ORIGINAL ARTICLE

JIACM 2026; 27 (3): 186-92

Aetiological and Haematological Profile of Patients of Pancytopenia at a Tertiary Care Hospital in Northern India

Bishakha Swain, Ratnakar Sahoo**, Vijay Kumar***, Priyanka Verma*****

Abstract

Background: Pancytopenia is characterised by simultaneous decrease in all the 3 cell lines: erythrocytes, leucocytes and platelets. Its aetiology is as variable as its presentation ranging from nutritional deficiencies to haematological malignancies. It may be completely treatable; however, in some cases, it requires extensive investigations for diagnosis and prolonged treatment. Our aim was to study the aetiological and haematological profile in patients of pancytopenia for early diagnosis, to avert catastrophic consequences by timely treatment.

Materials: This cross-sectional, observational study included 100 patients presenting with pancytopenia to the medical emergency and outpatient department of the Department of Medicine at ABVIMS and Dr. RML Hospital between November 2019 and March 2021.

Results: Mean age of patients was 37.92 ± 15.2 years with maximum number of patients (32%) in the age group of 21 - 30 years. 50 subjects were males and 50 were females. Megaloblastic anaemia (MA) accounted for 49% cases of pancytopenia. Other causes were infections (16%), hypersplenism (13%), drugs (6%), autoimmune disorders (5%), aplastic anaemia (AA) (4%), malignancy (3%), haemophagocytic lymphohistiocytosis (3%) and Sheehan syndrome (1%). 51% of patients had macrocytic anaemia, 35% had normocytic normochromic anaemia and 14% had dimorphic anaemia. The commonest bone marrow finding was hypercellularity with megaloblastic reaction and dyserythropoiesis (39.43%). Hypercellular, normocellular and hypocellular marrow were observed in 70.43%, 15.49% and 14.08% subjects, respectively.

Conclusion: This study elucidated causes and haematological profile of pancytopenia in Northern India. Megaloblastic anaemia was the most common aetiology in this study.

Key words: Pancytopenia, megaloblastic anaemia, bone marrow aspiration, peripheral blood smear, hypercellular bone marrow.

Introduction

Pancytopenia is not an illness but manifestation of an underlying disease. In simple language, it means lack of all cells (pan -all; cyte - cell; penia - poverty)[1]. It is characterised by decline in all three blood cell lines- erythrocytes, leucocytes and platelets[2]. Pancytopenia is encountered in daily clinical practice and is more common than realised due to its varied aetiology and variable presentation. Patients with pancytopenia present with symptoms of anaemia, leukopenia and thrombocytopenia, which if not diagnosed early, may be lethal. Pancytopenia is caused by failure of blood cell production, marrow suppression by drugs or infection, infiltration of bone marrow by malignant cells or fibrosis, ineffective erythropoiesis and dysplasia, peripheral sequestration of blood cells by overactive reticuloendothelial system, and immune or non-immune mediated increased destruction of blood cells[3]. Megaloblastic anaemia due to deficiency of vitamin B12/ folate, is a predominant cause of pancytopenia in India because of high prevalence of nutritional anaemia in the Indian subcontinent. Other causes include hypersplenism (congestive splenomegaly, chronic liver disease, malaria, and leishmaniasis), aplastic anaemia, myelodysplastic syndrome, subleukaemic leukaemias, military tuberculosis, multiple myeloma, paroxysmal nocturnal haemoglobinuria[3]. Genetic mutations and varied geographical distribution also affect the incidence and aetiology of pancytopenia. A careful examination of peripheral blood picture is the first step in the workup of pancytopenia and helps in planning relevant investigations. Bone marrow (BM) examination is a window to the diagnosis of pancytopenia as all blood cells are formed in it. It helps in the final diagnosis but may not always be conclusive. Having a knowledge of aetiology of pancytopenia is essential for prognosis and its management. Additional testing and clinical evaluation can be recommended when bone marrow studies are inconclusive[4]. This study was undertaken to evaluate the haematological parameters and aetiology of pancytopenia.

Assistant Professor, Department of Clinical Immunology and Rheumatology, KIMS Hospital, Bhubaneshwar - 751 024, Odisha, **Director Professor, Department of Medicine, ***Director Professor and Head, Department of Pathology, ABVIMS and Dr Ram Manohar Lohia Hospital, Baba Kharak Singh Marg, New Delhi - 110 001, ****Assistant Professor, Department of Medicine, UCMS and Guru Teg Bahadur Hospital, Dilshad Garden, Delhi - 110 095.

