st, 2021. Ethical approval was obtained from the Institutional Ethical Committee (IEC no. 2020/711), ensuring compliance with all ethical regulations.

Inclusion criteria: The study included indoor (admitted) patients aged 18 years and above from the Medicine and Surgery Inpatient Departments (IPD) with a platelet count greater than 450,000 cells/μL.

Exclusion criteria: Patients below 18 years of age and pregnant females were excluded from the study.

Study Procedure

Data was collected prospectively. Relevant Complete Blood Count (CBC) data was obtained from the laboratory records of indoor patients in the Surgical and Medicine wards. Clinical and laboratory data of patients with a platelet count greater than 450,000 cells/μL were collected. The following secondary variables were recorded: age, sex, clinical diagnosis, platelet count (150,000-450,000 cells/μL), Total Leucocyte Count (TLC) (4,000-11,000 cells/μL), absolute neutrophil count, Neutrophil Lymphocyte Ratio (NLR), haemoglobin levels (12-16 gm/dL), C-reactive protein (CRP) levels (less than 0.6 mg/L), and ferritin levels (24-336 ng/mL). Based on clinical details, bone marrow findings, and genetic abnormalities, cases were classified into primary and secondary thrombocytosis. The World Health Organisation (WHO) defines essential thrombocytosis as a platelet count greater than 450,000 cells/μL and the presence of either a Janus Kinase 2 (JAK2), Calreticulin (CALR), or Myeloproliferative Leukemia virus oncogene (MPL) mutation, with no clonal or reactive causes [30]. Secondary thrombocytosis refers to a high platelet count caused by another underlying disease or condition.

STATISTICAL ANALYSIS

Appropriate statistical analyses were carried out to study the possible correlation of each laboratory and clinical parameter with the presence of thrombocytosis, as defined above. Univariate and multivariate analyses were assessed using Pearson's Correlation test in SPSS software version 26.0. A significance level of p-value <0.05 was considered statistically significant for all analyses.

RESULTS

A total of 194 patients with a platelet count >450,000 cells/cumm were observed during the study period. Out of the total population of 2,280 patients admitted to the Surgery and Medicine wards, 194 patients were identified to have thrombocytosis, accounting for 8.50% of the total population. Among these, 113 patients were from the Medicine Inpatient Department (IPD) and 81 patients were from the Surgery IPD. The distribution of males and females in the sample population was 128 and 66, respectively, as shown in [Table/Fig-1]. The age of the study population ranged from 18 to 90 years, with an average age of 53±14 years. The average age for males was 51.78 years and for females was 57.27 years.

Parameters		Values
Age (years) (Mean, Range)		53.59, 18-90
Sex (n)	Male	128
	Female	66

[Table/Fig-1]: Mean age group and gender distribution.

Primary thrombocytosis was identified as the cause of platelet elevation in only 2 (1.03%) patients, while secondary thrombocytosis was the most frequent cause, accounting for 98.96% (192 patients) of the study group, as shown in [Table/Fig-2]. The two patients with primary thrombocytosis had essential thrombocytosis and polycythemia vera, respectively. Among the patients with secondary thrombocytosis, 51 (26.56%) patients had diabetes, 3 (1%) patients had a history of recent surgery, and 2 patients had underlying infections. A total of 21 patients had tissue damage and inflammatory conditions, while 13 patients had a history of myocardial infarction, 4 patients had non Myeloproliferative Neoplasm (non MPN) type malignancies, and 5 patients had gastroenteritis. Inflammatory conditions included rheumatoid arthritis, sarcoidosis, psoriasis, and inflammatory bowel disease, as shown in [Table/Fig-2].

