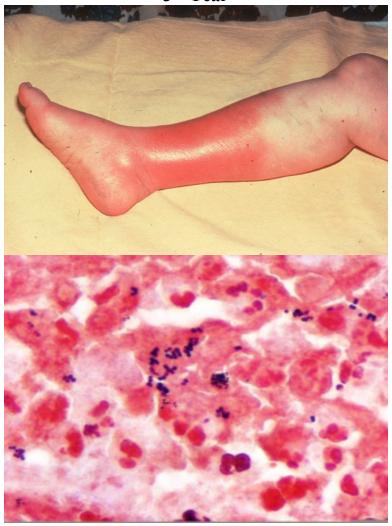
AIK Medical College Muzaffarabad



Inflammation, Healing & Immunology (IHI)

3rd Year



Module Code: 0203
Module Duration: 4 Weeks

DEPARTMENT OF MEDICAL EDUCATION

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Introduction to case



Objectives



Critical questions



Assessment



Laboratory sessions



Resourcematerial

Keywords

Rationale

The normal cell is able to handle physiologic stresses and some pathologic stimuli and can maintain a steady state called *homeostasis* by adaptive responses like an increase in the size of cells and functional activity, an increase in their number a decrease in the size and metabolic activity of cells or a change in the phenotype of cells The cells get injured when they are stressed so severely that they are no longer able to adapt or when cells are exposed to inherently damaging agents or suffer from intrinsic abnormalities cell injury results when cells are stressed so severely that they are no longer able to adapt or when cells are exposed to inherently damaging agents or suffer from intrinsic abnormalities. Injury may progress through a reversible stage and culminate in cell death.

The animals and human beings can survive only if they have the ability to get rid of damaged or necrotic tissues and foreign invaders, such as microbes. The host response that accomplishes these goals is called *inflammation*. This is fundamentally a protective response, designed to rid the organism of both the initial cause of cell injury (e.g., microbes, toxins) and the consequences of such injury (e.g., necrotic cells and tissues). Without inflammation infections would go unchecked, wounds would never heal, and injured tissues might remain permanent festering sores. In the practice of medicine the importance of inflammation is that it can sometimes be inappropriately triggered or poorly controlled, and is thus the cause of tissue injury in many disorders. Injury to cells and tissues sets in motion a series of events that contain the damage and initiate the healing process. This process can be broadly separated intoregeneration and repair Regeneration results in the complete restitution of lost or damaged tissue; repair may restore some original structures but can cause structural derangements. In healthy tissues, healing, in the form of regeneration or repair, occurs after practically any insult that causes tissue destruction, and is essential for the survival of the organism

The immune system is vital for survival, It in collaboration with the inflammatory response protects us from infectious pathogens. Immune deficiencies render individuals easy prey to infections. But the immune system is a double-edged sword. Although it normally defends us against infections, a hyperactive immune system may cause diseases that can sometimes be fatal.

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.

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 and radiological test investigation and interpretation, case analysis, diagnosis, deductions and management; all by the students and guided by the faculty.

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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 GIT Module:

Small Group Discussion (SGD):

Main bulk 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 cases related to the themes in groups. They will collect the information about the different facets of patient's disease and present to the whole class with the help of appropriate histopathological, 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, culture plates/media examination 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.

Table of Specifications (ToS)

1	Painful Swelling	35%
2	Persistent Cough	18%
3	Keloid	15%
4	Paralyzed Child	10%
5	Lady with a Butterfly Rash	13%
6	A Man with Recurrent Infection	9%

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Learning Objectives:

