

Tutor guide

M5- 2024-2025

First term

- Contents:

- 1-Guide lines (why P.B.L. “Problem Based Learning”) added to integrated system in October 6 university faculty of medicine (what the student & tutor will do this term) , (modules in this term & their general objectives)
- 2-Schedule for lectures , practicals , cases (small group teaching) , skill lab , & exams
- 3-Rubrics for grading assignments and presentations
- 4-Portfolio template (to be completed by each student and tutors and department members put the marks and be submitted to tutors by end of each module)
- 5-Cases (with objectives in tutor guide and without objectives in student guide)

- PBL Philosophy:

In a world where available information is growing exponentially, we believe that the most important thing a student needs to know is how to learn. So the main learning goals of the PBL are a framework for looking at concepts, skills, and abilities and help guide the creation of personalized student curriculum. PBL offers unique environments where students can flourish as individuals within a community of learners.

- PBL Process:

The core of the PBL process is the tutorials that will be held once weekly beside the practical sessions and the interactive lectures. In each tutorial there will be a case scenario that is delivered to the students, where they collaborate together through the seven jumps process to point out the possible problems present in the case and to find out the intended learning objectives need to be known through this case. In the second tutorial, they will discuss the objectives of the case after self study, and a new case will be delivered. In PBL process the role for lectures aim at clarification of complicated areas of information or to integrate different areas of information. Practical sessions and clinical skill lab are included as educational activities in BPL. They act as tools for the students to gain the needed psychomotor skills and to attain the professional attitude and behavior.

- Student role:

The student is the center of the learning process in PBL. **Students will depend on themselves in finding out the learning objectives by brain storming in the case study session. Then they will go home and study and search in the texts for the information of the objectives they got. Then the following session they should try to present the information they gazed and summarized to their students in an easy palatable way.** In PBL the students have to work hard, prepare themselves well for every tutorial group meeting, collaborate with their colleagues and practice team work. They also will have their reflection about the process, their colleagues and the tutor.

- Tutors role:

- The tutor will work as a facilitator more than traditional teacher who delivers all the information to the students. Tutors role is to stimulate and motivate the students to learn and to search for the information and knowledge. During the case they will guide the students and redirect them towards the intended learning objectives. The tutors share in the assessment process. Moreover, the tutor together with the students has the responsibility of setting the roles of the tutorial session.
- **The tutor will receive guide information for the objectives in each case from the departments at least one week before the case is to be discussed, he should read them and then in the discussion of the case he should see if the students had fulfilled all the needed items so as to approve their work or they need to search more for certain items and get them so as to complete their work completely or they got**

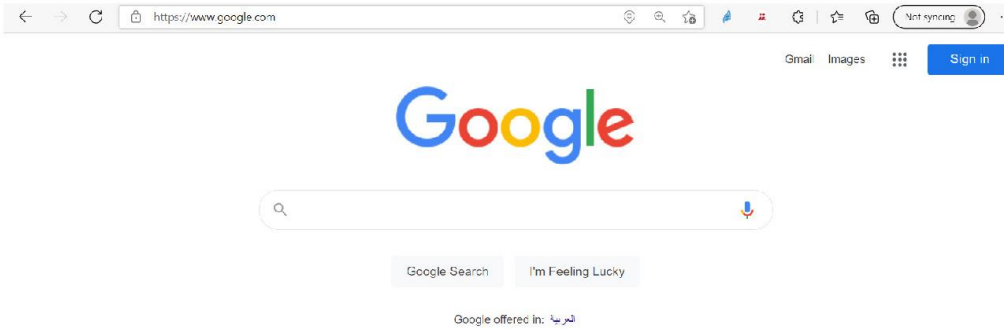
- more or un needed items they should discard them. By the end of the cases of the module students will have their hand out covering all items needed in the objectives they searched for
- All staff members should have their official mails done by the beginning of the academic year so as good communication may be applicable and to facilitate uploading of their lectures every Wednesday of each week
 - Concerning the module (URS) which is the beginning module for M5 the academic year 2022-2023.
 - In each session one of the students will be the reader (the one who reads the case) and another one will be the writer (the one who writes the objectives on the board after brain storming of the students with the tutor and collect them after that)
 - In session (1) (week 1)
 - One case will be red by the students
 - They make brain storming with each other and with the tutor to get the objectives the case is talking about. They will go home to search for them and make presentation about them the coming session.
 - Weeks for reading of the cases and discussion of the objectives are written above each case.
 - The presentation have certain rubrics the tutor try that the students stick more and more to them each presentation then at the last presentation of the module they will have certain mark among their portfolio total mark about:
 - The presentation they showed along the module and their share in the discussions and preparation of the work needed (see professional behavior sheet included) (the mark is given by the tutor)
 - After they finish the presentation in each session they will read the following case and brain storm to get the objectives that they will go home to prepare them as presentation in the coming case session and so on all the sessions
 - If the case is long its presentation by the students may take two weeks not one week to ensure that the students presented the objectives in the case in a good way
 - - All students are to make their Emails in the first week try to login to thr LMS so as to be able to reach the following:
 - Lectures
 - Videos
 - Presentation
 - On line exams formative questions

- Scoring Rubric for Presentations:

