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Anemia: Detection and Management by RBC Indices – A Retrospective Analysis of 100 Cases

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Abstract

Background: Anemia is highly prevalent in India, especially in children, pregnant females, and women of reproductive age, amounting to an average of 40%. Though the etiology is multifactorial, major factors are nutritional deficiencies and blood loss. A fall in hemoglobin is synonymous with anemia and Hb and PCV are considered the common values in a hemogram. The red cell indices indicate the size and content of the red cells and are highly valuable in narrowing the etiology and plan line of management. This study stresses the values of MCV, MCH, MCHC and RDW as equally important contributors as Hb and PCV.

MATERIALS & METHODS

Study Design: Retrospective clinical observational study.

Study Period: May 2026 – July 2026.

Study Material: Case records of one hundred patients from medical, Obstetric and Gynecology wards during the study period with complete hemogram available on admission.

Study Sample: A convenient sample of 100 cases was included for analysis. ***OBSERVATIONS AND RESULTS***: With the view of classifying the type of anemia and identifying the etiology, the values of Hb, PCV, MCV, MCHC and RDW were analysed for 100 patients. As per WHO criteria, Hb less than 12 g/dL was considered anemia in females; <11.9 g/dL was classified as mild anemia, <9.9 g/dL as moderate anemia and Hb <7 g/dL as severe anemia. According to cell size or mean corpuscular volume (MCV), the cells were considered normocytic if MCV was 80–100 fL, microcytic if <80 fL and macrocytic if >100 fL. According to hemoglobinisation or MCHC (mean corpuscular hemoglobin concentration), a value less than 32 g/dL was considered hypochromic anemia. The prevalence of anemia in our cohort was 70% and more than 70% was contributed by obstetrics and gynecology departments. In this study, normocytic normochromic anemia was the largest group, indicating a chronic nutritional deficiency or chronic blood loss. Majority of patients were categorised as mild anemia. MCH and MCHC were comparable in anemic and non-anemic patients, while Hb, PCV and MCV were low and RDW was higher than non-anemic population. Hb and PCV had strong correlation ($r = 0.94$, $p < 0.001$), while RDW rises as MCV and MCH fall, as expected, with increasing anisocytosis in microcytic anemia ($p < 0.05$). In this cohort, prevalence of anemia was the same irrespective of the departments. The MCV–MCHC scatter map offers a clear triaging of anemia as iron deficiency or folic acid deficiency as a simple bedside visual. If RDW is raised more than 15%, even if mean corpuscular volume is normal, iron deficiency anemia may be considered. RDW is negatively correlating with Hb, MCV and PCV, with a significant p value ($p < 0.001$) with MCV. ***CONCLUSION***: MCV is important in morphological classification of anemia. MCV and RDW correlate towards evaluation of anemia. MCV and MCHC offers a clear triaging of anemia as to iron deficiency or folic acid deficiency. RDW negatively correlates with Hb, significantly ($p < 0.001$). Thus, the RBC indices could offer valuable supplement to Hb and PCV towards etiological and management of anemia and need equal and parallel correlation with Hb and PCV.

Keywords: Hemoglobin (Hb), Packed Cell Volume (PCV), Mean Corpuscular Volume (MCV), Mean Corpuscular Hemoglobin (MCH), Mean Corpuscular Hemoglobin Concentration (MCHC), Red Cell Distribution Width (RDW).

INTRODUCTION

The prevalence of anemia is quite high with regional, age and gender variations. 40% of children aged 6–59 months, 37% of pregnant women and 30% of women aged 15–49 years are affected by anemia according to WHO Anemia Fact Sheet 2023. Northern and Eastern India have high pooling of anemia.\

The size, shape and content of erythrocytes constitute the red blood cell indices. The red blood cells are the largest population in blood and their deficiency in quality and quantity affect tissue oxygenation. Anemia is the commonest presentation as a comorbidity, playing a vital role in tissue oxygenation, surgical-related wound healing, sepsis and the course of the disorder for which the patient is admitted into the hospital. Thus, management of anemia is essential for an optimal anaesthetic and surgical outcome. In this study, we intended to analyse the erythrocyte indices from case records of one hundred patients and analyse their importance towards diagnosis and management of anemia.

Aim

To evaluate, understand and apply erythrocyte indices for detection and management of anemia.

Objectives

1. To understand RBC indices namely hemoglobin (Hb), packed cell volume (PCV), mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH), mean corpuscular hemoglobin concentration (MCHC), and red cell distribution width (RDW).
2. To classify the type of anemia with the values of the indices and find the main causative factor.
3. To aid in decision-making regarding blood, iron, vitamin or mineral therapy against blood and component transfusion.

MATERIALS AND METHODS

Study Design

Retrospective clinical observational study.

Study Period

May 2026 – July 2026.

Study Material

Case records of one hundred patients from medical, Obstetric and Gynecology wards during the study period with complete hemogram available on admission.

Study Sample

A convenient sample of 100 cases was included for analysis.

Methods

The complete hemograms on admission of patients admitted to medical, gynecological and obstetric wards were included for the study. Hemoglobin (Hb in grams percent), PCV (packed cell volume in percentage), MCV (mean corpuscular volume in femtolitres [fL]), MCHC (mean corpuscular hemoglobin concentration [g/dL]), MCH (mean corpuscular hemoglobin [pg]) and red cell distribution width (RDW) were noted for hundred patients along with patient demographics such as age, gender and admission area. The results were tabulated and analysed.