Corresponding Author: Dr Bishakha Swain, Assistant Professor, Department Clinical Immunology and Rheumatology, KIMS Hospital, Bhubaneshwar - 751 024, Odisha. Tel: 9730248779, E-mail: bshakha@gmail.com

Material and Methods

This was a cross-sectional, observational study conducted in the Department of Medicine at ABVIMS and Dr RML Hospital, New Delhi from November 2019 to March 2021 after obtaining permission from the institutional ethics committee. A total of 100 patients who presented to the outpatient and emergency department after taking proper informed consent were enrolled. The inclusion criteria were patients aged 18 years and above, having pancytopenia defined as haemoglobin level - below 9 g/dL, total leucocyte count - below 4000/mm^3, and platelet count - below 1,00,000/μL^3. Patients who were on chemotherapy, or had a history of radiotherapy and recent blood transfusion were excluded.

A thorough history, physical examination and relevant investigations were obtained and recorded. The investigations conducted were: Complete blood count (CBC), peripheral smear, reticulocyte count, serum vitamin B12 and folate levels, Serum Lactate Dehydrogenase (LDH) levels, kidney function test, liver function test, Anti-Nuclear Antibodies (ANA), Coomb's test, Human immunodeficiency virus- enzyme-linked immunosorbent assay (HIV ELISA), Anti Hepatitis C antibody, Hepatitis B surface Antigen (HBsAg), urinalysis, chest X-ray and electrocardiogram, ultrasound-abdomen. Patients were screened for Malaria, Dengue, Enteric fever, Chikungunya and Sepsis when suspected as per the clinical presentation. Special investigations were done when routine investigations were inconclusive. These included rk39 antigen, serum protein and immunofixation electrophoresis, spleno-portal axis doppler, Computed Tomography (CT) scan and Magnetic Resonance Imaging (MRI), Viral deoxyribonucleic Acid (DNA) Polymerase Chain Reaction (PCR) for Cytomegalovirus (CMV) and Epstein Barr Virus (EBV), Hormonal profile (Growth Hormone, Follicular Stimulating Hormone, Luteinizing Hormone, Thyroid Stimulating Hormone, FreeT3, FreeT4), procalcitonin levels, serum ferritin, serum fibrinogen and serum triglyceride level. Bone marrow aspiration and biopsy was done in 71 subjects.

Results

In this study, 50 patients were males and 50 were females. Mean age (years) of study subjects was 37.92 ± 15.2. Majority (32%) of patients belonged to age group 21 - 30 years and least patients were in the age group >70 years. Age-wise aetiological distribution is given in Table I.

49% patients had pancytopenia secondary to megaloblastic anaemia (MA) due to nutritional deficiency of vitamin B12 (28%) or both vitamin B12 and folate (21%). Other causes were: Infections (16%), hypersplenism (13%), drugs (6%), autoimmune disorders (5%), aplastic anaemia (4%), malignancy (3%), Haemophagocytic lymphohistiocytosis (HLH) (3%) and Sheehan syndrome (1%) (Fig. 1).