Aetiology	n (%)
1. Primary thrombocytosis	2 (1.03)
• Essential thrombocytosis	1 (50)
• Polycythaemia vera	1 (50)
2. Secondary thrombocytosis	192 (98.96)
• Infections	73 (38.02)
• Diabetes	51 (26.56)
• Inflammatory conditions	27 (14.06)
• Tissue damage	21 (10.93)
• Mycoradial infarction	13 (6.77)
• Maligancy	4 (2.08)
• Postsplenectomy	3 (1.56)

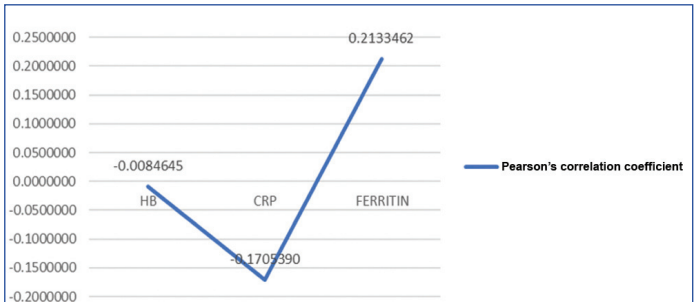
[Table/Fig-2]: Etiological distribution in various disease conditions.

The lowest platelet count observed in the patients was 454,000 cells/cumm, while the highest was 855,000 cells/cumm. The average platelet count among males was 507,948 cells/cumm, while among females it was 520,909 cells/cumm. CRP levels ranged from negative (less than 0.6 mg/L) to 9.6 mg/L, while serum ferritin levels ranged from 4 to 632 ng/mL, as shown in [Table/Fig-3].

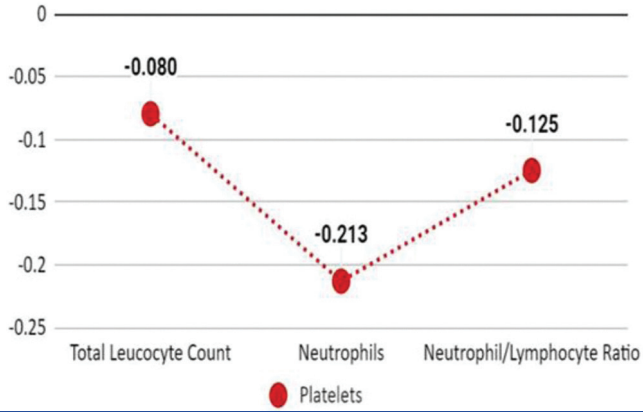
Haematological parameters	Mean value	Range
Platelet count (cells/cumm)	517310	454000-855000
TLC (cells/cumm)	11382	1490-56330
Neutrophills (cells/cumm)	9124.52	715-50133
NLR	10.14	1.06-96
Haemoglobin (g/dL)	12.2	6.1-16.8
CRP (mg/L)	6.0	0.6-9.6
Ferritin (ng/mL)	82.12	4- 632

[Table/Fig-3]: Mean value of haematological parameters.

The correlation of thrombocytosis with blood parameters (CRP, ferritin) is shown in [Table/Fig-4a]. A statistically significant correlation was observed between thrombocytosis and ferritin (p-value=0.032). [Table/Fig-4b] shows the correlation of TLC, absolute neutrophil count, and NLR. Statistically significant correlations were found



[Table/Fig-4a]: Correlation coefficients of CRP (r-value=0.17, p-value=0.065), haemoglobin (r-value=0.0085, p-value=0.98) and ferritin (r-value=0.21, p-value=0.032) with thrombocytosis (significant p-value <0.05).



[Table/Fig-4b]: Correlation of thrombocytosis with TLC (r-value=-0.080, p-value=0.786), Neutrophils (r-value=-0.213, p-value=0.023), NLR (r-value=-0.125, p-value=0.047).

with absolute neutrophil count and NLR, with p-values of 0.023 and 0.047, respectively. No statistically significant association was found between thrombocytosis and TLC, haemoglobin (Hb), and CRP. Among the patients, 47% had haemoglobin levels less than 12.5 gm/dL. The positive correlation indicates that thrombocytosis patients may have elevated ferritin levels. However, in anaemic patients, the correlation between thrombocytosis and ferritin levels was strongly negative (r-value=-0.81), whereas in the whole study group the correlation was positive (r-value=0.21) [Table/Fig-4a].