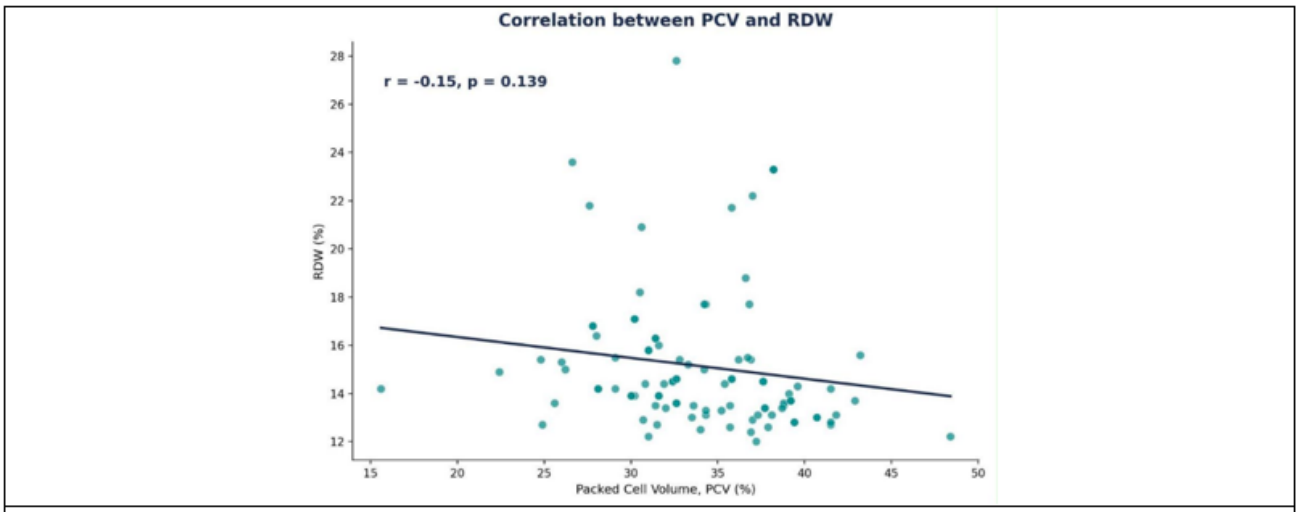
RESULTS

With the view of classifying the type of anemia and identifying the etiology, the values of Hb, PCV, MCV, MCHC and RDW were analysed for 100 patients. As per WHO criteria, Hb less than 12 g/dL was considered anemia in females; <11.9 g/dL was classified as mild anemia, <9.9 g/dL as moderate anemia and Hb <7 g/dL as severe anemia.

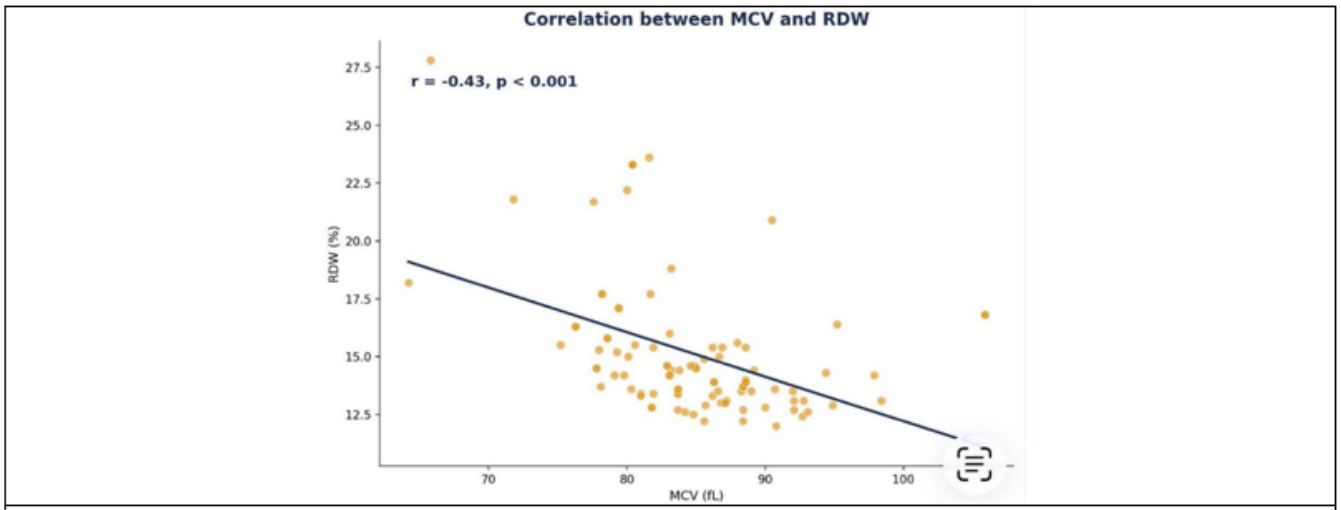
According to cell size or mean corpuscular volume (MCV), the cells were considered normocytic if MCV was 80–100 fL, microcytic if <80 fL and macrocytic if >100 fL. According to hemoglobinisation or MCHC (mean corpuscular hemoglobin concentration), a value less than 32 g/dL was considered hypochromic anemia. The prevalence of anemia in our cohort was 70% and more than 70% was contributed by obstetrics and gynecology departments.

In this study, normocytic normochromic anemia was the largest group, indicating a chronic nutritional deficiency or chronic blood loss. Majority of patients were categorised as mild anemia. MCH and MCHC were comparable in anemic and non-anemic patients, while Hb, PCV and MCV were low and RDW was higher than non-anemic population. Hb and PCV had strong correlation ($r = 0.94$, $p < 0.001$), while RDW rises as MCV and MCH fall, as expected, with increasing anisocytosis in microcytic anemia ($p < 0.05$). In this cohort, prevalence of anemia was the same irrespective of the departments.

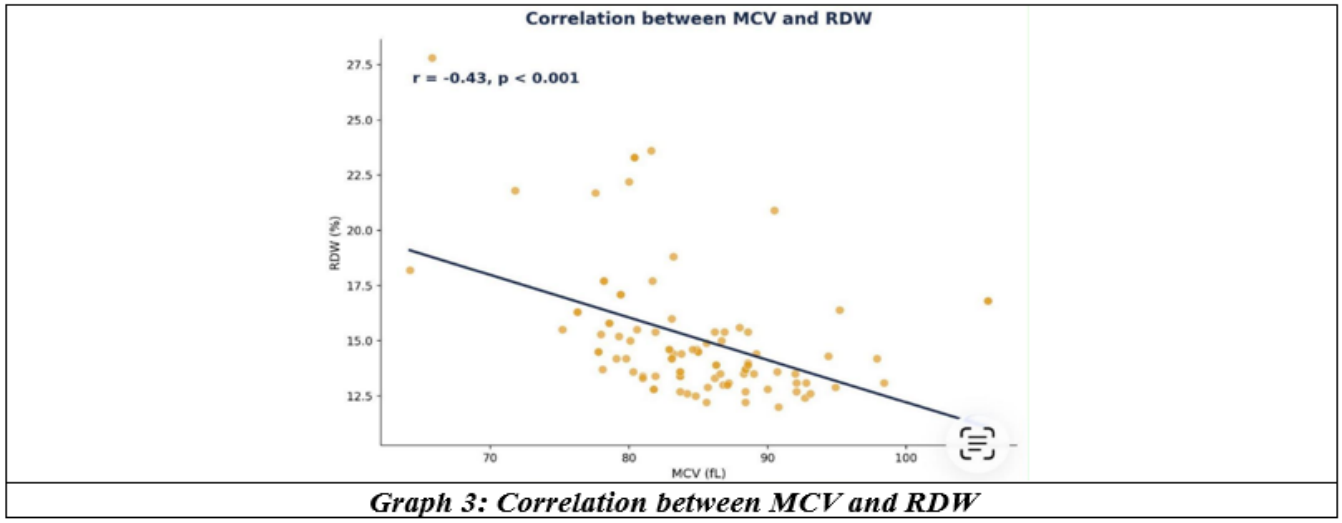
The MCV–MCHC scatter map offers a clear triaging of anemia as iron deficiency or folic acid deficiency as a simple bedside visual. If RDW is raised more than 15%, even if mean corpuscular volume is normal, iron deficiency anemia may be considered. RDW is negatively correlating with Hb, MCV and PCV, with a significant p value ($p < 0.001$) with MCV.



