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Bone Marrow Findings in Cases of Thrombocytopenia.

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ABSTRACT

Background: Thrombocytopenia is commonly encountered in a wide range of haematological and non-haematological disorders. Bone marrow examination plays a major role in diagnosis. Aims: The aim of our study was to analyse the etiology and to study megakaryocytic series in different cases of thrombocytopenia, irrespective of age or sex. **Methods:** This prospective bone marrow aspiration study was done in 100 cases of thrombocytopenia, which were diagnosed on peripheral blood film, to analyse the causes of thrombocytopenia. Megakaryocytes were examined in terms of their number and morphology. Bone marrow aspiration smears were stained with Leishman stain andexamined under light microscope. **Results:** Megaloblasticanemia was the most common cause with 33 (33%) cases followed by acute leukemia with 21 (21%) cases, immune thrombocytopenic purpura with 11 (11%) cases, aplastic anemia with 9 (9%) cases, dimorphic anemia and chronic lymphoproliferative disorders with 6 (6%) cases each, systemic causes with 5(5%) cases, myelodysplastic syndrome with 3 (3%) cases, chronic myeloproliferativeleukemia with 2 (2%) cases and 1 (1%) case each of plasmodium vivax + kalaazar,kalaazar, microfilaria and multiple myeloma. Maximum numbers of dysplastic megakaryocytes were seen in 100% cases of MDS and CML. Most common morphological change seen was micromegakaryocytes. **Conclusion:** Bone marrow examination is an important step to arrive at the probable diagnosis of wide varieties of haematological disorders in the cases of thrombocytopenia. Correlation with clinical, peripheral blood film findings and bone marrow findings provide valuable insight into the etiology of thrombocytopenia.

Keywords: Thrombocytopenia, bone marrow aspiration, Leishman stain, megaloblasticanemia, micromegakryocyte.

INTRODUCTION

Thrombocytopenia is the defined as the fall in the number of platelets below 150,000/µL. The normal range of platelets is between 150,000 and 450,000 cells per µl of blood. The main reasons of thrombocytopenia are decrease in platelet production, increase in destruction of platelets and abnormal platelet distribution (peripheral pooling of platelets). Thrombocytopenia separates three stages as numerical. Mild: 100,000 - 150,000/µL,Moderate: 50,000 - 100,000/µL, Severe: < 50,000/µL. However, thrombocytopenia is not usually detected clinically until the platelet count has fallen to levels below 100,000/µL.[1]

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Platelets are formed and released into the bloodstream by precursor cells called megakaryocytes (MK) that are derived from haematopoietic stem cells (HSCs), which evolve from the multipotential cell. Mature MKs give rise to circulating platelets by the acquisition of the cytoplasmic structural and functional characteristics necessary for platelet action,[2,3] reaching cell sizes 50–100 microns in diameter and ploidy ranging up to 128 N.[4,5] Normal maturation and platelet formation results from megakaryocytic deoxyribonucleic acid (DNA) replication that occurs without cell division resulting in large lobulated, polypoid nucleus.[6] The production of platelets by megakaryocytes requires an intricate series of remodeling events that result in the release of thousands of platelets from a single megakaryocyte. The stages of maturation are megakaryoblast, promegakaryocyte, granular megakaryocyte, mature megakaryocyte & platelets. Defect in any of these stages of megakaryocytopoiesis can lead to dysmegakaryocytopoiesis and thrombocytopenia.

Thrombocytopenia can be classified pathophysiologic ally into four major groups

1. Artifactual thrombocytopenia
2. Accelerated platelet destruction
3. Deficient platelet production
4. Abnormal platelet distribution or pooling

1. Artefactual Thrombocytopenia

Artifactual thrombocytopenia or falsely low platelet counts, occurs ex vivo when platelets are not counted accurately. This should be considered in patients who have thrombocytopenia but no petechiae or ecchymosis. It can be caused due to:

1. Platelet clumping caused by anticoagulant dependent immunoglobulin (psuedothrombocytopenia)
2. Platelet satellitism.[7]
3. Giant platelets.[8]

2. Accelerated Platelet Destruction

Accelerated platelet destruction leads to stimulation of thrombopoiesis and consequently to an increase in number, size and rate of maturation of precursor megakaryocytes.[9] When the rate of platelet destruction exceeds this compensatory increase in platelet production, thrombocytopenia develops. Platelet destruction may result from both intracorpuscular defects and extracorpuscular abnormalities.

