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Clinico-Hematological Profile of Megaloblastic Anemia at a Tertiary Care Center in North India: A Cross-Sectional Study

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Abstract

Background: Megaloblastic anemia (MA) remains a prevalent yet underdiagnosed nutritional disorder in India, particularly among vegetarian populations. Despite its treatability, patients often present late with multisystem involvement.

Objective: To describe the clinico-pathologic profile of megaloblastic anemia at a tertiary care center in North India.

Methods: This cross-sectional observational study enrolled 80 patients with confirmed megaloblastic anemia over 18 months. Diagnosis was established on the basis of peripheral smear findings, bone marrow examination, and serum vitamin B12 and folate assays. Clinical, hematological, and biochemical parameters were recorded and analyzed.

Results: The majority of patients were in the 20-40 year age group, with a slight male preponderance. Pallor (100%), weakness (96.3%), and glossitis (68.8%) were the most frequent findings. Jaundice was observed in 28.7% of patients. Pancytopenia was present in 44% of cases. Vitamin B12 deficiency was the predominant etiology (72.5%), followed by combined B12 and folate deficiency (18.7%). Most patients were vegetarians (70%).

Conclusion: Megaloblastic anemia predominantly affects young adults on vegetarian diets in North India. Jaundice, though often overlooked, occurs in a clinically significant proportion and should prompt consideration of this diagnosis. Early recognition and supplementation are essential to prevent irreversible complications.

Keywords: Megaloblastic Anemia; Vitamin B12 Deficiency; Folate Deficiency; Jaundice; Pancytopenia; Bone Marrow

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Introduction

Megaloblastic Anemia (MA) represents a morphologically and biochemically distinct form of anemia characterized by defective DNA synthesis during erythropoiesis, resulting in the emergence of abnormally large erythrocyte precursors - megaloblasts - in the bone marrow and macrocytic red cells in the peripheral circulation [1]. The term "megaloblastic" denotes a state of nuclear-cytoplasmic asynchrony in which cytoplasmic maturation outpaces nuclear development, a consequence of impaired purine and thymidylate synthesis [2].

The most frequent etiological basis is deficiency of vitamin B12 (cobalamin) or folic acid, either individually or in combination [1, 3]. In contrast to Western nations, where autoimmune gastritis causing pernicious anemia is the predominant etiology, Indian data consistently demonstrate that nutritional deficiency - driven by strict vegetarian or lacto-vegetarian diets - accounts for the majority of cases [4-6]. The Indian subcontinent, with its widespread cultural and religious preference for plant-based diets devoid of animal-source foods, bears a disproportionate burden of cobalamin deficiency [7].

Beyond the erythroid lineage, impaired DNA synthesis in megaloblastic anemia involves granulocyte precursors and megakaryocytes, resulting in hypersegmented neutrophils, leukopenia, and thrombocytopenia. In advanced disease, all three cell lines are affected, producing pancytopenia that may mimic aplastic anemia or myelodysplastic syndrome [1, 8]. This diagnostic overlap makes the clinico-pathologic characterization of MA particularly important in a tertiary care setting.

Clinically, MA presents an insidious course; patients frequently remain asymptomatic until hemoglobin falls to critically low levels [1]. On presentation, constitutional symptoms predominate, alongside characteristic signs such as pallor, glossitis, lemon-yellow tinge of skin, and, in cobalamin-deficient cases, subacute combined degeneration of the spinal cord [3]. Jaundice, arising from intramedullary hemolysis, occurs in a subset of patients and is a clinically important yet frequently underappreciated manifestation. Prior Indian studies have reported jaundice in 24-35% of MA patients, emphasizing its diagnostic relevance [9, 10].

Despite being one of the most treatable causes of severe anemia, MA continues to be under-recognized, particularly in settings where serum vitamin assays are not routinely accessible. In resource-limited tertiary care hospitals in India, peripheral smear and bone marrow examination retain central diagnostic importance [11, 12]. Characterizing the clinical, hematological, and biochemical profile of MA in this context not only informs clinical practice but also guides targeted public health interventions.

This study was undertaken to describe the clinico-pathologic profile of megaloblastic anemia among patients presenting to a tertiary care center in North India, with particular attention to the prevalence and clinical correlates of jaundice, the pattern of cytopenia, and the etiological distribution of vitamin deficiency.

