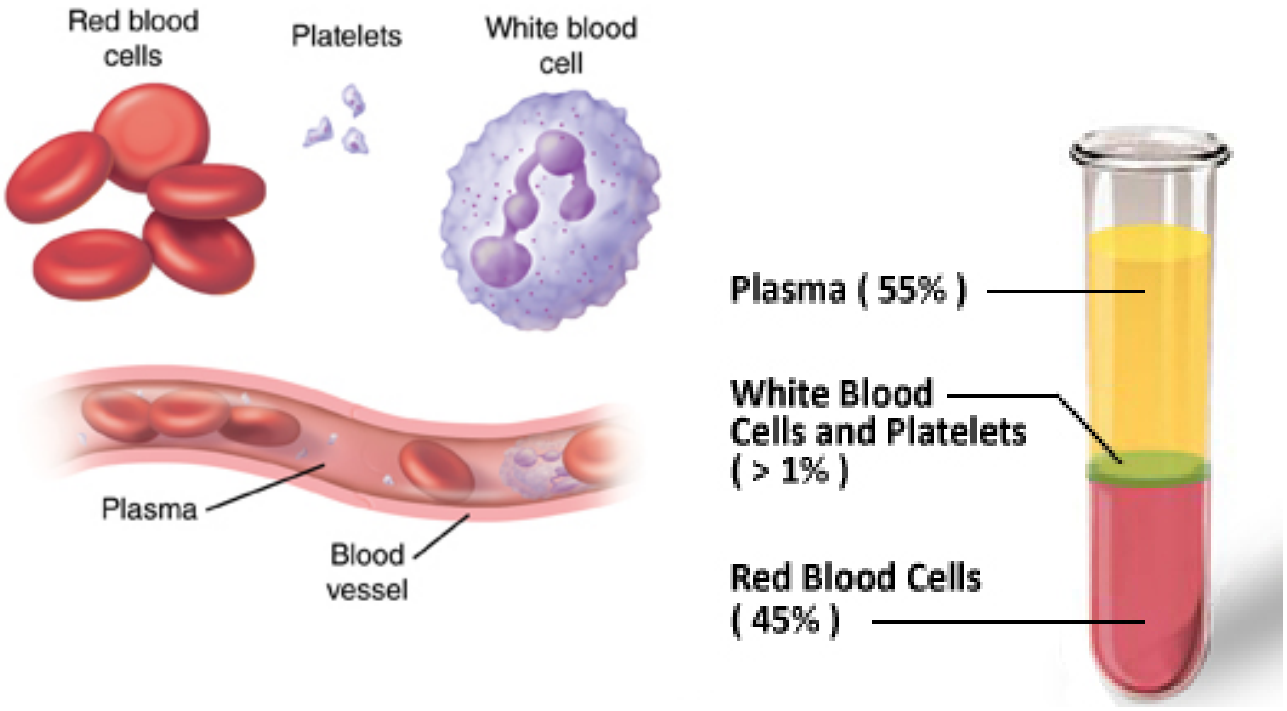
AJK Medical College, Muzaffarabad

STUDY GUIDE

Hematology Module

4th Year



Module Code: 0303
Duration: 2 Weeks
Starting on:

DEPARTMENT OF MEDICAL EDUCATION

CONTENTS

Module Team

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Rationale

Blood is a wonderful hybrid of cellular and fluid components. All the components are highly specialized in structure and perform very important role in delivering nutrients, removing waste products, regulating body pH and temperature, body defense and hemostasis.

The newborn has a unique hematopoietic profile that evolves with maturation. Moreover physiology of pregnancy stresses the hematopoietic system. We will look at how these factors influence the interpretation of laboratory investigations.

Host defense depends on specialized blood cells that identify foreign microorganisms/molecules and utilize oxidants, proteases, and other moieties to protect the host. We will see how these functions can go wrong and what affects they produce on human body.

Bone marrow is the factory for production of cellular blood components. The consequences of bone marrow failure leading to pancytopenia include anemia, infection propensity and bleeding. The students will learn, how these effects are produced and what management principles are?

