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A REVIEW: PAROXYSMAL NOCTURNAL HEMOGLOBINURIA, APLASTIC ANEMIA, PANCYTOPENIA AND MYELODYSPLASTIC SYNDROMES: CLINICAL SIGNIFICANCE OF LYMPHOCYTOSIS, ELEVATED LDH AND MULTILINEAGE CYTOPENIAS

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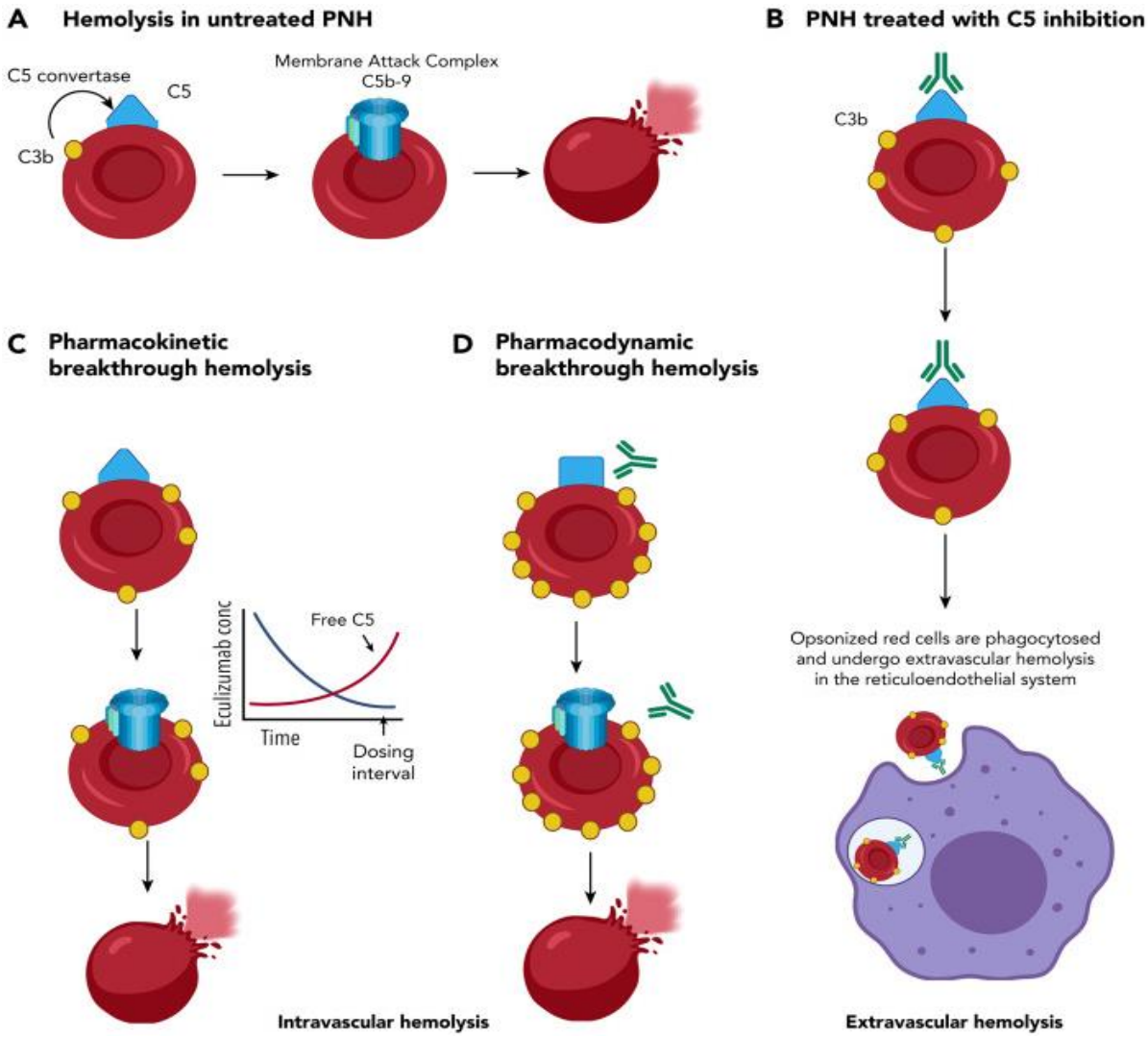
ABSTRACT