1. Theme 1: Painful Swelling (Acute Inflammation, Microorganisms)

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

- > Define inflammation in terms of its etiology, purpose, outcome and its benefits and hazards
- > Describe important mediators of inflammation and their interactions
- > Diagrammatically explain cellular and vascular events of inflammation
- Enumerate the cardinal signs of inflammation and explain their basis
- > Define Acute Phase Reactants and their individual roles
- > Explain Erythrocytes Sedimentation Rate and C Reactive Protein
- > Compare and contrast acute inflammation, chronic inflammation and chronic granulomatous inflammation
- Analyze inflammation as a basic reaction in terms of the mechanism of the vascular and cellular responses, the structure and function of inflammatory cells, the role of factors which modify the response, the classification of types 'of lesions produced and the mechanisms of systemic response.
- Name the major components of inflammation and cite common examples of beneficial and harmful inflammatory reactions.
- List chemical mediator s of the vascular and cellular responses and compare them in terms of source, chemical nature, mechanism of release and effect.
- ➤ Diagram the interrelationship s between the following as they occur in atypical acute inflammatory response:
 - release of chemical mediators
 - capillary dilatation
 - increased capillary permeability
 - components of exudate (cells, proteins, solutes, fluid)
 - chemotaxis
- > cardinal signs of inflammation movement of neutrophil s and monocytes
- Compare various types of inflammatory cells in terms of:
 - morphology normal location
 - source of renewal function in inflammation
 - time of appearance in inflammation
- List general factors relating to causative agents which influence the nature, extent, site and duration of inflammation.
- List types of local and systemic host factors that modify inflammatory responses.
- Compare the following types of exudates on the basis of composition, causative mechanism and common sites of occurrence:
 - ♣ Catarrhal, purulent, fibrinous, serous, sanguineous
 - Compare acute inflammation, chronic inflammation and granulomatous inflammation on the basis of cellular composition, duration and common examples.
- > Compare the appearance and common locations of:
 - ulcer
 - erosion
 - phlegmon
 - abscess
 - cellulitis
 - pseudomembranous inflammation
 - empyema
 - furuncle
 - carbuncle
- > Describe the pathogenesis of fever in terms of:
 - mechanisms of body heat regulation relationship of inflammation to fever mechanisms and stages of fever physiologic effects of fever, role of pyrogens
- Describe the mechanisms of and enlist major causes of leukocytosis and leukopenia
- Discuss the mechanism and significance of elevations in erythrocyte sedimentation rate in terms of gamma globulin and fibrinogen levels
- > Describe the appearance and indicate the significance of lymphangitis andlymphadenitis.

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- ➤ Define and use in proper context:
 - ♣ Transudate, exudate, opsonin
 - Agranulocytosis, pathogenicity, virulence,
- > Define mechanical injuries
- Classify mechanical injuries
- > Mechanism of production of mechanical injuries
- Medicolegal Aspects mechanical injuries
- > Age of mechanical injuries
- Describe Traumatic & microbial inflammation.
- > Define inflammatory response syndrome and how it is managed after major surgery and trauma
- > Describe surgical stress response and how unnecessary aspects of surgical stress response can be prevented?
- > Definition of SSIs, classify of surgical wounds and Infection control guidelines to decrease SSIs
- Describe morphological features, pathogenesis, spectrum of diseases and laboratory methods for diagnosis of Staphylococci
- Definition of MRSA, Infection control measures for MRSA
- Classify Streptococci; describe morphological features, pathogenesis, spectrum of diseases and laboratory methods for diagnosis of Strep pneumoniae
- > Know the morphological features, pathogenesis, spectrum of diseases and laboratory methods for diagnosis of Strep pyogenes
- > Morphological Features cultural characters antigenic structure and Pathogenicity of Neisseria Meningitides.
- > Know the laboratory diagnosis of meningitis
- Know the morphological Features cultural characters antigenic structure and pathogenicity of Neisseria Gonorrhea.
- Lab diagnosis of Gonorrhea
- Morphological Features cultural characters antigenic structure and Pathogenicity of Corynebacterium diptheriae
 - ➤ Classify NSAID'S with examples.
 - ➤ What is mechanism of action of NSAID'S
 - Classify various cyclooxygenase inhibitors.
 - Explain pharmacokinetics, mechanism of action and pharmacological effects of NSAIDs.
 - What are the features of NSAIDs toxicity and how it is managed
 - Write down pharmacokinetics and pharmacodynamics of selective COX-2 inhibitors.
 - What are clinical indications, adverse effects of NSAIDs
 - > Write contraindications/precautions and drug interactions of NSAIDs.
 - Write pharmacokinetics, mechanism of action, clinical uses and adverse effects of Paracetamol
 - ➤ Write contraindications/precautions and drug interactions of Paracetamol
 - > What is the mechanism and features Paracetamol toxicity and how it is managed
 - ➤ What is the significance of combination therapy with DMARDs
 - Enlist drugs used in acute and chronic gouty arthritis
 - ➤ Write down pharmacokinetics of drugs used in gout
 - > What is the mechanism of action , pharmacological effects, therapeutic indications of drugs used in gout
 - > Enlist adverse effects, contra-indications /precautions and drug interactions of drugs used in gout
- ➤ Understand signs & symptoms of uveitis
- Differentiate signs & symptoms of retinopathy and its principles of treatment

