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RESEARCH ARTICLE

Study on the Causes and Clinical Features of Thrombocytopenia in a Tertiary Care Centre

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Abstract: ***Background:*** Thrombocytopenia refers to a reduction in platelet concentration to below 150,000 per microliter of blood. It may arise from diverse etiologies and presents with a wide spectrum of clinical features. The present investigation focuses on identifying the underlying causes, categorizing the degree of platelet reduction, and assessing how the condition varies with respect to age and gender. Objective: To determine the range of causes contributing to thrombocytopenia, classify its severity according to platelet count thresholds, and evaluate its occurrence across different age brackets and between sexes. Methods: A retrospective review was conducted on 200 patient records from Saveetha Medical College between October and December 2023. Blood counts were obtained using an automated hematology analyzer, and only samples showing platelet levels under 150,000/μL were included for further study. Confirmation of thrombocytopenia was performed by preparing and reviewing peripheral blood smears. Results: The leading cause identified was acute febrile illness, followed in frequency by megaloblastic anemia and dengue infection. The condition was observed more often among male patients, with the highest incidence in individuals aged 21–40 years. The majority of affected cases fell into the Grade 1 category of thrombocytopenia. Conclusion: Infectious diseases, notably dengue, remain the primary contributors to reduced platelet counts. Acute febrile conditions also play a major role in disease burden. The disorder predominantly affects males and is most common in the 21–40 year age range, with mild cases being the most frequent presentation.

Keywords: Thrombocytopenia, Acute febrile illness, Megaloblastic anemia, Dengue fever, Platelet count, Hematology

INTRODUCTION

Thrombocytopenia denotes a pathological decrease in circulating platelet concentration, with values falling beneath 150,000 per microliter generally regarded as the physiological lower threshold. As illustrated in Figure 1, this hematologic abnormality can arise from insufficient megakaryocyte activity in the bone marrow, nutritional deficiencies, excessive splenic sequestration, or accelerated peripheral destruction due to immune, infectious, or drug-induced causes [1]. The impairment may stem from either congenital abnormalities or conditions acquired later in life. Such a decline disrupts the integrity of primary haemostasis by hindering platelet plug formation, which prolongs bleeding time and manifests clinically as petechial eruptions, purpuric patches, or hemorrhage from mucosal and cutaneous sites. Morphological evaluation through bone marrow aspiration or biopsy enables assessment of megakaryocyte abundance - whether reduced, within normative limits, or increased -thereby contributing critical diagnostic context. Often, a detailed clinical history and comprehensive physical examination can delineate the probable etiology [2].

The clinical behavior of thrombocytopenia spans a broad spectrum, from subclinical reductions to fulminant presentations with catastrophic bleeding. Diagnostic refinement in certain instances necessitates specialized laboratory assays, for example in suspected paroxysmal nocturnal hemoglobinuria or systemic lupus erythematosus. Within tropical epidemiologic settings such as India, infectious agents dominate the etiological profile, typically accompanied by febrile episodes [3]. Predominant contributors include microbial infections, pharmacologically induced platelet loss, autoimmune pathologies, splenic hyperactivity, disseminated intravascular coagulation, and vector-borne diseases such as malaria, leptospirosis, rickettsioses, septicemia, typhoid fever, brucellosis, arboviral infections, visceral leishmaniasis, and thrombotic microangiopathies including thrombotic thrombocytopenic purpura and hemolytic uremic syndrome [4].
Frequently, platelet deficiency is identified incidentally during routine hematologic screening in otherwise asymptomatic individuals. Symptomatic cases may present with ecchymoses, purpura, petechial rashes, epistaxis, or gingival hemorrhage. Critically diminished platelet levels -approximately 5,000 per microliter -carry high risk for bleeding into vital sites such as the central nervous system, gastrointestinal tract, or genitourinary system. Generally, values exceeding 100,000/μL are considered hemostatically competent[5,6].

Severity stratification is conventionally defined as:
Grade 1: 100,000–150,000/μL