The acute leukemias are aggressive malignancies that originate in a hematopoietic stem cell and are rapidly fatal without immediate treatment. Moreover there are clonal hematopoietic stem cell disorders in which the progenitor cells of one or more of the hematopoietic lineages proliferate excessively. Similarly erythrocytosis, or an increase in hematocrit, can be caused by a variety of disorders. Stem cell transplantation is a treatment options for many patients with hematologic diseases. Students will learn how these disorders occur and go through modalities of management.

Activation of the coagulation system is essential to keep us from bleeding to death from minor trauma. On the other hand blood coagulation is a potentially explosive process that could, in the absence of precise regulation, quickly lead to massive thrombosis and death.

Patients with a wide variety of inherited and acquired bleeding disorders are encountered in nearly all subspecialties of medicine.The screening tests of hemostasis were developed to help identify patients with hemostatic defects that could cause excessive bleeding. These tests are sufficiently sensitive to be abnormal in most patients with a hemostatic defect severe enough to cause bleeding. However, they occasionally are overly sensitive and may be abnormal due to disorders that do not cause hemorrhage. Students will become familiar with diagnosis and management principles of these disorders.

Blood transfusion is a modality used rather excessively to treat a variety of disorders. Recipients of red cell transfusions may have (or may form) antibodies to donor red cells. Such serological incompatibility is important because transfusion of incompatible blood can kill people. Knowledge of the contents of blood components and derivatives, and knowledge of the effects of irradiation and filtering of blood components, is essential to selecting appropriate transfusion therapy.

Appropriate selection and use of plasma, coagulation components and blood derivatives can be life-saving. Inappropriate use of blood components is costly, wastes a scarce resource, and can expose patients to unnecessary hazards

In spite of stringent donor screening and extensive laboratory testing, blood still can transmit infectious diseases.We will critically look into all these aspects of transfusion medicine.

The core contents of this module are organized into 6 themes and clinical cases have been provided to achieve our learning objectives logically, coherently and lucidly. Timeline and learning strategies are complemented.

Competencies:

- Medical knowledge
- Patient care
- Communication skills

Organization of Module:
The module consists of six themes, and; each based on a real life situation. Each theme has its explicit Learning Objectives (LOs). The module will employ different modes of instruction, briefly described below. Major emphasis will be on real life patient examination, discussion, laboratory investigation and interpretation, case analysis, diagnosis, deductions and management; all by the students and guided by the faculty.
Each theme in this module is augmented with a clinical scenarios. The clinical presentation of themes will give you a clue that how a patient presents in a real life situation and to draw a conclusion from the information given by the patient and signs elicited by your clinical examination. All this information is included in the respective clinical cases. Your daily activities would be divided into different slots. Please refer to time table for more details regarding organization of learning activities.

Teaching Strategies:
The content of this module will be delivered by a combination of different teaching strategies. These include small group discussions (SGD), large group interactive sessions (LGIS), history taking, patient examination, laboratory investigations and tests interpretation, clinicopathological conferences (CPCs), discussions and journal club. Entire curriculum will be delivered by clinical case scenarios each covering a theme. Read the cases and the objectives of the theme which you are supposed to encounter next day, understand and explain the case to yourself and study the relevant information. The students will present clinical cases based on scenarios themselves and display the relevant radiological and pathological features. Following learning/teaching strategies will be used in Hematology Module:

Small Group Discussion (SGD):
Part of the course content will be delivered in small group sessions. Each theme has an associated case. The case will be centered around which learning will take place. Every group will have a facilitator assigned to it. The facilitator will be there to keep you on track, giving you maximum liberty to discuss and achieve the objectives as a group. Small groups will be followed by a wrap up session to standardize learning. Rest of the information will be in the schedule/ time table.

Large Group Interactive Sessions (LGIS):
LGIS will be employed at times to augment small groups. By and large they will be used to pass on general concepts regarding the theme. Large group instruction will be employed at times sparingly. Attend large group sessions with the following focus:

- Identify important points.
- Ask questions on concepts not well understood in the text books.
- Measure your learning comprehension

Clinicopathological Conferences (CPCs):
The students will be required to present case related to the theme in group. They will collect the information about the different facets of patient's disease and present to the whole class with the help of appropriate hematological, radiological and clinical slides. It will be followed by question, answer and discussion.

Practical Skills:
Selection of tests, collection of the specimen, examination and interpretation of specimens/test reports, microscopic slides, and radiological images.