It can be:

1. Caused by immunologic processes
   a) Autoimmune
      i. Idiopathic
      ii. Secondary : Infections, pregnancy, collagen vascular disorders, lymphoproliferative disorders, drugs, miscellaneous
   b) Alloimmune
      iii. Neonatal thrombocytopenia
      iv. Post transfusion purpura
2. Caused by nonimmunologic processes Thrombotic microangiopathies
   i. Disseminated intravascular coagulation
   ii. Thrombotic thrombocytopenic purpura
   iii. Hemolytic-uremic syndrome
3. Platelet damage by abnormal vascular surfaces
4. Miscellaneous
   i. Infection
   ii. Massive blood transfusions

3. Deficient Platelet Production

It can be caused due to:

1. Hypoplasia of megakaryocytes
2. Ineffective thrombopoiesis
3. Disorders of thrombopoietic control
4. Hereditary thrombocytopenias such as Wiskott-Aldrich syndrome.[10]

4. Abnormal Platelet Distribution or Pooling:

Abnormal pooling or abnormal in vivo distribution of an essential normal total platelet mass may produce thrombocytopenia. It can be caused due to:

1. Disorders of the spleen (neoplastic, congestive, infiltrative, infectious, of unknown cause)
2. Hypothermia
3. Dilution of platelets with massive transfusions

MATERIALS AND METHODS

The present study was conducted in the Pathology department of Government Medical College, Patiala on 100 cases of thrombocytopenia. The age and sex of the patient were no criterion for selection of cases. A written consent was taken for every case. All the relevant clinical data were collected.

All the cases of thrombocytopenia which were diagnosed by peripheral smear were taken up for the study (platelet count <1,50,000). Bone marrow aspirations were done from posterior superior iliac spine using Salah's bone marrow aspiration needle following standard technique. 0.2 to 2 ml of bone marrow particles was aspirated. Slides were prepared and stained with Leishman stain and were examine under light microscope. Leukaemia cases were further categorised into acute myeloid leukaemia and acute lymphoid leukaemia by cytochemistry (Myeloperoxidase).

The number of megakaryocytes were categorised as normal (one megakaryocytes/ one to three low power field), increased (more than two megakaryocytes/ low power field), decreased (one megakaryocytes/ five to ten low power field) and absent (zero/ ten low power field).

At least 30 megakaryocytes were evaluated for megakaryocytic alterations including both dysplastic and non-dysplastic features. Dysplastic alterations were considered significant only when 10% or more of the megakaryocyte observed show the change.

Dysplastic features of megakaryocytes are multiple separated nuclei, micro megakaryocytes (size <15 µm, nucleus that of a size of small lymphocyte with a single or a bilobed nucleus) and hypo granular forms (megakaryocyte with water clear or pale grey cytoplasm and sparse or no granules). Non-dysplastic features include immature forms, emperipolesis and bare nuclei.

RESULTS

Out of 100 cases of thrombocytopenia, maximum number of cases 29 (29%) were in age group of 16-30 years followed by 21 (21%) in age group 0-15 years, 18 (18%) in age group 31- 35 years, 15 (15%) in age group 46-60 years, 14 (14%) in age group 61 - 75 years and 3 (3%) in age group 76-90 years. The youngest patient was of 1 year and oldest was 90 years of age.

Majority of patients were males 61 (61%) and females were 39 (39%) only. Male to female ratio being 1.6:1.

Table 1: The underlying cause distribution of 100 cases of thrombocytopenia.

Probable disease	No. of cases	Percentage
Megaloblastic Anemia	33	33%
Acute Leukemia	21	21%
Immune thrombocytopenic Purpura	11	11%
Aplastic Anemia	9	9%
Dimorphic Anemia	6	6%

Chronic Lymphoproliferative Disorders	6	6%
Systemic	5	5%
Myelodysplastic syndrome	3	3%
Chronic myeloproliferative leukemia	2	2%
Plasmodium vivax + Kala Azar	1	1%
Kala Azar	1	1%
Microfilaria	1	1%
Multiple myeloma	1	1%
Total	100	100

Table 2: Bone marrow cellularity in 100 cases of thrombocytopenia.

Cellularity	No. of cases	Percentage
Hypercellular	79	79%
Hypocellular	12	12%
Normocellular	9	9%
Total	100	

Table 3: Distribution according to adequacy of megakaryocytes (number per low power field).