Materials and Methods

Study design and setting

This was a cross-sectional observational study conducted over 18 months in the Department of Medicine at our medical college, a tertiary referral center serving western Uttar Pradesh and adjoining regions. The study period extended from June 2024 to December 2025. The study protocol was approved by the Institutional Ethics Committee of our medical college. Written informed consent was obtained from all participants prior to enrolment.

Study population

Patients of either sex, aged 14 years and above, admitted with or diagnosed to have anemia and subsequently confirmed to have megaloblastic anemia were enrolled. Patients with concurrent hepatic disease, hemolytic anemia of other etiology, malignancy, renal failure, or those on cytotoxic or antifolate medications (e.g., methotrexate, hydroxyurea) were excluded to avoid confounding of hematological or biochemical parameters.

Diagnostic criteria

Megaloblastic anemia was diagnosed on the basis of the following [8]:

1. Hemoglobin below 13 g/dL in males and 12 g/dL in females, with MCV (mean corpuscular volume) >100 fL.

2. Peripheral blood smear showing macrocytosis, macro-ovalocytes, anisocytosis, poikilocytosis, and hypersegmented neutrophils (≥5 lobes in ≥5% of neutrophils).

3. Bone marrow aspiration revealing megaloblastic erythroid hyperplasia with nuclear-cytoplasmic asynchrony, giant metamyelocytes, or hypersegmented megakaryocytes, wherever clinically indicated.

4. Serum vitamin B12 level <200 pg/mL and/or serum folate <3.0 ng/mL, measured by chemiluminescence immunoassay.

The severity of anemia was classified as mild (Hb 10-12.9 g/dL in males / 10-11.9 g/dL in females), moderate (Hb 7-9.9 g/dL), and severe (Hb <7 g/dL) per WHO criteria [13].

Data collection

A pre-designed, structured proforma recorded demographic details (age, sex, socioeconomic status, dietary habits), presenting symptoms (duration, nature), physical signs on examination, and the results of all laboratory investigations. Dietary habit was classified as pure vegetarian (no fish, poultry, eggs, or meat) or mixed diet.

Laboratory investigations

Complete blood count was performed using an automated hematology analyzer. Peripheral smear examination was performed by trained laboratory personnel and reviewed by a senior pathologist. Bone marrow aspiration was performed in cases with diagnostic uncertainty, pancytopenia, or suspicion of an alternate marrow disorder. Liver function tests, serum LDH (lactate dehydrogenase), indirect serum bilirubin, reticulocyte count, and urine urobilinogen were measured in all patients.

Statistical analysis

Data were entered into Microsoft Excel and analyzed using SPSS version 25.0. Categorical variables were expressed as frequencies and percentages. Continuous variables were expressed as mean ± standard deviation (SD). Chi-square test and Fisher's exact test were used for categorical comparisons where appropriate. A p-value of <0.05 was considered statistically significant.

Results

Demographic profile

A total of 80 patients with confirmed megaloblastic anemia were enrolled over the study period. The mean age was 34.6±14.2 years. The majority of patients, 38 (47.5%), fell in the 20-40 year age group, followed by 21 (26.3%) in the 41-60 year group and 14 (17.5%) in the 14-20 year group. Seven patients (8.7%) were above 60 years of age. Males accounted for 46 (57.5%) of cases and females for 34 (42.5%), yielding a male-to-female ratio of 1.35:1. Regarding dietary habit, 56 patients (70%) were pure vegetarians and 24 (30%) consumed a mixed diet. The majority, 68 (85%), belonged to lower or lower-middle socioeconomic strata. Sixty-two (77.5%) patients came from peri-urban or rural backgrounds (Table 1).

Table 1: Demographic profile of study patients (n=80).

Parameter	Category	Number (n)	Percentage (%)
Age group (years)	14–20	14	17.5
	21–40	38	47.5
	41–60	21	26.3
	>60	7	8.7
Sex	Male	46	57.5
	Female	34	42.5
Dietary habit	Vegetarian	56	70.0
	Mixed diet	24	30.0
Socioeconomic status	Lower/lower-middle	68	85.0
	Middle and above	12	15.0
Residence	Rural/peri-urban	62	77.5
	Urban	18	22.5

Table 2: Clinical symptoms and signs (n=80).