Self-Directed Learning(SDL):
A task will be given in SDL regarding the theme to be discussed before PBL. This will help to prepare you a bit before the theme is under discussion. A few SDLs have been added in between to create an environment for you to search literature as well as to deduce and synthesize information from different sources to meet the learning objectives.

Assessment:
In this module, you will have formative and summative assessment. This will give you an idea about the format of the examination that you will go through at the end of the year. This will be followed by feedback on your performance in the exam. Marks obtained in the module examination will contribute 30% (internal assessment) towards end of year Professional University Examination.**There is no re-sit exam for module written assessment and block IPE** under any circumstances. If you miss them, your internal assessment will be recorded as zero. No excuse of any kind is permissible for absence in module or IPE assessment.

RECOMMENDEDLISTOFICONS

INTRODUCTION TO CASE

FOR OBJECTIVES

CRITICALQUESTIONS

ASSESSMENT

LABORATORY SESSIONS

RESOURCEMATERIAL

KEYWORDS

Table of Specifications (TOS)

Themes/ Core content

1	A Patient with Pancytopenia	15%
2	Raised TLC with & without immature looking cells	25%
3	Polycythemia	10%
4	Fever Cough, Back Pain and Fatigue	10%
5	Myeloproliferative Disorder	15 %
6	Abnormal bleeding	25 %

Theme 1: A Patient with Pancytopenia

At the end of the theme students will insha Allah be able to:

1. Illustrate the mechanisms (production, destruction, and sequestration) and consequences of pancytopenia.
2. illustrate the causes, clinical features, and treatment principles of neutropenia
3. Identify the pathophysiologic mechanisms of bone marrow aplasia.
4. Explain clinical course and diagnosis of aplastic anemia
5. Give three treatment options for aplastic anemia.
6. Understand causes and effects of splenomegaly.
7. Describe disorders of granulocytes, including congenital neutropenia and chronic granulomatous disease.
8. Describe epidemiology, prevalence, preventive measures and investigations of malaria, dengue fever, leishmaniasis and filariasis.

Theme 2:Raised TLC with & without Immature Looking Cells

At the end of the theme students will insha Allah be able to:

1. Differentiate between benign and malignant causes of leukocytosis
2. Describe the epidemiological morphological and clinical features of infectious mononucleosis.
3. List the differential diagnosis for neutrophil leukocytosis and to define the phrase "left shift".
4. Define the leukemoid reaction and list specific causes of this phenomenon.
5. List the differential diagnosis for eosinophilia.
6. Identify the age and gender distribution of patients with ALL.
7. List common symptoms/signs and common laboratory findings in a patient presenting with ALL.
8. Briefly describe two tests that can be used to distinguish leukemic blast cells of ALL from leukemic blast cells of AML.
9. Describe phases of Therapy of ALL .
10. Describe one complication that leads to mortality in ALL.
11. Define "myeloblast" and describe the consequences of excess myeloblasts in the bone marrow and in the circulation.
12. List at least three disease features which result from replacement of marrow cells with myeloblasts or circulation of myeloblasts.
13. Describe the predominant cell types seen in the peripheral blood and/or bone marrowin major categories of leukemia.
14. Describe the unique features of acute promyelocytic leukemia (AML M3), including the morphologic appearance of the promyelocyte, hemorrhagic complications, chromosomal abnormality, and treatment with induction therapy with all trans retinoic acid.
15. List two favorable and two adverse cytogenetic abnormalities in AML
16. Identify the complications of chemotherapy for AML. List which complications contribute to mortality.
17. Compare and contrast AML and ALL in terms of age of patients, central nervous system involvement, treatment, and outcome.
18. List the indications for hematopoietic stem cell transplant and the rationale for this treatment choice.
19. Describe the different types and sources of hematopoietic stem cells.
20. Describe principles of pre-transplant conditioning and the role for post-transplant immunosuppression.
21. Describe graft versus host disease and graft versus tumor effect.
22. Describe the pathophysiologic basis for acute and chronic graft versus host disease.
23. Describe the changes in humoral and cellular immunity following stem cell transplant and how they relate to infectious complications.
24. Dermatological manifestations of various leukemias

Theme 3:Polycythemia

At the end of the theme students will insha Allah be able to:

1. Describe the pathophysiologic differences between absolute and pseudo- polycythemia (erythrocytosis).
2. List the primary and secondary causes of polycythemia.
3. Describe the mechanisms for cellular oxygen sensing and to identify reasons for which increased oxygen delivery is necessary.
4. Describe the mechanisms, specific causes and consequences of an elevated erythropoietin level.

