

**‏1437**

**King Saud University**

**College Of Medicine**

**Renal Block**

## http://www.ucalgary.ca/utoday/files/utoday/styles/utoday_general_photo/public/istock_000004440655xxlarge3.jpg?itok=294FDKFaC:\Documents and Settings\bms292\My Documents\My Pictures\Kidney\Kidney%20cross%20section.jpg

Renal System Student's

Guide

|  |
| --- |
|  **BLOCK BOOK AND STUDENT GUIDE****OF****RENAL SYSTEM** |
| Academic year 1436-1437 (10 April– 19 May 2016)Female group |

## C:\Documents and Settings\bms292\My Documents\My Pictures\Kidney\Kidney%20cross%20section.jpg

**Copyright Statement**

*This material is protected by copyright laws. For any other purposes other than teaching and research in the King Saud University, no part may be reproduced or copied in any form or by any means without prior permission of the King Saud University.*

**

*© King Saud University, Saudi Arabia, 2012*

|  |
| --- |
| **TABLE OF CONTENTS** |
| General Information | 5 |
| Faculty | 6 |
| List of Problem – based Learning Cases | 7 |
| Block General Objectives | 8 |
| Teaching and Learning Modes | 8 |
| Specific lecture objectives by Department : |
| * Department of Anatomy
 | 9-17 |
| * Department of Pathology
 | 18-22 |
| * Department of Biochemistry
 | 23-27 |
| * Department of Physiology
 | 28-38 |
| * Department of Immunology
 | 39-40 |
| * Department of Microbiology
 | 41-49 |
| * Department of Pharmacology
 | 50-57 |
| Picture5Time Table / Block Schedule | 58-61 |
| Assessment of the students in the Block | 62-64 |
| References | 65-67 |
| Academic Support Team | 68 |
| Feedback to students on PBL Performance | 69 |
| Assessment of Student’s Performance in PBL | 70 |
| Students’ evaluation of their PBL tutor  | 71 |
| Student Rating of lecture | 72 |

**Message from the dean**

We are pleased with your progress in the medical program and your achievements. Being a first year medical students is a great opportunity for you to consolidate what you have learnt in the preparatory year and prepares you for the clinical skills and competencies needed in the clinical years. The Department of Medical Education through its different units is working hard to create an integrated and innovative curriculum that builds on the changes introduced in the preclinical years and enforces best teaching/learning approaches in the design of the new medical curriculum. As you are aware, the College of Medicine at King Saud University is one of the best colleges not just in the Kingdom of Saudi Arabia but proved to be one of the best in the gulf region, and the Middle East. It also has its international influence among the best colleges of medicine worldwide. This makes us proud of our achievements and provides you with an insight about the quality of teaching and research that we have reached and our continuous work to maintain our standards.

Therefore, the medical curriculum aims at preparing you and equipping you with the best training and clinical skills to become a medical graduate that fulfils the highest international standards. Therefore, the focus of the curriculum is to enhance a number of skills such as case-based learning, critical thinking, self-directed learning, deep understanding of concepts, application of knowledge learnt, and how to make decisions on the basis of evidence. The curriculum also aims at enhancing your skills in areas such as professionalism, e-learning, task-based learning, and preparing you for life-long learning. The design of the curriculum encourages small group learning, use of cases for discussion, lectures, student-led seminars, bed-side teaching, task-based learning, use of multimedia and e–learning as modes for teaching and learning. The use of wide range of teaching and learning modes and small group discussion will help you to become active learners, and work with other students in your group as a team.

I wish you all the best during your academic year and would encourage all of you to get the best out of the teaching and learning opportunities provided to you during this year. Our teaching staff and clinicians would be very happy to help you on any issue that you need help with.

**Dr. Fahad Abdullah AlZamil**

**Dean, College of Medicine and the Supervisor of University Hospitals**

 **Message from Renal Block Chair**

I would like to welcome you, to the renal block as part of your first year curriculum. As you know, the first two years curriculum is organized in an integrated manner based on organ systems.

The renal block is four weeks long, which was designed to introduce you to the renal system in a weekly theme fashion, Acute Kidney Injury (AKI), urinary tract infection, nephrotic/ nephritic syndrome and Chronic Kidney Disease (CKD) respectively, the Material is integrated across multiple disciplines including Immunology, pathology, physiology, microbiology, pharmacology, anatomy, biochemistry, and radiology.

The learning formats are lectures, laboratories, small group discussion and self-study. Your evaluation is based on two written exams and small group discussion participation.

Finally, do not hesitate and feel free to provide me with your feedback directly in person or by e mail.

I’m looking forward to an exciting, rich and smooth four weeks and I wish you all the best.

**Mohammed Abdulrahman Al Ghonaim MBBS, FRCP (C), FACP**

**Chairman**

**Renal Block**

****

**General information**

***Block Title : Renal Block***

***Block Code & Number : Ren114***

***Credit Hour : 4***

******

***Block Duration : 4 Weeks***

***Block Dates : 10 April 2015 – 19 May 2016***

***Block Chairman : Dr. Mohammed Al-Ghonaim***

***Committee Members : Dr. Mohammed Vohra***

 ***Dr. Rana Hasanato***

 ***Dr. Hala Kfoury***

 ***Prof. Zahid Shakoor***

 ***Dr. Malak Mohsen El Hazmi***

 ***Dr. Ishfaq Bukhari***

 ***Dr. Ahmed Fahim Ahmeda***

**Faculty staff**

|  |  |  |  |
| --- | --- | --- | --- |
| **Name** | **Department** | **Extension** | **E-mail** |
| **Dr. Jamilah El-Medany** | Anatomy | 91696 | galmadani@ksu.edu.sa  |
| **Dr. Rana Mohammad Walid Hasanato** | Biochemistry | 79093 | ranamomen@yahoo.com rhasanato@ksu.edu.sa  |
| **Dr. Sumbul Fatma** | Biochemistry | 71321 | sumbulfatma@gmail.com  |
| **Dr. Reem Mohammad Salam** | Biochemistry | 71339 | rsallam\_10@hotmail.com sallam@ksu.edu.sa  |
| **Dr. Sanaa Al Shaarawi** | Embryology | 77094 | salsharawi@ksu.edu.sa  |
| **Dr. Hend Alotaibi**  | Immunology | - | Dr\_halotaibi@yahoo.com |
| **Dr. Raeesa Abdultawab** | Histology | 77094 | drraeesama@gmail.com  |
| **Prof. Hanan Habib** | Microbiology | 72456 | hahabib@ksu.edu.sa  |
| **Dr. Malak El-Hazmi** | Microbiology | 72693 | melhazmi@KSU.EDU.Sa  |
| **Dr. Fawzia Al-Otaibi** | Microbiology | 71088 | ofawzia04@ksu.edu.sa ofawzia@ksu.edu.sa  |
| **Dr. Shaesta Zaidi** | Pathology | - | snz24@hotmail.com snz24@yahoo.com  |
| **Picture5Dr. Hala Kfoury** | Pathology | 71889 | halakfoury@hotmail.com hkfoury@ksu.edu.sa  |
| **Dr. Sufia Husain** | Pathology | - | sufiahusain@hotmail.com  |
| **Dr. Mona Soliman** | Physiology | 91178 | msoliman1@KSU.EDU.SA  |
| **Dr. Manan Alhkbany**  | Physiology | 56121 | malhakbany@gmail.com  |
| **Prof. Hanan Hagar** | Pharmacology | 71342 | hananhhagar@yahoo.com  |
| **Dr. Abdulrahman Al Eissa**  | Radiology | Pager 0936 | Aaleisa90@gmail.com  |

|  |
| --- |
| **List of the Problem-Based Learning Cases** |

****

The table below summarizes the PBL cases to be discussed in the Renal Block.

|  |  |  |
| --- | --- | --- |
| **Week** | **Case No.** | **Case title** |
| **Week 1(Sunday & Wednesday)** | Case 1 | “Postoperative reduction in urine output…” |
| **Week 2 (Sunday & Wednesday)** | Case 2 | “Painful micturation...” |
| **Week 3 (Sunday & Wednesday)** | Case 3 | “My body is swollen…” |
| **Week 4 (Sunday & Wednesday)** | Case 4 | “I feel tired…” |

**Instructions:**

The cases listed above will be discussed by students in their small groups. Each group is about 8 to 12 students. Each case will be discussed in two tutorials, on Sunday and Wednesday. Each tutorial is two hours long.

**Attendance of Small Group Learning tutorials:**

Students must attend all small group learning tutorials. If a student is not well, he/she needs to provide a medical certificate from their family doctor. If a student misses out to attend four tutorials without acceptable reason, he/she might not be allowed to attend the final examination.

**Students Roles in Small Group Learning Tutorials:**

The design of the curriculum encourages small group discussion and student-centered learning. To achieve these goals there is a need for establishing good group dynamics, interpersonal skills, and effective communication. These elements will ensure that learning is an enjoyable process and rewarding to each member in the group. Therefore, students play a vital role in making a difference in their groups.

.

|  |
| --- |
| **Block General Objectives** |

**By the end of the course, students should be able to:**

* Understand the relationship between the structure of the different components of the renal system and their functions.
* Discuss the pathology, microbiology, pathogenesis and factors contributing to the development of most common diseases affecting renal system
* Use basic science to explain patient’s sign and symptoms; interpret investigation results, and provide justification for their views.
* Develop communication skills and explore biopsychosocial and ethical issues in their assessment of the case.
* Use clinical cases to apply knowledge learnt, generate hypotheses, build an enquiry plan, and use evidence to refine their hypothesis, justify different views.
* Design a brief management plan, and understand the pharmacological basis of drugs used in the management of common diseases affecting the renal system.
* Enhance their communication skills, and practice with the help of simulation patients to improve their communication in relation to respiratory case scenarios.

**Teaching and Learning Modes:**

In an integrated curriculum like our curriculum, we use a wide range of teaching and learning strategies to ensure that learning meets the different needs of the students. These strategies include:

* Small group discussion
* Lectures
* Student-led seminars
* Practical classes.
* Clinical skills
* Independent learning
* Writing an essay or mini thesis.

**Specific Lecture Objectives by Department**

|  |
| --- |
| **Department of Anatomy** |

**Lecture 1 ANATOMY OF KIDNEY**

**Objectives:**

*By the end of the lecture, the student should be able to:*

* Describe the anatomical features of the kidneys (position, extent, relations, hilum, peritoneal coverings).
* Describe the internal structure of the kidney, segments, blood supply and lymphatics).

**Introduction:**

The kidneys are retroperitoneal structures on the posterior abdominal wall and oriented with the hilum directed medially. The hilum is the site of entry and exit of the renal blood vessels and the ureter. They produce urine that is conveyed by the ureters to the urinary bladder in the pelvis.

**Key Outlines:**

1. Position and extent of kidney
2. Coverings of kidney (perinephric fat, renal fascia & paranephric fat)
3. Peritoneal reflection
4. Relations
5. Structures at hilum and their arrangements
6. Internal structure of kidney
7. Blood supply and vascular segments
8. Lymphatic drainage

**Take home message:**

Knowledge of normal anatomy, relations, blood supply and internal features of kidney is essential to understand the abnormalities of kidney.

**Recommended Books:**

* Drake RL, Vogl W and Mitchell AWM . Gray’s Anatomy for Students. Philadelphia: Elsevier Churchill Livingstone.
* Snell RS. Clinical Anatomy for Medical Students.. Philadelphia: Lippincott Williams & Wilkins.

**Lecture 2 ANATOMY OF URETERS, BLADDER AND URETHRA**

**Objectives:**

*By the end of the lecture, the student should be able to:*

* Describe the anatomical features of the ureter.
* Describe the anatomical features of the urinary bladder
* Describe the anatomical features of the male & female urethra
* Describe the blood supply, nerve supply and lymphatic drainage of urinary bladder & urethra.

**Introduction:**

The ureters are muscular tubes that convey urine from the kidneys to the urinary bladder. They descend retroperitoneally into the pelvis and penetrate the bladder wall obliquely from the posterolateral angle. The urinary bladder is a temporary reservoir for urine located in pelvis. In infants and young children, the urinary bladder lies in abdomen. The urine is expelled from the bladder through muscular tube called urethra.

**Key Outlines:**

1. Position and course of ureters
2. Constrictions of ureters
3. Location and relations of urinary bladder
4. Internal structure of urinary bladder; trigone
5. Differences between male and female urethra
6. Blood supply of ureters, urinary bladder and urethrae
7. Lymphatic drainage of ureters, urinary bladder and urethrae

**Take home message:**

Knowledge of normal anatomy, relations and blood supply of ureters, urinary bladder and urethra is essential to understand the abnormalities of these organs.

**Recommended Books:**

* Drake RL, Vogl W and Mitchell AWM. Gray’s Anatomy for Students. Philadelphia: Elsevier Churchill Livingstone.
* Snell RS. Clinical Anatomy for Medical Students. Philadelphia: Lippincott Williams & Wilkins.