Feature	Number (n)	Percentage (%)
Symptoms		
Weakness/fatigue	77	96.3
Anorexia	51	63.7
Breathlessness on exertion	44	55.0
Palpitations	36	45.0
Pedal swelling	30	37.5
Weight loss	22	27.5
Tingling/numbness	18	22.5
Gait unsteadiness	8	10.0
Physical signs		
Pallor	80	100.0
Glossitis	55	68.8
Hyperpigmentation of knuckles	46	57.5
Pedal edema	28	35.0
Splenomegaly	26	32.5
Jaundice	23	28.7
Hepatomegaly	22	27.5
Neurological signs	16	20.0
Koilonychia	12	15.0
Cardiac decompensation	14	17.5

Clinical presentation

Symptoms: Generalized weakness and easy fatigability were the most frequent presenting complaints, reported by 77 patients (96.3%) (Table 2). Anorexia was present in 51 (63.7%), breathlessness on exertion in 44 (55%), palpitations in 36 (45%), and swelling of feet in 30 (37.5%). Significant weight loss was noted by 22 patients (27.5%). Tingling or numbness in the extremities was reported by 18 patients (22.5%), and difficulty walking or gait unsteadiness by 8 (10%).

Physical signs: Pallor was universal, observed in all 80 patients (100%). Glossitis - manifesting as a smooth, beefy-red, depapillated tongue (Hunter's glossitis) - was present in 55 (68.8%). Jaundice was detected clinically in 23 patients (28.7%), presenting as mild-to-moderate icteric discoloration of sclera and skin, consistent with the predominantly indirect hyperbilirubinemia of intramedullary hemolysis. Hyperpigmentation of knuckles was a notable finding in 46 patients (57.5%). Pedal edema was present in 28 (35%), and koilonychia in 12 (15%). Splenomegaly was detected in 26 patients (32.5%) and hepatomegaly in 22 (27.5%).

Neurological signs - including impaired vibration sense, loss of proprioception, positive Romberg's sign, or brisk deep tendon reflexes - were elicitable in 16 patients (20%), though overt symptomatic neuropathy was clinically apparent in 18 (22.5%). Evidence of cardiac decompensation in the form of tachycardia with cardiomegaly was present in 14 patients (17.5%) (Table 2).

Hematological findings: The mean hemoglobin at presentation was 5.8±1.9 g/dL. Severe anemia (Hb <7 g/dL) was the most common severity category, present in 42 patients (52.5%). Moderate anemia was found in 30 (37.5%) and mild anemia in 8 (10%). The mean MCV was 114.3±12.6 fL, and all patients had MCV >100 fL.

Peripheral blood smear analysis demonstrated macrocytosis in all 80 patients (100%), anisocytosis in 74 (92.5%), macro-ovalocytosis in 66 (82.5%), and hypersegmented neutrophils in 78 (97.5%). Basophilic stippling was seen in 18 (22.5%) and Howell-Jolly bodies in 10 (12.5%).

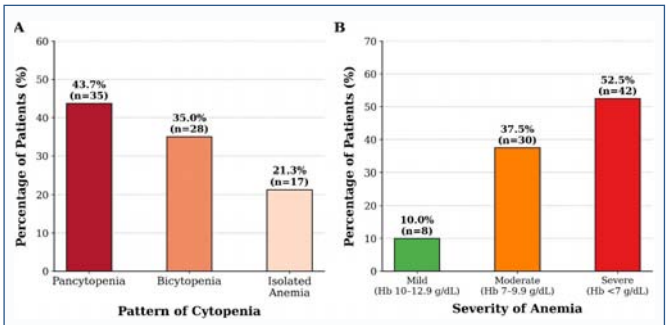

Figure 1: (A) Distribution of cytopenia patterns among patients with megaloblastic anemia (n=80). **(B)** Severity of anemia at presentation classified per WHO criteria. Values above bars represent percentage and absolute number of patients.
Hb: hemoglobin

Table 3: Hematological findings (n=80).