Category	Scoring Criteria	Total Points	Score
Organization (15 %)	Were the main ideas presented in a clear manner?	5	
	Information is presented in a logical sequence.	5	
	Presentation appropriately cites requisite number of references.	5	
Content (45 %)	- The Introduction is attention-getting, - It lays out the problem well, - It establishes a framework for the rest of the presentation.	5	
	Technical terms are well-defined in language that is appropriate for the target audience.	5	
	The Presentation contains accurate information.	10	
	The material included is relevant to the overall message/purpose.	10	
	Appropriate amount of material is prepared, and the points made reflect well their relative importance.	10	
	There is an obvious conclusion summarizing the presentation.	5	
Presentation (40 %)	Speaker maintains good eye contact with the audience and is appropriately animated (e.g., gestures, moving around, etc.).	5	
	Speaker uses a clear, audible voice.	5	
	Delivery is poised, controlled, and smooth.	5	
	Good language skills and pronunciation are used.	5	
	Visual aids are well prepared, informative, effective, and not distracting.	5	
	Length of presentation is within the assigned time limits.	5	
	Information was well communicated.	10	
Score %	Total Points	100%	

Steps to register on the Moodle e-learning website for Faculty of Medicine

1. Open any browser e.g. Chrome, Firefox, Edge, Safari

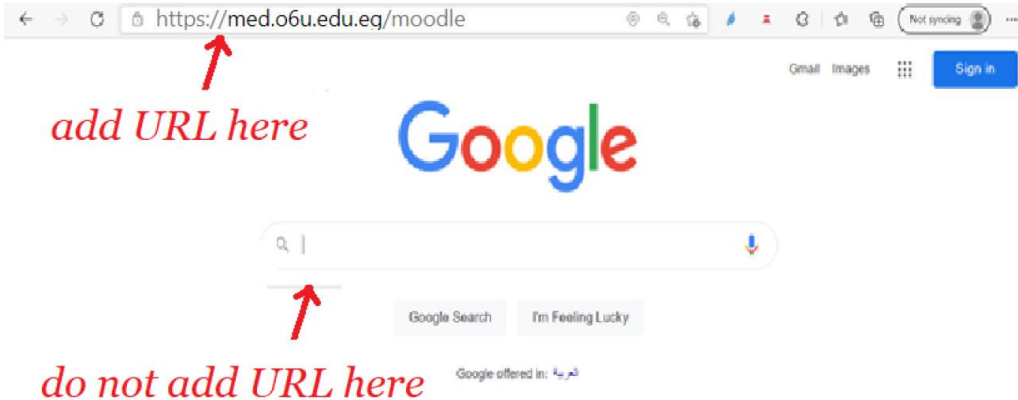


2. Then copy & paste this address in the URL box

<https://med.o6u.edu.eg/moodle>

N.B.

- It is https & not http
- There is no www in the address



3. Press "Enter" Key

o6u-med English (en) You are not logged in, (Log in)



الهيكـل الإداري لجامعة 6 أكتوبر- كلية الطب



رئيس مجلس الأمناء الأستاذ الدكتور أحمد زكي بدر

4. Click on "log in" in the upper right corner of the screen.



Forgotten your username or password?

Cookies must be enabled in your browser ?

Some courses may allow guest access

Username

Password

Remember username

Log in

Log in as a guest

5. Write your ID number twice: in the “Username” & in the “Password” here is an example:



20022792

.....

Remember username

Log in

Forgotten your username or password?

Cookies must be enabled in your browser ?

Some courses may allow guest access

Log in as a guest

6. Then click on “Log in” below. You will be asked to change your Password:

You must change your password to proceed. ×

Change password

Username 20022792

The password must have at least 8 characters, at least 1 digit(s), at least 1 lower case letter(s), at least 1 upper case letter(s), at least 1 non-alphanumeric character(s) such as *, -, or #

Current password ⓘ

New password ⓘ

New password (again) ⓘ

Save changes

There are required fields in this form marked ⓘ .

The new password must have at least 8 characters, at least 1 digit(s), at least 1 lower case letter(s), at least 1 upper case letter(s), at least 1 non-alphanumeric character(s) such as *, -, or #

Very Important: DO NOT FORGET THE NEW PASSWORD

How to enroll yourself in a Module?

Some modules need an “enrolment key” to enter it for the first time. Ask you teacher for this key.

Enrolment options

 Pediatrics-5

Teacher: Manar Aref

Teacher: Eman Sharaf

▼ Self enrolment (Student)

Enrolment key

[Enrol me](#)

Professional Behavior of student in the case checklist

Students Name:

Date: End of module (Summative):

Module title:

Student's Signature :..... Tutor's Name:.....

Criteria	Scale: 1 and 2 is unsatisfactory, 3, 4 and 5 is satisfactory performance					Comments
<u>Preparation:</u> Is well prepared with relevant information, uses a variety of references and summarizes key points	1	2	3	4	5	
<u>Critical thinking:</u> Identifies problem, analyzes problem, suggests possible reasons for the problem, helps group to formulate learning objectives	1	2	3	4	5	
<u>Participation:</u> Participates actively, talks on turn and listens attentively to others	1	2	3	4	5	
<u>Communication Skill & Group Skills:</u> Respects tutor and colleagues, communicates well uses appropriate language, accepts feedback and responds appropriately.	1	2	3	4	5	
Contributes to group learning, shares information with others, demonstrates sensitivity to views and feeling of others, takes on assigned tasks willingly						
<u>Presentation skills:</u> Presents the information relevant to the learning objective of the case, explains clearly the reasoning process with regard to solving the problem	1	2	3	4	5	

SATISFACTORY

UNSATISFACTORY

-The students portfolio (October 6 university - faculty of medicine - 2024 - 2025):

Portfolio :

It is a collection of student work , reflections , and educational experience done and arranged by the student for documentation and assessment.

Purpose of portfolio :

It is formed to monitor the student progress ,, assess the student achievement , and determine the student grades.