**Lecture 3 DEVELOPMENT OF KIDNEYS & URETERS**

**Objectives:**

*By the end of the lecture, students should be able to:*

* Identify *the embryological origin of kidneys & ureters.*
* Differentiate between *the 3 systems of kidneys* during development.
* Describe *the development of collecting & excretory parts of permanent kidney.*
* Describe *the fetal kidney* & identify the pre- and postnatal changes that occur in the kidney.
* Enumerate *the most common anomalies of kidneys & ureters.*

**Introduction:**

Kidney develops from the intermediate mesoderm. It develops in three consecutive phases: pronephros, mesonephros and metanephros. Metanephros develops in the pelvic region and gives the permenant kidney which becomes functional in 9th week. The excretory parts of kidney develops from metanephric blastema and the collecting part develops from the ureteric bud which is derived from mesonephric duct. Later the kidney ascends to lie in the abdominal cavity.

**Key Outlines:**

* Formation of intermediate mesoderm
* Formation of pro-, meso- and metanephros
* Formation of metanephric blastema and its derivatives
* Formation of ureteric bud and its derivatives
* Formation of glomeruli and functioning of kidneys
* Ascent of kidney and changes in blood supply
* Rotation of hilum
* Postnatal changes in kidneys
* Common anomelies related to development of kidney

**Take home message:**

Knowledge of the development of kidneys & ureters helps the students to understand the related abnormalities.

**Recommended Books:**

* More KL (2002). The Developing Human. Philadelphia: Saunders WB.
* Sadler TW. (2005) Langman’s Essential Medical Embryology. Philadelphia: Lippincott Williams & Wilkins.

**Lecture 4 DEVELOPMENT OF URINARY BLADDER & URETHRA**

**Objectives:**

By the end of the lecture the student is able to;

* Describe the cloaca and the formation of the urogenital sinus.
* Discuss the division of the urogenital sinus into various parts and name the adult organs that are derived from each part.
* Describe how the caudal parts of the mesonephric ducts and ureters are absorbed into the urogenital sinus and the significance of this embryonic event.
* Discuss the position of the urachus and its significance and fate.
* Describe the various anomalies concerned with the urinary bladder and urethra.

**Introduction:**

Urinary bladder develops from the urogenital sinus, a division of the cloaca. The primitive urogenital sinus divides into the cranial, vesical part (that forms most of the bladder), the middle, pelvic part (that forms main part of male urethra and entire female urethra) and the caudal, phallic part grows towards genital tubercle. The trigone is derived from the absorbed caudal end of the mesonephric ducts

**Key Outlines:**

* Division of cloaca and the formation of the urogenital sinus.
* Division of the urogenital sinus into various parts and derivatives of each part
* Urachus and its significance and fate.
* Anomalies related to the development of urinary bladder and urethra.

**Take home message:**

Knowledge of the development of urinary bladder and urethra helps the students to understand the related abnormalities.

**Recommended Books:**

* More KL. The Developing Human. Philadelphia: Saunders WB.
* Sadler TW. Langman’s Essential Medical Embryology. Philadelphia: Lippincott Williams & Wilkins.

**Lecture 5 HISTOLOGY OF THE KIDNEY**

**Objectives:**

By the end of the lecture the student should be able to:

* Identify the different zones of the kidney.
* Describe the detailed microscopic structures of the renal cortex and medulla.
* Describe the microscopic structure of the renal calyces and pelvis.
* Describe the microscopic structure of the juxtaglomerular apparatus

**Introduction:**

The kidney is formed of cortex and medulla in addition to the minor and major calyces and the renal pelvis. The structural and functional unit of the kidney is the nephron which is formed of renal corpuscle, proximal tubule, distal tubule (which share in the formation of juxtaglomerular apparatus together with the afferent arteriole) and a loop of Henle. The renal corpuscle is formed of renal or Bowman’s capsule and the glomerulus. The medulla is condensed with renal corpuscles, proximal and distal tubules in addition to thin vascular interstitial tissue, while the medulla is condensed with loop of Henle and collecting ducts that lead to the papillae of the medullary pyramids.

**Key Outlines:**

* Microscopic structure of the renal cortex and medulla.
* Histology of renal corpuscle, proximal distal tubules, loop of Henel, and collecting tubules & ducts.
* Histological structure of juxtaglomerular apparatus.

**Take home message:**

Knowledge of the microscopic structure of renal cortex and medulla as well as the calyces and the wall of renal pelvis helps the students to interpret the histopathological changes in these structures in different kidney lesions.

**Recommended Books:**

* Gartner LP and Hiatt JL. Color Textbook of Histology. 2nd ed. Philadelphia: Saunders WB.
* Young B, Lowe JS, Stevens A and Heath JW. Wheater’s Functional Histology. 5th ed. London: Churchill Livingstone.

**Lecture 6 HISTOLOGY OF THE URINARY PASSAGE**

**Objectives:**

By the end of the lecture the student should be able to describe the microscopic structures of the wall of:

* Ureter.
* Urinary bladder.
* Male and female urethra.

**Introduction:**

The microscopic structure of the wall of the ureter and urinary bladder is formed of three layers; mucosa, muscle layer and adventitia and/ serosa. Mucosa is formed of transitional epithelium and CT. lamina propria. The muscle layer is formed of smooth muscle that arranged as inner longitudinal and outer circular layers, in the wall of the bladder there is a third layer of smooth muscle. The male urethra is longer than that of the female and is lined with different types of epithelia ranging from transitional to stratified squamous epithelium.

**Key Outlines:**

* Microscopic structure of the renal pelvis and ureter
* Microscopic structure of the urinary bladder
* Microscopic structure of the urethra

**Take home message:**

Knowledge of the microscopic structure of the wall of different part of the urinary passage enable the students to interpret the histopathological changes confronted in the different urinary passage diseases.

**Recommended Books:**

* Gartner LP and Hiatt JL. Color Textbook of Histology. Philadelphia: Saunders WB.
* Young B, Lowe JS, Stevens A and Heath JW. Wheater’s Functional Histology. London: Churchill Livingstone.

**PRACTICAL SESSIONS**

**Practical 1: ANATOMY OF KIDNEY**

**Objectives:**

*By the end of the practical session, the student should be able to:*

* Demonstrate and compare the position, extent and relations of both kidneys
* Demonstrate and compare the peritoneal reflection in both kidneys
* Demonstrate and compare the anatomical features (shape, borders & hilum) of both kidneys.
* Identify the structures at the hilum and their arrangement (VAU)
* Identify the cortex and medulla in a longitudinal section of the kidney and demonstrate the following structures: renal column, renal pyramid, renal paplilla, minor & major calyces and renal pelvis.
* Demonstrate the renal sinus and its contents
* Identify the renal vessels and demonstrate their distribution inside the kidney
* Demonstrate the renal vascular segments

**Recommended Books:**

* McMinn RH. McMinn’s Color Atlas of Human Anatomy. Fifth Edition. Mosby Publisher, UK.
* Netter FH. Atlas of Human Anatomy. 4th ed. Philadelphia: Saunders WB.
* Agur AMR and Dalley AF. Grant’s Atlas of Anatomy. 11th ed. Philadelphia: Lippincott Williams & Wilkins.

**Practical 2: ANATOMY OF URETER, URINARY BLADDER AND URETHRA**

**Objectives:**

*By the end of the practical session, the student should be able to:*

* Demonstrate the position, extent and relations of both ureters
* Describe the course of ureter & identify the site of ureteric constriction
* Demonstrate the position and relations of urinary bladder
* Demonstrate the anatomical features (shape, surfaces, angles) of urinary bladder
* Demonstrate the peritoneal reflection in urinary bladder
* Demonstrate the internal structure of urinary bladder (mucosal folds, ureteric and urethral orifices, trigone, uvula vesicae)
* Demonstrate and differentiate between male & female urethra regarding length, structure, course & function.
* Identify the blood supply of ureter, urinary bladder and urethra

**Recommended Books:**

* McMinn RH. McMinn’s Color Atlas of Human Anatomy. Fifth Edition. Mosby Publisher, UK.
* Netter FH. Atlas of Human Anatomy. 4th ed. Philadelphia: Saunders WB.
* Agur AMR and Dalley AF. Grant’s Atlas of Anatomy. 11th ed. Philadelphia: Lippincott Williams & Wilkins.

**Practical 3: HISTOLOGY OF KIDNEY**

**Objectives:**

*By the end of the practical session, the student should be able to:*

* Identify a section of kidney under the microscope
* Demonstrate the microscopic structure of the renal cortex and medulla.
* Demonstrate the histology of renal corpuscle, proximal distal tubules, loop of Henel, and collecting tubules & ducts.
* Demonstrate the histological structure of juxtaglomerular apparatus.

**Practical 4: HISTOLOGY OF URETER, URINARY BLADDER AND URETHRA**

**Objectives:**

*By the end of the practical session, the student should be able to:*

* Demonstrate the microscopic structure of the renal pelvis and ureter.
* Demonstrate the microscopic structure of the urinary bladder and male and female urethra
* Relate the anatomical structure of the urinary tract with its functions

|  |
| --- |
| **Department of Pathology** |

**Lecture 1 ACUTE KIDNEY INJURY**

**Objectives:**

Upon completion of this lecture the students will be able to:

1. Describe the guidelines for performing renal biopsy.
2. Recognize the different types of acute kidney injury.
3. Recognize the clinical manifestations of acute kidney injury.
4. Describe the pathological findings in acute kidney injury.

**Introduction:**

Acute kidney injury (AKI) may occur in ambulant and hospitalized patients. The renal biopsy is not performed in all cases of acute kidney injury, especially when ischemia or drugs are suspected to be the cause of the kidney injury and the patient is treated conservatively, however, in complicated conditions, the renal biopsy will disclose in the majority of the situations, the cause of the acute kidney injury. (glomerular, interstitial, vascular or tubular origin) , hence the lecture will emphasize the importance of the renal biopsy in AKI.

**Key Outlines:**

1. Brief review of the normal anatomy and histology of the kidney and urinary tract.
2. Terminology used in renal diseases.
3. Etiology of acute kidney injury.
4. Pathophysiology of acute kidney injury.
5. Clinical manifestations with diagnostic approach.
6. Pathological evaluation: Gross and histological findings in cases of acute renal injury and failure.
7. Conclusion and “take home” messages.

**Summary:**

Acute kidney injury is related in the majority of cases to acute tubular necrosis, however, there are other factors which may cause AKI, such as crescentic glomerulonephritis, vasculitis, acute tubulo-interstitial nephritis etc…. The biopsy will help to recognize the cause of the AKI, and outline for the clinician the adequate therapeutic regimen for each patient.

**Take home message:**

The student will be able to define AKI, recognize the causes and the pathogenetic mechanisms involved in AKI. The student will be able to describe the different histopathological features seen in cases of AKI. Furthermore, the student will understand the different steps involved in the diagnosis and treatment of AKI.

**Recommended Books:**

* Pathologic Basis of Disease, Robbins and Cotran

**Lecture 2 PATHOLOGY OF THE RENAL ALLOGRAFT**

**(IMMUNE AND NON-IMMUNE MEDIATED INJURIES)**

**Objectives:**

At the end of the lecture the students will be able to:

1. Recognize the concept of renal allograft.

Describe the pathology of rejection and differentiate acute cell-mediated and antibody-mediated rejection.

1. Differentiate between acute and chronic rejection.
2. Brief account on principal opportunistic infections and drug toxicity encountered in renal transplant recipients.

**Introduction:**

The renal biopsy for allograft dysfunction is an integral part of the diagnostic tools used to recognize the underlying mechanisms to the graft abnormality, and hence represent a solid basis for the selection of the therapeutic regimen to be given. The lecture is meant to be a practical guide to the approach towards renal allograft pathology with emphasis on the immune and non-immune causes of graft failure.

**Key Outlines:**

1. Adequacy of the renal allograft biopsy.
2. Acute T-cell mediated rejection.
3. Acute antibody-mediated rejection.
4. Pathology of chronic rejection.
5. Pathology of the principal infections of the renal allograft: CMV-polyoma viruses.
6. Pathology of acute and chronic drug toxicity.

**Summary:**

Renal allograft failure may be secondary to a variety of causes. Renal allograft biopsy will help in the recognition of the different types of rejection if and when present like: Acute versus Chronic, cell-mediated versus antibody- mediated. The biopsy will also help in the diagnosis of any drug toxicity, (acute versus chronic) and in the recognition of the histological abnormalities secondary to infections.

**Take home message:**

The student will learn the pathogenetic mechanisms involved in renal allograft rejection. The student has to recognize the importance of the biopsy in differentiating between immune related dysfunction (rejection) and other non-immune causes (i.e infections, drug toxicity…) as well as to the treatment to be given in such cases.

**Recommended Books:**

* Pathologic Basis of Disease, Robbins and Cotran

**Lecture 3 TUMORS OF THE KIDNEY AND URINARY BLADDER**

**Objectives:**

At the end of the lecture the students will be able to:

1. Recognize common benign tumors of the kidney.
2. Describe the pathological features of renal cell carcinoma and Wilm’s tumor.
3. Recognize the predisposing factors and features of transitional cell and squamous carcinoma of the urinary bladder.

**Introduction:**

A remarkable variety of benign and malignant tumors arise in the kidneys and urinary tract of adults and children. The diagnostic challenges that these lesions pose to the pathologist are made greater by the wide spectrum of appearances of the benign and more specifically malignant tumors of the kidneys and urinary tract. The lecture will address the different histopathological variants of these tumors arising in the upper and lower urinary tract. Special emphasis on clear cell carcinoma of the kidney and transitional cell/squamous carcinomas of the urinary bladder will be considered.