Theme 4:Fever Cough, Back Pain and Fatigue

At the end of the theme students will insha Allah be able to:

1. Describe concept of monoclonal gammopathy.
2. Illustrate atypical plasma cells.
3. Describe hyper viscosity syndrome

4. List the major criteria used to diagnose multiple myeloma.
5. Describe at least five complications that may occur in patients with multiple myeloma.
6. Describe the pathophysiology of renal failure in patients with multiple myeloma.
7. Describe the pathophysiology, x-ray appearance, complications, and treatment of bone abnormalities in multiple myeloma.
8. Describe indications for therapy, treatment, and prognostic indicators for patients with multiple myeloma.
9. Define the diagnostic criteria, incidence, and clinical course of patients with monoclonal gammopathy of unknown significance

Theme 5:Myeloproliferative Disorder

At the end of the theme students will insha Allah be able to:

1. Diagram the chromosomal translocation that generates the Philadelphia chromosome, identify the genes involved and the protein created by this translocation.
2. Describe how the bcr-abl fusion protein causes leukemia and provides a target for effective therapy in CML.
3. Describe the presenting features of CML, including age at presentation, the most common symptoms, one physical exam finding, and a typical CBC.
4. Describe the typical findings on the blood smear in patients with CML, emphasizing the number and types of leukocytes seen in the blood.
5. For a patient with chronic phase CML, understand goals and modalities of treatment.
6. Describe the presenting features of CLL, including the typical age at presentation, the most common symptoms, two major physical exam findings and typical blood counts.
7. Describe the predominant leukemic cell in the blood of patients with CLL, and distinguish this from the leukemic cells that can be seen in the blood of patients with ALL, AML, and CML.
8. Describe the staging of CLL, and features that correlate with a better or worse prognosis.
9. Describe complications of CLL that exemplify the immune dysfunction associated with this disease.
10. List at least four symptoms and/or complications of CLL that are an indication for treatment.
11. Compare and contrast CLL and CML in terms of molecular mechanism, age at onset, symptoms, physical exam findings, typical blood counts, treatment, and outcome. Describe the typical age and classic peripheral blood findings of patients with myelodysplasia.
12. Describe the bone marrow findings and cytogenetics seen in patients with myelodysplasia.
13. Describe three laboratory determinants of prognosis in patients with myelodysplasia.
14. List three treatments used in patients with myelodysplasia, the goals of such therapy, and the results of each treatment in terms of response rate, cure, and/or impact on survival.
15. Describe the typical age and classic peripheral blood findings of patients with myelodysplasia.
16. Describe the bone marrow findings and cytogenetics seen in patients with myelodysplasia.
17. Describe three laboratory determinants of prognosis in patients with myelodysplasia.
18. List three treatments used in patients with myelodysplasia, the goals of such therapy, and the results of each treatment in terms of response rate, cure, and/or impact on survival.
19. List the four major myeloproliferative disorders and describe the pathophysiologic features shared by these disorders.
20. List four causes of reactive or secondary thrombocytosis.
21. List the two major complications of essential thrombocythemia.
22. Describe the typical physical exam, and blood and bone marrow findings in patients with chronic idiopathic myelofibrosis.
23. Describe the common mutation associated with polycythemia vera and its biological consequences.
24. Describe the clinical features and complications of polycythemia vera.
25. Describe treatment approaches to polycythemia vera.
26. Describe the pathophysiologic differences between absolute and pseudo- polycythemia (erythrocytosis).
27. List the primary and secondary causes of polycythemia.
28. Describe the mechanisms for cellular oxygen sensing and to identify reasons for which increased oxygen delivery is necessary.
29. Describe the mechanisms, specific causes and consequences of an elevated erythropoietin level.

Theme 6: Bleeding

At the end of the theme students will insha Allah be able to:

1. Interpret values of PT/INR, PTT, TT (thrombin time), fibrinogen concentration, and platelet count, for diagnosis of possible disorders giving rise to these abnormalities.
2. Interpret values of various clotting factor and will predict which screening tests of coagulation will be abnormal.
3. Explain how a 1:1 mixing study can distinguish a clotting factor deficiency from an inhibitor of coagulation.