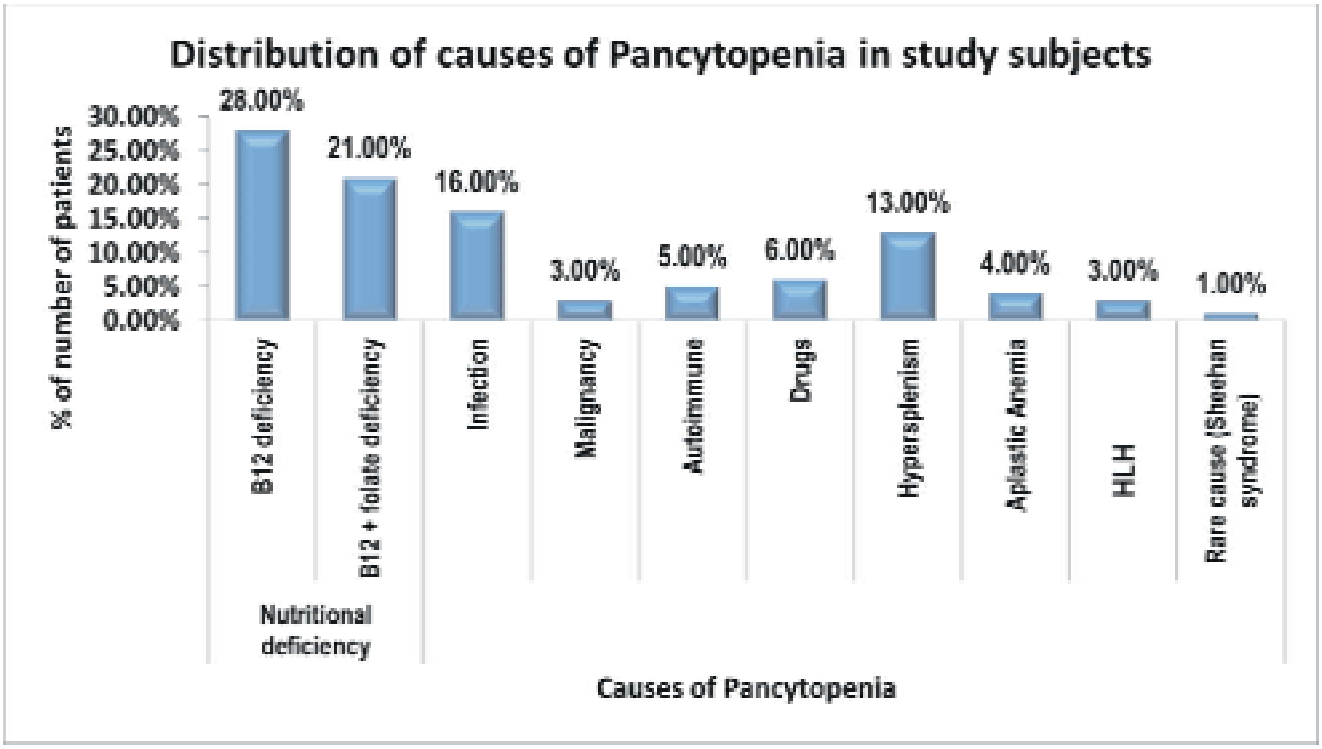


Fig. 1: *Distribution of causes of pancytopenia in study subjects.*

Table I: Age-wise distribution of aetiology of Pancytopenia.

Aetiology	<20 y	20 - 39 y	40 - 59 y	>60 y	Total
B12/folate deficiency	7	21	19	2	49
CLD/hypersplenism	0	5	6	2	13
Auto-immune	0	4	1	0	5
Drug Induced	0	5	1	0	6
Infections	1	12	3	0	16
Aplastic anaemia	0	0	2	2	4
Malignancy	0	0	1	2	3
Rarer diseases (HLH) (Sheehan syndrome)	1	2	 1		3 1
Total	9	49	34	8	100

Amongst B12 deficient individuals, dietary deficiency (89.29%) predominated followed by alcohol (7.14%) and 1 case was idiopathic (3.57%). The commonest cause of combined B12 and folate deficiency was dietary deficiency (66.67%) followed by alcohol use (23.81%) and malabsorption (Celiac disease) and idiopathic in only 1 out of 21 patients (4.76%). Thirty of the 49 subjects suffering from megaloblastic anaemia were vegetarian while 7 were vegans.

Out of 16 cases of infection, the commonest was malaria seen in 31.25% of patients (with infection) followed by disseminated tuberculosis (25%) and visceral leishmaniasis (12.5%). Cause of infection was HIV, CMV, EBV, sepsis (Klebsiella) and Dengue in only 1 out of 16 patients (6.25%) each.

Pancytopenia was attributable to hypersplenism in 13 cases. Chronic Liver Disease (CLD) was seen in majority (84.62%) whereas splenic abscess and portal hypertension (cause unidentified) caused hypersplenism in 1 patient (7.69%) each. The commonest cause of CLD was Alcoholic liver disease (7 cases), with 2 cases of Non-Alcoholic Fatty Liver Disease (NAFLD), one each of cryptogenic CLD and extrahepatic portal vein obstruction in a hepatitis B affected patient with CLD.

In the study, 6 patients were on myelotoxic drugs; 2 (33.33%) were taking Methotrexate (MTx). azathioprine, meropenem, carbimazole and cyclophosphamide were taken by only 1 out of 6 patients (16.67%) each. Haematological malignancies were seen in 3% cases; 1 patient each of Non-Hodgkin Lymphoma (NHL), Multiple Myeloma (MM) and Acute erythroid Leukaemia (AML-M6). Other causes of pancytopenia were aplastic anaemia seen in 4 patients, HLH in 3 (2 of which had tuberculosis and 1 remained unknown) and Sheehan syndrome in 1 patient.

Mean value of haemoglobin (g/dL), total leucocyte count (cells/mm^3), platelet count (/µL), packed cell volume (%) and corrected reticulocyte count (%) of study subjects was 5.53 ± 1.87, 2625.6 ± 824.86, 50100 ± 24174.24, 16.8 ± 5.57 and 1.73 ± 1.38, respectively.

Mean value of mean corpuscular volume (MCV) (fL), mean corpuscular haemoglobin (MCH) (pg) and mean corpuscular haemoglobin concentration (MCHC) (g/dL) of study subjects were 102.78 ± 16.6, 30 ± 2.12 and 33.73 ± 1.44 respectively.