**Key Outlines:**

1. Benign tumors of the kidney: Adenoma and Angiomyolipoma.
2. Renal Cell Carcinoma: Incidence, Clinical Presentation, Gross and Histological Features and Prognosis.
3. Wilm’s tumor (nephroblastoma): Incidence, Clinical Features, Genetic and Histological Characteristics.
4. Transitional Cell and Squamous Carcinoma: Predisposing Factors, Incidence, Clinical Pathological Features and Prognostic Indicators (Grade and Stage).

**Summary:**

Renal cell carcinoma in adults and Wilm’s tumor in children, as well as the transitional cell carcinoma of the urinary tract including the bladder are entities to be recognized in the proper clinical setting and associated symptoms and signs. The association of squamous cell carcinoma with schistosomiasis is a well-known occurrence. The prognosis of all malignant tumors of the kidney and urinary tract is related to the grade, stage and appropriate treatment.

**Take home message:**

The student will become aware of the various types of benign and malignant kidney and urinary tract neoplasms, their occurrence as part of a syndrome together with the clinical and pathological features and the prognosis of these lesions.

**Recommended Books:**

* Pathologic Basis of Disease, Robbins and Cotran

**Lecture 4 Nephrotic /Nephritic syndrome**

**Objectives:**

At the end of the teaching sessions (2 lectures) the students will be able to:

1. Recognize the five major renal glomerular syndromes.
2. Describe the main differential pathological diagnosis for each syndrome.
3. Perform a clinicopathological correlation.
4. Describe the patterns of injury of each syndrome.
5. Brief account on congenital/inherited nephropathies.

**Introduction:**

The renal biopsy for non-neoplastic kidney diseases has gained tremendous importance in the last few decades. The lecture is meant to be a practical guide to the approach to renal biopsy in the five major clinically outlined glomerular syndromes. The patterns of injury in the nephrotic, nephritic, rapidly progressive ,asymptomatic hematuria/proteinuria and chronic renal failure (chronic glomerulonephritis) will be addressed. The segregation of the pattern of injury in the glomeruli into focal ,diffuse, segmental or global will be emphasized. The assessment of the other components present on the renal biopsy will be considered. The evaluation of the kidney biopsy and correlation with the clinical findings is also of utmost importance.

**Key Outlines:**

1. The nephrotic syndrome: (Minimal change, FSGS, membranous, diabetes).
2. The nephritic syndrome: (Acute post streptococcal Glomerulonephritis GN, Membrano-proliferative GN, and renal manifestations of Systemic Lupus Erythematosus).
3. Rapidly progressive GN: (Crescentic GN)
4. Asymptomatic Hematuria / Proteinuria: IgA Nephropathy.
5. The Chronic Nephritic Syndrome: (Chronic Renal Failure).
6. A brief account on congenital nephropathies will also be given.

**Summary:**

The five major clinical glomerular syndromes are associated with different patterns of injury. The approach to a renal biopsy taken in the setting of any of these clinical syndromes is systematic, starting with assessment of the renal biopsy adequacy to the analysis, the pathological features of the four components of the renal biopsy and the drawing of the final conclusion (diagnosis).

**Take home message:**

The student will investigate the approach to a kidney biopsy in a given setting and into the logical steps to be taken to reach a diagnosis either in the context of a disease primarily involving the kidney or in a situation where involvement of the kidney is part of a systemic disease. Furthermore, the student should recognize the pathological findings related to chronicity.

**Lecture 5** **PATHOLOGY OF THE INFECTIONS OF THE KIDNEY AND**

**URINARY TRACT**

**Objectives:**

At the end of the two lectures the students will be able to:

1. Recognize the predisposing factors for infections of the kidney and urinary tract.
2. Describe the different types of infections in the kidney and urinary tract.
3. Recognize the clinicopathological features of acute and chronic pyelonephritis.
4. Describe the causes of urinary tract obstruction.
5. Recognize drug induced nephritis.

**Introduction:**

Urinary tract infections are common diseases seen in the medical practice. Systemic as well local factors play a major role in the initiation, recurrence and outcome of these infections. The predisposing factors including the congenital as well as the acquired causes will be discussed. A pathological description of the different types of infection will be considered. An overview of the cystic diseases of the kidney as well as medications related nephritis will be considered.

**Key Outlines:**

1. Urinary Tract Obstruction: causes and clinical manifestations in children and adults.
2. Infections of the Urinary Tract: Predisposing Factors and Clinical Manifestations.
3. Pathology of Acute and Chronic Pyelonephritis including causes and complications of urolithiasis.
4. Drug induced interstitial nephritis and renal papillary necrosis (necrotizing papillitis).

**Summary:**

Urinary tract infections induce a variegated histopathological spectrum depending on the location, type of infectious agent, predisposing factors as well as the associated systemic conditions. An overview of the different patterns will be considered with clinical correlation.

**Take home message:**

The student will investigate the causes and pathogensis of kidney and urinary tract infections and the histological findings related to the upper and lower urinary tract infections.

**Recommended Books:**

* Pathologic Basis of Disease, Robbins and Cotran

|  |
| --- |
| **Department of Biochemistry** |

**Lecture 1 Chemical examination of urine**

**Objectives:**

Upon completion of this lecture, students should be able to:

* differentiate between normal and abnormal constituents of urine including: Proteins, sugars, ketone bodies, nitrite, bile pigments, blood etc.
* know the clinical conditions in different types of proteinuria, blood urea and glycosuria etc.

**Introduction:**

Urine is obtained after glomerular filtration and has normal and abnormal constituents. Abnormal constituents in urine such as, protein, glucose, ketone bodies, blood, nitrite etc are helpful in the diagnosis of different clinical conditions.

**Key Outlines:**

Urine is a fluid obtained through renal glomeruli with considerable changes which occurs in this filtrate before it is excreted as urine. Normal urine contains about 1.5 liters of water per day. Normal Urine contains organic and inorganic solids, chief inorganic solids include, Sodium Potassium, Chlorides, Small amounts of Ca, Mg, S & phosphates, Traces of Fe, Cu, Zn and I2. Whereas, Chief organic solids in normal urine includes, non protein nitrogen (NPN) compounds, Organic acids, Sugars, Traces of proteins, vitamins, hormones and pigments.

Proteins are normally less than 200 mg protein is excreted in the urine daily; more than this level leads to a condition called “Proteinuria”. There are 3 different types of protein urea and will be discussed in detail with clinical conditions. Other abnormal constituents in urine such as glucose, ketone bodies and blood will be discussed and their clinical conditions.

**Take home message:**

* Normal and abnormal constituents of urine.
* Clinical conditions in different type of proteinurea.
* Abnormal constituents present in urine in different clinical conditions.

**Recommended Books:**

Lecture notes: Clinical Biochemistry, 8th edition, Geoffrey Beckett, Simon Walker, Peter Rae, Peter Ashby. January 2010, © 2010, Wiley-Blackwell.

Key words:

Urine, constituents of urine, proteinurea, glycosuria, hematuria, hemoglobin urea, ketone urea.

**Lecture 2 Kidney Stones**

**Objectives:**

Upon completion of lectures, students should be able to:

* list the general physiological and pathological factors that favor kidney stones formation.
* identify the various chemical constituents of kidney stones with reference to the characteristics of each type that help in their clinical diagnosis, and etiological factors that should be considered in medical treatment and prevention of recurrence.

**Introduction:**

Kidney stones formation is favored by several factors. One or more of these factors might be responsible for the formation of certain types. It is critical to know the etiological factors in order to properly diagnose and treat a currently available stone in addition to prevention of further stones. Kidney stones can be differentiated according to chemical constituents. Studying the physical and chemical nature of each kind may help in investigating the stone. A patient with a kidney stone has to be thoroughly investigated by an objective protocol.

**Key Outlines:**

In the beginning of the lecture, a definition of kidney stones is given. Factors helping the formation of kidney stones are discussed in brief. Next, the most important types of kidney stones are discussed regarding etiological factors, laboratory and radiological diagnosis and medical treatment. At the final stage of the lecture, a summary of the management of a patient of a kidney stones is discussed.

**Take home message:**

Kidney stones are formed due to many factors that have to be carefully understood in order to proper investigation and treatment

**Recommended Books:**

Clinical Chemistry and metabolic Medicine. 7th edition, 2006

Keywords:

Kidney stones, factors for formation of kidney stones, investigation of a kidney stone

**Lecture 3 Inborn errors of amino acid metabolism**

**Objectives:**

Upon completion of this lecture, students should be able :

1. Identify the amino acid degradation and synthesis of non-essential amino acids
2. recognize the metabolic defects in amino acid metabolism that lead to genetic diseases

**Introduction:**

Amino acid metabolism is part of the whole body nitrogen metabolism. Nitrogen enters the body as amino acids in proteins and leaves the body as urea, ammonia etc. All 20 amino acids present in proteins are required for health. 12 out of the 20 amino acids can be synthesized by the body and the rest are supplied by diet. Errors in the amino acid metabolism can result in irreversible brain damage and early mortality.

**Key Outlines:**

In the first lecture, we will discuss how the body maintains its amino acid pool by degradation and synthesis. During degradation the α-amino group of amino acid is removed with the help of different enzymes like transaminases and deaminases and the remaining C-skeletons are is degraded to 7 products- pyruvate, acetyl CoA, acetoacetate, α-KG, succinyl CoA, fumarate and oxaloacetate. Ammonia obtained from deamination gets converted to urea in liver via urea cycle. Depending upon the end product of carbon skeletons, the amino acids are classified as glucogenic and ketogenic. The essential amino acids are provided in the diet and the non-essential amino acids are synthesized in the body. We will discuss the disease conditions when some of the non-essential amino acids become essential and how they can be targeted during the treatment. We will also be discussing the other diseases related to the elevated levels of amino acids or products of amino acids metabolism like role of homocysteine in atherosclerosis and brth defects like spina bifida.

In the second lecture, we will be discussing the hereditary disorders of amino acid metabolism. Inborn errors of metabolism are caused by the deficiency/loss of enzymes involved in the amino acid metabolism that leads to an accumulation of the product of that enzyme. The diseases discussed will include PKU, Maple Syrup Urine Disease, Alkaptonuria and Homocystinuria. The enzymes involved in the disorder, symptoms and treatment/management of the different conditions will be discussed.

**Take home message:**

* Degradation of amino acids involves removal of α-amino group followed by degradation of leftover carbon skeleton by various ways to products that can either enter the biosynthetic pathways or TCA
* Glucogenic amino acids whose catabolism yields pyruvate or one of the intermediates of TCA
* Ketogenic amino acids whose catabolism yields either acetoacetate or one of its precursor.
* The essential amino acids are provided in the diet and the non-essential amino acids are synthesized in the body
* Deficiency of the enzymes involved in amino acid metabolism leads to hereditary disorders like phenylketonuria, alkaptonuria etc.

**Recommended Books:**

Colored Atlas of Biochemistry (material given to students by tutors)

Illustrated Reviews of Biochemistry by Lippincott 4th edition.

Key words:

Transaminases, deamination, essential and non-essential amino acids, ketogenic, glucogenic

**Lecture 4 Renal Function Tests**

**Objectives:**

Upon completion of lectures, students should be able to:

* know the physiological functions of the kidney.
* describe the structure and function of the nephron.
* Identify the biochemical kidney function tests with special emphasis on when to ask for the test, the indications and limitations of each kidney function tests.
* interpret the kidney function tests properly.

**Introduction:**

The kidney is an important organ in the regulation of body homeostasis. Many diseases affect kidney functions. The biochemical kidney function tests e.g. plasma creatinine, creatinine clearance and serum urea are important for the diagnosis, prognosis, and follow up of treatment of renal diseases.

**Key Outlines:**

The kidney is an important organ on the regulation of water, electrolyte, acid-base balance and blood pressure, the kidney has an addition endocrine function. The routine kidney function tests including plasma creatinine, creatinine clearance and serum urea will be fully discussed including the indications, advantage, disadvantages and limitation of each one of the kidney function tests. Cockroft-Gault formula for the estimation of glomerular filtration rate (GFR) will be mentioned as an alternative to the 24 hours urine collection.

**Take home message:**

* The physiological functions of the kidney
* The importance of biochemical kidney function tests.
* Plasma creatinine is the most accurate kidney function test.
* Creatinine clearance is important for the diagnosis of the early (minimal) renal disease.
* Proper collection of urine for measurement of creatinine clearance is a problem
* Serum urea is affected by diet, protein catabolism and dehydration.

**Recommended Books:**

Bishop Duben-Engelkirk Fody, Clinical chemistry principles, procedure, correlations (fourth edition).

Key words: plasma creatinine, creatinine clearance, serum urea, GFR, Co.

|  |
| --- |
| **Department of Physiology** |

**Lecture 1 RENAL FUNCTIONS & GLOMERULAR FILTRATION**

**Objectives:**

At the end of this session, the students should be able to:

* Enumarate general functions of the kidney
* Identify and describe that the nephron is the structural and function Unit of the kidney
* Explaiin glomerular filtration membrane & filtration forces
* Describe mechanism of filtration & composition of the glomerular filtrate
* Calculate the net filtration pressure using parameters of Starling forces

**Introduction:**

Kidneys perform important functions to maintain homeostasis. The first renal process is filtration. In this lecture we will explain glomerular filtration membrane & filtration forces that favor or impair filtration.