DISCUSSION

Thrombocytosis, also known as thrombocythemia, is a condition characterised by a platelet count exceeding 450,000 cells/μL. It can be classified into two groups: primary thrombocytosis and secondary (or reactive) thrombocytosis [7]. The increasing use of automated haematology analysers has made platelet count a routine part of the complete blood count (CBC) work-up, leading to the detection of thrombocytosis as an unexpected finding [9-11]. This unexpected finding poses a diagnostic challenge, particularly for primary caregivers. Platelets are acute phase reactants, so their count increases in response to various stimuli, including systemic infections, inflammatory conditions, tumors, and bleeding [24-26, 30-32]. This type of thrombocytosis is known as reactive or secondary thrombocytosis, and it is a benign, nonclonal form. Thrombocytosis is driven by the overproduction of thrombopoietin, interleukin-6, other cytokines, or catecholamines in inflammatory, infectious, neoplastic conditions, or situations of stress [33,34]. In iron deficiency anaemia, elevated platelet count is due to megakaryocyte proliferation, while in asplenia, decreased platelet sequestration leads to thrombocytosis. Secondary thrombocytosis has been found to serve as a marker for hidden diseases, such as undrained focus of infection or other occult comorbidities [20,21]. On the other hand, clonal thrombocytosis (primary or essential thrombocytosis) is an abnormality of platelet production caused by unregulated clonal expansion of bone marrow progenitor cells [30,35].

Secondary thrombocytosis is usually identified through routine laboratory evaluation, as most patients are asymptomatic. However, patients may exhibit symptoms related to the primary condition that precipitated the thrombocytosis, such as infection, surgery, or inflammatory disease [20-22,36]. When the clinical presentation does not clearly differentiate between primary and secondary thrombocytosis, further tests are required to exclude or confirm a diagnosis of disorders causing clonal thrombocytosis. In the present study, the frequency of patients with secondary thrombocytosis was much higher than primary thrombocytosis, which is consistent with the findings of studies conducted by Saadia A et al. (99.4% cases with secondary thrombocytosis) and Griesshammer M et al. (87.7% patients with secondary thrombocytosis), emphasising the prevalence of thrombocytosis in adults [11,12].

The difference in the average level of thrombocytosis between males (507,948 cells/cumm) and females (520,909 cells/cumm) was nearly the same and contrary to previous research findings [13]. The underlying aetiology of secondary thrombocytosis among the patients in the present study was mostly related to underlying infections or inflammatory conditions. Out of the 63% of patients with secondary thrombocytosis, the majority had an infection (including respiratory tract infections like COVID-19, with 37 cases), while others had chronic inflammatory conditions (13.91%) such as osteoarthritis, tuberculosis, type 2 diabetes mellitus, obesity, etc., or tissue damage (9.79%). This observation is supported by findings from other studies, including the study by Griesshammer M et al., which showed that the most frequent causes of secondary thrombocytosis were tissue damage (42%), infection (24%), malignancy (13%), and chronic inflammation (10%), indicating that these conditions are causative factors for secondary thrombocytosis [9,12-14]. However, more research is needed to establish a firm conclusion regarding the association between COVID-19 and thrombocytosis. Another contributing factor to secondary thrombocytosis was anaemia (iron deficiency) in the patients, with the majority being females. The present study found that anaemic patients with thrombocytosis had lower levels of ferritin compared to non anaemic patients. This could be due to different underlying mechanisms of thrombocytosis, as in anaemic patients, thrombocytosis is a result of megakaryocyte activation and increased platelet production, rather than an acute phase response associated with high ferritin levels. This result is supported by Park MJ et al. [37]. The present study showed that only 1% (2 patients) of the sample had primary thrombocytosis, which was due to an underlying myeloproliferative disorder (MPN), contrary to other research studies on thrombocytosis that have a significant cohort of primary thrombocytosis with MPN as the aetiology [15-18]. One possible reason for such variation could be that the research was conducted in a semi-rural hospital, with most patients presenting with such conditions being referred to higher centers.