Goals

- Show learning progress over time
- Provide greater motivation for students
- Increase self assessment skills
- Encourage reflective learning
- Increase tutor student collaboration

Module :

Student name :

ID:

Level:

Academic year :

First term / Second term :

Task	Monitored by	Signature	Mark
-CV of student -Attendance -List of Cases taken or seen in the module -Objectives detected by brain storming -Presentation in front of colleagues against rubrics -your reflection concerning the sessions, cases , objectives , and presentation	Tutor		20%
-Topics of Lectures taken in the module	-Department member		10%

<p>-topics of practicals and laboratories taken in the module</p> <p>-Skills achieved in this module</p> <p>-Number of formative exams done</p> <p>-Your reflection concerning the lectures , practicals , skill labs , and formative exams</p>	<p>-Department member</p> <p>-professor in the skill lab</p> <p>-professor in the lecture</p> <p>-Department member</p>		
<p>Task needed by department or assignments or research work or video making</p>	-Department member		Mark for all departments sharing in module 25%
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<p>Points of strength you had in this module (<i>what you need to do using what you knew or what could have been better in your work after your knowledge</i>)</p>	Tutor		10%
<p>Points of weakness in this module and your sight to correct them</p>	Tutor		5%
<p>Describe your study day (<i>what you tried to learn , how your learning influenced your practice , the</i></p>	Tutor		10%

<i>most important thing you learnt in this module)</i>			
Meeting with staff member (<i>what was the objective and what was the result)</i>	Staff member		5%
Have you visited Alex (the talking patient robot , and SECTRA table) (<i>If YES please say how was your journey and if NO please say why)</i>	Tutor		3%
Describe your group work with your colleagues (<i>Team Based Learning)</i>	Tutor		10%
Any activities you have done	Tutor		2%

FIFTH YEAR	Mid module	Continuous assessment	End module	OSPE	OSCE
OBSTETRICS	27 marks electronic	10 marks total 1.5 attendance sections 1.5 attendance cases 4 presentation 3 portfolio	50 marks total 40 MCQ 10 SAQs electronic	38 marks total -slides electronic	OSCE
GIT SM502	50 marks electronic	10 marks total 1.5 attendance small groups 1.5 attendance cases 4 presentation 3 portfolio	80 marks total 70 MCQ 10 SAQs electronic	60 marks total -slides electronic	OSCE
URINARY SM503	27 marks electronic	10 marks total 1.5 attendance sections 1.5 attendance cases 4 presentation 3 portfolio	50 marks total 40 MCQ 10 SAQs electronic	38 marks total -slides electronic	OSCE

General objectives for the modules included in this term
Obstetrics module
Obstetrics

<p>A-Knowledge and understanding:</p>	<p>Graduates completing OB/GYN courses should :</p> <p>-IN OB/GYN:</p> <ol style="list-style-type: none"> 1. Terms and definitions in obstetrics and gynecology and their equivalent in Arabic and slang. 2. Basic theories and principles that govern ethical decision making in OB/GYN and the major ethical dilemmas in the field, particularly those that arise at the beginning and the end of life and from the rapid expansion of medical knowledge and technology with respect to the Islamic code of medical ethics. <p>-IN OBSTETRICS:</p> <ol style="list-style-type: none"> 3-Demonstrate knowledge of the clinical course of normal pregnancy, delivery and puerperium and the more common abnormalities found in a general practice: <p>-PREGNANCY</p> <ol style="list-style-type: none"> 4.Be able to outline the multi-system physiologic changes associated with pregnancy and its effects on common laboratory and diagnostic studies. 5.Know the basic concepts of fetal-placental physiology and function. 6. Know how to diagnose pregnancy by history, physical exam, and investigations. 7. Know how to conduct a routine antenatal clinic. 8. Know the various techniques for assessment of fetal wellbeing and their indications. 9. Know the etiology, diagnosis and management of the common obstetric problems. 10. Know how to identify a high risk pregnancy and to recognize the need for the referral of such a case. 11. Be able to describe potential consequences of common medical and surgical conditions in pregnancy. 12. Know the teratogenic potential of the commonly used medications . 13.know the indications and effects of immunizations during pregnancy. <p>- NORMAL LABOR AND DELIVERY</p> <ol style="list-style-type: none"> 14. Be able to describe the onset, stages, mechanisms and management of normal labor and delivery 15. Be able to identify common problems in obstetrics. 16. Be able to identify the deviation from the normal process of labour. <p>C- ABNORMAL LABOUR</p> <ol style="list-style-type: none"> 17. Know the diagnosis and management of abnormal labour and when to refer. 18.Know etiology, diagnosis and outline management of the common obstetric emergencies. <p>D- NORMAL AND ABNORMAL PUERPERIUM</p> <ol style="list-style-type: none"> 19. Demonstrate knowledge of the physiology of normal puerperium and changes which occur during it and its management. 20. Describe routine postpartum care in an uncomplicated pregnancy including breast-feeding. 21. Know the commonly used medications which are acceptable or unacceptable
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	<p>to use.</p> <p>22. Know evaluation and management of common puerperal complications.</p> <p>-PROCEDURES FOR OBSTETRICS/GYNECOLOGY</p> <ol style="list-style-type: none"> a. Know Basic components of pre-operative evaluation. b. Know components of routine post-operative care. c. Know common post-operative complications. d. Know The basics of wound healing and closure. e. Know Commonly used diagnostic and therapeutic modalities
<p>B- Intellectual Skills:</p>	<ol style="list-style-type: none"> 1. Formulate a differential diagnosis. 2. Integrate laboratory and diagnostic studies; 3. Accurately interpret the clinical impact of laboratory and diagnostic studies on case management. 4. Combine the clinical and investigational database, with the evidence based knowledge. 5. Choose possible treatment options; and monitor the patients response to the treatment plan. 6. Perform patient education and counseling; 7. Plans for follow up and continuity; and develop prognosis for an individual patient. 8. Develop a plan for any necessary further investigation 9. Consider economic, psychosocial and ethical issues. 10. Develop management strategies (both diagnostic and therapeutic) for patients with common obstetrics - gynecology conditions, and applying