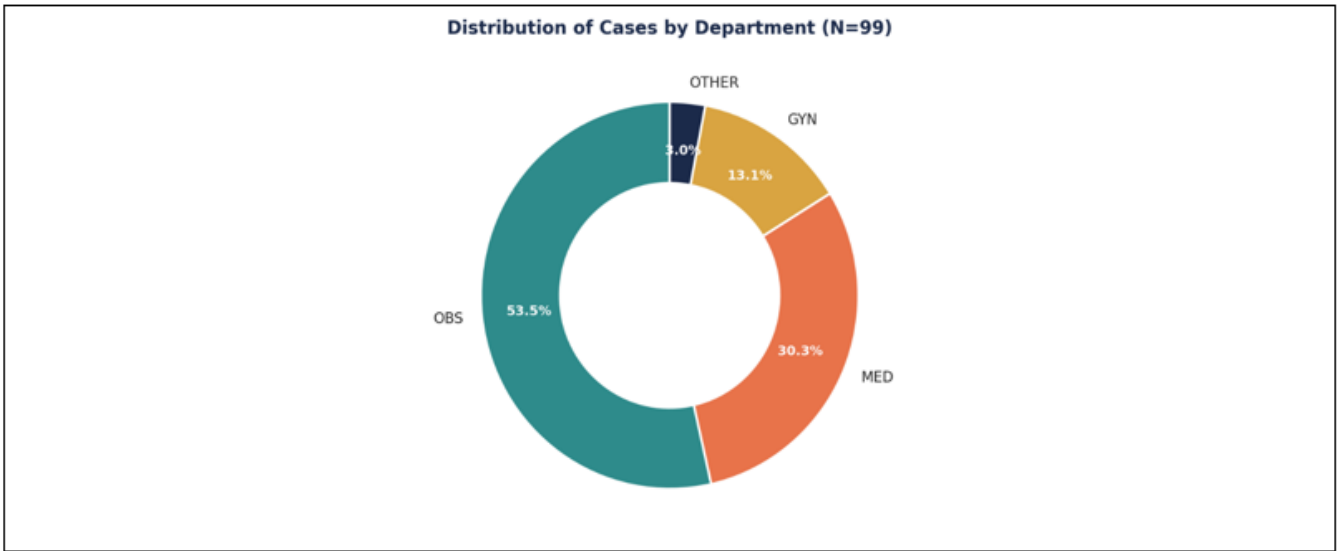
Graph 1: Correlation between PCV and RDW



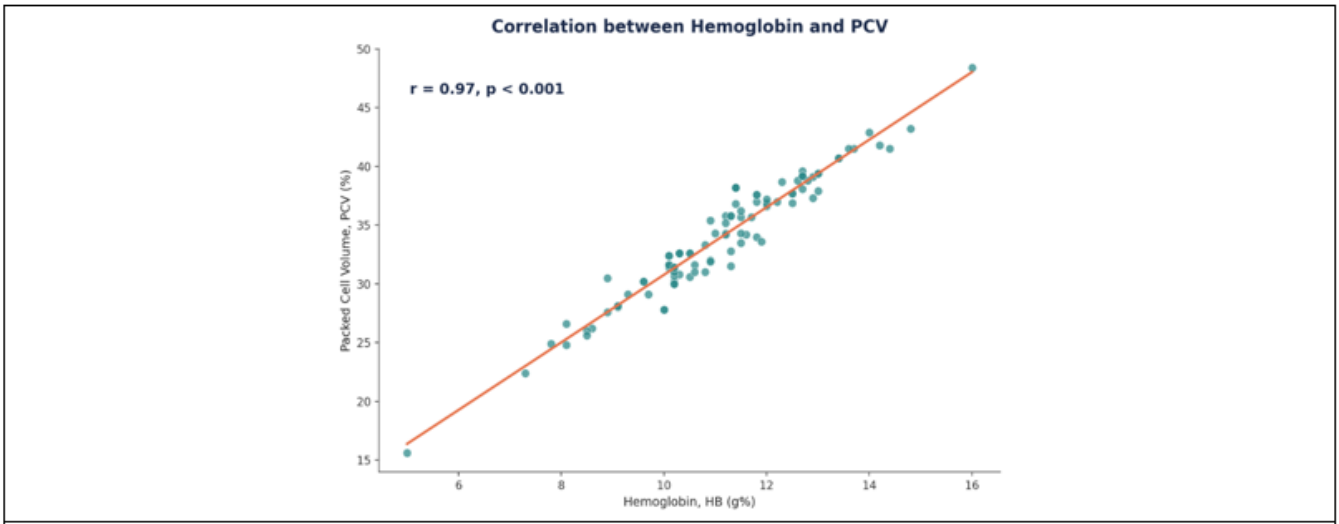
Graph 2: Correlation between MCV and RDW



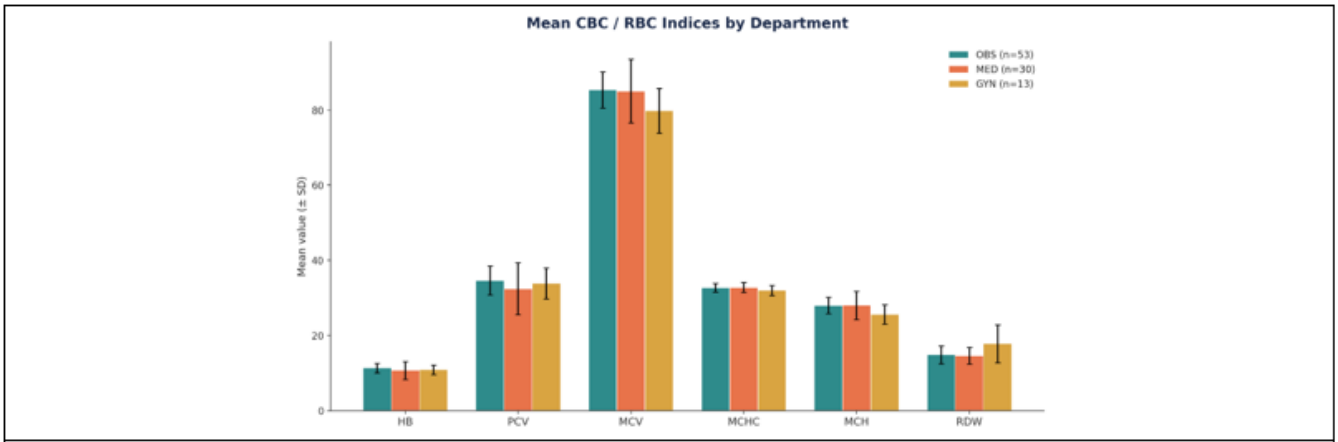
Graph 3: Correlation between MCV and RDW