**Key Outlines:**

* General functions of the kidney
* three basic renal processes; glomerular filtration, tubular reabsorption and tubular secretion
* Mechanism of filtration & composition of the filtrate
* glomerular filtration membrane & filtration forces

**Summary:**

We discussed the mechanism of filtration & composition of the glomerular filtrate. We can calculate the net filtration pressure using parameters of Starling forces.

**Take home messages:**

Filtration membrane, Hydrostatic & osmotic pressures in filtration and calculation of the net filtration pressure.

**Key Words:**

nephron, glomerular filtration, tubular reabsorption and tubular secretion, capillary hydrostatic pressure, glomerular filtration membrane, filtrate.

**Recommended Books:**

Guyton & Hall Textbook Of Medical Physiology 11th Ed

**Lecture 2 REGULATION OF GLOMERULAR FILTRATION**

**Objectives:**

At the end of this session, the students should be able to:

* Describe that the mechanism of urine formation include three basic processes; glomerular filtration, tubular reabsorption and tubular secretion
* Define GFR and quote normal value
* Identify and describe the factors controlling GFR in terms of starling forces, permeability with respect to size, shape and electrical charges and ultra-filtration coefficient
* Describe Intrinsic and extrinsic mechanism that regulate GFR
* Describe autoregulation of GFR & tubuloglomerular feedback mechanism

**Introduction:**

Kidneys perform important functions to maintain homeostasis. The mechanism of urine formation include three basic processes; glomerular filtration, tubular reabsorption and tubular secretion. The factors controlling GFR are starling forces, permeability with respect to size, shape and electrical charges and ultra-filtration coefficient.

**Key Outlines:**

* GFR and factors controlling GFR
* Autoregulation of GFR
* Intrinsic and extrinsic mechanism that regulate GFR

**Summary:**

Intrinsic and extrinsic mechanisms regulate GFR. The factors controlling GFR are starling forces, permeability and electrical charges and ultra-filtration coefficient.

**Take home messages:**

Definition of GFR and factors that regulate GFR.

**Key Words**

GFR, Filtration membrane, filtration fraction, autoregulation

**Recommended Books:**

Guyton & Hall Textbook Of Medical Physiology 11th Ed

**Lecture 3 RENAL CLEARANCE**

**Objectives:**

At the end of this session, the students should be able to:

* Describe the concept of renal plasma clearance
* Use the formula for measuring renal clearance
* Use clearance principles for inulin, creatinine etc. for determination of GFR
* Explain why it is easier for a physician to use creatinine clearance Instead of Inulin for the estimation of GFR
* Describe glucose and urea clearance
* Explain why we use of PAH clearance for measuring renal blood flow

**Introduction:**

We will discuss the concept of renal plasma clearance which is an important renal function. Renal plasma clearance is an important test to assess renal functional capability.Inulin, **C**reatinine clearance, PAH, glucose and urea clearance, will be described in detail.

**Key Outlines:**

* Concept of renal clearance & its formula
* Inulin, creatinine clearance for determination of GFR
* PAH clearance for measuring renal blood flow

**Summary:**

We will discuss the concept of renal plasma clearance and use of the formula for measuring renal clearance. Clearance principles for inulin, creatinine, urea, glucose, PAH etc. for determination of different renal concepts are discussed.

**Take home messages:**

Renal plasma clearance is an important test to assess renal function. We use creatinine instead of inulin to measure clearance. PAH clearance is used for measuring renal blood flow

**Key Words**

renal plasma clearance, inulin, PAH, creatinine, renal blood flow

**Recommended Books:**

Guyton & Hall Textbook Of Medical Physiology 11th Ed

**Lecture 4 PHYSIOLOGY OF MICTURITION**

**Objectives:**

At the end of this session, the students should be able to:

* Identify and describe the Functional Anatomy of Urinary Bladder
* Describe the mechanism of filling and emptying of the urinary bladder
* Cystometrogram
* Appreciate neurogenic control of the mechanism of micturition and its disorders.

**Introduction:**

Mechanism of filling and emptying of the urinary bladder. We will discuss neurogenic control of micturition and also disorders of micturition.

**Key Outlines:**

* Functional Anatomy of Urinary Bladder
* Describe the mechanism of filling and emptying of the urinary bladder
* Cystometrogram

**Summary:**

Describe the mechanism of filling and emptying of the urinary bladder with Cystometrogram will be discussed. Moreover, neurogenic control of the mechanism of micturition and its disorders are covered.

**Take home messages:**

Mechanism of filling of urinary bladder, cystometrogram and neurogenic control of the mechanism of micturition and its disorders.

**Key Words**

Urinary Bladder, micturition, neurogenic control

**Recommended Books:**

Guyton & Hall Textbook Of Medical Physiology 11th Ed

**Lecture 5 TUBULAR REABSORPTION**

**Objectives:**

At the end of this session, the students should be able to:

* Define tubular reabsorption, tubular secretion, transcellular and paracellular transport.
* Identify and describe mechanisms of tubular transport
* Describe tubular reabsorption of sodium and water
* Revise tubulo-glomerular feedback and describe its physiological importance
* Identify and describe mechanism involved in Glucose reabsorption
* Study glucose titration curve in terms of renal threshold, tubular transport maximum, splay, excretion and filtration
* Identify the tubular site and describe how Amino Acids, HCO3-, P04- and Urea are reabsorbed

**Introduction:**

Tubular reabsorption and tubular secretion are the second and third important renal processes in urine formation. Mechanisms of tubular transport with examples of some important substances like glucose, amino acids and some electrolytes will be discussed.

**Key Outlines:**

Nephron regional specificity to describle Tubular reabsorption and tubular secretion. Study glucose titration curve in terms of renal threshold, tubular transport maximum, splay, excretion and filtration Identify the tubular site and describe how Amino Acids, HCO3-, P04- and Urea are reabsorbed

**Take home messages:**

How Tubular reabsorption and tubular secretion interact to regulate body homeostasis.

**Key Words**

tubular reabsorption and tubular secretion, tubulo-glomerular feedback, Glucose reabsorption, Urea, Amino Acids, HCO3-, P04-

**Recommended Books:**

Guyton & Hall Textbook Of Medical Physiology 11th Ed

**Lecture 6 TUBULAR SECRETION**

**Objectives:**

At the end of this session, the students should be able to:

* Describe tubular secretion with PAH transport and K+
* Identify and describe the characteristic of loop of Henle, distal convoluted tubule and collecting ducts for reabsorption and secretion
* Identify the site and describe the influence of aldosterone on reabsorption of Na+ in the late distal tubules.

**Introduction:**

Tubular reabsorption and tubular secretion are important tubular transport mechanisms in urine formation.

**Key Outlines:**

Nephron regional specificity to describle Tubular reabsorption and tubular secretion. Tubular secretion of PAH and K+

**Take home messages:**

How Tubular reabsorption and tubular secretion interact to regulate body homestasis.

**Key Words**

PAH transport, K+ secretion

**Recommended Books:**

Guyton & Hall Textbook Of Medical Physiology 11th Ed

**Lecture 7 RENAL REGULATION OF BODY FLUID**

**Objectives:**

At the end of this session, the students should be able to:

* Identify and describe the role of the Sensors and Effectors in the renal regulation of body fluid volume & osmolality
* Describe the role of the kidney in regulation of body fluid volume & osmolality
* Understand the role of ADH in the reabsorption of water and urea
* Identify the site and describe the influence of aldosterone on reabsorption of Na+ in the late distal tubules.

**Introduction:**

Kidneys perform important functions to maintain homeostasis by regulating body fluids and electrolytes.

**Key Outlines:**

Role of the Sensors and Effectors in the renal regulation of body fluid volume

& osmolality

**Summary:**

We will describe the role of the Sensors and Effectors in the renal regulation of body fluid volume & osmolality. How the kidney helps the body in regulation of extracellular fluid volume & osmolality. The role of ADH in the reabsorption of water and urea will also be explained.

**Take home messages:**

Kidneys perform regulation of body fluid volume & osmolality via Sensors and Effectors.

**Key Words**

Sensors, body fluid volume, electrolytes, osmolality

**Recommended Books:**

Guyton & Hall Textbook Of Medical Physiology 11th Ed

**Lecture 8 URINE CONCENTRATING MECHANISMS**

**Objectives:**

At the end of this session, the students should be able to;

* Identify and describe that the loop of Henle is referred to as countercurrent multiplier and the loop and vasa recta as countercurrent exchange systems in concentrating and diluting urine
* Explain what happens to osmolarity of tubular fluid in the various segments of the loop of Henle when concentrated urine is being produced.
* Explain the factors that determine the ability of loop of Henle to make a concentrated medullary gradient
* Differentiate between water diuresis and osmotic diuresis
* Appreciate clinical correlates of diabetes mellitus and diabetes insipidus

**Introduction:**

Kidneys perform important functions to maintain homeostasis by making a dilute or concentrated urine. The concept of counter current multiplier and exchangers will be discussed.

**Key Outlines:**

* countercurrent multiplier and countercurrent exchangers
* Change in osmolarity of tubular fluid in the various segments of the loop of Henle
* Explain the factors that determine the ability of loop of Henle to make a concentrated medullary gradient
* diabetes insipidus & diabetes mellitus

**Summary:**

We will describe the role of countercurrent multiplier and countercurrent exchangers in concentrating the renal medullary interstitium. We will also explain that what happens to osmolarity of tubular fluid in the various segments of the loop of Henle when concentrated urine is being produced.

**Take home messages:**

Kidneys perform regulation of body fluid volume & osmolality by making a dilute or concentrated urine.

**Key Words**

Countercurrent multiplier, countercurrent exchangers, diabetes insipidus, diabetes mellitus, osmolality

**Recommended Books:**

Guyton & Hall Textbook Of Medical Physiology 11th Ed

**Lecture 9 BASICS OF ACID BASE PHYSIOLOGY**

**Objectives:**

### At the end of this lecture the student should be able to:

* Define: acid and base.
* Explain what is meant by strong and weak acids and bases
* List and identify the names/formulas for the common strong acids and strong bases.
* To explain the role of Henderson-Hasselbalch equation in acid-base regulation

**Introduction:**

Strong and weak acids and bases and their formulas. Importance of Henderson-Hasselbalch equation.

**Key Outlines:**

Importance of Henderson-Hasselbalch equation in acid-base regulation and pK.

**Summary:**

The concept of strong and weak acids and bases will be discussed. We will explain the role of Henderson-Hasselbalch equation in acid-base regulation.

**Take home messages:**

Acid base concepts. Differences between strong and weak acids and bases. Henderson-Hasselbalch equation and its clinical value.

**Key Words**

Strong and weak acids and bases, Henderson-Hasselbalch equation

**Recommended Books:**

Guyton & Hall Textbook Of Medical Physiology 11th Ed

**Lecture 10 BUFFER SYSTEMS**

**Objectives:**

**At the end of this lecture the student should be able to:**

* To define buffer system and discuss the role of blood buffers and to explain their relevant roles in the body
* To describe the role of kidneys in the regulation of acid-base balance
* To describe the role of lungs in the regulation of acid-base balance

**Introduction:**

It is important to know about Interpretation of blood gas analysis and diagnosis of various acid base disorders in medical practice. Different buffer system in the body will be discussed. The role of kidneys and lungs in the regulation of acid-base balance will be explained.

**Key Outlines:**

The role of blood buffers and to explain their relevant contribution in the body How kidneys help in the regulation of acid-base balance and how does it compare with the role of lungs in the regulation of acid-base balance.

**Summary:**

Different buffer system in the body will be discussed. The role of kidneys and lungs in the regulation of acid-base balance will be explained.

**Take home messages:**

Kidneys help in excretion of non volatile acids while lungs get rid of volatile acids.

**Key Words**

Kidneys, lungs, acid-base balance, regulation, buffer system, non volatile acids

volatile acids

**Recommended Books:**

Guyton & Hall Textbook Of Medical Physiology 11th Ed

**Lecture 11 ACID BASE DISORDERS**

**Objectives:**

**At the end of this lecture the student should be able to:**

* To explain the principles of blood gas and acid-base analysis
* To interpret blood gas analysis and diagnose various acid base disorders
* Describe causes of acid base disorders
* Understand use of acid base nomograms

**Introduction:**

It is important to know about Interpretation of blood gas analysis and diagnosis of various acid base disorders in medical practice.

**Key Outlines:**

Interpretation of blood gas analysis and diagnosis of various acid base disorders

**Summary:**

Interpretation of blood gas analysis and its value in the diagnose various acid base disorders will be discussed. Causes of various acid base disorders will be explained. You will also be taught how to use acid base nomograms.

**Take home messages:**

Interpretation of blood gas analysis and diagnose various acid base disorders.