	<p>principles of best evidence medicine.</p> <p>11. The student will be able to create a differential diagnosis of the "acute abdomen" in women of reproductive age, including care for a patient having acute abdominal pain.</p>
<p>C- Professional Skills:</p>	<p>THE PATIENT ENCOUNTER – CLINICAL SKILLS</p> <p>Student acquire a satisfactory clinical skill with regards to the following aspects:</p> <ul style="list-style-type: none"> - Develop the skill in taking a relevant history from an obstetric patient . - To carry out proper physical examination taking into consideration the peculiarity of a female patient. -To develop specific skills and basic clinical procedures in the area of obstetrics and to be able to undertake antenatal, intrapartum and postpartum care. - To diagnose common diseases and acute emergency cases and to administer initial management before being referred . - Showing an acceptable conduct and communicational skill to manage patients. <p>These goals will be achieved through the following objectives:</p> <ol style="list-style-type: none"> 1.Perform both comprehensive and problem-focused histories and physical examination: 2.Performing antenatal examinations. 3.Documenting clinical histories and examination findings. 4. Provide intrapartum care including: <ul style="list-style-type: none"> <input type="checkbox"/>performing at least 2 vaginal examinations <input type="checkbox"/>conducting at least 5 vaginal deliveries 5.Comply with infection control guidelines and applying standard protective precautions and proper surgical scrub technique and practicing sterile procedures and demonstrate proper hygienic practices when examining the patient. 6.Be able to perform ongoing bedside assessment of a pregnant patient and basic bedside procedures (e.g. placement of IV lines, Foley placement.) . 7.Able to use basic "tools" e.g: Stethoscope, Thermometer, sphygmomanometer ,measuring tape and Reflex hammer....etc. 8. Documenting clinical histories and examination findings. 9.Perform an emergency directed examination for patients with common obstetrics emergencies. 10. Accurately eliciting common physical findings and seek to correlate physical exam with expected disease process. 11. Accurately interpret patient responses and physical findings . 12.Create, prioritize and generate a problem list and synthesize the data obtained from the history and physical examination to arrive at a diagnosis and management plan. 13.Be able to provide basic antenatal care.

	<p>16. The student will observe and may assist in the operating and the delivery room.</p> <p>17. Demonstrate the ability to apply the principles of postpartum care and to perform an adequate post partum examination.</p> <p>18. Demonstrate the ability to accurate assessment and undertake care of the neonate during the hospital stay .</p> <p>19. Observe a demonstration of neonatal resuscitation, including intubation, of the neonate and perform mouth to mouth breathing when indicated .</p> <p>20. Communicate and demonstrate to the mother the technique and advise on breastfeeding immediately after delivery.</p> <p>21. Recognize the particular emotional needs of the mother and family in the postnatal and subsequent period.</p>
<p>D- General Skills:</p>	<p>In all health care settings, the Student should be able to:</p> <p>1- Recognize the particular emotional needs of the mother and family in the postnatal and subsequent period.</p> <p>2- Advise on subsequent family planning, Communicate recent knowledge of reproductive health to women.</p> <p>3- Demonstrate the ability to interact with the patient and families to gain her confidence and cooperation and assure her comfort and modesty.</p> <p>4- Communicating verbally and in writing with patients and colleagues.</p> <p>5- Create and sustain therapeutic and ethically sound relationships with patients and families utilizing a patient-centered approach.</p> <p>6- Establish rapport with patients and listen attentively to patients ,patient's relatives and other caregivers.</p> <p>7- Demonstrate a sensitivity to communicating with people from different cultural, community and religious backgrounds.</p> <p>8- Demonstrate the ability to address sensitive issues with compassion and demonstrate sensitivity to human differences and understanding of the impact of gender, ethnic, cultural, socioeconomic and other social factors.</p>