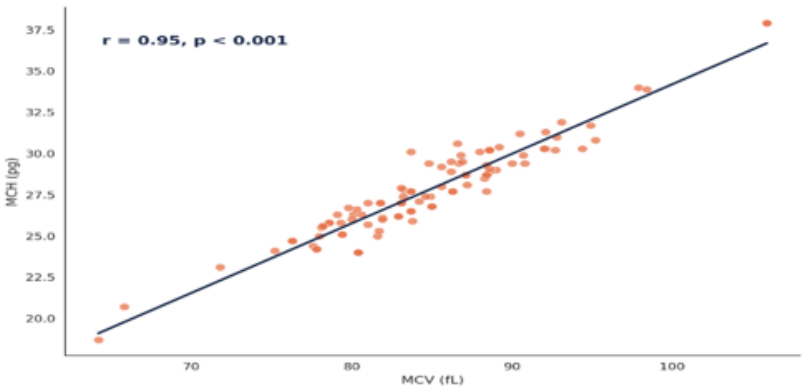
Graph 4: Distribution of Cases by Department



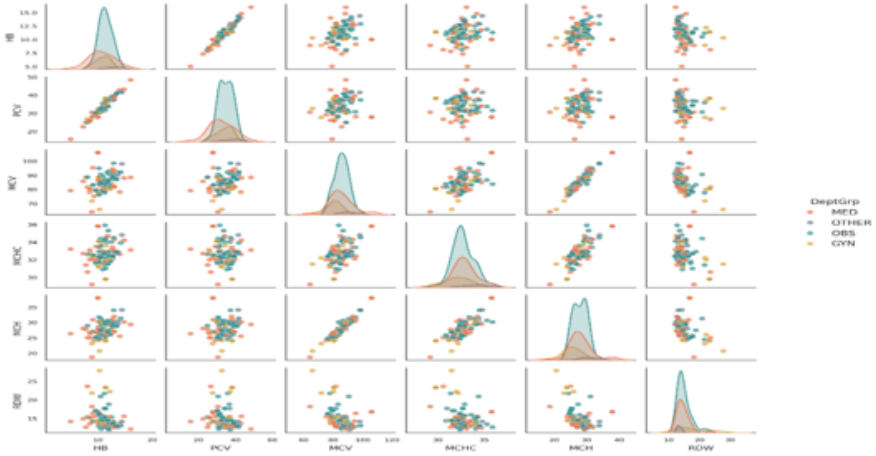
Graph 5: Correlation between Hemoglobin and PCV



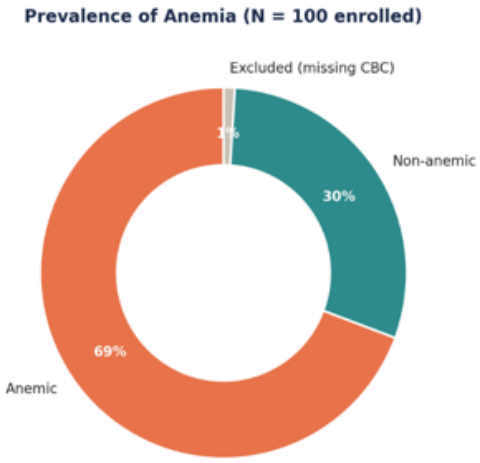
Graph 6: Mean CBC/ RBC Indices by Department



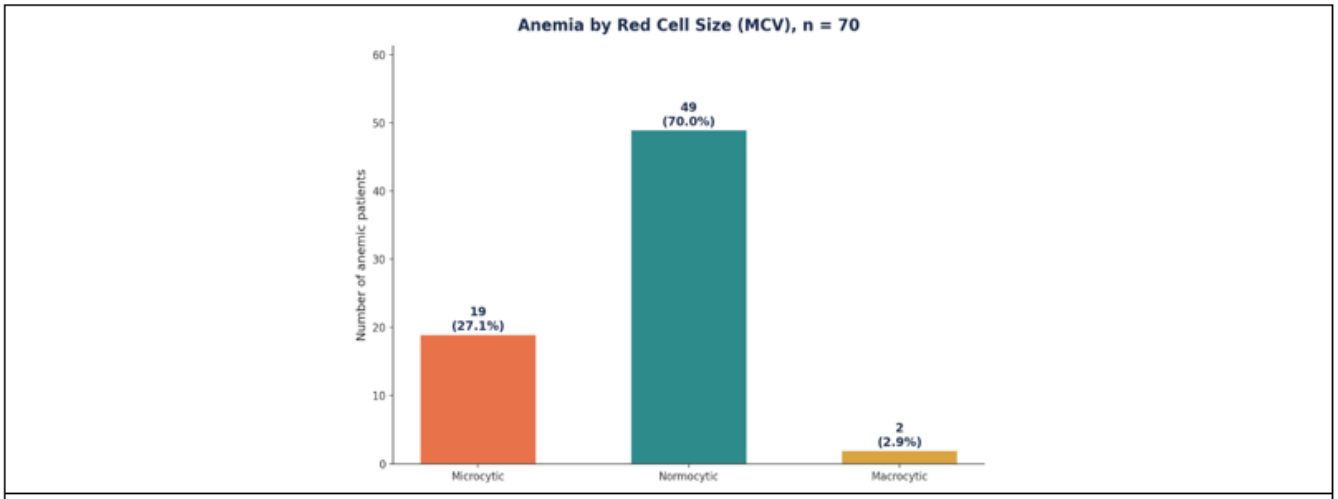
Graph 7: Correlation between MCV and MCH