Parameter	Finding	Number (n)	Percentage (%)
Severity of anemia	Mild (Hb 10–12.9 g/dL)	8	10.0
	Moderate (Hb 7–9.9 g/dL)	30	37.5
	Severe (Hb <7 g/dL)	42	52.5
Mean Hb (g/dL)	5.8 ± 1.9	—	—
Mean MCV (fL)	114.3 ± 12.6	—	—
Peripheral smear findings	Macrocytosis	80	100.0
	Hypersegmented neutrophils	78	97.5
	Anisocytosis	74	92.5
	Macro-ovalocytosis	66	82.5
	Basophilic stippling	18	22.5
	Howell-Jolly bodies	10	12.5
Cytopenia pattern	Pancytopenia	35	43.7
	Bicytopenia	28	35.0
	Isolated anemia	17	21.3
Leucopenia	Present	48	60.0
Thrombocytopenia	Present	52	65.0

Hb: hemoglobin; MCV: mean corpuscular volume; fL: femtolitres; g/dL: grams per decilitre

Regarding cytopenias, pancytopenia (concurrent anemia, leukopenia, and thrombocytopenia) was the presenting pattern in 35 patients (43.7%). Bicytopenia (anemia with either leucopenia or thrombocytopenia) was seen in 28 (35%), while isolated anemia without significant co-existing cytopenia was noted in 17 (21.3%) (Figure 1).

The mean total leukocyte count was $3.2\pm1.4\times10^3/\mu L$, with leucopenia ($<4.0\times10^3/\mu L$) in 48 patients (60%). Thrombocytopenia ($<1.5\times10^5/\mu L$) was present in 52 patients (65%). Reticulocyte count was low-for-degree-of-anemia in all cases, ranging from 0.4% to 1.7% (Table 3).

Biochemical findings: Serum LDH was elevated (>240 U/L) in 72 patients (90%), reflecting extensive intramedullary destruction of

Table 4: Biochemical and etiological profile (n=80).

Parameter	Finding	Number (n)	Percentage (%)
Elevated serum LDH	Present	72	90.0
Elevated indirect bilirubin (among jaundiced)	Present	23	100 of jaundiced
Raised urine urobilinogen (among jaundiced)	Present	20	87.0 of jaundiced
Mild transaminase elevation	Present	6	7.5
Etiology			
Isolated vitamin B12 deficiency		58	72.5
Combined B12 + folate deficiency		15	18.7
Isolated folate deficiency		7	8.75

LDH: lactate dehydrogenase

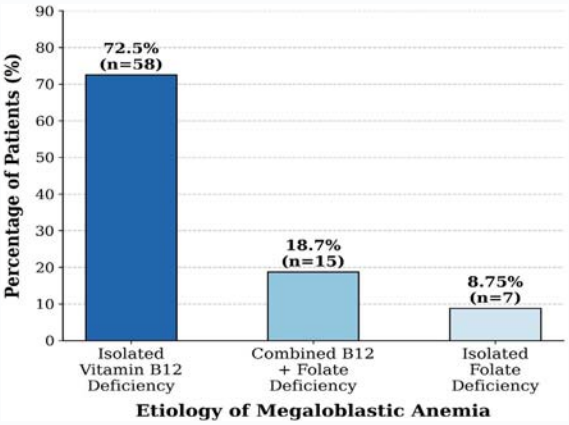

Figure 2: Bar chart depicting the etiological distribution of megaloblastic anemia among study patients (n=80). Values above bars represent the percentage and absolute number of patients in each category.
Hb: hemoglobin; B12: vitamin B12.

erythroid precursors. Among the 23 patients with clinical jaundice, indirect serum bilirubin was elevated in all, with values ranging from 2.1 to 5.6 mg/dL; direct bilirubin was within normal limits in all but two patients with minor hepatic component. Urine urobilinogen was raised in 20 of the 23 jaundiced patients (Table 4).

Serum ALT, AST, and alkaline phosphatase were within normal limits in the vast majority; mild transaminase elevation was noted in 6 patients (7.5%), attributable to anemic hepatocellular injury rather than primary hepatic disease.

Etiological profile: Vitamin B12 deficiency alone was identified in 58 patients (72.5%). Combined B12 and folate deficiency was found in 15 (18.7%). Isolated folic acid deficiency accounted for 7 (8.75%) of cases (Figure 2). Among patients with B12 deficiency, strict vegetarian dietary habit was the most common predisposing factor (67 of 73 B12-deficient patients, 91.8%) (Table 4).