4. Explain the utility and derivation of the INRBe able to compare and contrast three tests of platelet function - bleeding time, PFA-100, and platelet aggregation studies.
5. diagram the formation of the D-dimer and explain its utility in diagnosis of venous thromboembolic disease
6. Identify the components of Virchow's triad and their pathophysiologic contribution to thrombosis.
7. describe at least three major clinical symptoms that occur when a patient suffers from an acute iliofemoral thrombosis of the leg, and indicate the pathophysiologic reason for each one (for example, dilated superficial veins of the calf due to obstruction of venous return in the occluded deep veins).
8. Compare and contrast the cause and mechanism of a thrombus occurring in the arterial circulation (such as acute coronary artery thrombosis) from one that develops in a deep vein of the leg. Include the instigating factor(s) and composition of the clot.
9. List 3 clinical clues suggesting an inheritedhypercoagulable disorder.
10. Describe (in one paragraph) at the molecular level the pathophysiologic reason that patients with deficiencies of antithrombin, protein C, or protein S, factor V Leiden or the prothrombin gene mutation are likely to have thrombosis. Explain what tests are used to identify these patients.
11. List at least three acquired disorders that are associated with recurrent venous or arterial thromboembolism.
12. Describe the clinical features and criteria for diagnosis of antiphospholipid antibody syndrome.
13. What is the KEY factor in determining how long someone should be anticoagulated for a venous thrombosis?
14. Describe five screening tests of hemostasis and list several causes of an abnormal result in each case.
15. Distinguish between signs and symptoms of primary hemostasis defects and plasma coagulation defects.
16. Explain why a marked deficiency of von Willebrand factor leads to excessive bleeding.
17. Recommend two potential forms of therapy for hemorrhage in a patient with type 1 von Willebrand disease and be able to explain a likely mechanism of its therapeutic effect in each case.
18. Predict the results of hemostatic screening tests (PT/INR, PTT, fibrinogen, platelet count, and bleeding time) in a patient with severe hemophilia A.
19. Explain why a patient with severe von Willebrand disease and a patient with hemophilia A may both have a prolonged PTT.
20. Using inheritance patterns, clinical history and the results of laboratory tests, be able to distinguish hemophilia A (factor VIII deficiency), hemophilia B (factor IX deficiency) and moderate to severe von Willebrand disease
21. Briefly describe the pathogenesis, diagnostic tests, and therapeutic approach to patients with the following acquired disorders who are actively bleeding:
    a. end stage liver disease
    b. acquired factor VIII inhibitor (auto-antibody against FVIII)
    c. severe DIC due to acute promyelocytic leukemia
    d. Vitamin K deficiency

22. Students will be able to demonstrate the following
    a. Importance of blood donors
    b. Problems with donors
    c. Donor Selection criteria
    d. Donor Rejection criteria
23. Blood components in terms of :
    a. Storage
    b. Handling
    c. Shelf life
24. Indications of blood components
25. Alternates to blood transfusion/ strategies to reduce the use of blood
26. Tests done on blood donors
27. Apharesis
28. Legal requirements of transfusion medicine (Blood Banking)
29. Ethics in blood banking.
30. List the four major blood types (phenotypes) in the ABO system.
31. List the two main "naturally occurring" antibodies to red cell antigens.
32. Be able to tell which of these two antibodies would be found in individuals of each ABO type, and briefly explain why ordinarily they would or would not be present.
33. Explain why the ABO system is the most important red cell blood group system for transfusion therapy.
34. Given the Rh phenotype of a mother and her fetus, be able to state whether the baby may be at risk of developing hemolytic disease of the newborn (HDN) due to anti-Rh antibodies, and why (or why not). State

the immunoglobulin class responsible for HDN, and give the reason that other classes of immunoglobulin do not cause HDN.