	<p>9- Demonstrate the ability to assess and counsel women for sex- and gender-appropriate reduction of risk, including lifestyle changes and genetic testing, in a manner that is sensitive to cultural beliefs.</p> <p>10- Demonstrate the ability to discuss social and healthy policy aspects of women's health, including ethical issues surrounding sterilization, domestic violence, adolescent pregnancy, access to health care ... etc.</p> <p>11- Demonstrate the ability to share knowledge effectively with peers: Communicate effectively with: Colleagues, Faculty, The community, and Other sectors and the media.</p> <p>12- Consult and perform appropriate referrals with other health care professionals to enhance the quality of care.</p> <p>13- Demonstrate basic skills and positive attitudes towards teaching others and demonstrate the effective use of educational principles to educate patients, families, and fellow health professionals about health care problems and develop a life long commitment to the education of others.</p> <p>14- Writing and presenting patient interviews.</p> <p>15- Demonstrate skills in reproductive and fertility counselling, in a manner that is sensitive to cultural and Islamic religious beliefs.</p> <p>16- Work cooperatively with the health care team.</p> <p>17- Establish rapport and be visible to faculty to be fairly evaluate.</p> <p>Management of Information/data and Information Skills The Student should be able to:</p> <p>18- Demonstrate the ability to use information technology to access OB/GYN information, critically assess current literature to support his own education and provide accessible educational information to patients.</p> <p>19- Practice evidence based medicine, utilizing biomedical information from electronic databases and other resources.</p> <p>20- Display the ability to use feedback to identify areas for improvement.</p> <p>21- Medical reporting and presentation skills</p> <p>22- Demonstrate the ability to communicate the results of the OB/GYN history and physical examination by well organized written notes and oral reports.</p> <p>Critical thinking and research skills</p> <p>23- Problem Solving Skills :Identify, formulate and solve OB/GYN patients' problems using scientific thinking and based on obtained and correlated information from different sources .</p> <p>24- Students will develop proficiency at problem-solving using both deductive and inferential reasoning as well as pattern or syndrome-recognition .</p> <p>25- Decision Making Skills: Understand the roles of complexity, uncertainty and probability in decisions in OB/GYN.</p> <p>26- Demonstrate skills to write and present a concise review article.</p> <p>ATTITUDINAL ILOs Professional Values, Attitudes, Behavior and Ethics ILOs. Students should:</p> <p>27. Show a respect to the sanctity of human life including intrauterine life.</p> <p>28. Demonstrate through the period of undergraduate medical education a pattern of responsible behaviors consistent with the highest ethical standards of the profession.</p> <p>29. Actively participate in learning opportunities.</p> <p>30. Appreciate that each patient is an individual human being with special needs and to be sensitive to those needs; and respect for their privacy, dignity and beliefs.</p>
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	<p>31. Demonstrate personal integrity, ethical behavior and altruism.</p> <p>32. Exhibit dependability and responsibility.</p> <p>33. Acknowledge and accept the limitations in his knowledge and clinical skills.</p> <p>34. Practice medicine, relate to colleagues, and conduct research according to the highest standards of ethics with respect to Islamic code of medical ethics.</p> <p>35. Demonstrate the ability to develop effective therapeutic relationships with OB/GYN patients.</p> <p>36. Appreciate the ethical, social and economic factors affecting OB/GYN patients and the profession.</p> <p>37. Obtain informed consent from patients before involving them in any aspect of training.</p> <p>38. Develop humanistic attitudes of honesty, fairness, chastity and compassion towards OB/GYN patients, peers and other members of the health care professions.</p> <p>39. Demonstrate ethical responsibilities in dealing with special circumstances as: Reproductive issues, Fertility, Contraception, Abortion, Genetics issues, diagnostic testing, pre symptomatic screening. ...etc.</p> <p>40. Respect the staff who teach and assist them in their clinical training and respect his colleague, nursing staff, and secretarial staff;</p> <p>41. Respect for the roles of other healthcare professionals in the care of the patient.</p> <p>42. Display responsibilities of the medical professional towards the local and global community.</p> <p>43. Commitment to clinical competence and lifelong education with recognition of the importance of self-assessment and of continuing medical education and a willingness to teach others.</p> <p>44. The student will be familiarized with the importance of teamwork among those committed to the improvement of health and health care of OB/GYN patients.</p> <p>45. Commitment to self-care and personal development.</p> <p>46. Exhibiting and displaying a professional image (professional" look) in manner, dress, speech and interpersonal relations that is consistent with the medical profession's accepted standards in the community and following the Islamic code medical ethics.</p> <p>47. Recognize the following issues that could affect a patient's management and modify management as appropriate:</p> <ul style="list-style-type: none">i. Legal issues (such as informed consent and malpractice).ii. Ethical issues (such as confidentiality).iii. Conflict of values between the patient and the community.iv. Psychosocial issues.v. Religious issues.
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<p>4- Course Content:</p>	<p>Obstetrics :-</p> <p>I: APPROACH TO THE PATIENT (OB/GYN) 1-History, Personal Interaction and Communication Skills. 2-Examination (including basic anatomy and embryology), Diagnosis and Management Plan. 3-Preventive Care and Health Maintenance, Pap Smear and Cultures, Cervical Cytology Screening, Abnormal Cervical Cytology 4-Legal and Ethical Issues in Obstetrics and Gynecology. 5-Premarital / Preconception care.</p> <p>II : NORMAL PREGNANCY 6-Maternal-Fetal Physiology, fertilization, implantation and early development of the fetus, placenta and cord. Placental function and abnormalities of placenta, Formation and function of liquor amnio, fetal circulation, physiology of pregnancy. 7- Antenatal care, Lifestyle Issues in Pregnancy: Health Education i. Antenatal Care First Trimester. ii. Antenatal Care Second Trimester. iii. Antenatal Care Third Trimester.</p> <p>III: ABNORMAL PREGNANCY: 8- Hyperemesis gravidarum. 9- Bleeding in early pregnancy : Abortion. 10- Bleeding in early pregnancy : Ectopic Pregnancy. 11- Bleeding in early pregnancy : Vesicular mole. 12- Medical and Surgical Conditions in Pregnancy (Abnormal Glucose Tolerance including Diabetes Mellitus – Anemia - Infections in Pregnancy: Rubella, Hepatitis B, Urinary Tract Infections (UTI), Other Relevant Infections in Pregnancy, Specific Labour Infections: Chorioamnionitis, Syphilis and Pregnancy, HIV/AIDS and Pregnancy 13- Hypertensive complications in pregnancy , preeclampsia- eclampsia syndrome. 14- Isoimmunization (Alloimmunization). 15-High risk pregnancy. 16-Second and Third-Trimester Bleeding :Antepartum Hemorrhage (APH). 17-Oligohydramnios and polyhydramnios 18- Multifetal Gestation. 19- Fetal Growth Abnormalities. 20-Fetal Death and Stillbirth.</p> <p>IV : NORMAL LABOUR 21- Normal Labour and Intrapartum Care , Basic anatomy of female bony pelvis and fetal skull, The Birth Plan, Pain Relief during Labour. 22- Antepartum and Intrapartum Fetal Surveillance, Assessment of Fetal Well Being.</p> <p>V: ABNORMAL LABOUR 23- Occipito posterior. 24- Face and Brow presentations. 25-Breech presentation. 26- Shoulder presentation. 27- Cord presentation and prolapse.</p>
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28-Complex presentation.
29-Preterm labour.
30-Preterm Prelabour Rupture of the Membranes (PPROM).
31-Postterm Pregnancy (Prolonged Pregnancy)
32- Abnormal uterine action.
33- Contracted pelvis and cephalopelvic disproportion.
34-Soft tissue Dystocia.
VI: COMPLICATED LABOUR
35-Obstructed labour.
36-Rupture uterus.
37- Genital tract injuries during labour.
38- Post Partum complications : Postpartum Hemorrhage (primary and secondary)
39- Other complications of labour:
_Amniotic fluid embolism
_Retained placenta
_Acute inversion of uterus
_Obstetric shock and Collapse
VII: THE NEWBORN INFANT
40- Immediate Care of the Newborn.
41-Fetal birth injuries.
42- Respiratory distress syndrome.
VIII: NORMAL AND ABNORMAL PUERPERIUM
43- Normal puerperium and Postpartum Care.
44-Lactation.
45-Postpartum Infection, Puerperal Pyrexia and puerperal sepsis.
46-Psychiatric aspect of pregnancy, labour and puerperium.
IX: PROCEDURES IN OBSTETRICS.
47-Ultrasound in Obstetrics.
48-Induction of labour.
49-Instrumental delivery in modern obstetrics.
50-Cesarean section.
51- Episiotomy.
52- Other Obstetric procedures.
X: VITAL STATISTICS IN OBSTETRICS.
53- Maternal and perinatal mortality.