Grade 2: 50,000–100,000/µL
Grade 3: <50,000/µL

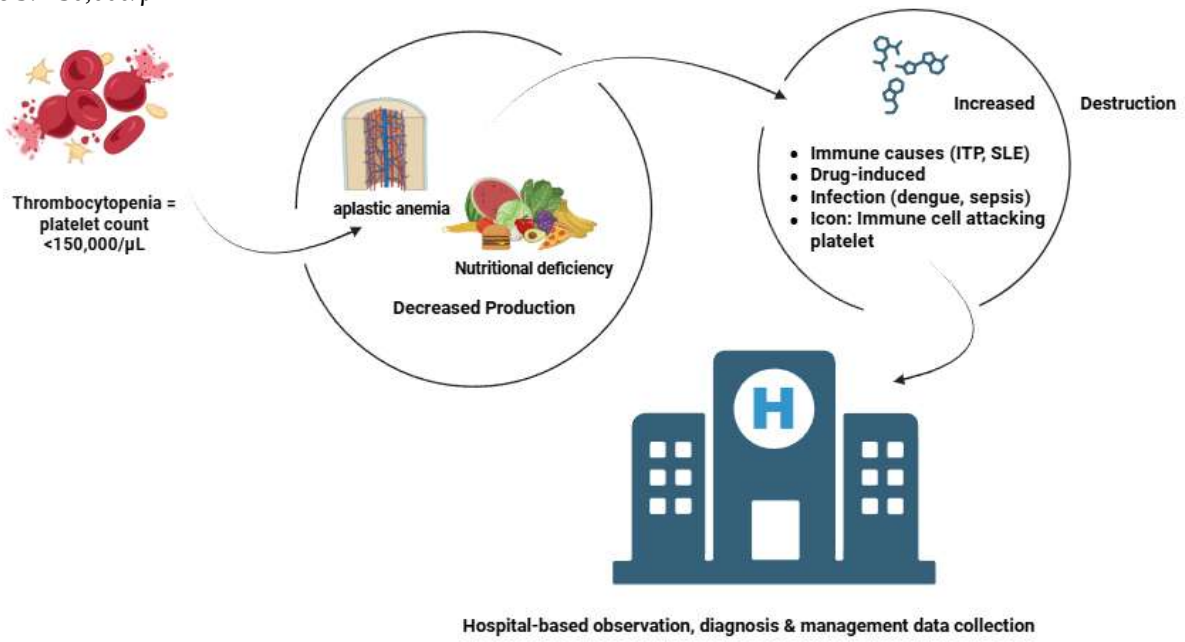

Figure 1: Overview of thrombocytopenia causes, showing decreased production (aplastic anemia, nutritional deficiencies) and increased destruction (immune, drug-induced, infectious).
Illustrates hospital-based observation, diagnosis, and management pathways for affected patients.

Multiple disease states may underlie this disorder, ranging from acute systemic infections and hematologic malignancies to aplastic anemia, pernicious anemia [7], viral exanthems, splenomegaly, scarlet fever, tuberculosis, idiopathic purpura, and metabolic disorders like Gaucher's disease. Hemorrhagic risk correlates inversely with platelet count -spontaneous mucocutaneous bleeding is typically observed at counts below 20,000/µL, whereas petechiae and purpura emerge more often within the intermediate range of 50,000–100,000/µL. Etiologically, thrombocytopenia can be categorized into four principal mechanisms: suppression of platelet synthesis, heightened peripheral destruction, splenic sequestration, and dilutional depletion secondary to massive transfusion or fluid replacement [8].

MATERIAL AND METHODS

This investigation employed a retrospective, observational design and was carried out at Saveetha Medical College and Hospital over a three-month period, from October to December 2023. Hematologic profiles were assessed using the SYSMEX XN-1000, a fully automated six-part differential analyzer, enabling evaluation of multiple hematological indices. Clinical records and laboratory findings were reviewed for adult patients whose platelet counts were measured at below 150,000 cells/µL [9]. Relevant data were compiled into a Microsoft Excel database for analysis. **Inclusion criteria** encompassed all hospitalized patients, irrespective of age, provided their platelet concentration fell under the 150,000 cells/µL threshold. The study ultimately comprised 100 confirmed cases of thrombocytopenia. Key parameters examined included patient age, sex, provisional or final clinical diagnosis, and absolute platelet count [10].

RESULTS AND OBSERVATIONS:

Table 1: Sex-wise Distribution of Cases

Sex	Number of Cases	Percentage (%)
Male	68	68%
Female	32	32%
Total	**100**	**100%**

Table 2: Age Group-wise Distribution

Age Group (Years)	Number of Cases	Percentage (%)
< 20	20	20%
21–40	42	42%
41–60	24	24%
> 61	16	16%
Total	**100**	**100%**

Table 3: Distribution of Grades

Grade	Number of Cases	Percentage (%)
Grade 1	42	42%
Grade 2	28	28%
Grade 3	30	30%
Total	**100**	**100%**

Table 4: Aetiology of Cases

Aetiology	Number of Cases	Percentage (%)
AFI	22	22%
Megaloblastic Anemia	18	18%
Dengue Fever	16	16%
Others	15	15%
Sepsis	10	10%
CKD	8	8%
Iron Deficiency Anemia (IDA)	5	5%
Leukemia	3	3%
Malaria	3	3%
Total	**100**	**100%**

DISCUSSION

In the present investigation, infectious pathologies emerged as the predominant drivers of thrombocytopenia within the study cohort. The most frequently encountered etiologies were acute febrile syndromes and megaloblastic anemia, followed by dengue virus infection. In dengue, thrombocytopenia is a well-recognized hematologic hallmark across both uncomplicated and severe disease states. Platelet concentrations commonly fall below 150 × 10^3/µL, and in more advanced stages may decline beneath 40 × 10^3/µL between the third and seventh febrile day. Profound cytopenia at these levels can necessitate transfusional support [11, 12] . The pathogenesis involves dual mechanisms-suppression of megakaryopoiesis within the bone marrow and accelerated peripheral destruction coupled with reticuloendothelial clearance [13].