In the present study, peripheral smear of 51% of patients was found to be macrocytic followed by normocytic normochromic (35%) and dimorphic (14%). Other features were hypersegmented neutrophils (35%) (Fig. 2), polychromatophils (14%), hemoparasite (5%), special features like rouleaux formation with lymphoplasmacytic forms (1%) and normocytic normochromic anaemia with 3 - 4% erythroblasts (1%).

71 patients were subjected to BM examination with variable findings (Fig. 4). 70.43% patients revealed a hypercellular marrow while normocellular and hypocellular marrow were observed in 15.49% and 14.08% patients respectively. 39.43% of patients had hypercellular marrow with megaloblastic reaction with dyserythropoiesis (MA) (Fig. 3), 18.30% had erythroid hyperplasia with normoblastic maturation with hypercellular marrow (Hypersplenism), 8.45% of patients had hypocellular marrow (drug induced pancytopenia), 7.04% of patients had normocellular marrow with normal maturation/mild dyserythropoiesis (autoimmune aetiology), 5.63% of patients had a hypocellular marrow replaced by fat (AA), 4.22% of patients had histiocytes and haemophagocytosis (HLH) (Fig. 5A), 5.63% of patients had normocellular marrow with granulomas and giant cells (disseminated Tuberculosis), mild dyserythropoiesis - 2.81% – was seen in patients with malaria without any haemoparasite and cellular marrow

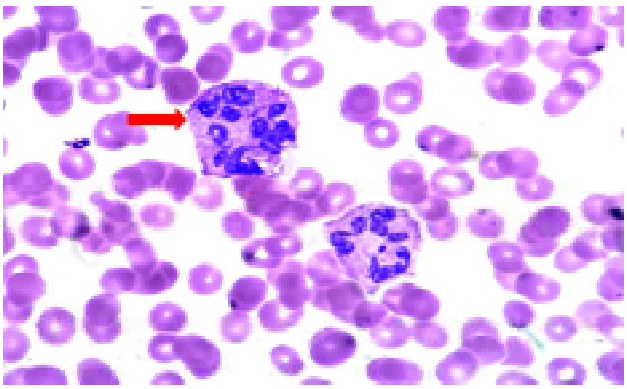


Fig. 2: *400X magnification. Leishman Stain. Megaloblastic anaemia. Peripheral Blood Smear showing hypersegmented neutrophils.*

with reactive changes was seen in 1.40 % patients (CMV infection). Specific findings were found in Visceral Leishmaniasis, i.e., LD bodies (2.81%) (Fig. 5B), Non-Hodgkin lymphoma- CD20 positive abnormal mononuclear cells (1.40%), AML M6- proerythroblasts witch block like PAS positivity (1.40%) and multiple myeloma CD 138 positive plasma cells (1.40%).

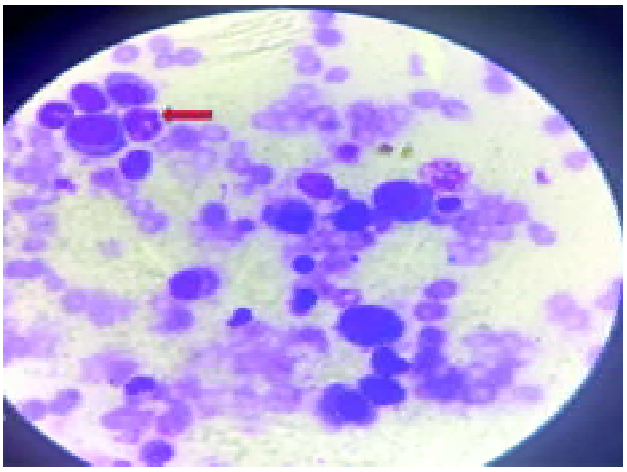


Fig. 3: *200X magnification. Giemsa stain. Bone marrow aspirate shows erythroid hyperplasia with megaloblasts (Red arrow).*

Aetiology	Bone marrow findings	Frequency (n %)
Megaloblastic anaemia	Hypercellular marrow with megaloblastic reaction with dyserythropoiesis	28 (39.43%)
Autoimmune	Normocellular marrow with normal maturation/mild dyserythropoiesis	5 (7.04%)
Drug induced	Hypocellular marrow	6 (8.45%)
Aplastic anaemia	Hypocellular marrow replaced by fat	4 (4.63%)
Haemophagocytic Lymphohistiocytosis	Histiocytes Haemophagocytosis	3 (4.22%)
Hypersplenism	Erythroid hyperplasia with normoblastic maturation	13 (18.30%)
Visceral leishmaniasis	Erythroid hyperplasia mild dyserythropoiesis/Leishmania Donovani (LD) bodies seen	2 (2.81%)
Disseminated tuberculosis	Normocellular with granulomas with langhans gaint cells	4 (5.63%)
Non Hodgkin's lymphoma	Abnormal mononuclear cells (CD 20 positive) with high NR ratio and opened up chromatin	1 (1.40%)
Multiple myeloma	Cellular with palsma cells (CD138 positive)	1 (1.40%)
Acute Myeloid Leukaemia	ME ratio 1:12/Erythroid hyperplasia/ >30% proerythropoiesis - No positivity	1 (1.40%)
Malaria	Mild dyserythropoiesis parasite	2 (2.81%)
Cyto megalovirous	Cellular Marrow with Reactive changes - No Inclusions	1 (1.40%)