Theme 2: Persistent Cough (Chronic Inflammation, Treatment & Microorganisms)

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

- > Draw a typical granuloma and explain how and why it is formed
- Enumerate the conditions giving rise to granuloma formation
- Explain the differences between caseating, non caseating, naked or bare and foreign body granulomas
- ➤ Bacteria causing Chronic Granulomatous inflammation.
- Morphological features ,and cultural characteristics pathology and Pathogenicity of Mycobacterium Tuberculosis
- > Immune response of the body against Mycobacterium Tuberculosis
- ➤ Laboratory diagnosis of Tuberculosis.

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- > Morphological featuresand cultural characteristics of Mycobacterium Laprae.
- ➤ Laboratory diagnosis of leprosy
- ➤ Enumerate the parasites which can cause chronic cough.
- > Salient morphological features, life cycle, pathogenicity and laboratory diagnosis of Ascariasis
- > Salient morphological features, life cycle, pathogenicity and laboratory diagnosis of hook worm disease
 - ➤ Define and classify autacoids.
 - ➤ Write down the pharmacokinetic properties of histamine.
 - Enlist various histamine receptors subtypes with their mechanism of action.
 - > What is the mechanism of action of histamine. Also write its organ system effects and clinical uses.
 - > Write down the adverse effects, contraindications / precautions and drug interaction of histamine.
 - > Classify histamine receptor antagonists.
 - ➤ What are the pharmacokinetic properties of H1-blockers.
 - ➤ How H1 –Blockers act and write their organ system effects with their therapeutic uses.
 - > Enumerate the adverse effects, contraindications/precautions and drug interactions of H1-receptor blockers.
 - ➤ Difference between first generation and second generation H1-antagonists.
 - ➤ Write down the pharmacokinetic properties of H2- blockers.
 - ➤ What is the mechanism of action, organ system effects and clinical uses of H2 –blockers.
 - > Enlist the adverse effects, contraindications /precautions and drug interactions of H2-antagonists
 - ➤ Give pharmacokinetic properties of serotonin.
 - Name various serotonin receptor subtypes and write down their distribution.
 - > Write the mechanism of action, pharmacological effects and clinical uses of serotonin
 - What are various side effects, contraindications/precautions and drug interactions of serotonin?
 - ➤ Enlist the drugs causing serotonin and other hyperthermic syndromes. How these patients present and how you will treat them?
 - ➤ What do know about role of serotonin agonist in migraine headache?
 - > Enumerate various serotonin receptor agonists and antagonists with their pharmacokinetics and pharmacodynamics.
 - ➤ What are ergot alkaloids? Write down their effects on several receptors.
 - Explain clinical pharmacology of ergot alkaloids.
 - Enlist various vasoactive peptides that are used clinically.
 - ➤ Write down the biosynthesis of Angiotensin.
 - ➤ Give a brief review actions of Angiotensin -II
 - > Write down various angionensin receptors with their mechanism of action.
 - ➤ Give the pharmacological inhibition of Renin-Angiotensin system.
 - ➤ Write down the biosynthesis of kinins.
 - ➤ What are various physiological and pathological effects of kinins?
 - > Enumerate different kinin receptors with their mechanism of action?
 - ➤ How kinin are metabolized in the body.
 - ➤ Enlist various drugs affecting the kallikrein-kinin system.
 - ➤ Write down the clinical application of vasopressin antagonists.
 - ➤ How different natriuretic peptides are used clinically?
 - ➤ Enlist different vasopeptidase inhibitors with their clinical uses.
 - Write down the names of various endothelins synthesized in the body. Also write their actions.
 - What are the inhibitors of endothelinsynthesis. Also write their physiological and
 - > pathological role.
 - ➤ Give a brief review on Vasoactive intestinal peptide, Substance Neurotensin , Calcitonin gene-related peptide. Adrenomedullin, Neuro peptide Y & Urotensin.
 - ➤ How Eicosanoids are synthesized in the body?
 - ➤ Write down the receptor mechanisms and effects of eicosanoids, synthesized by both COX and lipoxygenase enzyme pathways.
 - ➤ Write down the clinical pharmacology of Eicosanids.
 - ➤ What are various Eicosanoids synthesis inhibitors. Write down their clinical uses
 - > How nitric oxide is synthesized in the body. Write down its signaling mechanisms and its inactivation?
 - ➤ What are various inhibitors and donors of nitric oxide?
 - > Write down the role of nitric oxide in various diseases