Pathobiological research implicates aberrant platelet activation and qualitative dysfunction in the genesis of thrombo-occlusive sequelae seen in Dengue

Hemorrhagic Fever and Dengue Shock Syndrome. Early-phase viremia is frequently accompanied by heightened expression of platelet surface P-selectin and apoptotic signaling markers, such as caspase upregulation and externalized phosphatidylserine [14].
Thrombocytopenia is also frequently documented in Plasmodium falciparum and Plasmodium vivax infections. In the current series, the detection of low platelet counts served as a supportive diagnostic pointer toward malaria [15]. Published prevalence rates for platelet counts under 150 × 10^3/mm^3 in malarial cases span from 24% to 94%. The underlying mechanism is thought to involve adsorption of malarial antigens onto platelet membranes, thereby triggering binding of antimalarial antibodies and subsequent immune complex deposition.

In our dataset, dengue accounted for 15% of cases, which contrasts with higher frequencies reported by Paramjit (27.7%) and Lakum (35.4%). Notably, malaria accounted for only 3% of cases in our study, despite being reported as a significant contributor in other series. Megaloblastic anemia ranked as the second most prevalent cause [16]
.
Hepatic disease–associated thrombocytopenia arises through multifactorial processes. Dysfunction of the platelet glycoprotein GPIIb–IIIa complex-pivotal for fibrinogen-mediated aggregation and signal transduction-has been implicated. In renal insufficiency, circulating peptides containing the Arg–Gly–Asp sequence of fibrinogen can accumulate, competitively inhibiting receptor engagement and impairing platelet bridging [17].

In uraemia, bleeding diathesis arises from multiple platelet-related derangements:
1. Impaired adhesion via GPIb–V–IX–von Willebrand factor interactions, which may improve with increased vWF availability.
2. Secretory defects linked to heightened prostacyclin and nitric oxide synthesis, elevating intraplatelet cAMP.
3. Diminished platelet stores of ADP, serotonin, and thromboxane A_2, signifying an acquired storage pool defect secondary to uraemic toxicity.

Chronic liver disease–related thrombocytopenia not only heightens hemorrhagic risk but complicates clinical management. Contributory factors encompass hypersplenism with excessive platelet pooling, suboptimal thrombopoietin production relative to the degree of thrombocytopenia, autoimmune-mediated platelet destruction, and marrow suppression.

Symptomatically, the most prevalent presentations among our patients included generalized fatigue (70%), overt bleeding signs (60%), fever (50%), arthralgia (37%), splenomegaly (35%), headache (30%), dyspnea (23%), lymphadenopathy (22%), hepatomegaly (24%), and abdominal discomfort (12%). Comparative data from a 412-patient cohort identified fever in 79.3% and hemorrhagic manifestations in 11.2% of cases. Modi T et al. similarly described fever as the most common feature, followed by cephalgia, generalized myalgia, emesis, retro-orbital pain, nausea, arthralgia, abdominal tenderness, and respiratory complaints [18].
In our cohort, 60% of thrombocytopenic individuals demonstrated bleeding phenomena. Cutaneous manifestations-including petechiae, purpura, and ecchymoses-were documented in 30% of patients, gingival bleeding in 16.66%, subconjunctival hemorrhage in 15%, melena in 13.33%, and hematemesis in 11.66%. Less common events included hemoptysis (5%), hematuria (3.33%), and epistaxis (3.33%). A study by Patne SV et al. reported a 37.5% bleeding rate among thrombocytopenic cases, with skin and mucosal sites most frequently affected.

CONCLUSION

Thrombocytopenia is a frequently encountered hematological abnormality in routine medical practice and warrants comprehensive investigation to identify its secondary causes. In certain instances, the condition may progress to a critical state, necessitating urgent platelet transfusion. A clear understanding of the etiopathogenic factors is essential for improving clinical outcomes and minimizing both morbidity and mortality. In the present study, megaloblastic anemia emerged as the leading etiology of thrombocytopenia. Timely recognition of this deficiency disorder is pivotal in ensuring effective therapeutic intervention and favorable prognoses. Dengue fever constituted the second most prevalent cause, followed by other acute febrile illnesses. The detection of thrombocytopenia in clinical settings should therefore prompt consideration of these conditions and reinforce the need for early, targeted treatment in affected patients.

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