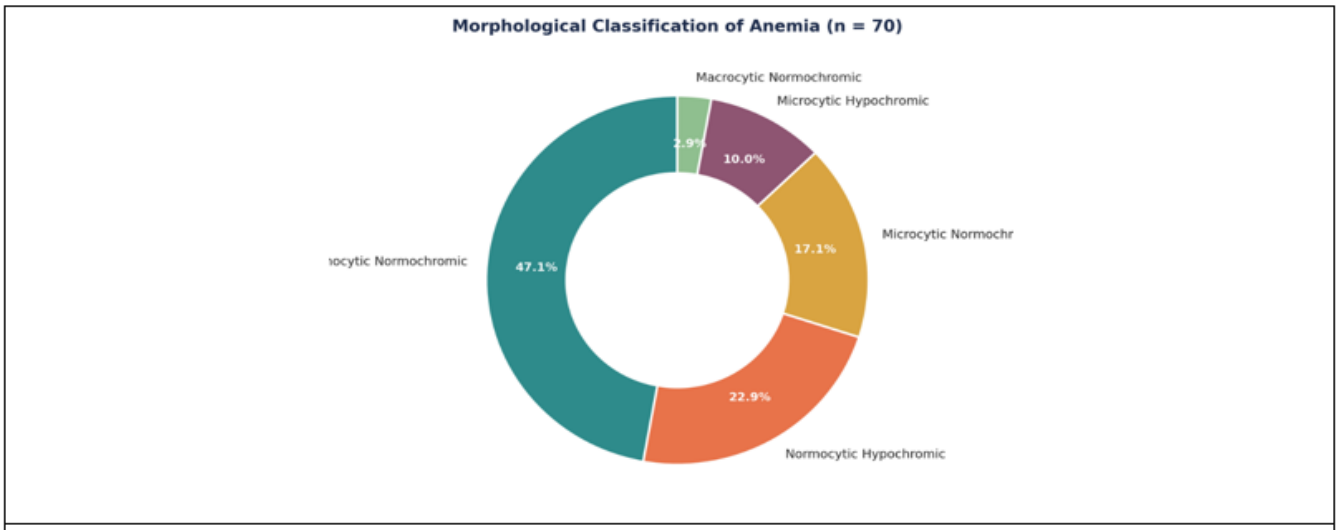
Graph 8: Pairwise Scatter Matrix of RBC Indices by Department



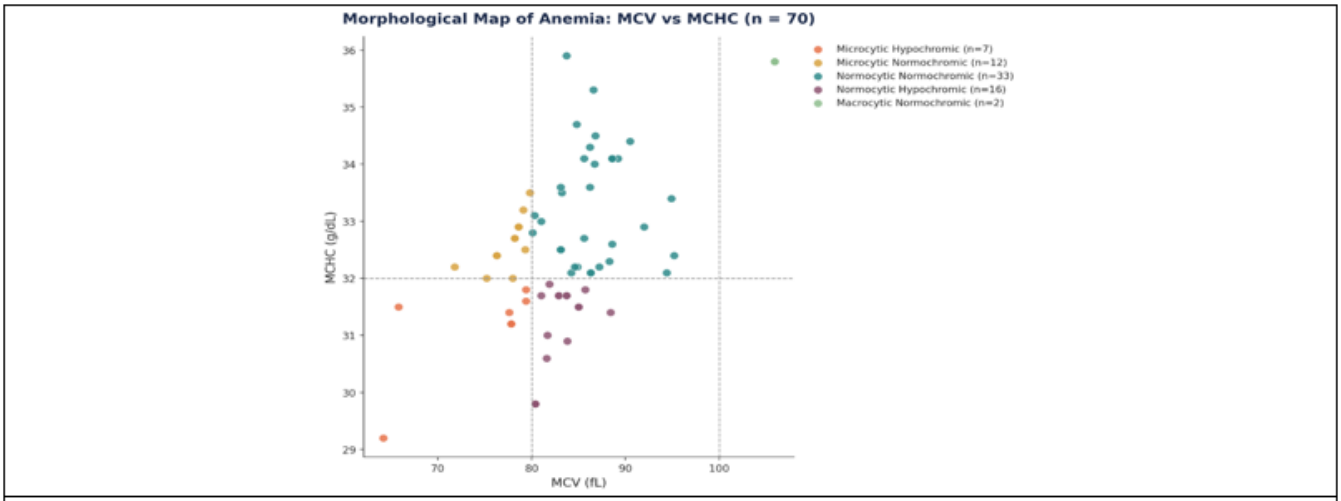
Graph 9: Prevalence of Anaemia



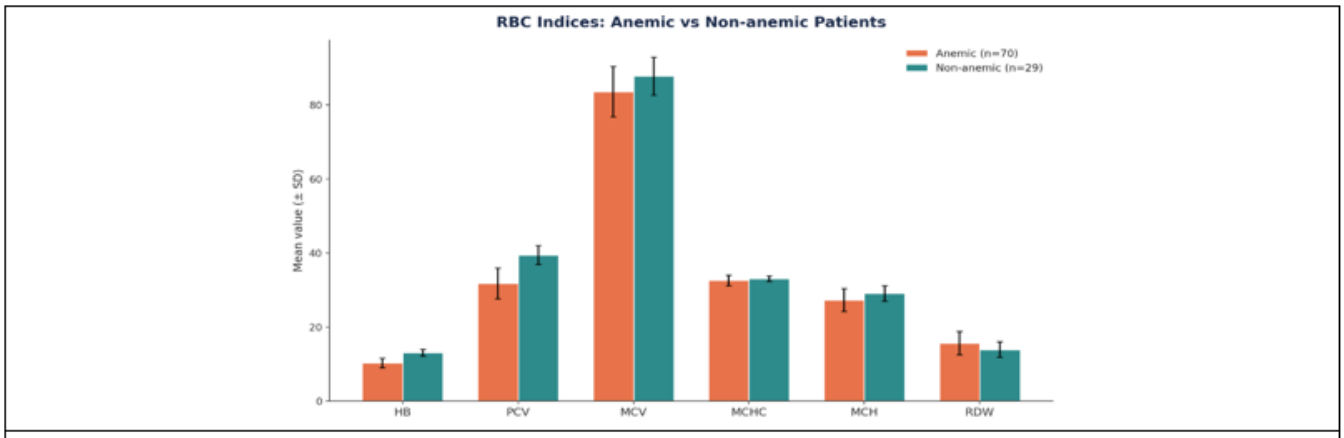
Graph 10: Anemia by Red Cell Size (MCV)