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Theme 3:Keloid (Wound Healing, Repair & Scar)

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

- Know the morphological features, pathogenesis, spectrum of diseases and laboratory methods for diagnosis of Pseudomonas
- Classify the Anaerobes, describe the morphological features, pathogenesis, spectrum of diseases and laboratory methods for diagnosis of Medically important Clostridia
- > Should be able to enumerate the bacteria responsible for causing infections of the road side accidents wounds and war wounds
- Enumerate the important gram positive anaerobic spore forming bacilli.
- > To know the morphological features and cultural characteristics of important Clostridia.
- Should know the laboratory diagnosis of road side and war wounds
- Define Healing
- > Define Primary and secondary healing primary and secondary healing
- > Compare and contrast Define inflammation in terms of its etiology, purpose, outcome
- Describe factors affecting healing and repair primary and secondary healing
- Define Hyperplastic scar and keloid
- Define and classify normal healing and what are the factors affecting the healing?
- Define and classify wounds. Describe the management of wounds of different structures & of different sites.
- Describe disordered wound healing leading to chronic wounds.
- Describe various types of scars and their Management
- Describe preoperative, operative and postoperative surgical care in immune compromised patient

Theme 4: Paralyzed Child

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

- ➤ Classify the PICORNA Viruses
- > Understand the general properties and replication of PICORNA Viruses.
- ➤ Know the general properties of Enteroviruses.
- Know general properties, multiplication, antigenic structure, pathogenesis and Pathology and Laboratory diagnosis of Polio Virus.

Theme 5: Lady with a Butterfly Rash (Autoimmune Disorders)

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

- > Define innate and acquired immunity.
- > Enumerate cells and mechanisms of both defense system
- > Discuss the mechanism of normal immune response
- > Enumerate the types and discuss the mechanism of abnormal immune responses in the body
- > Define hypersensitivity, enumerate its types & discuss pathogenesis of each type
- > Define histocompatibility complex
- Relate mechanism of transplant rejection with major histocompatibility complex in the body
- ➤ Define autoimmunity and discuss role of genetic & environmental factors in relations to the following:
 - Rheumatoid arthritis
 - **♣** SLE
 - Sjogren's disease
 - Diabetes mellitus,
 - Autoimmune thyroid disease.
 - Myastenia gravis,
 - Multiple sclerosis,
 - ♣ Autoimmune hemolytic anemia
- ➤ Define Immunodeficiency, enumerate is types & discuss pathogenesis of primary immunodeficiencies of lymphocytes, phagocyte disorders & complement disorders
- > Discuss various types of acquired immunodeficiencies and enlist its complications
- > Define amyloid, discuss pathogenesis of amyloidosis, enumerate its types and briefly illustrate its diagnostic tests
- > Describe the applied anatomy of the nose.
- Describe etiology and pathology of nasal allergy
- > Enumerate managements steps of allergic rhinitis