35. Diagram the direct antiglobulin test (the Coombs test), indicating the main components and their source (patient vs. reagent).
36. State what the direct antiglobulin test is capable of detecting. Be able to diagram the indirect antiglobulin test and state the major purpose for the indirect antiglobulin test.
37. List the three essential steps in blood compatibility testing, and the purpose of each step.
38. Indicate what kind of blood is given, if necessary in emergency situation , before typing is complete, and what kind of blood is given, if necessary, , before cross-matching is complete
39. Give three reasons why blood component therapy is preferable to whole blood therapy.
40. List the clinical indication for red cell transfusion.
41. State two methods of platelet product preparation commonly used.
42. What is meant of platelet alloimmunization, how it can be prevented, and how it can be managed if it occurs.
43. Give one clinical indication for CMV negative, irradiated, and / or filtered blood.
44. List two indications for transfusing fresh frozen plasma (FFP).
45. List three major therapeutic constituents of cryoprecipitate, and name a clinical indication for its use.
46. List at least one common indication for each of the following blood derivatives: factor VIII concentrates, prothrombin complex concentrates, albumin, intravenous immune globulin.
47. List the main coagulation abnormalities that occur after massive transfusion, and outline the appropriate treatment for each
48. List the major clinical effects of intravascular hemolytic transfusion reactions.
49. Identify the most effective method known to prevent the majority of acute hemolytic transfusion reactions.
50. List the clinical symptoms and laboratory findings of delayed hemolytic transfusion reactions.
51. List three major clinical situations in which Rh Immune Globulin should be given to prevent HDN.
52. Describe the clinical presentation of Transfusion-Associated Acute Lung Injury (TRALI) and what causes it.
53. Explain what transfusion-associated graft-versus-host disease is, who is at risk, and how to prevent it
54. List the blood component most likely to cause bacterial sepsis, and explain the reason why.
55. Identify the two major causes of post-transfusion hepatitis, frequency ofoccurrence in the Pakistani population, and relative risk of transmission in blood transfusions.
56. Give the approximate risk of HIV transmission per unit of blood
57. Explain the meaning of "window period" in the context of transmission of virus in blood or transplanted tissues.
58. Explain why directed donation (blood given by relatives or friends) should not be regarded as safer than blood from a regular volunteer donor.

PBL-1A

A 59 years presents with insidious onset, tiredness, fatigue lymph node enlargement since 1 months. He has repeated chest infections which were diagnosed as pneumonia. In between ha also had herpes simplex labialis. He also complains of early satiety and abdominal discomfort. He also has mucocutaneous bleeding and petechiae.More recently he has symptoms of weight loss, fever, night sweats, muscle wasting.

On physical examination he has generalized lymphadenopathy, as well splenomegaly, hepatomegaly, petechiae and pallor. Complete blood count (CBC) with differential shows absolute lymphocytosis, with more than 25000 B-lymphocytes/μL. Microscopic examination of the peripheral blood smear confirmed lymphocytosis and showed the presence of smudge cells. A few large atypical cells, cleaved cells, and prolypmhocytes were also noted. Peripheral blood was subjected to flow cytometry. Serum quantitative immunoglobulin levels were depressed.

PBL 2

A 65 years presents withsymptoms of fatigue and malaise. He has history of anemia for 5 years. He is diagnosed as suffering from macrocytic anemia without evidenceof B12, folate deficiency. Two years later he was found to be having mild thrombocytopenia and neutropenia. In between he had attacks of Fever, cough, dysuria. On examination he showed pallor of the skin and mucosal membranes and evidence of, tachycardia, and congestive heart failure. His peripheral blood showed anemia with macroovalocytes, a dimorphic population consisting of microcytes admixed with

macrocytes and punctate basophilia. White cell series showed, bilobed or unsegmented nuclei (Pseudo–Pelger-Huet abnormality) and a few hypersegmented neutrophils with (6-7 lobes) similar to megaloblastic diseases. Platelet counts were decreased. His bone marrow was aspirated that depicted dysplastic changes.