**Key Words**

Acid base disorders, blood gas and acid-base analysis

**Recommended Books:**

Guyton & Hall Textbook Of Medical Physiology 11th Ed

|  |
| --- |
| **Department of Immunology** |

Lecture 1: **Immunology of Renal Transplant**

**Objectives:**

1. To understand the diversity among human leukocyte antigens (HLA) or major histocompatibility complex (MHC)
2. To know the role of HLA antigens in transplant rejection
3. To be familiar with types of immune responses medicating transplant rejections and the importance of tissue matching
4. To understand the principles of management after transplantation

**Take home messages:**

1. HLA or MHC molecule miss-match can stimulate humoral and cell mediated immunity which is the main cause of rejection of transplants.
2. Cell mediated immune responses play a major role in transplant rejection
3. Tissue matching particularly for HLA-D antigens is important for successful transplantation
4. Immuno-suppresive therapy is usually required after transplantation

Lecture 2: **Immune Complex Nephritis**

**Objectives:**

1. To understand the importance of immune complexes in the pathogenesis of renal injury
2. To learn that the immune complexes form in the circulation and may get deposited in different tissues.
3. To understand the dynamics of immune complex deposition depending upon the size and the rate of immune complex formation
4. To identify different types of renal diseases based on the site of deposition of immune complexes.

**Take home messages:**

1. Deposition of immune complexes in kidneys appears to be the underlying cause in the pathogenesis of majority of glomerulo-nephritides.
2. Activation of the complement system is an integral part of the immune complex mediated nephritis and measurement of the complement proteins help in diagnosis and follow up of patients.
3. Diagnosis of different types of immune complex mediated glomeruloneprits is usuall made by detection of immune complexes in the renal tissue by immunofluoresence.

|  |
| --- |
| **Department of Microbiology** |

**Lecture 1**   **Cystitis**

**Prepared by : Prof .Hanan Habib**

**Objectives:**

By the end of the lecture ,students are expected to :

1-To define the term cystitis and who commonly get cystitis.

2- To describe the pathogenesis and risk factors of cystitis.

3- To know the most common causative organisms of cystitis

4- To recognize different types of cystitis ( infectious and non-infectious).

5- To recognize that venereal diseases can present with cystitis.

6- To understand the laboratory diagnostic of cystitis ( specimen collection, microscopic examination, chemical screening tests, and urine culture).

7-To know the antimicrobial agents suitable for the treatment and prevention of cystitis.

**Introduction:**

Cystitis is an acute infection of the urinary bladder ,common in women . the infection is localized to the bladder and usually there is no bacteremia. There are a number of reasons why women are susceptible , including short urethra ,sexual intercourse,pregnancy, decreased production of estrogen during menopause. In men, the most common cause of cystitis is persistence bacterial infection of the prostate .In both sexes, the presence of bladder stone, urethral stricture ,catheterization of the urinary tract and diabetes mellitus.

Pathogenesis :Cystitis is produced by frequent irritation of the mucosal surface of the urethra and the bladder. Infection results when the bacteria got access to the urinary bladder. These are usually flora of the large intestine. Toxins may be produced by uropathogens.

The most common etiologic agents are bacterial species, and 90% of these are *E.coli*, others include; *Klebsiella pneumonia ,Proteus* spp. ) Pseudomonas and gram positive bacteria ( *Enterococcus fecalis* , group *B Streptococcu*s & *Staphylococcus saprophyticus* [ honeymoon cystitis]) are increasingly frequent causes . *Candida* species isolated from diabetic or catheterized patients receiving antibiotics . Parasitic infection caused by *Schistosoma hematobium* in endemic areas.

symptoms of cystitis are usually of acute onset and these include ; painful sensation of urination (dysuria ) , frequency ,urgency and hematuria in 50% of patients. Venereal diseases ( gonorrhoea,*Chlamydia* ) can present with symptoms similar to cystitis. Cystitis can be differentiate it from urethritis in that it is of acute onset with dysuria, may hematuria and pubic pain. Urine in cystitis is cloudy ,malodorous and may be bloody.

**Differential diagnosis** ( types of cystitis): several distinct types of cystitis that have no infectious causes . These are;

1-Traumatic cystitis which common in females,

2-Interstitial cystitis with unknown cause ,probably autoimmune attack of the bladder

3- Eosinophilic cystitis may be attributed to *Schistosoma hematobium* or certain medications.

4- Hemorrhagic cystitis can occur as a result of radiotherapy or chemotherapy.

**Laboratory diagnosis**:

1-**specimen collection** : based on the collection of clean catch urine specimen ( **midstream urine sample[MSU]**) to bypass contamination by fecal flora. Other specimens such as supra-pubic aspiration or catheterization in children may be used .Cather urine should not be used .

2- **microscopic examination**: about 90% of patients with acute cystitis have >10 WBCs/mm3

Gram stained smear of uncentrifuged urine is more sensitive and specific. The presence of at least one organism per oil-immersion field is always indicative of infection. In addition microscopy of a urine sample can demonstrate the presence of white blood cells ,parasites or other substances.

3- **chemical screening tests**: Urinary dipstick test is performed for the rapid diagnosis of cystitis by detecting of nitrites that are released by bacterial metabolism and leukocyte esterase from inflammatory cells. These tests are nonspecific. .

4-**urine culture** : is important to identify the causative bacteria and its antimicrobial susceptibility pattern. Quantitative culture ( >10 5/cmm) is typical of UTI. however, lower counts ( 103 or less than 105 bacteria /mm3 ) may be considered indicative of cystitis if the patient is symptomatic.

Recurrent cystitis ( 3 or more episodes per year) requires further investigations such as ; radiological investigations such as intravenous Urogram, ultrasound or CT to detect any obstruction or congenital deformity in children. In some cases Cystoscopy is required.

 **Treatment** empiric treatment is common and depends on the knowledge of local susceptibility pattern of the most common causative bacteria. Treatment is best guided by the susceptibility pattern of the causative bacteria. Antimicrobial agents commonly used are : Ampicillin ,Cephradine, Ciprofloxacin ,Norfloxacin , Gentamicin & TRM-SMX ). Duration of treatment is 3 days for uncomplicated cases. however, for complicated or recurrent cases 10-14 days is recommended. Preventive measures include; drinking plenty of fluids and prophylactic antibiotics ( nitrofurantoin ,TRM-SMX ).

**Take home messages:**

1-Cystitis is commonly caused by bacteria.

2- Cystitis is common in women particularly during reproductive age ,pregnancy and menopause.

3- Common symptoms are: dysuria, frequency and urgency .

4-Cystitis in men is due to recurrent infection of the prostate.

5- *E.coli* is the most common cause of cystitis. Among gram positive bacteria; *Enterococcus fecalis* and group *B Streptococcus* are common causes. *Candida* species is common among diabetics and catheterized patients on antibiotics..

6- venereal diseases can present as cystitis.

6- Cystitis can be caused by non-infectious causes.

6- Laboratory diagnosis is through examination of midstream urine specimen by microscopy ,chemical screening tests and quantitiative culture.

7- Treatment of cystitis by antimicrobial agents according to the causative organisms .

8- Prevention of cystitis includes;by drinking plenty of fluids and prophylactic antibiotics.

**Keywords**

Cystitis, dysuria, urgency, frequency, *E.coli, Enterococcus fecalis , Staphylococcus saprophiticus*, Interstitial cystitis, hemorrhagic cystitis ,venereal diseases, MSU ,microscopic examination, nitrite , leukocyte esterase, quantitative culture, empiric treatment, prophylaxis.

**Recommended Books:**

-***Sherris Medical Microbiology*** ,An Introduction to Infectious Diseases

Authors: Kenneth Ryan, C.G.Ray. chapter 66.

Publisher: Mc Graw Hill.

**Lecture 2 Treatment of Urinary Tract Infection**

**Objectives:**

**By the end of this lecture students are expected to :**

1- know the principal goal of urinary tract infection (UTI) management is to eradicate the offending organisms from the urine and tissues.

2- understand that management of UTI depends on several factors .

3-know that antibiotics are the main treatment of UTI.

4- know the management/treatment of different conditions of UTI ( cystitis, pyelonephritis, catheter associated UTI ,etc.)

**The Choices of antibiotic depends on the followings :**

 - Whether the infection is complicated or uncomplicated

 - Whether the infection is primary or recurrent.

 - Type of patient ( man or women, pregnant or non-pregnant, child , hospitalized or non-hospitalized, diabetic patient, ..etc)

 - Bacterial count

 - Presence of symptoms

**Summary:**

**Treatment of Uncomplicated UTIs**

UTI in low-risk women for recurrent infection ( who do not have vaginitis) can besuccessfully treated with **3-days** antibiotic without the need of urine test**.** Cure rate is about 94%.

**Antibiotic regimen commonly used ( depending on the susceptibility pattern in the hospital ):**

 **Amoxicillin** ( with or without clavulanate)

  **Cephalosporins** ( first or second generation, Cephradine or Cefuroxime , respectively) .

 **Fluoroquinolone** ( Ciprofloxacin or Norfloxacin) antibiotics. Pregnant women and children less than 8 years of age should not take these drugs.

 **Trimethoprim-Sulfamethoxazole** , commonly called TMP-SMX ( Bactrim, Septra, Cotrim).

*Other options:*

 **Nitrofurantoin**: should be given longer than 3-days.

***Relapsing infection***: caused by treatment failure or structural abnormalities or abscesses, should be treated similarly to a first infection but antibiotics are usually continued for 7-14 days.

***Treatment for recurrent infections***

Women who have two or more symptomatic UTIs within 6 months or three or more over the course of a year may need preventive therapy.

The patient should take the antibiotic as soon as she develops symptoms. However, if the infection occurs less than twice a year , a clean catch urine test should be taken for culture and usually treated with a single dose or as an initial attack with 3-days of antibiotic.

**A patient should NOT have 3-days self treatment and should consult the doctor under the following circumstances:**

 - If symptoms persist

 - If there is a change in symptoms

 - If the patient was pregnant

 - If the patient has more than 4 infections a year.

 - Patients with impaired immune system

 - Patients with previous kidney infections

 - Patients with structural abnormalities of the urinary tract

 - History of infection with antibiotic resistant bacteria.

***Postcoital antibiotics***

If recurrent UTI is clearly related to sexual activity and episodes recur more than two times within 6 months period, a single preventive dose taken immediately after intercourse is very effective.

Antibiotics for such cases include : TMP-SMX, Nitrofurantoin , Cephlexin or Ciprofloxacin.

***Prophylactic antibiotics***

This is an option for some patients who do not respond to other measures. It reduces recurrences by up to 95%. A low-dose antibiotics are taken continuously for 6 months or longer. For example: Nitrofurantoin, TMP-SMX or Cephalexin. Taking the antibiotic at bed time may be most effective.

**Treatment for Kidney Infections ( Pyelonephritis)**

**Treatment of uncomplicated pyelonephritis**: those patients are healthy, non pregnant women and do not have nausea or vomiting and show no symptoms of kidney involvement , they can be treated at home with oral antibiotics.

The standard treatment is a **14-day** course of oral antibiotics usually Cephalosporin, TMP-SMX or Ciprofloxacin. Some patients may receive first dose by injection. Oral Amoxicillin or Amoxicillin-Clavulanate ( Augmentin) may be prescribed for infection with gram-positive organisms, including *Enterococcus* species and *S.sprophyticus*) that do not respond to standard regimen.

A urine culture may be obtained within one week of completion of therapy and again 4 weeks later.

**Treatment of moderate to sever pyelonephritis**: those patients had symptoms or other complications , may need *hospitalization*. Antibiotics are given by intravenous **IV**) route for 3-5 days or until symptoms and signs are relieved for 24-48 hours.)

If fever and back pain continue after 72 hours of antibiotic administration, *imaging tests* are to be done to exclude abscesses, obstructions or other abnormalities.

**Treatment of chronic pyelonephritis**: those patients often treated with long-term antibiotics, even during periods when they have no symptoms.

**Treatment for specific populations**

**Pregnant women**: should be screened for UTIs, since they are at high risk for UTIs and their complications.

Antibiotics used during pregnancy include: Amoxicillin, Ampicillin, Cephalosporins and Nitrofurantoin. Pregnant women should not take Quinolones.

Pregnant women with asymptomatic bacteriuria ( *evidence of infection with out symptoms*) have a 30% risk for acute pyelonephritis in their second or third trimester. Screening and treatment are needed with a short course antibiotics (3-5 days).

For uncomplicated UTI , pregnant women may need **7-10 days** antibiotic treatment.

**Diabetic patients**: have more frequent and more sever UTIs than non diabetic patients. It is recommended that these patients treated for **7-14 days** with antibiotics even patients with an uncomplicated infections.

**Urethritis in men**: typically treated with **7-days** regimen of Doxycycline. A single dose Azithromycin may be effective as well but is not recommended to avoid spread to the prostate gland. These patients should also be tested for an accompanying sexually transmitted disease such as gonorrhea.

**Children with UTIs:** children usually treated orally with TMP-SMX or Cephalexin. Sometimes given as a shot or IV. As resistance to Cephalexin is increasing, some doctors recommend Aminoglycoside ( Gentamincin) which is given by IV route.

**Vesicoureteral reflux ( VUR**) is a concern for children with UTIs .It can lead to pyelonephritis which can cause kidney damage. **Long-term** antibiotics or surgery have traditionally been options to correct VUR and prevent infections.

Children with acute kidney infection are treated with oral Cefixime ( Suprax) or a short course (2-4 days) of an IV Gentamicin given in one daily dose. An oral antibiotic then follows the IV.

**Management of Catheter-Induced UTIs**

A very common problem and preventive measures are very important.