In the present study, it was observed that 2% of the population (four patients) had coexisting malignancy along with thrombocytosis. Two of these patients had gastrointestinal malignancy, one had lung cancer, and one had breast carcinoma. Previous studies, such as the one by Cozzi GD et al., have shown thrombocytosis in 20-50% of ovarian cancer cases, while the study by Bailey SE et al. reported associations between thrombocytosis and gastrointestinal malignancies, lung cancers, malignant lymphomas, and ovarian carcinoma [23,26]. Thrombocytosis can serve as an independent marker of outcome, including death, in various clinical situations [23,24]. It has also been linked to complications in different medical conditions, such as retinopathy in type 2 diabetes mellitus, invasiveness of fungal infections, intraventricular haemorrhage, myocardial infarctions, and postsplenectomy complications [25-31]. The finding of thrombocytosis in a patient should alert the physician to the need for careful and detailed evaluation, as well as potential further investigations. However, the accidental finding of thrombocytosis can create fear in physicians and lead to unnecessary referrals and investigations. Alternatively, primary care physicians may be unaware of the significance of this finding, resulting in a lack of further work-up when it is needed [31].

Limitation(s)

The study population was limited to indoor patients, so the findings cannot be generalised to the general population.

CONCLUSION(S)

Adult thrombocytosis is mostly secondary, with only a few cases of primary thrombocytosis. The most common causes of secondary thrombocytosis are infections, inflammatory conditions, and diabetes. It is usually a transient condition with no major clinical

implications. If no secondary cause of an increased platelet count is found or if it persists after treating the primary cause, a search for an underlying primary thrombocytosis should be conducted.