AJK Medical College, Muzaffarabad

Schedule for Hematology Module – (4th Year)

Week-1

DATE→					
TIME↓	**MONDAY**	**TUESDAY**	**WEDNESDAY**	**THURSDAY**	**FRIDAY**
8:00am-09:00am	**LGIS** Introduction to Module/ Peripheral smear and CBC **Dr. Malik Mehmood/Module Team**	**PBL-1A** **Dr. Malik Mahmood & Team-3**	**LGIS** Complication and management of blood transfusion reactions **(Dr. Malik Mahmood)**	**LGIS** Blood Sample Collection Procedure & Factors Affecting Quality of Samples **Dr. Malik Mahmood**	
9:00am-10:00am	**LGIS** Transfusion medicine **Dr. Malik Mehmood**	**LGIS** Hemorrhagic Fever **Dr. Bashir Ahmed Trunbu**	**LGIS** Inherited & acquired Bleeding Disorders **Dr. Mateen**	**LGIS** Blood Component Therapy **Dr. Malik Mahmood**	
10:00-10:30am	**CLINICAL ROTATION**	**CLINICAL ROTATION**	**CLINICAL ROTATION**	**CLINICAL ROTATION**	**B R E A K**
10:30am-12:30pm					**PBL-1B** **Dr. Malik Mahmood & Team-3**
12:30-1:30					**DSL**
1:30 - 2:00pm	**B R E A K**				
2:00pm-4:00pm	**LGIS** Ethics in Transfusion **Prof. Anwar ul Haque**	**LGIS** Massive blood transfusion **Prof. Nizam ud Din**	**SGD** Strategies for reducing the use of blood **Dr. Mehmood & Team-3**	**LGIS** Neutropenia and chronic granulomatous diseases of child hood **Dr. Malik Mahmood**	**SDL**
	SDL	**SDL**		**SDL**	

AJK Medical College, Muzaffarabad

Schedule for Hematology Module (4th Year)

Week-2 (6th - 10th March 2017)

DATE→					
TIME↓	**MONDAY**	**TUESDAY**	**WEDNESDAY**	**THURSDAY**	**FRIDAY**
8:00am-09:00am		**LGIS** Acute lymphocytic Leukemia **Dr. Mehmood Malik**	**LGIS** Hemolytic anemias **(Dr. Mahmood Malik)**	**LGIS** Myeloproliferative disorders and Multiple myeloma **(Dr. Mahmood Malik)**	**LGIS** Laboratory Evaluation of Anemia **(Dr. Mahmood Malik)**
9:00am-10:00am		**LGIS** Polycythemia & erythrocytosis **(Dr. Mahmood Malik / Dr. Khalid Awan)**	**LGIS** Anemias of Dimnished Erythopoiesis (iron Deficiency, megaloblastic Anemia) **Prof. Dr. Jawed Rathore**	**LGIS** Bone Marrow Transplantation **(Dr. Malik Mahmood)**	**LGIS** Acute myeloid leukemia **(Dr. Mahmood Malik)**
10:00-10:30am	**CLINICAL ROTATION**	**CLINICAL ROTATION**	**CLINICAL ROTATION**	**CLINICAL ROTATION**	**B R E A K**
10:30am-11:30pm					**LGIS** Transfusion case scenarios **Dr. Mahmood Malik**
11:30am-1:30pm					**PBL-2B**
1:30 - 2:00pm	**B R E A K**				
2:00pm-4:00pm	**PBL-2A Dr. Mehmood Malik & team**	**SGD** Leukocytosis & Eosinophilia **Team-3** **Wrap-up** **Dr. Mahmood Malik**	**LGIS** Hematological aspects of Pregnancy & neonatology **(Dr. Mahmood Malik)**	**LGIS** Hematology case scenarios **(Dr. Mahmood Malik)**	**DSL** Blood Dyscrasias
	LGIS Approach to bleeding patient **Dr. Ali Arshad**		**DSL** Classification of Leukemia	**DSL** Hazards of Transfusion	

Reference Books

- ROBBINS BASIC PATHOLOGY 8th ED
- ROBBINS AND CORTAN PATHOLOGY BASIS OF DISEASE WITH SEARCHABLE FULL TEXTONLINE 8th ED
- ROBBINS AND CORTAN ATLAS OF PATHOLOGY 2nd ED.
- ROBBINS AND COTRAN REVIEW OF PATHOLOGY 3rd ED
- ESSENTIALS OF HEMATOLOGY SHIRISH M KAWTHALKAR FIRST EDITION CLINICAL MEDICINE, 6TH EDITION, PERVEEN & KUMAR, PAGES 425 – 429.

Web Links

Following online medical dictionaries can be referred

WWW.NBT.gov
www.nlm.nih.gov
www.medterms.com
www.bloodmed.com
www.online-medical-dictionary.org
www.medscape.com
www.jpathology.com
www.cdc.com

Inquires & trouble shooting

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