*Catheters should not be used unless absolutely necessary and they should be removed as soon as possible.*

**Intermittent use of catheters:** if catheter is required for long periods, it is best to use it intermittently. Some doctors recommend replacing it every 2 weeks to reduce the risk of infection and irrigating the bladder with antibiotics between replacements.

**Daily hygiene**: and use of closed bag system to prevent infection.

**Antibiotics for catheter-induced infections**

Catheterized patients who develop **UTI with symptoms or at risk of sepsis** ,should be treated for each episode with antibiotics and the catheter should be removed, if possible. The organisms in catheter associated UTI are constantly changing. They are likely to be multiple species of bacteria, so an antibiotics that is effective against wide variety of microorganisms is indicated. Antibiotic use for prophylaxis is **NOT** recommended since high bacterial counts present in most patients and they do not develop symptomatic UTI . Antibiotic therapy has little benefit if the catheter is to remain in place for long period.

**Recommended Books:**

***Sherris Medical Microbiology***

**Lecture 3 Pyelonephritis**

**Objectives:**

**By the end of this lecture, the students should be able to:**

1. Define the term Pyelonephritis
2. Know the bacterial causes of Pyelonephritis
3. Know the pathogenesis of Pyelonephritis
4. Know the organisms that cause Pyelonephritis by ascending from urethra and those cause Pyelonephritis through the hematogenous spread.
5. Know the clinical features of acute Pyelonephritis and chronic Pyelonephritis.
6. State the laboratory diagnostic tests and the radiological test of the diagnosis Pyelonephritis.
7. State the complications of Pyelonephritis, mainly those of chronic Pyelonephritis.
8. Know the management of Pyelonephritis including nursing management and anti-microbial management.

**Introduction:**

Infection of the Urinary tract fall into two General anatomical categories.

Lower tract infection (Urethritis and Cystitis) upper tract infection (Pyelonephritis, prostitis and perinephritic abscess.

However these infections may occur separately or together, symptomatic or asymptomatic.

Infection of the urethra (Urethritis) bladder (Cystitis) are supposed to be mucosal or superficial infection where as Pyelonephritis signifies tissue and blood stream invasion. Fever is a characteristic of Pyelonephritis and so blood cultures can be helpful in the diagnosis of acute Pyelonephritis. Acute Pyelonephritis is characterized by a acute onset and acute clinical feature where as chronic Pyelonephritis is characterized by more insidious onset.

**Key Outlines:**

The definition of Pyelonephritis as infection of the renal pelvis, tubules and interstitial tissue of one or both kidney will be stated.

The pathogenesis as the means by which microorganisms reach the kidney will be explained. Stress will be put on the fact that most bacteria causing Pyelonephritis and other types of urinary tract infection like cystitis reach the kidney from colon to urethra then by the ascending way.

Some bacteria like *Staphylococcus aureus* and *Mycobacterium tuberculosis* reach the kidney by the blood strain i.e haematogenously: Organisms in each group will factors predisposing to be discussed to the

Urinary tract infection e.g. Pyelonephritis will be explained like Urinary tract obstruction, blondes lumber and prostate enlargement in elderly men.

The pathology of Pyelonephritis shall be explained as enlargement of kidney, scaring, abscess formation. The different methods of management of Pyelonephritis will be discussed. This will include explanation of nursing and anti-microbial treatment, and use of different anti-microbial agents will be explained.

**Take home messages:**

* Pyelonephritis means infections of Kidney pelvis, tubules and kidney parenchyma.
* The commonest bacterial causes are enterobacteriacae like *E. coli* which reach the Kidney through ascending route. Other bacteria like *Staphylococcus aureus* and *Mycobacterium tuberculosis* reach the Kidney through haematogeneous route.
* Acute Pyelonephritis clinically presents as acute febrile condition with loin pain, frequency and dysuria. Chronic Pyelonephritis presents as insidion condition.

**The commonest bacterial causes are:**

1. *E. coli*
2. Klebsiella
3. Proteus
4. Enterobacteria Ascending route
5. *Staphylococcus saprophyticus*
6. Enterococci

Staphylococcus aureus Hematogenous route

Mycobacterium tuberculosis

* Pyelonephritis maybe complicated by conditions like hypertension.

Pyelonephritis is treated by anti-microbial like

* Amoxicillin/ampicillin/ clavulanic
* Cotrimoxazole / sulphamexazole
* Cephalosporins
* Ciprofloxacin

Nitrofurantoin and Nalidixic acid which can be used for treatment of cystitis are not useful for treatment of Pyelonephritis.

|  |
| --- |
| **Department of Pharmacology** |

**Lecture 1 Diuretics (Carbonic anhydrase inhibitors and osmotic diuretics)**

**Objectives:**

*By the end of these lectures, student should be able to:*

* Identify major locations of ion and water exchange in the nephron
* Identify the different classes of diuretics
* Understand sites of action of carbonic anhydrase inhibitors and osmotic diuretics
* know molecular target of actions of carbonic anhydrase inhibitors and osmotic diuretics.
* Understand the pharmacokinetics and pharmacodynamics of each class of diuretics.
* Discuss the clinical applications of diuretics in special diseases.
* Know what are the major adverse effects of each group especially those of clinical importance.

**Introduction:**

Depending upon your background about the structure of the basic unit for kidney (nephron) and the main function of each part of the nephron in renal excretion. This will help understand the mechanism of action of each class of diuretics.

**Key outlines:**

Mechanism of action, kinetics, dynamics and uses of carbonic anhydrase inhibitors and osmotic diuretics. Special concern will be paid to the implication of carbonic anhydrase inhibitors in some application other than diuretic action as chronic open angle glaucoma, epilepsy, mountain sickness.

**Take home messages:**

* Osmotic diuretics as mannitol produce their diuretic actions via increasing osmotic pressure. Mannitol is not absorbed orally and should be given intravenously. They are used to effect water excretion rather than sodium excretion. They are the mainstay of treatment for patients with increased intracranial pressure or acute renal failure due to shock, drug toxicities and trauma. Dehydration and hypernatremia are the major side effects.
* Carbonic anhydrase inhibitors as acetazolamide produce its action by inhibiting carbonic anhydrase in the proximal convoluted tubules. It is given orally. Carbonic anhydrase inhibitors are the weakest diuretics. They are used for other pharmacologic actions rather for their diuretic effects as in chronic open angle glaucoma, epilepsy, mountain sickness. Alkaline urine, metabolic acidosis, hypokalemia, renal stone formation are its major side effects. It should be avoided in patients with hepatic cirrhosis.

**Recommended books:**

* Rang HP, Dale MM, Ritter JM, Moore PK (2007). Pharmacology. Six Edition. Churchill Livingstone, Elsevier, UK.
* Katzing BG (2008). Basic and Clinical Pharmacology. New York: McGraw Hill/Appleton & Lange.

**Key words:**

Acetazolamide, Mannitol, mechanism of action, uses, pharmacokinetics, pharmacodynamics and side effects.

**Lecture 2 Diuretics (Loop diuretics and thiazide diuretics)**

**Objectives:**

*By the end of these lectures, student should be able to:*

* Understand sites of action of loop and thiazide diuretics
* know molecular target of actions of loop and thiazide diuretics
* Understand the pharmacokinetics and pharmacodynamics of loop and thiazide diuretics
* Discuss the clinical applications of loop and thiazide diuretics in special diseases as hypertension, congestive heart failure and pulmonary edema.
* Know what are the major adverse effects of each group especially those of clinical importance.

**Introduction:**

Solid physiological background is required for understanding different mechanisms of action of various classes of diuretics.

**Key outlines:**

Mechanism of action, kinetics, dynamics and uses of loop diuretics and thiazide diuretics. Special concern will be paid to their implication in some clinical situations as hypertension, edema and heart failure.

**Take home messages:**

* Loop Diuretics as furosemide is most efficacious of the diuretics. Furosemide can be given orally or intravenously. It has rapid onset of action. It produces its effect on thick sscending loop of Henle by inhibiting Na+/K+/2 Cl- cotransporter in the luminal membrane. This group is reserved for emergency situation as acute pulmonary edema of heart failure. Ther are also useful inhyperkalemia and hypercalcemia. Hypokalemia, hyperuricemia, hypovolemia are their major side effects.
* Thiazide diuretics as hydrochlorothiazide produce their diuretic action by inhibiting Na+/ Cl\_ cotransporter on the luminal membrane of distal convoluted tubules. Clinically, they are the mainstay of antihypertensive medication. They are also useful in heart failure, hypercalciuria (nephrolithiasis) and diabetes inspidus. Hpokalemia, hypercalemia, hyperglycemia are their major side effects.

**Recommended books:**

* Rang HP, Dale MM, Ritter JM, Moore PK (2007). Pharmacology. Six Edition. Churchill Livingstone, Elsevier, UK.
* Katzing BG (2008). Basic and Clinical Pharmacology. New York: McGraw Hill/Appleton & Lange.

**Key words:**

* Loop diuretics, thiazide diuretics, mechanism of action, pharmacokinetics, pharmacodynamics, uses, side effects.

**Lecture 3 Renal excretion of drugs**

**Objectives:**

*By the end of these lectures, student should be able to:*

* Identify main and minor routes of excretion of drugs including renal elimination and biliary excretion
* Describe mechanisms involved in renal handling of drugs.
* Describe some pharmacokinetics terms including plasma half life, renal clearance of drugs.
* Describe the correlation between renal clearance and duration of actions of drugs.
* Discuss important factors affecting renal handling of drugs
* Be able to identify the influence of age and diseases on renal excretion of drugs.
* Be able to characterize what should be done upon prescribing drugs in patients with renal impairment.

**Lecture 4 Diuretics (Potassium sparing diuretics)**

**Objectives:**

*By the end of these lectures, student should be able to:*

* Understand sites of action of potassium sparing diuretics on nephron.
* know molecular target of actions of potassium sparing diuretics.
* Understand the pharmacokinetics and pharmacodynamics of potassium sparing diuretics.
* Discuss the clinical applications of potassium sparing diuretics
* Know what are the major adverse effects of potassium sparing diuretics

**Introduction:**

* Good background about the function of collecting tubules, aqueous channels, aldosterone receptors and sodium channels is a mandatory.

**Key outlines:**

How k-sparing diuretics produce their diuretic action, their kinetics, dynamics, side effects, uses and contraindication. Also the purpose of combination of this category with other classes of diuretics

**Take home messages:**

K-sparing diuretics as spironolactone produce its action by blocking action of aldosterone on collecting ducts. It is the only class of diuretics that can produce hyperkalemia. Spironolactone is clinically useful in the treatment of hepatic ascites. Gynecomastia and menstrual irregularities are its major side effects.

**Recommended books:**

* Rang HP, Dale MM, Ritter JM, Moore PK (2007). Pharmacology. Six Edition. Churchill Livingstone, Elsevier, UK.
* Katzing BG (2008). Basic and Clinical Pharmacology. New York: McGraw Hill/Appleton & Lange.

**Key words:**

* Potassium sparing diuretics-aldosterone antagonists, collecting ducts

**Lecture 5 & 6 Treatment of URINARY TRACT (Co-trimoxazole, nitrofurantoin and quinolones / Cephalosporins tetracyclines and aminoglycosides in UTI )**

**Objectives:**

At the end of the two lectures the students will be able to:

1. Recognize different groupd of antibiotics used urinary tract.
2. Describe their mechanism of action, pharmacokinetic properties and adverse effects.
3. Describe the use of antibitotic and their rational of combination of different antiobiotics.
4. Describe the spectrum of various antibiotics

**Introduction:**

Urinary tract infections are common diseases seen in the medical practice. Several group of antibitotics such as Co-trimoxazole, nitrofurantoin and quinolones / Cephalosporins tetracyclines and aminoglycosides are used alone or in combination. Each group fo antibiotics has its owen efficacy against specific bacteria, their use depend on the type of etiology and sveraity of infection.

**Summary:**

Antibiotics used in Urinary tract infections can prevent serious complication if used rationally. UTI can be effectively treated with different antibiotics.

**Take home message:**

The student will understand and recognize important clss of antibiotics used ins UTIs.

**Recommended books:**

* Rang HP, Dale MM, Ritter JM, Moore PK (2007). Pharmacology. Six Edition. Churchill Livingstone, Elsevier, UK.
* Katzing BG (2008). Basic and Clinical Pharmacology. New York: McGraw Hill/Appleton & Lange.