	<p>XIV: MANAGEMENT OF INFORMATION/DATA AND INFORMATION SKILLS, CRITICAL THINKING AND RESEARCH SKILLS</p> <p>37. Using information technology to access OB/GYN information.</p> <p>38. Practice evidence based medicine in OB/GYN.</p> <p>39. Application of scientific method to patient care and career-long learning.</p> <p>40. A concise review article.</p>
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Urinary SM 503

Objectives

1. Structure and function of the kidney

Study the different function of the kidney

2. Clinical pathology

Learn and interpret renal function tests in different renal diseases. Recognize causes and laboratory diagnosis of different types of acid base disorders.

3. Acute renal failure

Know the causes of acute renal failure

How to diagnose acute renal failure

Different modalities used in treatment of acute renal failure

4. Chronic renal failure

Know the causes of chronic renal failure

How to diagnose chronic renal failure

Different modalities used in treatment of chronic renal failure

How to differentiate between acute and chronic renal failure

5. Dialysis and renal transplantation

Know the different methods of dialysis, hemodialysis and peritoneal dialysis and complications associated with each of them

Study the Indications of renal transplantation and long term complication

6. GN Nephrotic syndrome

To study the causes of nephrotic syndrome, different causes, types, clinical picture and how to treat each of them

7. GN Nephritic syndrome

To know the causes of nephritic syndrome, different causes, types, clinical picture and how to treat each of them

To differentiate between nephrotic and nephritic syndrome

8. Pathology of GN

Define glomerular diseases and types. Discuss causes of nephritic syndrome. Describe pathology of kidney diseases. Recognize cause and types of nephrotic syndrome.

9. Interstitial nephritis, Renal tubular acidosis, Hypo & hypernatremia, Hypo & hyperkalemia

Study the different causes of acute and chronic interstitial nephritis and how to differentiate between them and how to treat each of them.

To know and study the causes of renal tubular acidosis

To study the causes of hyponatremia & hypernatremia, their manifestations and how to treat it.

To know the causes of hyperkalemia & hypokalemia, their manifestations and how to treat them

10. Diuretics

Differentiate between and answer questions about osmotic diuretics, carbonic anhydrase inhibitors, loop diuretics and potassium sparing diuretics.

11. History and physical Examination (urology 1/2)

Study what are the important questions should be asked to the patients, how to analyze the symptoms, how to guide the patient, how to examine the case, what are the important points in clinical examination

12. Urine retention (Acute, chronic) (urology 1/2)

Discuss the definition, causes, types of obstruction, complication and the Differential diagnosis. Treatment

13. Urinary tract stones (urology 1/2)

Renal and ureteric stone

Study the incidence, clinical presentation, differential diagnosis of the symptoms, site of the stones, investigations, different types of managements (surgical, medical and conservative)

Bladder stone

Study the etiology, types, investigations, and management

14. Types of stones (urology 1/2)

Study the different types of stones, etiology, and compositions

15. Urinary Tract Trauma (urology 1)

Renal injury

Bladder injury

Urethral injury

Scrotal injury

Testicular injury

Penile injury

Study the types of injuries, etiology, clinical picture, investigations, modalities of management

16. Urinary Tract Infection

Etiology and organisms (micro 1/2)

Discuss the etiology, epidemiology and pathogenesis

Bacterial and viral infection (micro 1/2)

Discuss the virulence mechanisms and correlate it with specific pathogens
Describe the laboratory diagnosis and antimicrobial testing methods for bacterial, viral, fungi.

Parasitic infection (para 1)

Study Schistosomiasis and its types, life cycle, pathogenesis, mode of infection, Clinical picture, investigations, types of treatment

Acute pyelonephritis

Study the definition, etiology, risk factors, clinical picture, diagnosis and treatment.

Chronic pyelonephritis

Study the definition, etiology, risk factors, clinical picture, diagnosis and treatment.

Pyonephrosis

Study the definition, etiology, risk factors, clinical picture, complication diagnosis and treatment.

Perinephric Abscess

Study the definition, etiology, clinical picture, diagnosis and treatment.

Cystitis (Acute, Chronic and interstitial cystitis)

Study the definition, etiology, risk factors, clinical picture, diagnosis and treatment.