Paroxysmal nocturnal haemoglobinuria (PNH), aplastic anaemia (AA), pancytopenia and myelodysplastic syndromes (MDS) represent important and biologically interconnected disorders of haematopoiesis. Although these conditions differ in their primary mechanisms, considerable clinical and molecular overlap exists, particularly at the level of hematopoietic stem and progenitor cells. PNH results from an acquired somatic defect affecting glycosylphosphatidylinositol (GPI)-anchored proteins, leading to complement-mediated destruction of susceptible blood cells. Aplastic anaemia is predominantly an immune-mediated bone-marrow failure syndrome, whereas MDS represents a heterogeneous group of clonal myeloid neoplasms characterized by ineffective and dysplastic haematopoiesis. Pancytopenia may be a common presenting laboratory manifestation of these disorders but is not itself a diagnosis. Elevated lactate dehydrogenase (LDH), particularly when associated with indirect hyperbilirubinemia, reticulocytosis and reduced haptoglobin, is an important biochemical marker of haemolysis and can be markedly elevated in PNH. Conversely, cytopenias caused by impaired marrow production may produce a different laboratory pattern. Lymphocytosis requires careful interpretation because relative lymphocytosis can occur simply as a consequence of neutropenia or leukopenia, while absolute and persistent lymphocytosis may reflect reactive infection, autoimmune disease or lymphoproliferative disorders. Contemporary diagnosis therefore requires integration of complete blood counts, peripheral blood morphology, haemolysis markers, high-sensitivity flow cytometry using FLAER and GPI-linked markers, bone-marrow morphology, cytogenetics and, where appropriate, molecular studies. This review summarizes the current understanding of these interconnected conditions and emphasizes a systematic approach to patients presenting with unexplained pancytopenia, lymphocyte abnormalities and elevated LDH.

KEYWORDS: Paroxysmal Nocturnal Haemoglobinuria, Pnh, Aplastic Anaemia, Pancytopenia, Myelodysplastic Syndrome, Mds, Ldh, Lymphocytosis, Flaer, Haemolysis, Bone Marrow Failure.

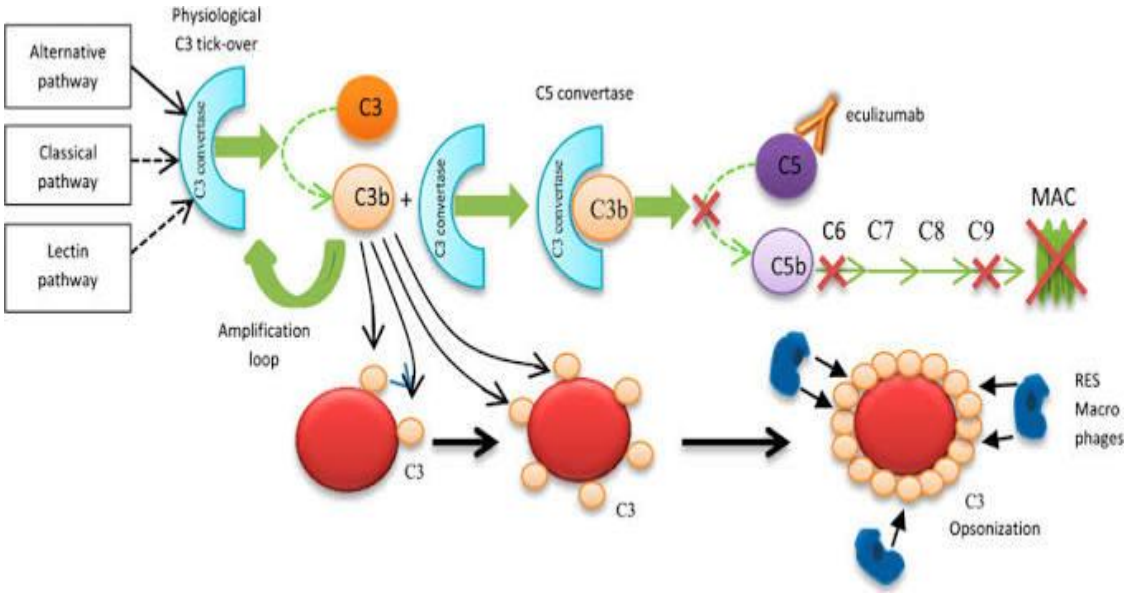
INTRODUCTION

The hematopoietic system maintains a continuous supply of erythrocytes, leukocytes and platelets through highly regulated activity of hematopoietic stem and progenitor cells (HSPCs). Disturbances in stem-cell survival, differentiation, immune regulation or clonal selection may result in anaemia, leukopenia, thrombocytopenia or pancytopenia.