Probable disease	Normal	Increased	Decreased	Absent	No. of cases
Megaloblastic Anemia	20 (60.7%)	3 (9.09%)	10 (30.3%)	0 (0%)	33
Acute Leukemia	1 (4.76%)	1 (4.76%)	10 (47.6%)	9 (42.8%)	21
Immune thrombocytopenic Purpura	0 (0%)	11 (100%)	0 (0%)	0 (0%)	11
Aplastic Anemia	1 (11.1%)	0 (0%)	3 (33.3%)	5 (55.6%)	9
Dimorphic Anemia	5 (83.3%)	0 (0%)	1 (16.7%)	0(0%)	6
Chronic Lymphoproliferative Disorders	2 (33.3%)	0 (0%)	4 (66.7%)	0(0%)	6
Systemic	4 (80%)	0 (0%)	1 (20%)	0(0%)	5
Myelodysplastic syndrome	1 (33.3%)	1 (33.3%)	1 (33.4%)	0(0%)	3
Chronic myeloproliferative leukemia	1 (50%)	0 (0%)	1 (50%)	0(0%)	2
Plasmodium vivax + Kala Azar	1 (100%)	0 (0%)	0 (0%)	0(0%)	1
Kala Azar	1 (100%)	0 (0%)	0 (0%)	0(0%)	1
Microfilaria	1 (100%)	0 (0%)	0 (0%)	0(0%)	1
Multiple myeloma	1 (100%)	0 (0%)	0 (0%)	0(0%)	1

Table 4: Distribution according to prevalence of dysplastic & non- dysplastic changes in 100 cases of thrombocytopenia.

Probable disease	Dysplasia	Non-dyspalsia	No. of cases
Megaloblastic Anemia	15 (45.4%)	18 (54.6%)	33
Acute Leukemia	5 (23.8%)	16 (76.2%)	21
Immune thrombocytopenic Purpura	5 (45.4%)	6 (54.6%)	11
Aplastic Anemia	3 (33.3%)	6 (66.7%)	9
Dimorphic Anemia	2 (33.3%)	4 (66.7%)	6
Chronic Lymphoproliferative Disorders	0 (0%)	6 (100%)	6
Systemic	0 (0%)	5 (100%)	5
Myelodysplastic syndrome	3 (100%)	0 (0%)	3
Chronic myeloproliferative leukemia	2 (100%)	0 (0%)	2
Plasmodium vivax + Kala Azar	0 (0%)	1 (100%)	1
Kala Azar	0 (0%)	1 (100%)	1
Microfilaria	0 (0%)	1 (100%)	1
Multiple myeloma	0 (0%)	1 (100%)	1

Table 5: Various morphological changes in megakaryocytes in 13 different causes of thrombocytopenia.

Probable disease	Immature (No./Perc.)	Bare (No./Perc.)	Micromegakaryocyte (No./Perc.)	Hypogranular (No./Perc.)	Hypolobated (No./Perc.)	No. of cases
Megaloblastic Anemia	1 (3%)	4 (12.1%)	8 (24.2%)	2 (6%)	2 (6%)	33
Acute Leukemia	0 (0%)	3 (14.2%)	5 (23.8%)	0 (0%)	2 (9.5%)	21
Immune thrombocytopenic Purpura	7 (63.6%)	1(9%)	2 (18.1%)	2 (18.1%)	1 (9%)	11
Aplastic Anemia	0 (0%)	0 (0%)	0 (0%)	0 (0%)	3 (33.3%)	9
Dimorphic Anemia	0 (0%)	1 (16.6%)	1 (16.6%)	1 (16.6%)	1 (16.6%)	6
Chronic Lymphoproliferative Disorders	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	6
Systemic	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	5
Myelodysplastic syndrome	0 (0%)	1 (33.3%)	2 (66.6%)	0 (0%)	2 (66.6%)	3
Chronic myeloproliferative leukemia	1 (50%)	1 (50%)	2 (100%)	1 (50%)	1 (50%)	2
Plasmodium vivax + Kala Azar	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	1
Kala Azar	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	1
Microfilaria	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	1
Multiple myeloma	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	1

Various morphological features studied were immature megakaryocytes, bare megakaryocytes, micromegakaryocytes, hypogranular megakaryocytes and hypolobated megakaryocytes.

Immature megakaryocyte is defined as young forms with scant bluish cytoplasm and lacking lobulation of the nucleus which occupied most of the cell. Normal megakaryocytes are considered to have four to sixteen nuclear lobes, those having less than four lobes were considered hypolobated.