Prostatitis (Acute, chronic)

Study the definition, etiology, risk factors, clinical picture, diagnosis and treatment.

17. Urinary tract tumors (pathology 1) + Urology 1)**Renal cell carcinoma (Adult)**

Study the incidence, etiology, risk factors, pathogenesis, classification, clinical pictures, diagnosis and management.

Wilms' tumor (children)

Study the incidence, etiology, risk factors, pathogenesis, classification, clinical pictures, diagnosis and management.

Bladder carcinoma

Study the incidence, etiology, risk factors, pathogenesis, classification, staging clinical pictures, diagnosis and management.

Renal pelvis tumour

Study the incidence, etiology, risk factors, pathogenesis, classification, staging clinical pictures, diagnosis and management.

Testicular tumor

Study the incidence, etiology, risk factors, pathogenesis, classification, staging clinical pictures, diagnosis and management.

18. Hematuria (Urology)

Discuss the causes (renal, ureteric, bladder and urethra) , investigation

19. Prostate (pathology 1/2 +Urology 1/2)

Begin prostatic hyperplasia

Study the definition, gross and microscopic picture, clinical picture, diagnosis, complication (general and local), differential diagnosis, different modalities of treatment.

Prostatic carcinoma

Study the incidence, etiology, risk factors, pathogenesis, classification, staging clinical pictures, diagnosis and management.

20. Scrotal diseases (Urology 1)

Inflammation (Orchitis, epididymitis)

Study the definition, causes, clinical picture, and treatment

Scrotal torsion

Study the definition, causes, clinical picture, and treatment

D.D Scrotal swelling

Study the causes and differential diagnosis of scrotal swelling

Varicocele

Study the definition, clinical picture, complication and treatment

Hydrocele

Study the definition, clinical picture, complication and treatment

21. Congenital Abnormalities (Urology -1)

Hypospadias

Discuss the incidence, types, treatment

Epispadias

Discuss the incidence, types, treatment

UPJ Obstruction

Discuss the incidence, types, clinical picture, D.D and treatment

Ectopic testes

Study the incidence, types, Clinical picture, D.D, and treatment

Posterior urethral valve

Study the incidence, types, Clinical picture, D.D, and treatment

Gastrointestinal SM 502

Introduction and Gastrointestinal symptoms and signs and investigations of Gastrointestinal diseases

Objectives

The student will

- 1) Know the various symptoms of Gastrointestinal diseases
- 2) Understand the basic mechanism underlying each of the symptoms of gastrointestinal tract
- 3) Understand the basic diagnostic tools for Gastrointestinal diseases

THE ESOPHAGUS

Objectives

The student will

- 1) Know the Symptomatology of Esophageal diseases.
- 2) Understand the clinical manifestation , diagnostic approach and treatment of acid related esophageal diseases
- 3) Understand the clinical manifestation , diagnostic approach and treatment of motility disorders affecting the esophagus
- 4) Understand the clinical manifestation , diagnostic approach and treatment of cancer Esophagus .

THE STOMACH AND DUODENUM

Objectives

The student will

- 1-Understand the clinical manifestation , diagnostic approach and treatment of acid related gastric and duodenal diseases
- 2-Understand the clinical manifestation , diagnostic approach and treatment of peptic ulcer
- 3-Understand the, diagnostic approach and treatment of Helicobacter pylori
- 4- Understand the clinical manifestation , diagnostic approach and surgical management of complications of peptic ulcer
 - 1- Understand the clinical manifestation , diagnostic approach and management of acute and chronic gastrointestinal bleeding
 - 2- Understand the clinical manifestation , diagnostic approach and management of cancer stomach

3- Treatment of Peptic Ulcer Pharma Dr Hanan

DIARRHEA AND MALABSORPTION

Objectives

The student will

- 1) Know the definition of acute and chronic diarrhea

- 2) Understand the basic mechanisms and causes underlying acute and chronic diarrhea
- 3) Know the clinical presentation of various forms of acute and chronic diarrhea
- 4) Know the diagnostic studies for acute and chronic diarrhea
- 5) Understand the basic treatment options for acute and chronic diarrhea
- 6) Understand the basic concepts and mechanisms for malabsorption.
- 7) Know the various causes of malabsorption
- 8) Know the clinical features of various types of malabsorption
- 9) Know the diagnostic tools for malabsorption

Know the basic treatment options for different types of malabsorption

Foodborne illness Prof Mona Gharib

Parasitology Prof Nancy BLOOD FLUKES

INFLAMMATORY BOWEL DISEASE

Objectives

The student will

- 1) Understand the definition , basic concepts of inflammatory bowel disease
- 2) Understand the differences between types of inflammatory bowel disease
- 3) Know the clinical features of inflammatory bowel disease
- 4) Know the diagnostic tools for inflammatory bowel disease
- 5) Know the basic treatment options for inflammatory bowel disease

FUNCTIONAL GASTROINTESTINAL TRACT DISORDERS. IRRITABLE BOWEL SYNDROME

Objectives

The student will

- 1) Understand the concept of functional gastrointestinal tract disorders
- 2) Know the classification of functional gastrointestinal tract disorders
- 3) Know the clinical features of irritable bowel syndrome
- 4) Know the diagnostic tools for irritable bowel syndrome
- 5) Know the basic treatment options for irritable bowel syndrome

ACUTE ABDOMEN

Objectives

The student will

- 1) Understand the definition of acute abdomen

- 2) Know the various causes of acute abdomen, both medical and surgical causes
- 3) Know the clinical presentation of acute abdomen
- 4) Know the diagnostic studies needed to be done in acute abdomen

Dr Manal Pathology : GIT tumors

Cases for the fifth year students (first term 2024-2025)