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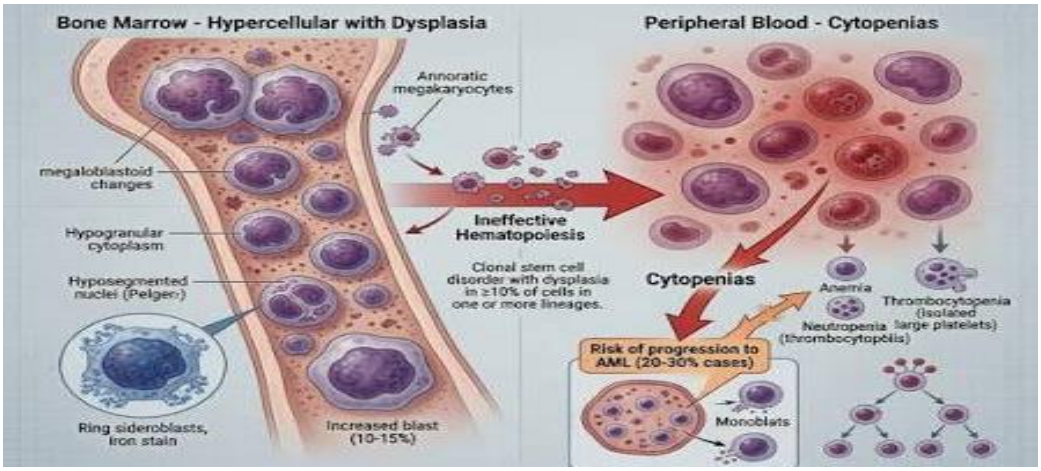
PNH, AA and MDS are particularly important because they may share clinical manifestations while having different underlying mechanisms. PNH is a clonal hematopoietic disorder caused by acquired abnormalities in the PIGA gene, resulting in deficiency of GPI-anchored proteins including CD55 and CD59. Their absence makes affected cells susceptible to complement-mediated destruction.



Representative Images

Aplastic anaemia is primarily characterized by failure of hematopoietic stem and progenitor cells, often through immune-mediated mechanisms, resulting in hypocellular bone marrow and peripheral blood cytopenias. PNH clones are frequently associated with acquired aplastic anaemia, supporting a biological relationship between the two disorders.

MDS, in contrast, comprises clonal myeloid neoplasms characterized by ineffective haematopoiesis, cytopenias and morphologic dysplasia, with variable risk of progression to acute myeloid leukaemia (AML).



Representative Images

Understanding these relationships is particularly important when a patient presents with the combination of pancytopenia, markedly elevated LDH and a detectable PNH clone.

Objectives of the Review

This literature review aims to

1. Describe the pathophysiological relationship between PNH, AA and MDS.
2. Explain the causes and clinical significance of pancytopenia.
3. Review the significance of markedly elevated LDH in PNH and other haematological conditions.
4. Discuss relative versus absolute lymphocytosis and its differential diagnosis.
5. Review the role of FLAER-based flow cytometry in PNH diagnosis.
6. Discuss the diagnostic distinction between aplastic anaemia and MDS.
7. Examine clonal evolution and the relationship between AA, PNH and secondary MDS/AML.
8. Provide a practical framework for laboratory and clinical evaluation.

METHODOLOGY

This is a narrative literature review based on peer-reviewed haematology literature and authoritative

medical sources. Literature was identified through searches of PubMed/PMC and major haematology publications using combinations of the terms:
"paroxysmal nocturnal hemoglobinuria," "PNH," "FLAER," "aplastic anemia," "pancytopenia," "myelodysplastic syndrome," "MDS," "LDH," "hemolysis," "lymphocytosis," "bone marrow failure," and "clonal evolution."
Priority was given to review articles, consensus literature and diagnostic studies addressing the relationship between PNH, AA and MDS.

Pathophysiology of PNH
PNH arises from an acquired somatic mutation involving the PIGA gene in a hematopoietic stem cell. PIGA is necessary for synthesis of the GPI anchor that attaches numerous proteins to the cell membrane.

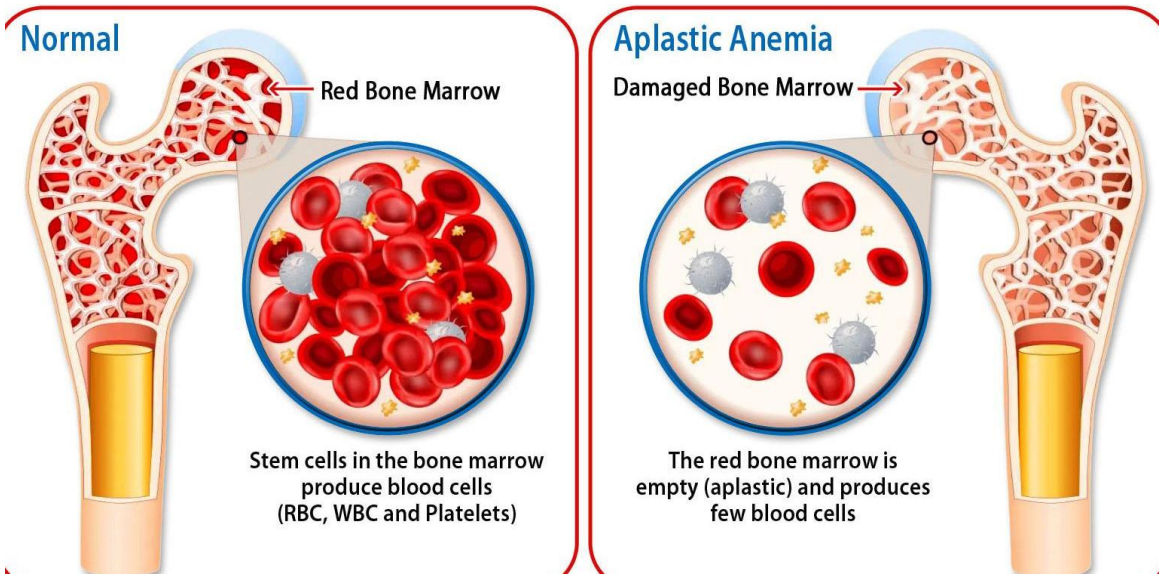
Important GPI-anchored complement-regulatory proteins include
* CD55
* CD59
CD55 contributes to regulation of complement activation, while CD59 protects cells from formation of the membrane attack complex.
Loss of these protective mechanisms makes PNH cells vulnerable to complement-mediated destruction.