Bone marrow findings: Bone marrow aspiration was performed in 52 patients (65%) - those with diagnostic uncertainty, all patients with pancytopenia, and cases in whom a primary marrow disorder was clinically suspected. Hypercellular marrow with erythroid hyperplasia was the dominant pattern, observed in 46 of 52 aspirates (88.5%). Nuclear-cytoplasmic asynchrony in erythroid precursors (megaloblastic change) was confirmed in all aspirated cases. Giant metamyelocytes were seen in 40 (76.9%) and hypersegmented megakaryocytes in 22 (42.3%). Three patients (5.7%) had normocellular marrow (Table 5).

Table 5: Bone marrow findings (n=52 aspirated).

Finding	Number (n)	Percentage (%)
Hypercellular marrow with erythroid hyperplasia	46	88.5
Megaloblastic erythroid change (nuclear-cytoplasmic asynchrony)	52	100.0
Giant metamyelocytes	40	76.9
Hypersegmented megakaryocytes	22	42.3
Normocellular marrow	3	5.7

Treatment response: All patients received intramuscular cyanocobalamin 1000 μg daily for 7 days followed by monthly maintenance, with oral folic acid 5 mg daily where deficiency was established or suspected. Reticulocytosis was the first response to therapy, observed within 5–8 days of initiation. Hemoglobin recovery to acceptable levels (>10 g/dL) was achieved in 70 patients (87.5%) within 8 weeks. Jaundice resolved within 2-3 weeks in all 23 affected patients. Blood transfusion was required in 12 patients (15%) presenting with very severe anemia (Hb <4 g/dL) or hemodynamic compromise.

Discussion

Megaloblastic anemia remains one of the most prevalent and clinically significant nutritional deficiency disorders in the Indian subcontinent. In the present study, 80 patients presenting to a tertiary care center in Agra were found to have a clinico-pathologic profile broadly concordant with, yet distinctively nuanced from, reports across other Indian centers.

The predominance of the 20-40 year age group (47.5%) in our series aligns with findings from Sharma *et al*., from central India, who similarly found the highest burden in patients aged 16-30 years, attributing it to the economic dependence on vegetarian staple diets in young adults [5]. Kaur *et al*., from a military tertiary center also found MA most common in adults in their third to fifth decades [6]. The modest male preponderance (57.5%) in our study is consistent with reports from Unnikrishnan *et al*., who noted a 65% male predominance in macrocytic anemias, potentially reflecting a higher rate of healthcare-seeking among male patients in this region [12].

The overwhelming predominance of pure vegetarians (70%) among our patients corroborates the well-established etiological link between plant-based diets and vitamin B12 deficiency in India [7]. Animal-derived foods - meat, fish, eggs, and dairy - are the sole reliable dietary sources of cobalamin, and strict avoidance of these foods without supplementation creates a physiological basis for progressive depletion of hepatic B12 stores over two to five years [3, 7]. Sharma *et al*., reported 68.2% vegetarians in their series, and Sagari *et al*., found 81% vegetarians among affected children in Hyderabad [5,14].

Pallor was universal in our cohort (100%), as expected given the severity of anemia at presentation. Glossitis, present in 68.8%, reflects the widespread mucosal involvement of nutritional deficiency and was reported in 35.7% of patients by Pokharel et al. from Nepal, though their small series (n=14) may have underrepresented this finding [10]. Hyperpigmentation of knuckles was found in 57.5% of our patients, consistent with the observation of Mulani et al. and Sagari et al. that this is a common cutaneous marker in megaloblastic anemia in South Asian patients [9, 14].