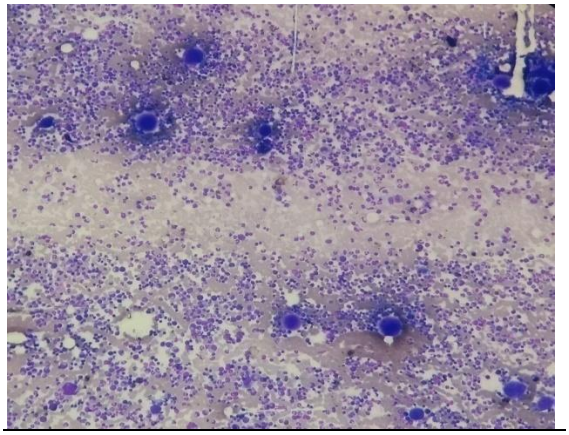

Figure 1: Bone marrow showing increased number of megakaryocytes in a case of immune thrombocytopenic purpura. (Leishman stain X 100).

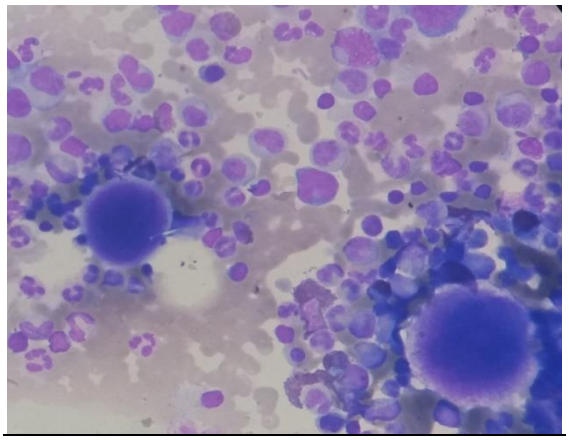

Figure 2: Bone marrow showing immature megakaryocytes (scant bluish cytoplasm and lacking lobulation of the nucleus which occupied most of the cellin a case of immune thrombocytopenic purpura. (Leishman stain X 400).

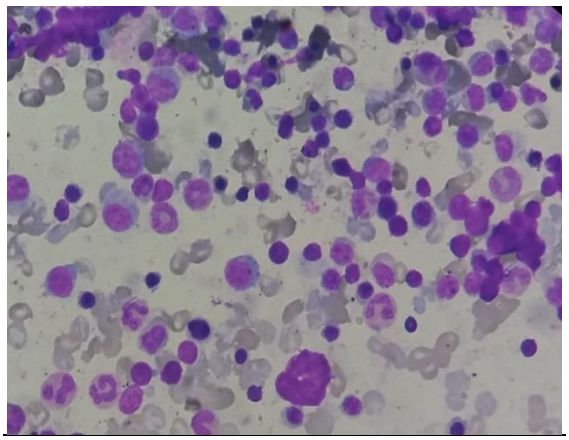

Figure 3: Bone marrow showing bare megakaryocyte in dimorphic anemia. (Leishman stain X 400).

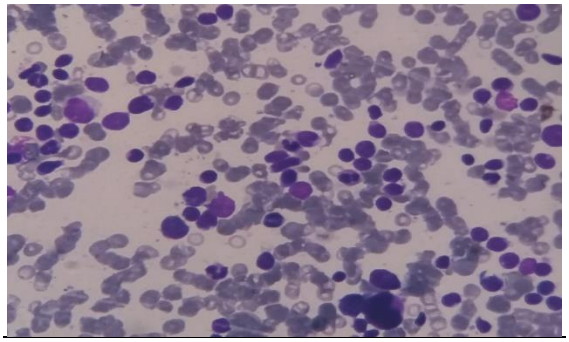

Figure 4: Bone marrow showing micromegakaryocyte in a case of acute leukemia. (Leishman stain X 400).

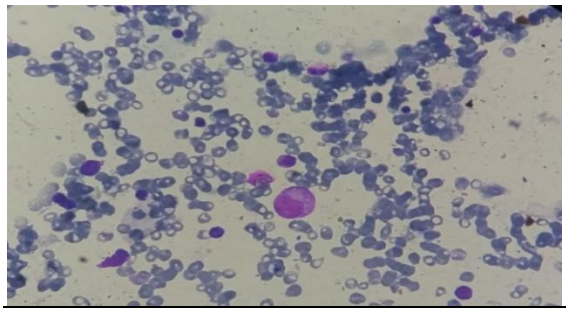

Figure 5: Bone marrow showing hypolobated megakaryocyte in a case of aplastic anemia. (Leishman stain X 400).