The disease therefore has three major clinical dimensions
Haemolysis
Complement-mediated intravascular destruction of RBCs.

Thrombosis
PNH is associated with potentially severe thrombotic complications.

Bone-marrow failure
Some patients predominantly present with cytopenias related to associated marrow failure rather than overt haemolysis.
This clinical heterogeneity is an important feature of PNH.

PNH and Aplastic Anaemia
The association between PNH and AA is well established.
Acquired aplastic anaemia is generally considered an immune-mediated bone-marrow failure syndrome involving destruction or suppression of hematopoietic stem and progenitor cells by aberrant immune mechanisms.



Representative Images

PNH clones can emerge in this environment. One proposed explanation is that GPI-deficient stem cells may possess a relative survival advantage under immune pressure, allowing them to expand clonally.
Thus:
Immune-mediated marrow injury → suppression of normal hematopoietic cells → relative survival/expansion of PNH clone
This model helps explain why PNH clones are frequently detected in patients with aplastic anaemia.

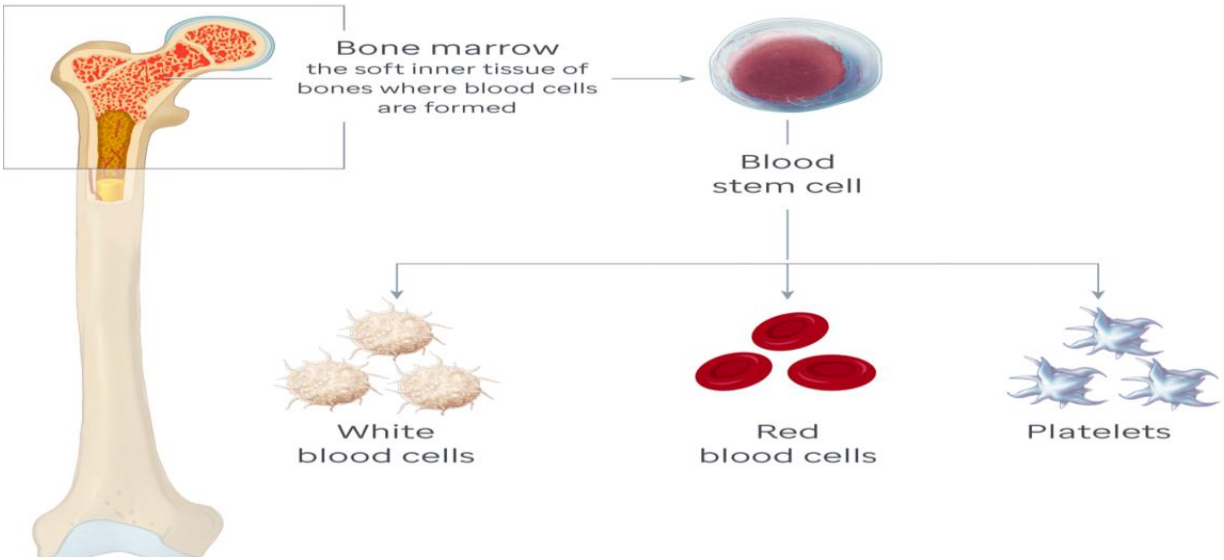
The clinical phenotype can range from
Small/subclinical PNH clone + marrow failure
to
Large PNH clone + overt haemolysis ± thrombosis.
Recent consensus literature describes small PNH clones as commonly occurring in patients with bone-marrow failure, while larger clones are more frequently associated with classical haemolytic PNH and increased thrombotic risk.

Pancytopenia

Pancytopenia refers to reduction of all three major peripheral blood cell lineages.

Haemoglobin/RBC ↓ + WBC ↓ + Platelets ↓

It is a laboratory finding rather than a specific diagnosis.