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- Define histocompatibility complex, autoimmunity and discuss role of genetic & environmental factors in relations to the following;
 - ♣ SLE
 - Rheumatoid arthritis
 - Sjogren's disease.
- > Define SLE and explain etiology, pathogenesis, clinical presentation and management of SLE.
- ➤ Enumerate various types of monoclonal antibodies and immunosuppressive drugs, their mode of action, clinical uses in SLE and RA
- > Define and classify osteoarthritis and explain etiology and pathogenesis and management of osteoarthritis
- > Define and classify RA and explain etiology and pathogenesis clinical presentation and management of RA
- Differentiate between gout and pseudo gout and explain etiology, pathogenesis and clinical management of acute and chronic gout.
- Take history, examination and clinical assessment of a case of arthritis (Osteo, RA, AS)
- Students should be able to appreciate common diseases of joints like
 - Rheumatoid Arthritis
 - Ankylosing Spondylitis
 - Septic Arthritis
 - Tuberculous Arthritis
 - Osteoarthritis
 - Degenerated Spondylitis
- Classify immunomodulators.
- Classify glucocorticoids.
- Write down pharmacokinetics of Glucocorticoids.
- What is the mechanism's of action, clinical uses and adverse effects of Glucocorticoids?
- > Briefly give the contraindications and precautions of Glucocorticoids. Also write their drug interactions.
- What are the pharmacokinetics of calcineurininhibitors
- What is the mechanism of action, clinical uses and adverse effects of tacrolimas
- Briefly describe pharmacokinetics, mechanism of action, clinical uses and adverse effects of mycophenolate mofetil.
- Write down the pharmacokinetics of thalidomide. Also give its mode of action, clinical uses and adverse effects.
- Enumerate various cytotoxic drugs.
- Write down the pharmacokinetic properties of cytotoxic drugs
- > By which mechanism cytotoxic drugs act? also give description of their clinical uses and adverse effects.
- > Enumerate various immunosuppressive antibodies.
- Write down the pharmackokinetics and mechanism of action of immunosuppressive antibodies. Also write their clinical applications and adverse effects.
- Enlist various types of monoclonal antibodies with examples.
- > What are the pharmacokinetics, mode of action, clinical uses and adverse effects of monoclonal antibodies?
- Enumerate the drug that cause various immunological reactions in human body along with brief description of such reactions.
- > Define urticaria and angioedema
- Classify urticaria.
- Enlist questions to be asked in history of patient with chronic urticaria.
- Enlist lab investigation in a patient with urticaria. patient with urticaria and angioedema.

Management of a

Theme 6:A Man with Recurrent Infection (Immunodeficiency)

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

- > Describe the general properties and classification of Lenti viruses
- > Describe the general properties, replication, Pathogenesis and pathology, and laboratory diagnosis of HIV disease.
- Describe the morphological features, cultural characters, pathogenicity and pathogenesis of candidiasis.
- Describe the lab Diagnosis of Candidiasis.
- Enumerate the opportunistic Fungi which cause disease in an immune compromised patient
- Write down the normal and abnormal human immune response
- Describe the organization of the immune System
- > Should be able to understand the humoral and the cell mediated immunity

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- > Should be able to describe the properties of different classes of antibodies
- > Should be able to understand and describe the different mode by which the antigens and antibodies react in vivo and in vitro.
- Understand and describe the different serological tests which can be used to detect the presence of antigens and antibodies in serum and other biological fluids.
- > Should be able to understand the complement system ,its organization ,its activation and the role it plays in inflammatory response and hypersensitity reactions.