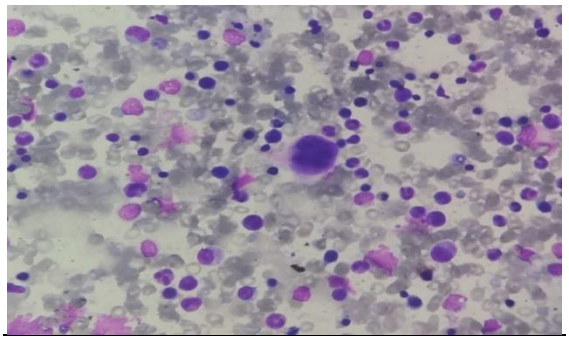

Figure 6: Bone marrow showing hypolobated megakaryocyte in megaloblasticanemia (Leishman stainX400).

DISCUSSION

Thrombocytopenia is a common clinical entity with varied differential diagnosis and presents in various clinical diseases. Thrombocytopenia, either persistent, isolated or in association with pancytopenia refractory to treatment is one of the commonly encountered hematological problems for which a bone marrow study is sought.

In the present study,out of 100 cases,maximum number of patients (29%) were seen in age group of 16-30 years which was comparable with the results of study by Choudhary PK et al where maximum number of patients (38.8%) were in the age group of 20-39 years and in the study of Gupta P et al maximum number were in the age group of 21-30 years which were included in the age group of 16-30 years.[11,12]

Male preponderance was seen in present study (61%) which was similar with the results of studies done by Sengupta M et al in which males were 65.9%,[13] Choudhary PK et al in which males were 61.9% and Gupta P et al in which males were 58%.[11,12]

Table 6: Comparison of the various studies of thrombocytopenia with respect to first and second most common etiology.

Author and year of study	Number of cases	First common cause	Second common cause
Muhury M et al [14] (2009)	144	Acute leukemia (31.3%)	Immune thrombocytopenic purpura (13.15%)
SenguptaM et al[13](2012)	85	Acute leukemia (25.9%)	Immune thrombocytopenic purpura (20%)
ChoudharyPK et al[11] (2013)	139	Megaloblastic anemia (31.7%)	Acute leukemia (27.3%)
Bhasin TS et al [15] (2013)	60	Dimorphic anemia (30%)	MDS (10%)
Gupta P et al[12](2015)	50	Immune thrombocytopenic purpura (34%)	Megaloblastic anemia (24%)
Present study	100	Megaloblastic anemia (33%)	Acute leukemia (21%)

Table 7: Comparison of prevalence of dysplastic changes with various studies.

Author and year of study	Megaloblastic anemia	Acute leukemia	ITP	Aplastic anemia	Dimorphic anemia	MDS	CML
Muhury M et al [14] (2009)	75%	24%	89%	12.5%	72%	77.7%	-
ChoudharyPK et al[11](2013)	52.3%	50%	21.2%	30.8%	-	-	-
Bhasin TS et al [15] (2013)	0%	0%	66.6%	0%	44.4%	40%	20%
Gupta P et al[12] (2015)	41.7%	0%	35.3%	75%	100%	-	100%
Present study	45.4%	23.8%	45.4%	33.3%	33.3%	100%	100%

In the present study most common cause of thrombocytopenia was megaloblastic anemia (33%) which was similar to the study conducted by Choudhary PK et al where megaloblasticanemia was seen in 31.7% of patients. Megaloblasticanemia was the commonest cause in our setup because most of the patients presented had nutritional deficiency. The second most common cause in our study was acute leukemia (21%) which was similar to the study conducted by Choudhary PK et al where it was seen in 27.3% of cases.[11] The most common clinical features in the present study was fatigue (87%) as compared to a study by Sengupta M et al where it was bleeding manifestation (60%).[13]

In the present study, bone marrow hypercellularity was seen in 79%, hypocellular in 12% and normocellular in 9% cases as compared to 48.2%, 27.1% and 24.8%, respectively in a study by Sengupta M et al.[13]

In the present study, in megaloblastic anemia increased numbers of megakaryocytes were seen in 9.09% of cases comparable to 8.3% by Muhury M et al and 11% by Sengupta M et al.[11,13] Increased megakaryocytes were also found in 34% cases by Choudhary PK et al and 58.3% by Gupta P et al.[11,12] Decreased numbers of megakaryocytes were seen in 30.03% of cases which was comparable to 33.3% by Gupta P et al.[12] Decreased megakaryocytes were also seen in various other studies, 16% in a study by Choudhary PK et al,[11] 16.6% by Muhury M et al and 77% by Sengupta M et al.[13,14]

In the present study, in acute leukemia decreased numbers of megakaryocytes were seen in 47.6% of cases which were comparable to 44% by Muhury M et al and in contrast to 87% by Choudhary PK et al and 18% by Sengupta M et al [11,13,14].