Representative Images

Important causes include

Bone-marrow failure

- Aplastic anaemia
- MDS
- Acute leukaemia
- Marrow infiltration
- Chemotherapy/radiotherapy
- Certain drugs and toxins

Nutritional disorders

- Vitamin B12 deficiency
- Folate deficiency
- Severe nutritional deficiency
- Copper deficiency in selected circumstances

Infections

- Viral infections
- HIV
- Hepatitis viruses
- Parvovirus B19
- Other systemic infections

Immune-mediated disorders

- Peripheral destruction/sequestration
- Hypersplenism
- Certain autoimmune conditions

MDS literature emphasizes that secondary causes of cytopenia must be carefully excluded before assigning a diagnosis of MDS.

Clinical Consequences of Individual Cytopenias

Anaemia

Reduction in haemoglobin decreases oxygen-carrying capacity and may produce

- Fatigue
- Weakness
- Pallor
- Exercise intolerance
- Dyspnoea
- Dizziness
- Tachycardia

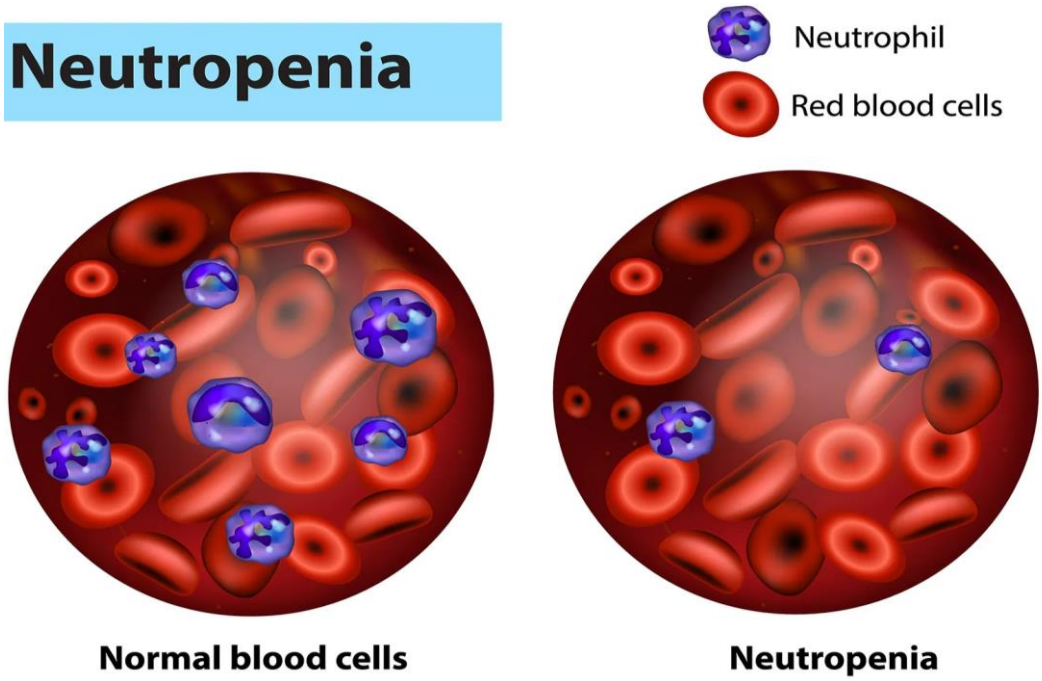
In PNH, anemia may result from both hemolysis and bone-marrow failure.

Leukopenia and Neutropenia

Reduced neutrophil numbers can compromise host defence.

Patients may develop

- Recurrent infections
- Fever
- Oral ulcers
- Respiratory infections
- Skin infections
- Severe systemic infection in profound neutropenia



Representative Images

The absolute neutrophil count (ANC) is therefore more clinically informative than total WBC count alone.

Thrombocytopenia
Low platelet counts can cause

- Petechiae
- Purpura
- Easy bruising
- Epistaxis
- Gum bleeding
- Menorrhagia
- Gastrointestinal or urinary bleeding

Severe thrombocytopenia can result in life-threatening haemorrhage.



Representative Images

LDH as a Marker of Hemolysis
LDH is a nonspecific marker of tissue injury but is particularly useful in evaluating hemolysis.
In PNH, intravascular RBC destruction releases LDH into the circulation, potentially producing marked elevation.

A more convincing biochemical pattern of haemolysis includes

- LDH ↑
- Indirect bilirubin ↑
- Reticulocytes ↑
- Haptoglobin ↓

The magnitude and trend of LDH can therefore help clinicians assess the activity of haemolysis in PNH.

Classical diagnostic approaches to PNH specifically recommend assessment of LDH, bilirubin, haptoglobin and reticulocyte count together with flow cytometry.

However
LDH elevation is not specific for PNH.