Fig. 4: *Distribution of bone marrow findings of study subjects with corresponding aetiology.*

Discussion

Our study included 100 patients (50 males, 50 females) with a mean age of 37.92 ± 15.2 years. Most patients (32%) were in the 3rd decade. A study by Jain *et al*[5] showed a clear male predominance likely due to sociocultural factors unlike our study. Both studies; however, showed a predominance of younger adults presenting with pancytopenia. While the comparison study reported most cases in the 3rd and 4th decades, our study found the highest proportion (32%) in the 21 - 30 years age group, suggesting a slightly earlier peak age of presentation in our cohort.

The aetiological profile of pancytopenia demonstrated a distinct age-related pattern in our cohort. The majority of patients were between 20 and 59 years of age (83%), with 49% belonging to the 20 - 39-year age group. Vitamin B12/ folate deficiency was the predominant aetiology overall (49%) and remained the leading cause across the 20 - 39 and 40 - 59-year age groups, accounting for 42.8% and 38.8% of cases, respectively. This finding is consistent with a similar study where maximum number of megaloblastic anaemia cases were seen in the age group 41 - 50[6].

Another study by Dasgupta *et al*, reported aplastic anaemia predominated in younger patients, aged 5 - 35 years, whereas megaloblastic anaemia became relatively more prominent with increasing age and was the only reported cause among patients >65 years along with leishmaniasis unlike our study where majority of patients had Megaloblastic anaemia between ages of 20 - 59 years. None of our patients aged <40 years had aplastic anaemia. Four cases were seen in elderly[7].

Another study reported evenly distributed cases across age groups (11 - 20 and 21 - 30 years) together accounting for only 32% of cases, with a comparatively higher proportion in the paediatric (<10 years: 26%) and elderly (>60 years: 21%) populations). Megaloblastic anaemia, was the commonest aetiology (25%) followed by a broader spectrum of marrow-based and malignant disorders (aleukaemic leukaemia, hypoplastic/aplastic marrow, myelodysplastic syndrome, myelofibrosis, and hairy cell leukaemia) accounting for 37% of cases) and visceral leishmaniasis (11.8%)[8].

These differences reflect variations in geographical setting, referral patterns, patient characteristics and the relative prevalence of nutritional, infectious and marrow disorders between the cohorts. Our study excluded paediatric patients which could partially account for some differences among the studies.

In our study the majority of admitted patients were diagnosed with megaloblastic anaemia (49%) as the cause

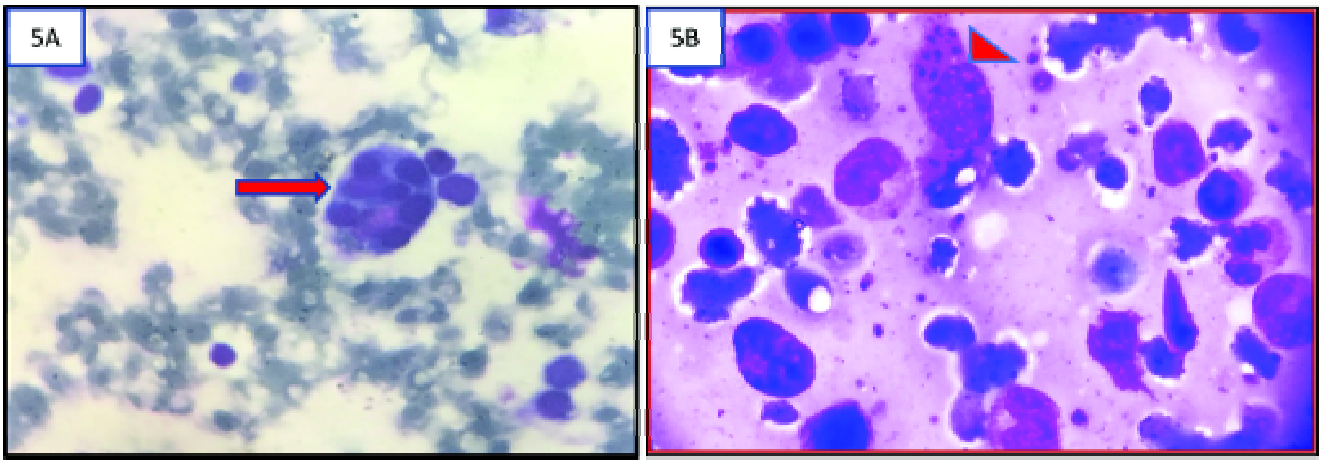


Fig. 5: *400X magnification. Giemsa stain. 5A) Haemophagocytic Lymphohistiocytosis. Histiocytic cells showing haemophagocytosis (Red arrow). 5B) Kala-azar. Bone marrow aspirate smear showing Leishmania Donovani (LD) bodies with amastigote forms (red arrow head) of the hemoparasite.*