**Key words:**

* Potassium sparing diuretics-aldosterone antagonists, collecting ducts

|  |
| --- |
| **WEEK 1 – RENAL BLOCK (Group-Female)** |
| **Week (1 ) Starting: 10/04/2016 to 14/04/2016 (03/07/1437 to 07/07/1437H)****Acute Kidney injury** |
| **CHAIR PERSON: Dr. Mohammed Al-Ghonaim** |
| **Sunday****10 April 2016** | **Monday****11 April 2016** | **Tuesday****12 April 2016** | **Wednesday****13 April 2016** | **Thursday** **14 April 2016** |
| **8:00 - 10:00 am****Problem-based** **Learning****Case 1 Tutorial 1** | **8:00 - 9:00 am****Self-directed Learning** | **8:00 - 9:00 am****Self-directed Learning** | **8:00 - 10:00 am****Problem-based** **Learning****Case 1 Tutorial 2** | **8:00 - 10:00 am****(Practical - 1)**Acute kidney injury**(Pathology)****Dr. Shaesta Zaidi/****Dr. Sufia Husain**  |
| **9:00 - 11:00 am****(Practical)**Glomerular filtration rate and renal clearance**(Physiology)****All staff** | **9:00 - 10:00 am**Renal function tests**(Biochemistry)** **Dr. Rana Hasanato** |
| **10:00 - 11:00 am**Anatomy of the kidney**(Anatomy)****Dr. Jamilah El Medany** | **10:00 - 11:00 am**Radiology of Renal System**( Radiology )** **Dr. Abdulrahman Al Eissa** | **10:00 - 11:00 am**Development of the kidney, ureters**(Embryology)****Dr. Jamilah El Medany**  | **10:00- 11:00 am****Self-directed Learning** |
| **11:00 - 12:00 pm**Histology of the Kidney**(Histology)****Dr. Raeesa Abdultawab** | **11:00 - 12:00 pm**Renal functions and glomerular filtration**(Physiology)****Dr.Manan Alhakbany**  | **11:00 - 12:00 pm**Chemical examination of urine**(Biochemistry)****Dr. Rana Hasanato**  | **11:00 - 12:00 pm****Self-directed Learning** | **11:00 - 12:00 pm**Renal clearance**(Physiology)****Dr.Manan Alhakbany**  |
| **Lunch****12:00 – 1:00pm** | **Lunch****12:00 – 1:00pm** | **Lunch****12:00 – 1:00pm** | **Lunch****12:00 – 1:00pm** | **Lunch****12:00 – 1:00pm** |
| **1:00 - 2:00 pm****Self-directed Learning** | **1:00 – 3:00 pm****(Practical)**Anatomy of the kidney**(Anatomy)****All Staff** | **1:00 - 2:00 pm****(Practical)**Histology of the kidney**(Histology)****All Staff** | **1:00 -3:00 pm****Salam** | **1:00 – 2:00 pm****Self-directed Learning** |
| **2:00 - 3:00 pm****Self-directed Learning** | **2:00 - 3:00 pm**Acute kidney injury**(Pathology)****Dr. Hala Kfoury**  | **2:00 - 3:00 pm**Regulation of glomerular filtration**(Physiology)****Dr.Manan Alhakbany**  |

|  |
| --- |
| **WEEK 2 - RENAL BLOCK (Group-Female)** |
| **Week (2 ) Starting: 17/04/2016 to 21/04/2016 (10/07/1437 to 14/07/1437H)****Urinary Tract Infections** |
| **CHAIR PERSON: Dr. Mohammed Al-Ghonaim** |
| **Sunday****17 April 2016** | **Monday****18 April 2016** | **Tuesday****19 April 2016** | **Wednesday****20 April 2016** | **Thursday** **21 April 2016** |
| **8:00 - 10:00 am****Problem-based** **Learning****Case 2 Tutorial 1** | **8:00 - 9:00 am****Cystitis****(Microbiology)****Prof. Hanan Habib** | **8:00 - 9:00 am****Management of UTI****(Microbiology)****Prof. Hanan Habib** | **8:00 - 10:00 am****Problem-based** **Learning****Case 2 Tutorial 1** | **8:00 - 9:00 am****Infection of the upper urinary tract****(Pathology)****Dr. Hala Kfoury** |
| **9:00 - 10:00 am****Self-directed Learning** | **9:00 - 10:00 am**Renal excretionof drugs**(Pharmacology)****Prof. Hanan Hagar** | **9:00 - 10:00 am****Infection of the lower urinary tract****(Pathology)****Dr. Hala Kfoury** |
| **10:00 - 11:00 am****Self-directed Learning** | **10:00 - 12:00 pm****(Practical)****Organisms causing UTI****(Microbiology)****Dr. Fawzia Al Otaibi** | **10:00 - 12:00 pm****(Practical)****Ureters, bladder, and urethra** **(Anatomy)****All Staff** | **10:00 - 11:00 am****Self-directed Learning** | **10:00am - 12:00 pm****(Practical - 2)****Infection of the urinary tract** **(Pathology)****Dr. Shaesta Zaidi/****Dr. Hala Kfoury** |
| **11:00 - 12:00 pm****Ureters, bladder and urethra****(Anatomy)****Dr. Sanaa Al Shaarawi** | **11:00 - 12:00 pm****Physiology of Micturation** **(Physiology)****Dr.Manan Alhakbany**  |
| **Lunch****12:00 – 1:00pm** | **Lunch****12:00 – 1:00pm** | **Lunch****12:00 – 1:00pm** | **Lunch****12:00 – 1:00pm** | **Lunch****12:00 – 1:00pm** |
| **1:00 - 2:00 pm****Histology of Urinary tract****(Histology)****Dr. Raeesa Abdultawab** | **1:00 - 2:00 pm****Co-trimoxazole, nitrofurantoin and quinolones in UTI****(Pharmacology)****Dr. Ishfaq Bukari**  | **1:00 - 3:00 pm****(Practical)****Histology of urinary** **Tract****(Histology)****All Staff** | **1:00 - 3:00 pm****Salam** | **1:00 - 2:00 pm****Development of bladder and urethra****(Embryology)****Dr. Sanaa Al Shaarawi** |
| **2:00 - 3:00 pm****Kidney stones** **(Biochemistry)****Dr. Reem Sallam** | **2:00 - 3:00 pm****Cephalosporins tetracyclines and aminoglycoside in UTI****(Pharmacology)****Dr. Ishfaq Bukari** | **2:00 - 3:00 pm****Self-directed** **Learning** |

|  |
| --- |
| **WEEK 3 - RENAL BLOCK (Group-Female)** |
| **Week (3 ) Starting: 24/04/2016 to 28/04/2016 (17/07/1437 to 21/07/1437H)****Nephrotic/Nephritic syndrome** |
| **CHAIR PERSON: Dr. Mohammed Al-Ghonaim** |
| **Sunday****24 April 2016** | **Monday****25 April 2016** | **Tuesday****26 April 2016** | **Wednesday****27 April 2016** | **Thursday** **28 April 2016** |
| **8:00 – 10:00 am****Problem-based** **Learning****Case 3 Tutorial 1** | **8:00 - 9:00 am**Tubular reabsorption**(Physiology)****Dr. Mona Soliman** | **8:00 - 9:00 am**Renal regulation of body fluid**(Physiology)****Dr. Mona Soliman** | **8:00 - 10:00 am****Problem-based** **Learning****Case 3 Tutorial 2** | **8:00 - 10:00am****(Practical- 3)**Rapid Progress Glomerular Nephritis/Chronic Kidney Disease**(Pathology)****Dr. Shaesta Zaidi/****Dr. Hala Kfoury** |
| **9:00 - 10:00 pm**Inborn errors of amino acid metabolism **(Biochemistry)****Dr. Sumbul Fatma** | **9:00 - 10:00 am**Loop and thiazide diuretics**(Pharmacology)****Prof. Hanan Hagar** |
| **10:00 - 12:00 pm****(Practical)**Renal tubular function**(Physiology)****All staff** | **10:00 - 12:00 pm****(Practical)**Tubular transportation **(Physiology)****All staff** | **10:00 - 11:00 am**Carbonic anhydrase inhibitors and osmotic diuretics**(Pharmacology)****Prof. Hanan Hagar** | **10:00 - 11:00 nn**Concentration of the urine **(Physiology)****Dr. Mona Soliman** | **10:00 – 11:00 am**Pyelonephritis**(Microbiology)****Dr. Fawzia Al Otaibi** |
| **11:00 – 12:00pm** Immune complex nephritis**(Immunology)****Dr. Hend Alotaibi** | **11:00 - 12:00 pm**Potassium sparing diuretics**(Pharmacology)****Prof. Hanan Hagar** | **11:00 - 12:00 pm****Self-directed** **Learning** |
| **Lunch****12:00 – 1:00pm** | **Lunch****12:00 – 1:00pm** | **Lunch****12:00 – 1:00pm** | **Lunch****12:00 – 1:00pm** | **Lunch****12:00 – 1:00pm** |
| **1:00 - 2:00 pm****Self-directed Learning** | **1:00 - 2:00 pm**Tubular secretion **(Physiology)****Dr. Mona Soliman** | **1:00 - 2:00 pm**Rapid Progress Glomerular Nephritis/ChronicKidney Disease **(Pathology)****Dr. Hala Kfoury** | **1:00 - 3:00 pm****Salam** | **1:00-3:00 pm****(Practical)**Urinalysis**(Biochemistry)****All Female Staff** |
| **2:00-3:00pm****Self-directed Learning** | **2:00-3:00pm****Self-directed Learning** | **2:00-3:00pm**Nephrotic/nephritic syndrome **(Pathology)****Dr. Hala Kfoury** |

|  |
| --- |
| **WEEK 4 - RENAL BLOCK (Group-Female)** |
| **Week (4 ) Starting: 01/05/2016 to 05/05/2016 (24/07/1437 to 28/07/1437H)****Chronic Kidney Disease** |
| **CHAIR PERSON: Dr. Mohammed Al-Ghonaim** |
| **Sunday****01 May 2016** | **Monday****02 May 2016** | **Tuesday****03 May 2016** | **Wednesday****04 May 2016** | **Thursday** **05 May 2016** |
| **8:00 - 10:00 am****Problem-based** **Learning****Case 4 Tutorial 1** | **8:00 - 9:00 am** **Self-directed Learning**  | **8:00 - 9:00 am****Self-directed** **Learning** | **8:00 - 10:00 am****Problem-based** **Learning****Case 4 Tutorial 2** | **8:00 -9:00 am****Self-directed** **Learning** |
| **9:00 - 10:00am****Self-directed Learning** | **9:00 - 10:00am****I**mmunology of renal transplant**(Immunology)****Dr. Hend Alotaibi** | **9:00 - 10:00 am** **Self-directed** **Learning** |
| **10:00 - 12:00 pm****Practical Revision**Organism causing UTI**( Microbiology)****Dr. Malak El Hazmi** | **10:00 - 11:00 am**Basics of acid base**(Physiology)****Dr. Mona Soliman** | **10:00 - 11:00 am****Self-directed** **Learning** | **10:00 – 11:00 am**Acid base disorders **(Physiology)****Dr. Mona Soliman** | **10:00 - 11:00 am****Self-directed** **Learning** |
| **11:00 – 12:00 pm**Buffer systems**(Physiology)****Dr. Mona Soliman** | **11:00 – 12:00 pm**Tumors of the kidney and urinary tract**(Pathology)****Dr. Hala Kfoury** | **11:00 - 12:00 pm**Pathology of renal allograft**(Pathology)****Dr. Hala Kfoury** | **11:00 – 12:00 pm****Self-directed** **Learning** |
| **Lunch****12:00 – 1:00pm** | **Lunch****12:00 – 1:00pm** | **Lunch****12:00 – 1:00pm** | **Lunch****12:00 – 1:00pm** | **Lunch****12:00 – 1:00pm** |
| **1:00 - 2:00 pm** **Self-directed Learning** | **1:00 – 3:00pm****(Practical)**Basics of Acid base **(Physiology)****All staff** | **1:00 - 2:00 pm****Self-directed Learning** | **1:00-3:00 pm****Salam** | **1:00 – 2:00 pm****Self-directed** **Learning** |
| **2:00 -3:00pm****Self-directed Learning** | **2:00 -3:00pm****Self-directed Learning** | **2:00 -3:00pm****Self-directed Learning** |

|  |
| --- |
| **Assessment of Students in the Block**  |

Dear Student, in order to pass the block, you must obtain a minimum final block grade of D (the grading guide attached as appendix¹), this grade is a composition from several block requirements, which can be subdivided as:

1. Attendance
2. Tutor assessment
3. Written Exams (MCQs, SAQs)
4. OSPE (Objective Structured Practical Examination)
* **Marks distribution for Blocks with Renal block**

The final grade is a composition of the grades obtained for the specified block requirements, calculated as follows:

* Continuous Assessment (Tutor Assessment in PBL sessions) : 5%
* Written Examinations:
	+ SAQs : 20%
	+ MCQ : 40%
* Final Block Exam (40%)
* OSPE : 35 %

**TOTAL : 100 %**

1. **Attendance :**

Students are required to attend not less than 75% of all educational activities during the block. These include small group teaching, lectures, practical sessions, skills training sessions and integrated clinical sessions.

Your attendance will be recorded during all sessions. Failure to meet this requirement without a valid explanation will result in exclusion from the final examination. On the other hand, your presence will be rewarded by assigned marks.

1. **Tutor Assessment in Large and Small groups (Continuous Assessment):**

During each session, your individual efforts will be evaluated by your tutor. The tutors are instructed to evaluate two aspects:

1. The extent to which you demonstrate that you study and prepare yourself thoroughly between the two sessions (i.e., preparation).
2. The extent to which you actively contribute during group discussion (i.e., participation). Your grade for each session depends upon both your preparation and your participation. The grade will be on the scale from “5”, “4”, “3”, “2”, or “1”. Which have the following general descriptors:

5 = Outstanding (Excellent)

4 = Very good

3 = Good

2 = Average

1 = Poor

The block contains two sessions each week, so the maximum amount of ‘participation points’ you are able to obtain will be from two sessions multiplied by the number of weeks. The total participation points will be recalculated according to the weight for each participation in the total assessment.