REFERENCES

[1] McPherson R, Pincus M. Henry's Clinical Diagnosis and Management by Laboratory Methods. Philadelphia.
[2] Bleeker JS, Hogan WJ. Thrombocytosis: Diagnostic evaluation, thrombotic risk stratification, and risk-based management strategies. Thrombosis. 2011;2011:536062.
[3] Charles M, Fontoura R, Sugalski G. Early recognition of intraventricular haemorrhage in the setting of thrombocytosis in the emergency department. Open Access Emergency Medicine: OAEM. 2016;8:29-33.
[4] Williams B, Morton C, Sica DA. Cerebral vascular accident in a patient with reactive thrombocytosis: A rare cause of stroke. The American Journal of the Medical Sciences. 2008;336(3):279-81.
[5] Mizuta E, Takeda SI, Sasaki N, Miake J, Hamada T, Shimoyama M, et al. Acute myocardial infarction in a patient with essential thrombocythemia. Circulation Journal. 2005;69(8):1000-02.
[6] Syed NN, Usman M, Khurshid M. Thrombocytosis: Age dependent aetiology and analysis of platelet indices for differential diagnosis. Indian Journal of Pathology & Microbiology. 2007;50(3):628-33.
[7] Harrison CN, Bareford D, Butt N, Campbell P, Conneally E, Drummond M, et al. Guideline for investigation and management of adults and children presenting with a thrombocytosis. British Journal of Haematology. 2010;149(3):352-75.
[8] Biino G, Santimone I, Minelli C, Sorice R, Frongia B, Traglia M, et al. Age-and sex-related variations in platelet count in Italy: A proposal of reference ranges based on 40987 subjects' data. PloS one. 2013;8(1):e54289.
[9] Kiro K, Ganjoo P, Saigal D, Hansda U. Incidental thrombocytosis: Should it concern the anesthesiologist? Journal of Anaesthesiology, Clinical Pharmacology. 2014;30(2):281-83.
[10] Schafer AI. Thrombocytosis: When is an incidental finding serious. Cleveland Clinical Journal of Medicine. 2006;73(8):767-74.
[11] Griesshammer M, Bangerter M, Sauer T, Wennauer R, Bergmann L, Heimpel H. Aetiology and clinical significance of thrombocytosis: Analysis of 732 patients with an elevated platelet count. Journal of Internal Medicine. 1999;245(3):295-300.
[12] Saadia A, Farhan S, Butt T, Mumtaz A. Thrombocytosis: Frequency and etiologic analysis. Pak J Med Health Sci. 2015;9:681-84.
[13] Biino G, Balduini CL, Casula L, Cavallo P, Vaccargiu S, Parracciani D, et al. Analysis of 12,517 inhabitants of a Sardinian geographic isolate reveals that predispositions to thrombocytopenia and thrombocytosis are inherited traits. Haematologica. 2011;96(1):96-101.
[14] Kaushansky K. The molecular mechanisms that control thrombopoiesis. The Journal of Clinical Investigation. 2005;115(12):3339-47.
[15] Essential Thrombocythemia Facts (No. 12 in a series providing the latest information for patients, caregivers and healthcare professionals), webpage of www.LLS.org (leukemia and lymphoma society).
[16] Thiele J, Kvasnicka HM. The 2008 WHO diagnostic criteria for polycythemia vera, essential thrombocythemia, and primary myelofibrosis. Current Hematologic Malignancy Reports. 2009;4(1):33-40.
[17] Wilkins BS, Erber WN, Bareford D, Buck G, Wheatley K, East CL, et al. Bone marrow pathology in essential thrombocythemia: Interobserver reliability and utility foridentifying disease subtypes. Blood, The Journal of the American Society of Hematology. 2008;111(1):60-70.
[18] Tefferi A, Barbui T. Polycythemia vera and essential thrombocythemia: 2017 update on diagnosis, risk-stratification, and management. American Journal of Hematology. 2017;92(1):94-108.
[19] Tchebiner JZ, Nutman A, Boursi B, Shlomai A, Sella T, Wasserman A, et al. Diagnostic and prognostic value of thrombocytosis in admitted medical patients. The American Journal of the Medical Sciences. 2011;342(5):395-401.
[20] Samra T, Pawar M. Reactive thrombocytosis in H1N1 infection. Journal of Laboratory Physicians. 2011;3(2):131-32.
[21] Ji S, Zhang J, Fan X, Wang X, Ning X, Zhang B, et al. The relationship between mean platelet volume and diabetic retinopathy: A systematic review and meta-analysis. Diabetology & Metabolic Syndrome. 2019;11(1):25.
[22] Prina E, Ferrer M, Ranzani OT, Polverino E, Cillóniz C, Moreno E, et al. Thrombocytosis is a marker of poor outcome in community-acquired pneumonia. Chest. 2013;143(3):767-75.
[23] Bailey SE, Ukoumunne OC, Shephard E, Hamilton W. How useful is thrombocytosis in predicting an underlying cancer in primary care? A systematic review. Family Practice. 2016;34(1):04-10.
[24] Mohamud M, Osborne L, Jones HG, Ahmed A, Beynon J, Harris DA, et al. Thrombocytosis as a marker for postoperative complications in colorectal surgery. Gastroenterology Research and Practice. 2018;2018:1978639.
[25] Harrison MT, Short P, Williamson PA, Singanayagam A, Chalmers JD, Schembri S. Thrombocytosis is associated with increased short and long term mortality after exacerbation of chronic obstructive pulmonary disease: A role for antiplatelet therapy? Thorax. 2014;69(7):609-15.
[26] Cozzi GD, Samuel JM, Fromal JT, Keene S, Crispens MA, Khabele D, et al. Thresholds and timing of pre-operative thrombocytosis and ovarian cancer survival: Analysis of laboratory measures from electronic medical records. BMC Cancer. 2016;16:612.
[27] Ramalimgam P, Ganapathri K, Jawahar B. Postsplenectomy thrombocytosis and managements. Open Access Blood Research & Transfusion Journal. 2018;1(5):96-99.
[28] Aydogan T, Kanbay M, Alİcİ O, Kosar A. Incidence and etiology of thrombocytosis in an adult Turkish population. Platelets. 2006;17(5):328-31.
[29] Rose SR, Petersen NJ, Gardner TJ, Hamill RJ, Trautner BW. Etiology of thrombocytosis in a general medicine population: analysis of 801 cases with emphasis on infectious causes. J Clin Med Res. 2012;4(6):415-23.
[30] Arber DA, Orazi A, Hasserjian R, Thiele J, Borowitz MJ, Le Beau MM, et al. The 2016 revision to the World Health Organization classification of myeloid neoplasms and acute leukemia. Blood, The Journal of the American Society of Hematology. 2016;127(20):2391-405.
[31] Vora AJ, Lilleyman JS. Secondary thrombocytosis. Archives of Disease in Childhood. 1993;68(1):88-90.
[32] Chiarello P, Magnolia M, Rubino M, Liguori SA, Miniero R. Thrombocytosis in children. Minerva Pediatrica. 2011;63(6):507-13.
[33] Tefferi A, Barbui T. Polycythemia vera and essential thrombocythemia: 2017 update on diagnosis, risk-stratification, and management. American Journal of Hematology. 2017;92(1):94-108.
[34] Cheung MC, Hicks LK, Pendergrast J. Thrombocytosis. The New England Journal of Medicine. 2004;350(24):2524-25.
[35] Araneda M, Krishnan V, Hall K, Kalbfleisch J, Krishnaswamy G, Krishnan K. Reactive and clonal thrombocytosis: Proinflammatory and hematopoietic cytokines and acute phase proteins. Southern Medical Journal. 2001;94(4):417-20.
[36] Rokkam VR, Kotagiri R. Secondary Thrombocytosis. StatPearls [Internet]. 2022 Jan.
[37] Park MJ, Park PW, Seo YH, Kim KH, Park SH, Jeong JH, et al. The relationship between iron parameters and platelet parameters in women with iron deficiency anaemia and thrombocytosis. Platelets. 2013;24(5):348-51.