of pancytopenia. The predominant cause for this was nutritional deficiency. This is similar to a study done by Kulkarni *et al*[9] wherein nutritional deficiency was the most common cause of pancytopenia. Rao *et al* [10] (74.04%), Dhooria *et al*[2] (29.5%) and Rohira *et al*[11] (50%) also report Megaloblastic anaemia as the commonest cause of pancytopenia in their studies. Dhooria *et al* showed that 55.9% pancytopenic subjects with Megaloblastic anaemia were vegetarians[2] while 61.2% were vegetarians in our study.

Viral infections are well documented causes of cytopenias and severe cases can also result in aplastic anaemia. In our study, infections were observed as the 2nd commonest cause of pancytopenia wherein malaria predominated (31.25%) followed by disseminated Tuberculosis (25%) and few cases of Visceral Leishmaniasis (12.50%). Viral illnesses with pancytopenia were observed in one case each (6.25%) of HIV, CMV, EBV, Dengue and 2 cases of hepatitis B.

Hypersplenism causes peripheral pooling or trapping and destruction of cells in an enlarged spleen resulting in cytopenias. It is also a frequent cause specially noted in alcoholic patients developing CLD with portal hypertension. In our study, hypersplenism (13%) was the third commonest cause of pancytopenia resulting from CLD (11% cases) and 1 case each of portal hypertension and splenic abscess. Dhooria *et al*[2] reported hypersplenism as the cause of pancytopenia in 4.5% cases in which hepatitis C and alcoholic CLD were the main causes.

Drug induced pancytopenia was observed in 6% cases. Maximum cases were caused by methotrexate followed by azathioprine. Similar finding was reported by Dhooria *et al*[2] where 21 patients (10.5%) were labelled as drug induced pancytopenia and methotrexate constituted 12 cases followed by azathioprine in 3 patients, combination of MTx and indigenous medication in 2 patients and 1 each of 6-Mercaptopurine, NSAIDS, Anti-retroviral therapy (ART) and combination of MTx and leflunomide.

In our study, only 3% cases constituted haematological malignancies with pancytopenia. Similar results were reported by Javalgi *et al*[12] where least common causes of pancytopenia were leukaemia (2.8%), myelofibrosis (0.9%), multiple myeloma (0.9%) and myelodysplastic syndrome (0.9%). Contrary to our study, Neha Sharma *et al*[13] (19%) and Dhooria *et al*[2] (14%) reported higher prevalence of malignancies causing pancytopenia.

Pancytopenia is seen as a frequent haematological involvement in various autoimmune diseases caused by defective hematopoiesis due to antibodies, infection/sepsis and immunosuppressant therapy. Our study reported only 5% cases of Systemic Lupus Erythematosus (SLE) which is comparable to Santra *et al*[14], where 3.7% cases of SLE caused pancytopenia out of 215 subjects.

Aplastic anaemia was seen in only 4% cases. This was corroborated by Jain *et al*[5] who reported 12 (4.8%) cases of hypoplastic/aplastic anaemia. But few studies[14,15,16] reported it as the commonest aetiology of pancytopenia.

HLH is a life-threatening disease characterised by uninhibited activation of immune cells. HLH could occur sporadically or genetically. The clinical presentation is typically triggered by infection, autoimmune conditions, or immunosuppression. HLH was established as a cause of pancytopenia by a comprehensive electronic literature search from January 1990 to July 2016 conducted by Gnanaraj *et al*[17]. We observed pancytopenia in 3 cases of HLH during this study.

One unusual cause of pancytopenia was a case of Sheehan syndrome who had presented with secondary

amenorrhoea which resolved after hormonal replacement; these findings are corroborated by a case report by Fatma *et al*[18] who observed the same in a Tunisian lady with post-partum pituitary apoplexy.