Your tutor can give you more information about the evaluation of your participation. The details of these evaluation also given in “Tutor Assessment of Student” form.

1. **Written Examination:**

40% for the renal block: at the end of the block in form of MCQs, that are prepared mainly from sessions and presented to the students. This exam will consist of 80 -100 MCQs that will assess factual knowledge too.

1. **Objective Structured Practical Examination** **(OSPE** **):**

This comprises 35% for the renal block. It is a practical examination at the end of the block. The OSPE examination will consist of 15-20 OSPE stations. Each station will take about 5 minutes, which contains a mix of slide show and some practical sessions. The purpose of the OSPE stations is to test your deeper understanding of the basic sciences. The OSPE will take place at the end of each block.

1. **Short Answer Questions (SAQs)**

This comprises 20% of the marks for renal block. It contains 3 to 5 cases which include 12-20 integrated SAQs. The time allocated for each question is 3 minutes. The purpose of SAQs is to test the knowledge and its application in integrated manner and to increase the validity and reliability of the written exam. The SAQ will take place at the end of each block.

**Block Evaluation**

The block evaluation uses the following three data sources:

1. Student Feedback
2. Tutor Feedback
3. Student Results

***Methods of student’s formative assessment:***

* Self evaluation
* Peer evaluation
* Tutor evaluation (both summative & formative)
* Assignments

******Reference**

The list below comprises the key textbooks and learning resources which have been prescribed and recommended for use in the undergraduate medical course at King Saud University. It is expected that you have your own copy of prescribed textbooks and use them as one of your main resources in learning. Before making any purchases, you might carefully examine all other recommended textbooks in an area and chose the text that matches with your needs and your learning style. Although all these texts are available in the Medical Library, you might need to purchase texts that you use frequently in these years as the demand upon library texts is usually high.

**Anatomy & Embryology (Lectures)**

*Recommended books:*

Drake RL, Vogl W and Mitchell AWM (2005). Gray’s Anatomy for Students. Philadelphia: Elsevier Churchill Livingstone.

Snell RS (2005). Clinical Anatomy for Medical Students. 7th ed. Philadelphia: Lippincott Williams & Wilkins.

More KL (2002). The Developing Human. Philadelphia: Saunders WB.

Sadler TW. (2005) Langman’s Essential Medical Embryology. Philadelphia: Lippincott Williams & Wilkins.

**Histology**

*Recommended books:*

Gartner LP and Hiatt JL. Color Textbook of Histology. 2nd ed. Philadelphia: Saunders WB.

Young B, Lowe JS, Stevens A and Heath JW. Wheater’s Functional Histology. 5th ed. London: Churchill Livingstone.

**Anatomy (Practical)**

*Recommended books:*

McMinn RH. McMinn’s Color Atlas of Human Anatomy. Fifth Edition. Mosby Publisher, UK.

Netter FH. Atlas of Human Anatomy. 4th ed. Philadelphia: Saunders WB.

Agur AMR and Dalley AF. Grant’s Atlas of Anatomy. 11th ed. Philadelphia: Lippincott Williams & Wilkins.

**Physiology**

*Recommended books:*

Guyton & Hall Textbook Of Medical Physiology 11th Ed

**Pharmacology**

*Prescribed textbook:*

Rang HP, Dale MM, Ritter JM, Moore PK (2007). Pharmacology. Six Edition. Churchill Livingstone,

Katzing BG (2008). Basic and Clinical Pharmacology. New York: McGraw Hill/Appleton & Lange.

**Biochemistry**

*Recommended books:*

Lecture notes: Clinical Biochemistry, 8th edition, Geoffrey Beckett, Simon Walker, Peter Rae, Peter Ashby. January 2010, © 2010, Wiley-Blackwell.

Colored Atlas of Biochemistry (material given to students by tutors)

Illustrated Reviews of Biochemistry by Lippincott 4th edition.

Clinical Chemistry and metabolic Medicine. 7th edition, 2006

Bishop Duben-Engelkirk Fody, Clinical chemistry principles, procedure, correlations (fourth edition).

**Microbiology & Parasitology**

Recommended Books:

-***Sherris Medical Microbiology*** ,An Introduction to Infectious Diseases

Authors: Kenneth Ryan, C.G.Ray. chapter 66.

Publisher: Mc Graw Hill.

**Pathology**

*Recommended books* :

Pathologic Basis of Disease, Robbins and Cotran

**Immunology**

*Prescribed textbook:*

Delves PJ, Martin SJ, Burton DR, Riott IM (2006). Riott’s Essential Immunology. Eleventh Edition. Blackwell Publishing, UK.

*Recommended textbooks:*

Male D, Brostoff J, Roth DB, and Roitt I. (2006). Immunology. 7th ed. Edinburgh: Mosby.

**Medicine**

Kumar P and Clark M (2010). Clinical Medicine. 7th ed. Edinburgh: Elsevier Saunders.

Edwards C and Bouchier IA. (2003). Davidson’s Principles and Practice of Medicine. 14th ed. Edinburgh: Churchill Livingstone.

*(In the preclinical years these two textbooks may help you in the preparation of your learning issues, you will also need them in the clinical years).*



**ACADEMIC SUPPORT TEAM**

The College of Medicine and the Department of Medical Education are working on ensuring that our students receive optimal support to their learning. The list of academics shown below represents the departments involved in the teaching and learning of this block. If a student needs help in their teaching and learning they might consult one academic from the list. He/she might email them and arrange a time to see them if needed, otherwise email might be of help.

|  |
| --- |
| **CHAIRPERSON :** Dr. Mohammed Abdulrahman Al Ghonaim – MBBS, FRCP , Assistant professor, Nephrology unit, Department of MedicineEmail: malghonaim@ksu.edu.saExtension: 71903Bleep: 2391 |
| **MEMBERS** | **DEPARTMENT** | **CONTACTS** | **E-MAIL ADDRESS** |
| Dr. Mohammed Vohra | Anatomy | Extension : 99329 | vohra@ksu.edu.sa |
| Dr. Rana Hasanato | Biochemistry | Extension : 79093 | ranamomen@yahoo.com ;rhasanato@ksu.edu.sa  |
| Prof. Zahid Shakoor | Immunology | Extension : 71229 | shakoor\_zahid@yahoo.com |
| Dr. Malak Mohsen El Hazmi | Microbiology | Extension : 72693 | melhazmi@ksu.edu.sa |
| Dr. Hala Kfoury | Pathology | Extension : 71889 | hkfoury@ksu.edu.sahalakfoury@hotmail.com |
| Dr. Ishfaq Bukhari | Pharmacology | Extension : 71325 | bukharirph@yahoo.comishfaqbukhari@yahoo.com |
| Dr. Ahmed Fahim Ahmeda | Physiology | - | aelsheek@yahoo.com  |



**Feedback to Students on PBL Performance**

**Renal Block**

**Year 1 (Academic Year 2015-2016)**

**Student’s ID no : …………………………………………………Group number:…………**

**Student’s name:………………………………………………………………………………..**

**Tutor’s name……………………………………………………………………………….......**

-------------------------------------------------------------------------------------------------------------------

You will receive feedback on your performance in PBL tutorials from your tutor. After completing the 2nd PBL case, your tutor will meet with each student in your group on individual basis. He or she will use the following criteria for providing feedback on your performance. Feedback items are grouped under two main headings.

1= Deficient/lacking/or poor; 2= Working on it; 3= showing some improvement; 4 = developed; 5=well developed (marks are allocated as follows: 1 for rank 1, 2 mark for rank 2, 3 marks for rank 3, and 4 marks for rank 4, and 5 marks for rank 5, maximum mark is 5 for each group)

**1. Learning and cognitive skills:**

Ability to: 1 2 3 4 5

* Identify problems in the case
* Generate hypotheses
* Build mechanisms
* Collect new information
* Interpret findings
* Identify learning issues
* Apply knowledge learnt

 Mark= /5

**2. Interaction and participate to the group function:**

Ability to: 1 2 3 4 5

* Work collaboratively with other members
* Take active roles such as scribing
* Communicate effectively
* Arrive to tutorials on time
* Demonstrate good manners
* Keep the group focused
* Share resources with others

 Mark = /5

Tutor’s Name: Signature: Total Mark= /10

Comments

………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………



**Assessment of student’s Performance in PBL**

**Renal Block**

**Year 1 (Academic Year 2015-2016)**

**Student’s ID no.: ............................................................................ Group number:………………**

**Student’s name:…………………………………………..........................................................................**

**Tutor’s name:………………………………………….............................................................................**

**1=Unsatisfactory ; 2=Poor; 3=Good, 4=Very good; 5=Excellent**

 **1. Preparation and participation:**

Ability to:

* Contribute actively to discussion 1 2 3 4 5
* Use evidence when debate an issue 1 2 3 4 5
* Demonstrate critical analysis skills 1 2 3 4 5
* Integrate knowledge 1 2 3 4 5
* Demonstrate deep understanding 1 2 3 4 5

**Total Marks = 25**

**2. Professional behaviour:**

Ability to:

* Come to tutorials on time 1 2 3 4 5
* Communicate effectively 1 2 3 4 5
* Demonstrate good manners 1 2 3 4 5
* Keep the group focused 1 2 3 4 5
* Give and receive feedback 1 2 3 4 5

 **Total marks = 25**

-------------------------------------------------------------------------------------------------------------------------------

**Tutor’s Name: Signature: Total maximum Marks for the case = 50 /10 = 5 marks**

**Comments**

………………………………………………………………………………………………………………….………………………………………………………………..……………………………………………………………………………………………………………………………………………………………………..................................................................................................................................................................................................................

****

**STUDENT’S EVALUATION OF THEIR PBL TUTOR**

**Date: \_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_**

**Tutor’s Name: \_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_ Group No.:\_\_\_\_\_\_\_\_**

**Student: Peer: Other: Name (Optional):\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_**

**How well did the tutor facilitate group process in the following regards? Please put a check (✓) in the box.**

|  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- |
| 1. Appropriately facilitated the brainstorming sessions.
 | **1** | **2** | **3** | **4** | **5** |
| 1. Appropriately facilitated the hypothesis reorganization sessions.
 | **1** | **2** | **3** | **4** | **5** |
| 1. Appropriately facilitated the reporting sessions.
 | **1** | **2** | **3** | **4** | **5** |
| 1. Appropriately manage the time flow.
 | **1** | **2** | **3** | **4** | **5** |
| 1. Help to keep the group focused on its task
 | **1** | **2** | **3** | **4** | **5** |
| 1. Provided a well balanced intervention within the group process, but avoided dominating.
 | **1** | **2** | **3** | **4** | **5** |
| 1. Intervened when chairman or reporter needed.
 | **1** | **2** | **3** | **4** | **5** |
| 1. Provided constructive positive and constructive feedback to the group as needed.
 | **1** | **2** | **3** | **4** | **5** |
| 1. Encouraged positive and constructive feedback within the group about its performance
 | **1** | **2** | **3** | **4** | **5** |
| 1. Showed enthusiasm.
 | **1** | **2** | **3** | **4** | **5** |
| 1. Helped to create a supportive group climate.
 | **1** | **2** | **3** | **4** | **5** |
| 1. Encouraged logical and critical thinking.
 | **1** | **2** | **3** | **4** | **5** |
| 1. Overall rating of the tutor.
 | **1** | **2** | **3** | **4** | **5** |

Number Code Values:

5- EXCELLENT 4- VERY GOOD 3-GOOD 2- FAIR 1- POOR



  **STUDENT RATING OF LECTURES**

**Date: \_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_Subject: \_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_Instructor:\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_**

**Purpose:**

This form is designed as an observation tool to rate the performance of each instructor in the different sessions. It is intended to provide a tool for lecturer improvement.

**Directions:**

Using the anchors below, check (✓) your rating for each item below. Check (✓) N/A for items that do not apply.

|  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- |
| **No.** | **Standard Procedure** | **5** | **4** | **3** | **2** | **1** | **N/A** |
| **1** | Started and ended class on time. |  |  |  |  |  |  |
| **2** | Presented overview of content and objectives. |  |  |  |  |  |  |
| **3** | Presented information according to objectives. |  |  |  |  |  |  |
| **4** | Used relevant examples and illustrations (graphs, etc.) to explain major ideas |  |  |  |  |  |  |
| **5** | Used alternative explanations when necessary. |  |  |  |  |  |  |
| **6** | Made efficient use of questions with students. |  |  |  |  |  |  |
| **7** | Covered all contents/objectives. |  |  |  |  |  |  |
| **8** | Exhibited enthusiasm. |  |  |  |  |  |  |
| **9** | Encouraged students to express themselves. |  |  |  |  |  |  |
| **10** | Asked questions prior to closure |  |  |  |  |  |  |
| **11** | Summarized major points/related contents to objectives. |  |  |  |  |  |  |
| **12** | Amount you learned in the class was: |  |  |  |  |  |  |

**Mention 3 strong points in this lecture:**

1. **\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_**
2. **\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_**
3. **\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_**

**Mention 3 points for Improvement:**

1. **\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_**
2. **\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_**
3. **\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_**

**Your name: (optional) \_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_­­­**