PBL-1A

A 21-year-old man came to the clinic with a chief complaint of a sore on his penis. The lesion began as a papule about 3 weeks earlier and slowly progressed to form the ulcer. It was painless, and the patient noticed no pus or discharge from the ulcer.

The patient was seen previously because of a sexually transmitted disease and was suspected of trading drugs for sex. The patient's temperature was 37 °C, pulse 80/min, respirations 16/min, and blood pressure 110/80 mm Hg. There was a 1-cm ulcer on the left side of the penile shaft. The ulcer had a clean base and raised borders with moderate induration. There was little pain on palpation. Left inguinal lymph nodes 1–1.5 cm in diameter were palpable.

PBL-2A

A 44-year-old man presented with a history of several weeks of intermittent fever accompanied at times by shaking chills. He had increased frequency of bowel movements without frank diarrhea but with occasional cramping and abdominal pain. There was no headache or cough. He had lost about 5 kg of body weight. The remainder of his medical history was negative.

His temperature was 38 °C, pulse 90/min, respirations 18/min, and blood pressure 110/70 mm Hg. He did not appear to be acutely ill. The tip of the spleen was palpable in the left upper abdominal quadrant 3 cm below the ribs (suggesting splenomegaly). Hepatomegaly and lymphadenopathy were not present, and there were no neurologic or meningeal signs. The balance of the physical examination was normal.

The patient's white count was stable at 3000/L (below normal). The hematocrit was 29% (below normal).

The chemistry panel was notable only for the liver enzyme alkaline phosphatase concentration of 210 units/L (normal, 36–122 units/L). Further evaluation of the cause of the patient's fever showed a normal urinalysis, negative routine blood cultures, and a normal chest radiograph. A serum cryptococcal antigen test was negative.

Tasks/Questions:

- 1. What more information do you need on this patient?
- 2. What investigations will you order?
- 3. What is the probable cause of this condition?
- 4. How will you manage this lady?
- 5. What pharmacological treatment you will suggest for this lady?
- 6. What is importance of buccal ulcer in this case?

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AJK Medical College, Muzaffarabad Schedule for IHI Module – (3rd Year) Week 1

Date→					
↓ TIME	MONDAY	TUESDAY	WEDNESDAY	THURSDAY	FRIDAY
8.00am - 9.00 am	Introduction to the Module (Module Team)	LGIS Streptococcal diseases Prof. Munir	LGIS Antigen Antibodies Reactions-I Prof. Munir	LGIS NSAIDS-III Dr Arif/Dr Inayat	LGIS Introduction to immunology Dr. Anwar
9.00am- 10.00 am	LGIS Gram Positive Cocci (Staphylococcal diseases) Prof. Munir	LGIS Acute inflammation Dr. Sarosh	LGIS Mediators of inflammatory response Dr Sarosh		LGIS Pathogenesis of fever, leukocytosis, ESR, Lymphangitis in Acute Inflammation Dr Wafa
10.00am - 10.30 am		В	reak		Break
10.30am - 11.30 am		LGIS Medicolegal aspects of lacerated and incised wounds Dr Humayun	SGD	CLINICAL ROTATION	LGIS Sore throat Swollen inflamed Tonsils ENT
11.30am - 12.30 pm	CLINICAL ROTATION	LGIS Warm , painful, swollen limb with difficulty in walking; Morphological Features of Acute Inflammation DR Anwar	NSAIDs-I Dr. Arif & Team Wrap up Dr Arif/Dr Inayat		LGIS Antigen Antibodies Reactions-II Dr. Munir
12.30pm - 01.30 pm		LGIS Polio Virus Prof. Munir	LGIS Humoral limb of immune system- Antigens and antibodies Dr Munir	LGIS NSAIDs-II Pharmacology	LGIS Histamine and antihistamine-I Dr Arif/Dr Inyat
01.30am - 02.0 pm	BREAK				
02.00pm- 3.00 pm	LGIS Staphylococcal diseases Prof. Munir	LGIS Streptococcal disease Rheumatic fever Prof. Munir	LGIS Immunity; EPI Community medicine team Dr. Ahmed Khan/Dr Murtaza Gilani	SGD NSAIDs-III Wrap up Dr Arif/Dr Inayat	SDL
3.00pm- 4.00pm	SDL	SDL	SDL		