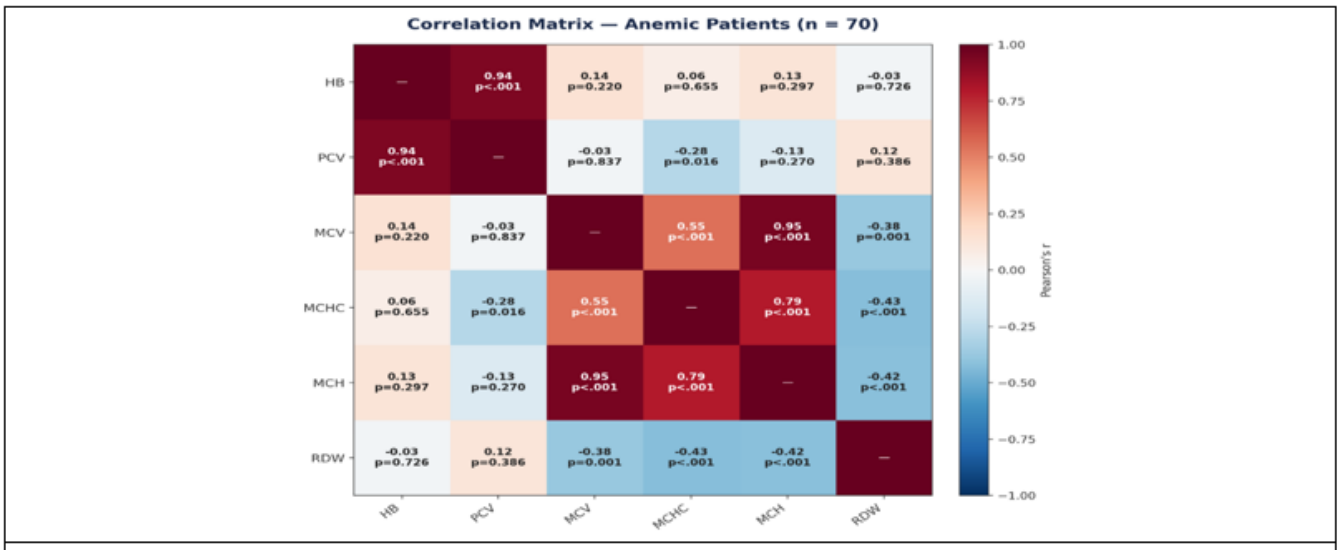
Graph 11: Morphological Classification of Anemia



Graph 12: Morphological Map of Anemia



Graph 13: Morphological Map of Anemia



Graph 14: Correlation Matrix – Anemia Patients

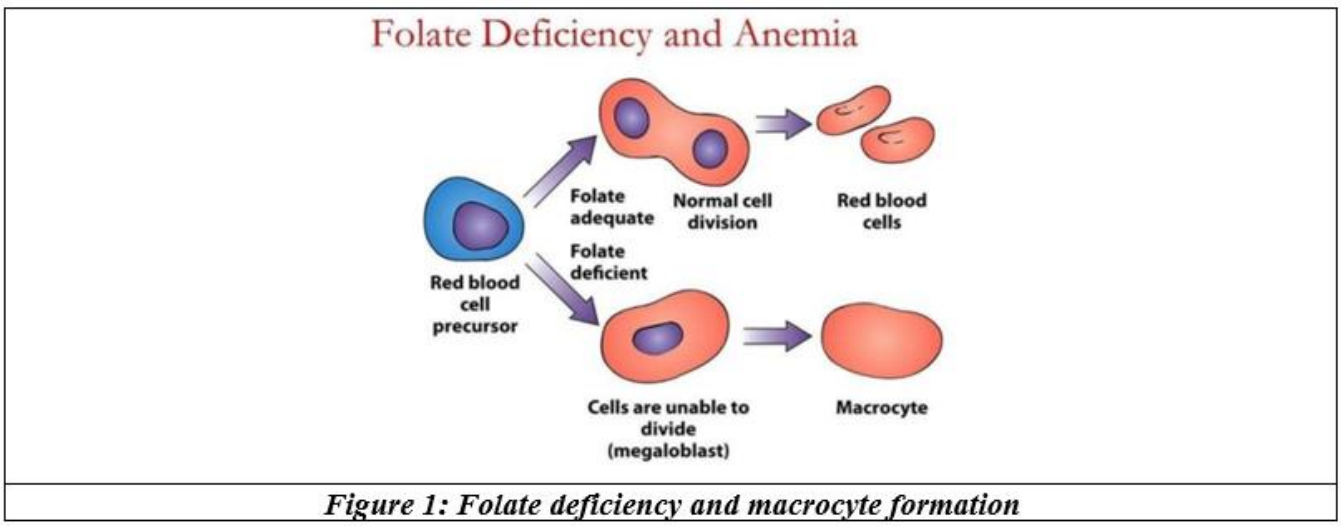


Figure 1: Folate deficiency and macrocyte formation

Macrocytic anemia (MCV >100 fl)	Microcytic anemia (MCV <80 fl)	Normocytic anemia (MCV 80–100 fl)
Megaloblastic anemia	Iron deficiency anemia	Reticulocyte production—normal
Nonmegaloblastic anemia	Thalassemias	• Recent blood loss
• Liver disease	Sideroblastic anemia	• Hemolytic anemia
• Hemolytic anemia	Anemia of chronic disease	Reticulocyte production—deficient
• Alcoholism		• Aplastic anemia
• Myelodysplastic syndrome		• Myelophthisic anemia
• Hypothyroidism		• Chronic renal failure
		• Anemia of chronic disease
		• Hypothyroidism

Table 1: Morphological classification of anemia

RDW	Red cell size as defined by MCV	
	Decreased (microcytic; low MCV)	Normal (normocytic; MCV = N)
Normal (little or no anisocytosis)	Heterozygous thalassemias	Poor iron utilization
		Acute blood loss
	Anemia of chronic disease (hypoproliferative)	Enzyme defects (e.g., G6PD deficiency)
		Acute hemolysis
		Liver disease
Increased (anisocytosis)	Iron deficiency	Early iron deficiency
	Red cell fragmentation	Early megaloblastic
		Sideroblastic
		Myelophthisis
		Combined deficiency
		Sickle cell anemia
		Immune hemolysis

Table 2: MCV and RDW in the evaluation of anemias

Reticulocyte response	Reticulocyte production index (Absolute reticulocyte count)	Causes
1. Appropriate for the degree of anemia	≥2% (>100,000/μL)	Hyperproliferative anemia (blood loss, hemolytic anemia)
2. Inappropriately low for the degree of anemia	<2% (<75,000/μL)	Hypoproliferative anemia (iron deficiency anemia, megaloblastic anemia, anemia of chronic disease, thalassemia, endocrine diseases, sideroblastic anemia, aplastic anemia, myelodysplasia)