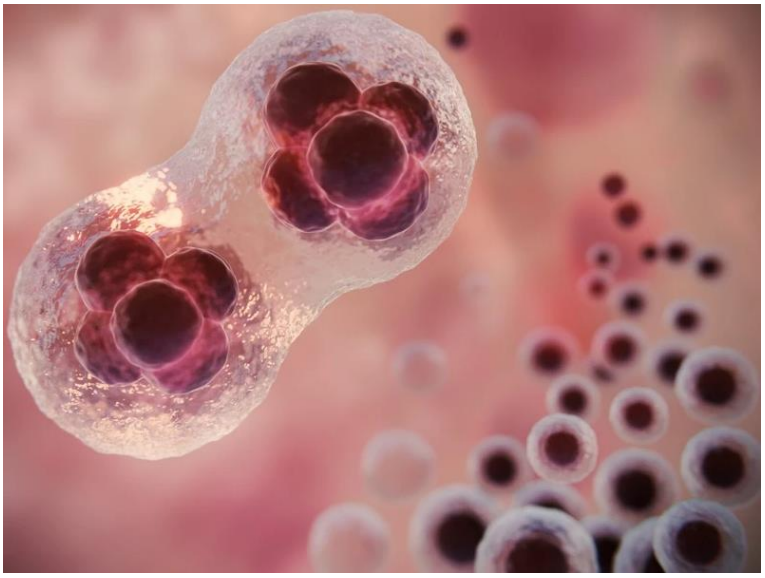
Other causes include
- Megaloblastic anaemia
- Haemolytic anaemia
- Liver disease
- Muscle injury
- Tissue ischemia
- Infection
- Malignancy
- Leukaemia/lymphoma

Therefore, LDH should always be interpreted in clinical context.

Lymphocytosis
Lymphocytosis can be either
Relative lymphocytosis
The percentage of lymphocytes increases because another leukocyte population, particularly neutrophils, decreases.

Absolute lymphocytosis
The absolute lymphocyte count itself is increased.
This distinction is especially important in patients with pancytopenia.

For example
WBC ↓ + neutrophils ↓ + lymphocyte percentage ↑
does not necessarily mean that lymphocytes are increased in absolute terms.



Representative Images

Therefore, interpretation should include
- Total WBC
- Absolute lymphocyte count
- Absolute neutrophil count
- Peripheral smear

Causes of Lymphocytosis
Common reactive causes include
Viral infections
- EBV
- CMV
- Influenza
- COVID-19
- Viral hepatitis
- Other viral infections

Certain bacterial infections
Pertussis and some chronic infections may produce lymphocytosis.

Post-infectious state
Lymphocyte elevation may persist temporarily after resolution of an infection.

Autoimmune/inflammatory disorders
Some systemic inflammatory and autoimmune diseases can be associated with lymphocyte abnormalities.



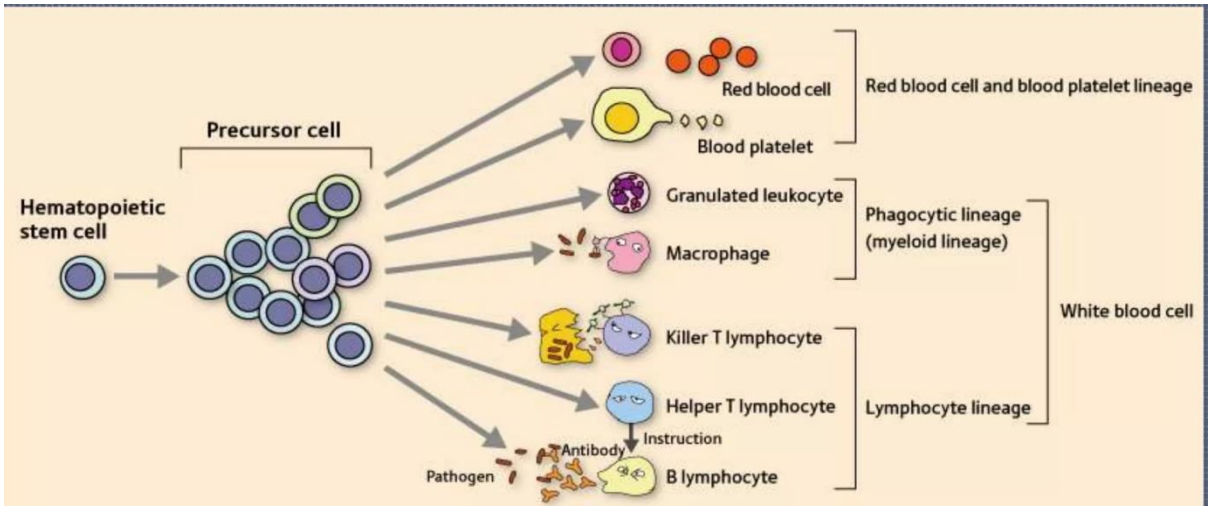
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Haematological disorders

Persistent absolute lymphocytosis without an adequate reactive explanation requires evaluation for:

- Chronic lymphocytic leukaemia
- Lymphoproliferative disorders
- Certain lymphomas/leukaemia's

Peripheral blood flow cytometry can be important when a clonal lymphoid disorder is suspected.