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AJK Medical College, Muzaffarabad Schedule for IHI Module – (3rd Year) Week 2

Date→			VV CCK 2		
↓ TIME	MONDAY	TUESDAY	WEDNESDAY	THURSDAY	FRIDAY
8.00am -9.00 am	Written Assessment CP&GAP	LGIS Diphtheria Dr Munir	LGIS Chronic inflammation II Dr Sarosh	LGIS Ergot alkaloids Dr Arif /Dr Inayat	LGIS War wounds: Gas Gangrene Dr Munir/Dr. Mumtaz
9.00am- 10.00 am	Module	PBL-1A Dr Munir and team 3	LGIS Gonorrhea Dr Munir		LGIS Gout Dr Khalid
10.00am - 10.30 am		BR	REAK		BREAK
10.30am - 11.30 am	CLINICAL ROTATION	LGIS Pneumococcal diseases Dr Munir	LGIS Wound healing Dr Adnan Mehraj	CLINICAL ROTATION	PBL 1 B Dr Munir and team
11.30am - 12.30 pm		SGD Serotonin agonists and antagonists	LGIS Road side wounds- Tetanus Dr Munir/Dr. Mumtaz		III
12.30pm - 01.30 pm	LGIS Chronic inflammation I Dr Sarosh	Dr. Arif & Team Wrap up Dr. Arif /Dr Inayat	LGIS Ergot alkaloids Dr Arif /Dr Inayat	LGIS Histamine and antihistamine-III Dr Arif /Dr Inayat	LGIS Anaphylaxis – Hypersensitivity Reaction Dr. Rubina
01.30am - 02.0 pm					
02.00pm- 3.00 pm	SGD Histamine and antihistamine-II	LGIS Serotonin agonists and antagonists Dr. Arif/Dr. Inayat	Practical acute inflammatory lesions Department of Pathology	LGIS Role of parasites in chronic cough Dr. Munir	SDL
3.00pm- 4.00pm	Dr. Arif & Team wrap up Dr Arif /Dr Inayat	LGIS Introduction to Cell Mediated Immunology Dr. Anwar	LGIS NSAIDS-III Dr Arif/Dr Inayat	LGIS Drug treatment of gout-I Dr Arif /Dr Inayat	SDL

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AJK Medical College, Muzaffarabad Schedule for IHI Module – (3rd Year) Week 3

Date→			week⊔3		
↓ TIME	MONDAY	TUESDAY	WEDNESDAY	THURSDAY	FRIDAY
8.00am - 9.00 am	LGIS Rheumatoid Arthritis Dr Javed Rathore	SGD Drug treatment of Rheumatoid Arthritis-I	LGIS Cell mediated immunity Dr Wafa/Dr Mahmood	PBI 2 A Team III	SGD Oral thrush in an elderly Immunodeficient
9.00am- 10.00 am		Dr. Arif & Team Wrap-Up Dr Arif /Dr Inayat	LGIS Drug treatment of Rheumatoid Arthritis-III Dr. Arif & Team Wrap-Up Dr Arif/Dr Inayat		patient Dr. Anwar & Team- 3 WRAP UP Dr Anwar
10.00am -	CLINICAL	В	reak		Break
10.30 am 10.30am - 11.30 am	ROTATION	SGD AIDS Dr. Anwar & Team-3	SGD Eicosanoides Dr. Arif & Team	Clinical Rotation	LGIS SLE Dr. Kamran Butt/ Dr. Rubina
11.30am - 12.30 pm		WRAP UP Dr Anwar/Dr Mehmood	<u>Wrap-Up</u> Dr Arif /Dr Inayat		SGD
12.30pm - 01.30 pm	LGIS Tuberculosis-I Dr Mumtaz	LGIS Tuberculosis II Dr Mumtaz	LGIS Medicolegal aspects of bruises Dr Hamayun	LGIS Immunodeficienc y Diseases in children Dr. Tahir Aziz	Immunomodulators-I Dr. Arif & Team Wrap-Up Dr Arif /Dr Inayat
01.30am - 02.0 pm			Break		
02.00pm- 3.00 pm 3.00pm- 4.00pm	SGD Drug treatment of Gout-II Dr. Arif & Team	SGD Drug treatment of Rheumatoid Arthritis-II Dr. Arif &	PRACTICAL Gram Staining Z- N staining	SGD Hypersensitivity Dr. Anwar & Team-3	SDL
	<u>Wrap-Up</u> Dr Arif /Dr Inayat	Team <u>Wrap-Up</u> Dr Arif /Dr Inayat	Dr Mumtaz/Dr Munir	WRAP UP Dr Anwar	