In our study, most cases were hypo-proliferative, with corrected reticulocyte count ranging from 0.2 - 6.8 %, the mean being 1.73 ± 1.38%. Many of these cases were related to megaloblastic anaemia with corrected reticulocyte count of patients <2% while a few were secondary to aplastic anaemia (corrected reticulocyte count <1%) and myelotoxic drugs. Reticulocyte count was raised in cases with hypersplenism and malaria. Priya *et al*[19] conducted a study to evaluate the role of Absolute Reticulocyte count (ARC) in patients of pancytopenia. They observed that ARC was severely reduced in aplastic anaemia whereas nutritional anaemias and myelodysplastic syndrome showed moderate decrease in ARC values. All marrow infiltrative disorders and systemic infections presented with high ARC.

In RBC indices, MCV revealed the widest variation with a range of 70 - 137.1 fL and mean of 102.78 ± 16.6 fL suggesting that most cases were macrocytic (51%). In the study conducted by Barik *et al*[20] MCV was increased in 49% cases. Dimorphic picture was seen in only 14% cases in our study. A study conducted by Rao *et al*[10] reported different features with predominant dimorphic anaemia (37.5%), followed by macrocytic anaemia (31.7%) and normocytic normochromic anaemia (15.3%).

Highest number of cases in our study had a hypercellular marrow (70.43%) with majority showing dyserythropoiesis and megaloblastic reaction (39.43%). Hypercellular bone marrow is indicative of infiltration by neoplasm, immune-dysregulation by inflammatory cells, dys-erythropoiesis by overwhelming infection and megaloblasts. Our study observed few cases (3%) of haematological malignancy. Visceral Leishmaniasis and HLH also revealed hypercellular bone marrow. Normocellular marrow was observed in 15.49% cases and hypocellular marrow in 14.08% cases. Special features observed were LD bodies in visceral leishmaniasis, giant cells and granulomas in disseminated Tuberculosis and haemophagocytes with histiocytosis in HLH. The bone marrow findings were reflective of the aetiology. A study conducted by Geetanjali *et al*[21] showed bone marrow hypocellularity in 18.7%, normocellularity in 32.8% and predominant hypercellularity in 48.5% with megaloblastic maturation in 23 cases (32.8%) which is akin to our study. In another comparable study by Javalgi *et al*[11] 68.8% cases had hypercellular marrow, followed by normocellular (15%) and hypocellular marrow (16.2%) and megaloblastic anaemia was found as the most common cause.

Pancytopenia was diagnosed in 29 patients without a bone marrow examination. Peripheral smear and CBC clinched the diagnoses in 24 patients. 20 patients had megaloblastic anaemia with a high MCV on CBC and hypersegmented neutrophils on peripheral smear, supplemented with vitamin B12 levels. Malaria led to pancytopenia in 3 cases with the peripheral smear showing schizonts of *plasmodium*. EBV DNA PCR and relative lymphocytosis diagnosed another case. Serology helped in diagnosing 2 cases (HIV Elisa, Dengue IgM). Ultrasound of the abdomen, hormonal profile and blood culture diagnosed hypersplenism (CLD), Sheehan syndrome and sepsis respectively.

It is notable that only 15 of the 100 patients needed bone marrow evaluation for a definitive diagnosis. These included disseminated TB (3), Aplastic anaemia (4), HLH (3), Leishmaniasis (2) and haematological malignancies (3).

The remaining 56 patients were diagnosed based on clinical history, examination, CBC and peripheral smear findings, along with serology and imaging. However, suggesting that bone marrow examination can be routinely avoided in the majority of patients with pancytopenia would be an oversimplification, given the possibility of underlying serious or unsuspected pathology. Moreover, diagnostic certainty is often easier to establish in retrospect.

Our study highlights that a thorough clinical history and examination, CBC, and peripheral smear remain the cornerstone of the initial evaluation of pancytopenia, guiding the need for further investigations, including bone marrow examination.

Limitations: The limitations of our study include the stringent definition of pancytopenia that may have resulted in under-representation of patients with early or milder cytopenias. Additionally, exclusion of patients with prior blood transfusion may have introduced selection bias, particularly in a tertiary-care setting, as pre-transfusion haematological parameters were not consistently available for retrospective assessment.

Conclusion

The aetiological profile of pancytopenia can be established with a thorough clinical history, physical examination and relevant haematological investigations. Early diagnosis of pancytopenia is pertinent as treatment varies with specific aetiology. In this study megaloblastic anaemia was found as the commonest cause followed by infections. Bone marrow examination was essential in few cases for diagnosis. Many cases of pancytopenia can be diagnosed without a bone marrow examination; however, for

confirmation the importance of a marrow examination cannot be underestimated.