Megakaryocytes were absent in 42.8% of cases which was similar to a study by Muhury M et al [14], 44%. This was in contrast 82% by Sengupta M et al [13] and 2.6% by Choudhary PK et al.[11]

In immune thrombocytopenic purpura increased numbers of megakaryocytes were seen in 100% of cases in the present same as by Sengupta M et al and comparable to 98%, 95.3%, 95%, 91% and 82.3% in studies by Shi XD et al,[13,16] Jubelirer SJ et al,[17] Muhury M et al,[14] Choudhary PK et al and Gupta P et al,[11,12] respectively. This was attributed this to stimulation of the marrow megakaryocytes to synthesize platelets at an increased rate due to immune-mediated platelet destruction in the spleen and other reticuloendothelial tissues.

In aplastic anemia, in the present study, megakaryocytes were absent in 55.6% cases as compared to 75% and 71% in studies by Muhury M et al and Sengupta M et al.[13,14] Megakaryocytes were decreased in 33.3% of cases in the present study which was comparable to studies by Sengupta M et al in which they were decreased in 29% cases,[13] 25 % each in a study by Gupta P et al [12] as well as by Muhury M et al.[12,14]

In megaloblasticanemia, in the present study dysplasia was seen in 45.4% of cases which was comparable to the results of other studies i.e. 41.7% by Gupta P et al.[12] In acute leukemia, dysplasia was seen in 23.8% cases which were almost similar to Muhury M et al.[14] In immune thrombocytopenic purpura,dysplastic megakaryocytes were seen in 45.4% cases of present study. They were also seen in other studies by Choudhary PK et al (21.2%), Gupta P et al (35.3%),[11,12] Bhasin TS et al (66.6%) and Muhuruy M et al (89%).[14,15]

In aplastic anemia,dysplastic megakaryocytes were seen in 33.3% cases in present study as compared to 30.8% by Choudhary PK et al .[11]

In megaloblasticanemia, immature megakaryocytes were seen in 3% of cases as compared to 41% in a

study by Muhury M et al,[14] bare in 12.1% as compared to 8% in Gupta P et al,[12] micromegakaryocytes were seen in 24.2% as compared to 25% in Muhury M et al and 50% in Gupta P et al,[12,14] hypogranular in 6% in contrast to 54% in Gupta P et al and hypolobated in 6% in contrast to 54% Gupta P et al.[12] The mechanism for such dysmegakaryocytopoesis characterized by abnormal morphology according to Wang C et al,[18] is based on the impaired DNA synthesis and methylation due to folate and vitamin B12 deficiency.

In acute leukemia, bare megakaryocytes were seen in 14.2% of cases, micromegakaryocytes in 23.8% and hypolobated in 9.5% as compared to 22%, 11% and 4.5%, respectively in Muhury M et al.[14]

In immune thrombocytopenic Purpura, immature megakaryocytes were seen in 63.6% of cases as compared to 100% in Muhury M et al and 12% in Gupta P et al.[12,14] Bare were seen in 9% cases of present study as compared to 12% in Gupta P et al and 84% in Muhury M et al,[1214], micromegakaryocytes in 18.1% cases of present study as compared to 12% in Gupta P et al.[12] Hypogranular megakaryocytes were seen in 18.1% as compared to 30% in Gupta P et al and hypolobated in 9% as comapred to 6% in Muhury M et al and 50% in Gupta P et al.[12,14]

In aplastic anemia, hypolobated megakaryocytes were seen in 33.3% of cases as compared to 13% in Muhury M et al which were in contrast to those of Tricot G et al where megakaryocytes were of normal morphology.[14,19]

CONCLUSION

Bone marrow examination is an important step to arrive at the probable diagnosis of wide varieties of haematological disorders in the cases of thrombocytopenia. Megaloblasticanemia was the commonest cause of thrombocytopenia in this study followed by acute leukemia, followed by immune thrombocytopenic purpura. Dysplastic megakaryocytes were commonly observed in megaloblasticanemia and immune thrombocytopenic purpura, which were seen in <10% of megakaryocytes.

Further studies on the evaluation of megakaryocytic alteration and their contribution to thrombocytopenia can provide growing knowledge to the pathogenesis of numerous hematopoietic disorders.

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