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AJK Medical College, Muzaffarabad Schedule for IHI Module – 3rd Year MBBS Week 4

Date→					
ΨTIME	MONDAY	TUESDAY	WEDNESDAY	THURSDAY	FRIDAY
8.00am - 9.00 am	LGIS Surgical problems in immunocomprised patients Dr Adnan Mehraj	SGD Immunomodulators-II Wrap up	SGD HIV disease; AIDS (Team -3) Wrap-Up Dr. Javed Rathore	LGIS Antigen Antibodies Reactions-II Dr. Munir	PBL-2B Dr Munir & Team-3
9.00am- 10.00 am		Dr Arif /Dr İnayat	Munir/Dr Dr. Anwar/Dr Mumtaz		
10.00am - 10.30 am		BRE	EAK		BREAK
10.30am - 11.30 am	CLINICAL ROTATION	SGD Skill Lab routes of administration of	LGIS Leprosy Dr. Sadaf	CLINICAL ROTATION	LGIS Child presenting with fever, stiff neck and a rash Dr Munir
11.30am - 12.30 pm		drugs Pharmacology	LGIS Tumor immunity Dr Mahmood		SGD Immunomodulators- III Team-III
12.30pm - 01.30 pm	LGIS Complement system Dr Wafa	Dr. Inayat	LGIS Syphilis Dr Munir	LGIS Autoimmune diseases Dr. Anwar	Wrap up Dr Arif
01.30am - 02.0 pm			Break		
	Practical	Practical	LGIS Oppurtunistic fungal infections Dr. Mumtaz	LGIS Histamine and antihistamine-I Dr Arif/Dr Inyat	
02.00pm- 4.00 pm	Chronic inflammatory lesions Department of pathology	lesions Department of Department of forensic Medicine	SGD HLA & Graft Rejection (Team-3) Wrap-Up (Dr. Anwar)	Practical KOh Preparation India ink Preparation Dr Mumtaz	SDL

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RESOURCE FOR LEARNING



Reference Books

- ROBBINS BASIC PATHOLOGY 8th ED
- ROBBINS AND CORTAN PATHOLOGY BASIS OF DISEASE WITH SEARCHABLE FULL TEXTONLINE $8^{\rm th}$ ED
- ROBBINS AND CORTAN ATLAS OF PATHOLOGY 2nd ED.
- ROBBINS AND COTRAN REVIEW OF PATHOLOGY 3rd ED
- BRS PATHOLOGY
- MEDICAL MICROBIOLOGY BY JAWETZ, MELNICK & ADELBERG'S 25TH EDITION
- CLINICAL PHARMACOLOGY BY KATZUNG
- RANGE AND DALE'S PHARMACOLOGY WITH ONLINE ACCESS 7th ED
- MCQs IN PHARMACOLOGY WITH EXPLANATORY ANSWER

Web Links

Following online medical dictionaries can be referred www.nlm.nih.gov www.medterms.com www.bloodmed.com www.online-medical-dictionary.org www.medscape.com www.jpathology.com www.cdc.com

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