References

1. Kar M, Ghosh A. Pancytopenia. *J Indian Acad Clin Med* 2002; 3 (1): 29-34.
2. Dhooria HP, Kaur S, Dhooria GS. Aetiological spectrum and clinical profile of patients admitted with pancytopenia. *J Adv Med Med Res* 2020; 32 (4): 56-65.
3. Garg AK, Agarwal AK, Sharma GD. Pancytopenia: Clinical approach. In: Bansode BR, editor. *Medicine Update.* 27th ed. New Delhi: Evangel Publishers; 2017; p. 450-54.
4. Weinzierl EP, Arber DA. The differential diagnosis and bone marrow evaluation of new-onset pancytopenia. *Am J Clin Pathol* 2013; 139 (1): 9-29.
5. Jain A, Naniwadekar M. An aetiological reappraisal of pancytopenia: Largest series reported to date from a single tertiary care teaching hospital. *BMC Hematol* 2013; 13 (1): 1-9.
6. Makheja KD, Maheshwari BK, Arain S. The common causes leading to pancytopenia in patients presenting to tertiary care hospital. *Pakistan J Med Sci* 2013; 29 (5): 1108.
7. Dasgupta S, Mandal PK, Chakrabarti S. Aetiology of Pancytopenia: An Observation from a Referral Medical Institution of Eastern Region of India. *J Lab Physicians* 2015; 7 (2): 90-5.
8. Chandra H, Gupta AK, Nath UK. Clinico-haematological study of pancytopenia: A single-center experience from north Himalayan region of India. *J Family Med Primary Care* 2019; 8 (12): 3944.
9. Kulkarni NS, Patil AS, Karchi SD. Study of pancytopenia in a tertiary care hospital in North Karnataka. *Int J Med Res Health Sci* 2017; 6 (3): 61-67.
10. Rao KS. Pancytopenia: A clinicohaematological study. *J Lab Physicians* 2011; 3 (1): 15-20.
11. Rohira N, Sawke GK, Sawke N. Pancytopenia associated megaloblastic anaemia: A clinicohaematological study in a tertiary care hospital. *Trop J Pathol Microbiol* 2019; 5 (2): 69-75.
12. Javalgi AP, Dombale VD. Clinicohaematological analysis of pancytopenia: A bone marrow study. *Natl J Lab Med* 2013; 2 (4): 12-7.
13. Sharma N, Bhatia PK, Kaul KK. A clinicohaematological study of pancytopenia: An experience of a tertiary care teaching hospital, Jammu, India. *Indian J Pathol Oncol* 2017; 4 (4): 632-7.
14. Santra G, Das BK. A cross-sectional study of the clinical profile and aetiological spectrum of pancytopenia in a tertiary care center. *Singapore Med J* 2010; 51 (10): 806-12.
15. Sitwat HA, Haque KA, Hussian G. Pancytopenia: Study for clinical features and aetiological pattern at tertiary care settings in Abbottabad. *Prof Med J* 2014; 21 (1): 60-5.
16. Rehmani TH, Arif M, Heraid S. Spectrum of pancytopenia. *Prof Med J* 2016; 23 (5): 620-6.
17. Gnanaraj J, Parnes A, Francis CW. Approach to pancytopenia: Diagnostic algorithm for clinical haematologists. *Blood Rev* 2018; 32 (5): 361-7.
18. Fatma M, Mouna E, Nabila R. Sheehan's syndrome with pancytopenia: A case report and review of the literature. *J Med Case Rep* 2011; 5 (1): 1-3.
19. Priya PP, Subhashree AR. Role of absolute reticulocyte count in evaluation of pancytopenia: A hospital-based study. *J Clin Diagn Res* 2014; 8 (8): FC01-FC03.
20. Barik S, Chandoke RK, Verma AK. A prospective clinicohaematological study in 100 cases of pancytopenia in capital city of India. *J Appl Haematol* 2014; 5 (2): 45-50.
21. Geetanjali N, Das S. Clinicohaematological profile and aetiological spectrum of pancytopenia: An institutional study. *Indian J Pathol Oncol* 2019; 6 (2): 302-8.

ADVERTISEMENT TARIFF

Journal, Indian Academy of Clinical Medicine

Advertisement Tariff effective January, 2020

Position	Single Issue	Consecutive Four Issues
(a) Back cover and Inside front cover	₹ 30,000/-	₹ 1,00,000/-
(b) Inside back cover	₹ 25,000/-	₹ 75,000/-
(c) Full page	₹ 20,000/-	₹ 60,000/-
(d) Half page	₹ 10,000/-	₹ 30,000/-

Note: Artworks/positives (processing)/art pulls of advertisements for Back cover, Inside front cover, Inside back cover and Full page should not exceed 28 cm (H) x 21 cm (W) – (for bleed); and 25 cm (H) x 18 cm (W) – (for non-bleed). For half page advertisements the artwork should not exceed 12 cm (H) x 18 cm (W).

Size of the Journal is 28 cm x 21 cm.

For advertisement assistance & queries, contact:
Mr. Yash Pal Satmukhi, Office Secretary, JIACM
Mobile: +91-9811107245