PARTICULARS OF CONTRIBUTORS:
1. Intern, Department of Pathology, MIMER Medical College, Talegaon Dabhade, Pune, Maharashtra, India.
2. Associate Professor, Department of Pathology, Zydus Medical College, Dahod, Gujarat, India.
3. Assistant Professor, Department of Pathology, MIMER Medical College, Talegaon Dabhade, Pune, Maharashtra, India.
4. Intern, Department of Pathology, MIMER Medical College, Talegaon Dabhade, Pune, Maharashtra, India.
5. Associate Professor, Department of Pathology, MIMER Medical College, Talegaon Dabhade, Pune, Maharashtra, India.
6. Professor and Head, Department of Pathology, MIMER Medical College, Talegaon Dabhade, Pune, Maharashtra, India.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:
Dr. Harsha Dangare,
Associate Professor, Department of Pathology, MIMER Medical College,
Talegaon Dabhade, Pune-410507, Maharashtra, India.
E-mail: dr.harshahj@gmail.com

PLAGIARISM CHECKING METHODS: [Jain H et al.]
- Plagiarism X-checker: Jan 06, 2023
- Manual Googling: Mar 23, 2023
- iThenticate Software: Apr 25, 2023 (13%)

ETYMOLOGY: Author Origin

EMENDATIONS: 9

AUTHOR DECLARATION:
- Financial or Other Competing Interests: None
- Was Ethics Committee Approval obtained for this study? Yes
- Was informed consent obtained from the subjects involved in the study? Yes
- For any images presented appropriate consent has been obtained from the subjects. NA

Date of Submission: **Jan 06, 2023**
Date of Peer Review: **Mar 03, 2023**
Date of Acceptance: **May 01, 2023**
Date of Publishing: **Jul 01, 2023**