Representative Images

MDS and Its Relationship With Pancytopenia

MDS represents a heterogeneous group of clonal hematopoietic disorders characterized by ineffective haematopoiesis and dysplasia.

Common laboratory manifestations include

- Anaemia
- Neutropenia
- Thrombocytopenia
- Pancytopenia

More than 90% of patients with MDS develop anaemia in reported series, and approximately half may present with pancytopenia.

However, cytopenia alone is not diagnostic of MDS.

The diagnosis requires integration of Clinical history

- CBC
- Peripheral smear
- Bone-marrow morphology
- Blast percentage
- Cytogenetics
- Molecular studies when appropriate
- Exclusion of alternative causes

Bone-marrow examination remains central to the diagnostic process.

Distinguishing Aplastic Anaemia From MDS

This is one of the most challenging areas in bone-marrow failure.

Aplastic anaemia
Typically characterized by
- Pancytopenia
- Markedly hypocellular bone marrow
- Reduced hematopoietic precursors
- Absence of significant dysplasia as the primary defining feature

MDS
Typically characterized by
- Persistent cytopenias
- Dysplastic haematopoiesis
- Clonal abnormalities
- Variable blast increase
- Abnormal cytogenetic/molecular findings

Some cases, especially hypoplastic MDS, may resemble aplastic anaemia. Therefore, careful morphological, cytogenetic and molecular assessment is required.

Clonal Evolution in Aplastic Anaemia and PNH
Modern sequencing has demonstrated that clonal haematopoiesis is an important component of acquired aplastic anaemia.
Some clones may represent adaptive survival under immune selection, whereas other genetic alterations can be associated with progression toward MDS/AML.
Studies have demonstrated a long-term risk of secondary MDS/AML following acquired AA. Risk is influenced by factors including disease duration, telomere biology and particular somatic mutations.
This emphasizes the importance of long-term surveillance rather than considering successful initial treatment as the end of clinical monitoring.

Diagnostic Role of FLAER Flow Cytometry
Flow cytometry is central to modern PNH diagnosis.
FLAER (fluorescently labelled aerolysin) binds to GPI anchors and therefore identifies cells with intact GPI-anchor expression. PNH leukocytes deficient in GPI-anchored proteins show reduced/absent FLAER binding.

Assessment commonly includes
- Granulocytes
- Monocytes
- RBCs

and GPI-linked markers such as
- CD55
- CD59
- CD14
- CD16
- CD24

The use of multiple lineages improves diagnostic confidence.
Importantly, RBC clone size can underestimate the underlying PNH clone because PNH RBCs are preferentially destroyed during haemolysis. Consequently, granulocyte and monocyte measurements can be particularly informative.

Interpretation of a Patient With a Large PNH Clone and High LDH
A patient demonstrating:
- Significant PNH clone on FLAER-based flow cytometry
- Markedly elevated LDH
- Anaemia
- Possible pancytopenia

requires evaluation for both
Haemolytic PNH
and
PNH associated with bone-marrow failure.
The distinction is clinically important because the therapeutic priorities differ.
A patient with active complement-mediated haemolysis requires assessment for complement directed treatment, whereas a patient with predominant marrow failure requires detailed assessment of aplastic anaemia/MDS and marrow reserve.
Modern reviews emphasize that PNH is clinically heterogeneous and that some patients have predominantly haemolytic disease while others have predominantly marrow failure.

Clinical Diagnostic Algorithm
A practical approach can be summarized as

Unexplained anemia / pancytopenia
↓
CBC + differential + reticulocyte count
↓
Peripheral blood smear
↓
Evaluate

Production failure?
→ Bone marrow examination

Haemolysis?
→ LDH + bilirubin + haptoglobin + reticulocytes

Possible PNH?
→ High-sensitivity FLAER-based flow cytometry

Possible MDS?
→ Bone marrow morphology + cytogenetics ± molecular testing

Persistent absolute lymphocytosis?
→ Peripheral smear + lymphoid flow cytometry where indicated

Possible nutritional/infectious causes?
→ Appropriate biochemical and infectious investigations
This integrated approach is more reliable than interpreting any single laboratory parameter.

Therapeutic Implications
Treatment must be individualized according to disease phenotype.

For clinically significant PNH with complement-mediated haemolysis, complement inhibition is an established therapeutic strategy. Modern management has therefore shifted from purely supportive treatment toward targeted inhibition of complement activation.
For aplastic anaemia, treatment depends on disease severity, age, donor availability and patient-specific factors and may include hematopoietic stem-cell transplantation or immunosuppressive approaches.
MDS treatment is determined by risk category, cytogenetic/molecular characteristics, age, comorbidities and transplant eligibility.

Supportive measures may include
- Red-cell transfusion when clinically indicated
- Platelet transfusion when indicated
- Infection management
- Appropriate nutritional correction
- Iron assessment
- Monitoring for renal complications
- Thrombosis assessment in PNH

DISCUSSION
The literature demonstrates that PNH, AA and MDS should not always be viewed as completely separate entities. They occupy an overlapping biological spectrum of hematopoietic stem-cell disorders.