Table 3: Classification of anemia according to the reticulocyte response

Anemias due to impaired red cell production	
1. Anemias due to deficiency of nutrients – Iron deficiency anemia – Megaloblastic anemia due to deficiency of folate or vitamin B_{12}	
2. Anemia of chronic disease	
3. Sideroblastic anemia	
4. Aplastic anemia and related disorders	
5. Anemia of chronic renal disease	
6. Anemia of liver disease	
7. Anemia in endocrine disorders	
8. Myelophthisic anemia (anemia due to replacement of marrow by metastatic carcinoma, leukemia, lymphoma, infections, storage disorders, etc.)	
9. Congenital dyserythropoietic anemia	
Anemias due to excessive red cell destruction (Hemolytic anemia)	
Abnormality intrinsic to red cells	**Abnormality extrinsic to red cells**
1. Defects in red cell membrane – Hereditary spherocytosis – Hereditary elliptocytosis	**1. Immune hemolytic anemias** – Autoimmune – Alloimmune – Drug-induced
2. Defects in hemoglobin – Quantitative: Thalassemias – Qualitative: Sickle-cell disease; Hemoglobin D, E, or C disease	**2. Mechanical hemolytic anemia** – Microangiopathic – Cardiac – March hemoglobinuria
3. Defects in enzymes – Glucose-6-phosphate dehydrogenase deficiency – Pyruvate kinase deficiency	**3. Direct action of physical, chemical, or infectious agents**
	4. Hypersplenism
Anemias due to excess blood loss	

Table 4: Etiological classification of anemia

DISCUSSION

The discussion on red blood cell indices revolves around erythropoiesis, the size, shape and content of RBCs, metabolism, oxygen affinity to hemoglobin, measurement and interaction of RBC indices, their predictive value and their clinical applications.

The physiological process of formation of blood cells is hematopoiesis. This proceeds through three stages: the mesoblastic stage, the hepatic stage and the myeloid (medullary) stage. Primitive hematopoiesis begins in the extra-embryonic yolk sac during the third week of embryonic life and continues until approximately the third month of intrauterine life. The hepatic stage extends from approximately the second month of gestation until birth. Some hematopoiesis also occurs in the spleen, particularly from the third to the seventh month of the prenatal period.[1]

The hierarchy of hematopoiesis begins with the pluripotent hematopoietic stem cell, which gives rise to the myeloid stem cell. The erythroid lineage subsequently develops through erythroid progenitors, including burstforming unit-erythroid (BFU-E) and colony-forming unit-erythroid (CFU-E), before morphologically recognizable erythroid precursors appear. The erythroid lineage proceeds towards erythroid precursor cells, while the hematopoietic stem/progenitor system also gives rise to the platelet lineage and individual leukocyte lineages.

The existence of hematopoietic microenvironments is suggested by the fact that formation of blood cells is restricted to bone marrow, especially in flat bones such as the sternum, ribs and skull, and in the proximal parts of long bones.

Erythropoiesis is the proliferation, survival and differentiation of erythroid precursors within adult bone marrow.[2] Hepcidin and erythropoietin are two important hormones involved in maintaining hemoglobin homeostasis.[3]

Hepcidin regulates iron availability through a negative-feedback mechanism involving dietary iron absorption and mobilisation of iron from body reserves.[4]

Erythropoietin (EPO) directly stimulates the bone marrow. Its synthesis is upregulated by reduced tissue oxygen availability. During pregnancy, EPO is also produced by the placenta, particularly during the second trimester. Higher erythropoietin concentrations among obese pregnant women have been proposed to relate to the antiinflammatory and cytoprotective role of EPO against hypoxia-induced inflammation in adipose tissue.[5]

The stages of development of erythropoiesis include proerythroblast, basophilic erythroblast, polychromatophilic erythroblast, orthochromatic erythroblast, reticulocyte and finally the mature red blood cell.

Mature RBCs are approximately the size of a mature lymphocyte nucleus. The erythrocyte lacks a nucleus and has a central pallor of approximately one-third of the cell diameter. Younger or immature RBCs are larger in size and earlier erythroid precursors contain nuclei.

Iron, vitamin B12 and folic acid are essential for proper erythropoiesis. Erythropoiesis is the major consumer of iron in the body. Iron combines with protoporphyrin in the mitochondria of late-stage erythroblasts as the final step in the formation of heme, the oxygen-carrying core of hemoglobin.

Division, differentiation and maturation of red-cell precursors from early to late stages are dependent on adequate iron availability. Modulation of the erythroid response to erythropoietin by erythroid precursors is also influenced by iron.

The surface of an erythrocyte is supported by a condensed cytoskeletal stroma beneath the membrane. Reduced hemoglobin content is associated with reduced cell size; therefore, defective hemoglobin synthesis can result in microcytosis, with RBCs measuring less than 80 fL.

Similarly, in folate deficiency, the late erythroblast persists as a megaloblast and is unable to divide normally, resulting in the formation of macrocytes.[6]

In 1929, Wintrobe first introduced the terms mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH) and mean corpuscular hemoglobin concentration (MCHC) to define the size and hemoglobin content of red cells.

Red cell indices can be calculated when the values of hemoglobin (Hb), hematocrit/packed cell volume (PCV) and RBC count are known. Variation in red-cell size can be quantified and expressed as RDW (red cell distribution width.[7]

MCV defines the size of red cells and is expressed in femtolitres (fL), equivalent to cubic micrometres (μm^3). The normal value is approximately 87 ± 7 fL.

MCV = [PCV (%) × 10] / [RBC count (millions/µL)]

MCH quantifies the amount of hemoglobin per red cell. The normal value is approximately 29 ± 2 pg.

MCH = [Hemoglobin (g/dL) × 10] / [RBC count (millions/µL)]

MCHC indicates the amount of hemoglobin per unit volume of packed red cells. The normal value is approximately 34 ± 2 g/dL.

MCHC = [Hemoglobin (g/dL) × 100] / [PCV (%)]

RDW represents the coefficient of variation of red-cell volume distribution and is expressed as a percentage. The normal value is approximately 13 ± 1.5%.

During erythropoiesis, erythroid maturation involves progressive condensation of nuclear chromatin and finally extrusion of the nucleus from the cell. Cytoplasmic maturation is characterized by hemoglobin synthesis. Nuclear extrusion, cell division and water loss result in a progressive reduction in cell size from a large normoblast to the mature erythrocyte.

Defects in nuclear maturation, as seen in folate or vitamin B12 deficiency, result in megaloblastic anemia with large oval erythrocytes (macro-ovalocytes), often with a relatively preserved hemoglobin concentration per cell.

In macrocytosis, MCV and MCH are increased while MCHC may remain normal. Anisocytosis is present and RDW is increased. In liver disease, macrocytosis may occur without a defect in nuclear maturation; the cells are large and round due to excess red-cell membrane, and RDW may remain normal.