Jaundice in our study was observed in 28.7% of patients - a

proportion that closely mirrors several Indian and South Asian studies. Mulani et al. from Maharashtra found jaundice in 28.7% (12 of 42 patients) of their MA cohort, and Pokharel et al. reported a prevalence of 35.7% in their Nepalese series [9, 10]. Sharma *et al*., documented jaundice in 24.3% of their central Indian patients [5]. The jaundice in MA is characteristically mild and of the indirect (unconjugated) type, attributable to intramedullary hemolysis - the premature destruction of dysplastic erythroid precursors within the bone marrow before they reach the peripheral circulation - rather than posthepatic obstruction or hepatocellular damage [2, 15]. In our study, this was corroborated by elevated indirect bilirubin, raised urine urobilinogen, markedly elevated LDH (90%), and normal or near-normal direct bilirubin in all jaundiced patients. The complete resolution of icterus within two to three weeks of commencing cobalamin supplementation further supports the hemolytic rather than hepatic basis of this finding. Katakam *et al*., also documented this pattern in an adolescent with vitamin B12 deficiency presenting with prominent jaundice [16]. Clinicians must be aware that MA is an important differential when indirect jaundice co-exists with macrocytic anemia, and failure to recognize this may delay treatment.

Pancytopenia was present in 43.7% of patients in our series, a figure comparable to the 45% reported by Kaur *et al*., [6]. Santra and Das found megaloblastic anemia to be an important reversible etiology among patients presenting with pancytopenia to a tertiary care center [11]. The presence of all three cytopenias in a patient with macrocytic anemia should prompt early bone marrow examination and serum vitamin assays to exclude treatable nutritional causes before attributing pancytopenia to primary marrow disorders such as aplastic anemia or myelodysplastic syndrome [17].

Regarding etiology, isolated vitamin B12 deficiency was the dominant finding (72.5%), followed by combined B12 and folate deficiency (18.7%). This pattern is consistent with Kaur et al., who found B12 deficiency in 40% and combined deficiency in 35% of patients [6], and with Unnikrishnan et al., who found 78.3% of megaloblastic anemias attributable to B12 alone and 21.7% to combined deficiency [12]. Isolated folate deficiency was relatively uncommon in our study (8.75%), a finding attributable to the availability of green leafy vegetables in the dietary pattern of this region.

Neurological involvement - peripheral neuropathy, subacute combined degeneration - was present in 22.5% of patients, underscoring the multisystem nature of cobalamin deficiency. This is comparable to the 35% overt and subclinical neuropathy rate reported by Kaur *et al*., [6] and the 21.4% paresthesia rate in Pokharel *et al*.,'s series [10]. Notably, neurological symptoms may manifest even at near-normal hemoglobin levels, and their presence should prompt serum B12 estimation regardless of MCV [3, 18].

The high prevalence of severe anemia (52.5%) in our study reflects the characteristic delayed presentation seen at tertiary care hospitals in India, where patients tolerate gradually worsening symptoms and seek care only when profoundly compromised. The low reticulocyte count despite severe anemia - a hallmark of ineffective erythropoiesis - was a consistent finding, reinforcing the utility of this parameter in distinguishing megaloblastic from hemorrhagic or hemolytic anemia.

Treatment response was gratifyingly prompt. The early reticulocytosis, resolution of jaundice, and hemoglobin recovery within eight weeks in the majority of patients highlight that MA, despite its potentially alarming presentation, is one of the most rewarding treatable causes of severe anemia.

Limitations of the Study

This study has certain limitations. The cross-sectional design precludes long-term follow-up of neurological outcomes. Anti-intrinsic factor and anti-parietal cell antibodies, which would facilitate identification of pernicious anemia, were not measured in all patients. Methylmalonic acid assay - a more sensitive marker of functional B12 deficiency - was unavailable. Despite these constraints, the findings add to the growing body of Indian literature characterizing MA in tertiary care settings and reinforce the need for broader nutritional surveillance and supplementation programs.

Conclusion

Megaloblastic anemia at this tertiary care center in North India predominantly affects young adults on strict vegetarian diets and presents with advanced, multisystem disease. Jaundice, arising from intramedullary hemolysis, was observed in approximately one-third of patients, a proportion consistent with previous Indian and South Asian studies. Pancytopenia is a frequent presenting pattern and should not preclude early consideration of nutritional etiology. Vitamin B12 deficiency, alone or in combination with folate deficiency, accounts for the majority of cases. Prompt recognition and parenteral supplementation result in rapid clinical and hematological recovery. The study underscores the need for dietary counseling, public awareness, and inclusion of B12 supplementation in national nutritional programs targeting at-risk vegetarian populations.

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