The relationship can be conceptualized as

Immune-mediated marrow injury
↓
Selective survival of hematopoietic clones
↓
PNH clone / clonal haematopoiesis
↓

Depending on biological characteristics
Marrow failure → AA phenotype
or
Complement-mediated haemolysis → Classical PNH phenotype
or
Clonal evolution/dysplasia → MDS phenotype
These pathways may coexist within the same patient.
This is why a patient with pancytopenia and a PNH clone should not automatically be categorized as having "pure PNH." Likewise, a patient with a PNH clone and marrow failure should be evaluated for aplastic anaemia, and persistent unexplained dysplasia should prompt consideration of MDS.

Clinical Significance of the Laboratory Triad
A particularly important clinical combination is
Pancytopenia + high LDH + PNH clone

This combination should raise three simultaneous questions
Question 1

Is there active haemolysis?
Assess
- LDH
- Indirect bilirubin
- Haptoglobin
- Reticulocytes
- Plasma-free haemoglobin

Question 2

Is there significant bone-marrow failure?
Assess
- ANC
- Platelets
- Reticulocyte production
- Bone-marrow cellularity

Question 3

Is there clonal evolution or an alternative marrow disorder?
Assess
- Bone marrow morphology
- Cytogenetics
- Molecular studies
- Clinical course

This three-dimensional assessment provides a more accurate picture than simply labelling the patient "PNH positive."

Limitations of Current Literature
Several important limitations remain.
First, PNH is rare, and many studies involve relatively small cohorts. Second, PNH clone size does not perfectly predict clinical phenotype. Third, molecular abnormalities in AA and MDS may have different biological meanings depending on the mutation, clone size and timing of acquisition. Fourth, cytopenias are nonspecific and may arise from nutritional, infectious, immune, malignant or treatment-related causes.
Consequently, no single laboratory parameter should be interpreted in isolation.

Future Research Directions
Future research should focus on
1. Better molecular characterization of PNH/AA/MDS overlap.
2. Identification of biomarkers predicting PNH clone expansion.
3. Better prediction of progression from AA/PNH to MDS/AML.
4. Integration of NGS with flow cytometry and marrow morphology.
5. Long-term monitoring strategies for small PNH clones.
6. Identification of patients most likely to benefit from complement inhibition.
7. Understanding immune mechanisms responsible for clonal selection.
8. Development of individualized risk-stratification models.

9. Evaluation of quality-of-life outcomes in chronic PNH.
10. Improved approaches for differentiating hypoplastic MDS from aplastic anaemia.

CONCLUSION

PNH, aplastic anaemia, pancytopenia and MDS represent clinically and biologically interconnected disorders of haematopoiesis. PNH is characterized by acquired GPI-anchor deficiency and complement-mediated haemolysis, whereas aplastic anaemia is predominantly a marrow-failure disorder and MDS is a clonal dysplastic hematopoietic neoplasm.

Pancytopenia is a manifestation rather than a diagnosis, and its underlying cause must be systematically established. Similarly, LDH elevation is a marker of cellular injury and, in the appropriate context, haemolysis but is not specific for PNH. Lymphocytosis should be differentiated into relative and absolute lymphocytosis, with persistent absolute lymphocytosis requiring appropriate evaluation for reactive and clonal causes.

FLAER-based flow cytometry has become a central diagnostic technique for PNH, while bone-marrow morphology, cytogenetics and molecular testing remain essential in evaluating suspected aplastic anaemia and MDS.

The modern approach should therefore integrate:

CBC + differential + reticulocyte count + peripheral smear + haemolysis profile + FLAER flow cytometry + bone-marrow examination + cytogenetic/molecular assessment where indicated.

Such an integrated strategy facilitates earlier diagnosis, appropriate risk assessment, recognition of PNH/AA/MDS overlap and individualized treatment planning.

Biography

Dr. A. K. Dwivedi, BHMS (Gold Medallist), MD, MBA, Ph.D. has been a Registered Homeopaths for over 30 years. He is a Professor & HOD: Department of Physiology S.K.R.P. Gujarati Homoeopathic Medical College, Indore. He is a Member of Executive Council, Devi Ahilya Vishwavidyalaya Indore, MP, INDIA, he is also a Member Central Ethical committee (CCRH) Ministry of Ayush, Govt of India. Member, Scientific Advisory Committee (NEAH) Shilong Meghalaya, Ministry of Ayush, Govt. of India, Member Academic Board Madhya Pradesh Medical Science University, Jabalpur MP (India). DIRECTOR,& CEO Advanced Homeo Health Centre & Homeopathic Medical Research Pvt.Ltd. Indore, Madhya Pradesh, India, EDITOR,"SEHAT EVAM SURAT"(Hindi Monthly Medical Magazine).

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