Defective hemoglobin synthesis results in small cells (low MCV), i.e. microcytosis. In heterozygous thalassemia, MCV is low but RDW may remain normal because there is relatively uniform microcytosis. In iron deficiency anemia, MCV is reduced and RDW is increased because of greater variation in red-cell size. The rise in RDW may precede marked microcytosis.

When nuclear maturation is abnormal and cell division is delayed, cell size increases despite impaired effective erythropoiesis, leading to macrocyte formation.

When there is defective or delayed synthesis of hemoglobin, continued cell division can lead to microcytosis. Red-cell indices are valuable in the morphological classification of anemias. Different etiological factors produce characteristic differences in red-cell morphology, structure and function. Anemias can therefore be classified morphologically according to RBC size and according to the reticulocyte response.

Anemia is defined as a reduction in the concentration of circulating hemoglobin, or the oxygen-carrying capacity of blood, below the level expected for healthy persons of the same age, sex and environment.

Morphological classification according to MCV is broadly divided into microcytic, normocytic and macrocytic anemia. MCV and RDW are useful together in the evaluation of anemia because they provide information about red-cell size and anisocytosis. RDW may become elevated before a major change in MCV in early iron deficiency.

Reticulocytes are young red blood cells that contain residual RNA remnants. The reticulocyte count is calculated from the number of reticulocytes divided by the number of red cells and multiplied by 100, giving the reticulocyte percentage. This assesses erythropoietic activity of the bone marrow in anemia.

The number of reticulocytes counted among 1000 red cells is expressed as a percentage. The normal reticulocyte count is approximately 0.5–2.5%; in newborns, the count is higher, approximately 2–5%

Another approach is the etiological classification of anemia. Anemia may occur because of impaired red-cell production, excessive red-cell destruction (hemolysis), or excessive blood loss.

Several metabolic pathways exist in the red cell for its structural maintenance and oxygen carrying functions. The Embden-Meyerhoff pathway is an anaerobic glycolysis pathway that generates 90% of ATP to provide energy for reactions like maintenance of membrane integrity, regulation of intra- and extracellular pumps, and maintenance of hemoglobin function. The Rapoport-Luebering shunt involves synthesis of 2,3-diphosphoglycerate (2,3-DPG), which is a determinant of the oxygen affinity of hemoglobin. The hexose monophosphate shunt provides 5 to 10% of ATP and protects hemoglobin from oxidant damage. This shunt is mediated by the enzyme glucose-6-phosphate dehydrogenase. Methhemoglobin reductase pathway maintains hemoglobin in the ferrous state by reducing NAD to NADH and prevents accumulation of methemoglobin in the red cell. In sepsis and acidosis, the anaerobic glycolysis is enhanced and the final product of the EM pathway, lactate, is increased, serving as a marker and indicator of acidosis. On the contrary, RL shunt increases 2,3-DPG synthesis to release more oxygen to the tissues to prevent hypoxia.

The red cell membrane is composed of lipids, complex network of proteins and small amount of carbohydrates. The membrane lipids include phospholipid, cholesterol and glycolipids. The red cell membrane proteins are either transmembranous or submembranous and constitute the cytoskeleton of the red cell. The membrane provides mechanical strength and flexibility to red cell to withstand the shearing forces of circulation. A cation pump maintains the red cell volume. This is affected in microcytic or macrocytic anemia.

Regarding the fate of RBCs, it is essential to understand the process of increased RBC destruction. The senile red cells are recognised by macrophages of reticuloendothelial system and destroyed by the spleen. Globin is converted to amino acids, heme is converted to iron and porphyrin. Iron is stored as ferritin or released to be taken up by transferrin and transported to bone marrow. Porphyrin is converted to bilirubin.

In hemolysis, the cell membrane integrity is lost; red cells are unable to withstand the shearing forces of circulation and excessive destruction. There is premature destruction of RBCs.

A normocytic normochromic anemia may still be present with iron deficiency. Erythropoiesis may lead to a microcytic anemia. A microcytic hypochromic anemia may be an iron deficiency anemia, anemia of chronic disease or β-thalassemia. RBC count is normal in β-thalassemia and iron-binding capacity and ferritin levels are all unaffected.

RDW may be normal in microcytic anemia of chronic disease and iron binding capacity is reduced in chronic disease while it is increased in iron deficiency anemia.[8]

Apart from MCV and RDW, which are sensitive red cell indices for iron deficiency anemia, a low serum ferritin, low serum iron and transferrin saturation, increased total iron binding capacity, increased soluble transferrin receptor have been advocated in the diagnosis of iron deficiency anemia.[9] The amount of iron needed by the patient is derived from the formula:
Body weight × 2.3 × Hb deficit + 500 mg.

Macrocytic anaemias commonly occur due to folic acid or B12 deficiency. Folic acid occurs as polyglutamates in nature. Conversion of polyglutamate to the active form is tetrahydrofolate, which is essential to participate in metabolic reactions. Folate is incorporated into red cells during erythropoiesis and its level remains constant throughout the life span of RBCs. A low red cell folate (<4 µg/dL) indicates megaloblastic anemia due to folate deficiency. About 50% of patients with B12 deficiency also have reduced folate levels.

Hence treatment should be a combined supplementation of both vitamins.[10] High-dose folate treatment with folate alone may precipitate subacute combined degeneration of spinal cord. Vitamin B12 deficiency caused neurological manifestations and was originally called 'pernicious' anemia as it was fatal. This vitamin B12 deficiency is known by several names such as pernicious anemia, Addisonian anemia, after Thomas Addison who described it in 1849 and intrinsic factor deficiency by William B Castle.

Vitamin B12 is the co-enzyme for two reactions: 1) synthesis of methionine from homocysteine and 2) conversion of methylmalonyl CoA to succinyl CoA. Elevation of methylmalonic acid is specific for B12 deficiency. Thus the megaloblastic anemia is diagnosed as macrocytic anemia with low Hb, raised MCV, reduced MCH, and raised RDW.[11] A malabsorption or malabsorption state elicits a mixed picture in the peripheral smear often called dimorphic anemia. Thus it is evident that apart from hemoglobin and packed cell volume, other RBC indices such as MCV, MCH, MCHC and RDW direct diagnosis and treatment.

CONCLUSION

Red cell indices are valuable in the morphological classification of anemias and helpful in planning the management as different etiological factors depict as different characteristic morphology. Electronically determined MCHC is normal in microcytic anemias but MCV and MCH are reduced with raised RDW (PL<0.005). Hemoglobin and packed cell volume are no doubt of foremost consideration but this study and discussion clarifies the importance and contribution of other red cell indices of size and content of the red cells namely MCV, MCH, RDW and MCHC which could direct the plan of etiology and management by pinpointing the diagnosis of anemias.

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