Urinary module

lower urinary tract infection

Male, 65 years old, diabetic for 10 years, taking oral hypoglycemics, and hypertensive for 25 years, taking beta blockers. The patient has been diagnosed with CKD for three years with a baseline creatinine level of 1.8 g/dl

The patient presented to the emergency room with abdominal pain, diarrhoea, and anuria

On physical examination, the patient was awake, alert, and oriented to time, place, and person; BP 90/50, HR 100/min, Temp 38.5C; decreased skin turgor; and loss of elasticity. The left lower limb was swollen, hot, red, and tender; the sole of the foot was contaminated with a diabetic ulcer

The abdomen exhibits epigastric tenderness, but all other findings are normal. The chest examination was normal, and the heart reveals a mild, blowing systolic murmur at the heart's apex

HB 6,2 g/dl, Plt 245000, TLC 8.5, creatinine 2,6 g/dl, and urea 225 g/dl are the results of the investigation. Na 128 meq/L, K 4.9 meq/L, CRP 157, albumin 3.5 g; liver enzymes and bilirubin normal.

- 1- What is the most probable diagnosis?
- 2- What is the next step to reach the diagnosis?
- 3- What is the suggested line of management?

Case 2: Red week 3 and discussed week 4

Hematuria

A 68-year-old man diabetic and hypertensive under medications presents with microscopic hematuria.

- What are questions of the medical and surgical history should be asked?
- What are the clinical examination (general and local)?
- What are the investigations should be done?
- What are the D.D of hematuria?
- What are the recommended treatment?

Objectives: to discuss and study the following items

- Definition
- Causes
- Differential diagnosis
- Investigations
- Treatment (medical, surgical)

Tutor guide :

- 1- Hematuria
- 2- History and clinical examination
- 3- UTI
- 4- Stones
- 5- Tumors

GIT module :

Case 1 . Ulcerative Colitis. Red week 10 and discussed week 11

A 28-year-old man visits the emergency room after experiencing abdominal pain and diarrhoea for two days. He describes his movements as 10 to 12 per day, small in volume, occasionally containing blood and mucous, and preceded by an urgent need to defecate. The crampy, diffuse, slightly severe abdominal discomfort is not alleviated by defecation.

In the past six to eight months, he has experienced comparable episodes of abdominal pain and loose, mucoid stools, but they were less severe and resolved within twenty-four to forty-eight hours. He has no additional medical history and does not take any medication. He has neither travelled outside of Egypt nor had any contact with individuals exhibiting similar symptoms. He is a certified public accountant who does not smoke or consume alcohol. No one in his family suffers from gastrointestinal (GI) issues.

His temperature is 38 degrees Celsius; his heart rate is 110 beats per minute, and his blood pressure is 118/74 millimetres of mercury. He appears distressed, is perspiring, and is reclining motionless on the stretcher. His sclerae are anicteric, and his oral mucosa is pink and ulcer-free. His chest is clear, and his heartbeat is regular and free of murmurs. His abdomen is soft and minimally distended, with hypoactive bowel sounds, minimal diffuse tenderness, and no rebound tenderness.

White blood cell (WBC) count of 15,800/mm³ with 82% polymorphonuclear leukocytes, haemoglobin level of 10.3 g/dL, and platelet count of 754,000 are significant findings from laboratory tests. Negative HIV (human immunodeficiency virus) test results. Normal renal and hepatic function tests were conducted. A conventional abdominal radiograph reveals a mildly dilated colon with an air-filled diameter of 4.5 cm and no pneumoperitoneum or air/fluid levels.

- a) What is your provisional diagnosis?

- b) What is the differential diagnosis?
- c) What are the needed diagnostic studies?
- d) How would you treat such a case?
- e) What are the complications of this condition?

Objectives

The student will.

- 1) Know the nature of inflammatory bowel disease.
- 2) The student will understand the basic classification of inflammatory bowel disease.
- 3) The student will know the clinical presentation of ulcerative colitis.
- 4) The student will understand the major differences between Crohn disease and ulcerative colitis.
- 5) The student will know the extraintestinal manifestations of inflammatory bowel disease.
- 6) The student will know the diagnostic tools for inflammatory bowel disease.
- 7) The student will know the major complications of ulcerative colitis.
- 8) The student will understand the various treatment options for ulcerative colitis.

Tutor guide:

INFLAMMATORY BOWEL DISEASE AND ULCERATIVE COLITIS

Definition

- Chronic inflammatory diseases of the gastrointestinal tract.
- They are diagnosed by a set of clinical, endoscopic, and histologic characteristics, but no single finding is absolutely diagnostic for one disease or the other.
- Moreover, some patients have a clinical picture that falls between the two diseases and are said to have indeterminate colitis.

Epidemiology

- The highest rates occur in white populations in northern Europe and North America, where the incidence for each disease is about 5 per 100,000 and the prevalence is approximately 50 per 100,000.
- The peak age at onset is between 15 and 25 years of age, with a second, lesser peak between 55 and 65 years.

Clinical features

Ulcerative Colitis

- The dominant symptom in ulcerative colitis is diarrhea, which is usually associated with blood in the stool.
- Other symptoms include fever and pain, which may be in either lower quadrant or in the rectum.

- The initial attack of ulcerative colitis may be fulminant with bloody diarrhea, but more commonly the disease begins indolently, with non-bloody diarrhea progressing to bloody diarrhea.

Crohn's Disease

- Crohn's disease is marked by one of three major patterns: (1) disease in the ileum and cecum (40% of patients), (2) disease confined to the small intestine (30%), and (3) disease confined to the colon (25%).
- The predominant symptoms are diarrhea, abdominal pain, and weight loss.
- In patients with colonic disease, especially with rectal involvement, diarrhea is of small volume and associated with urgency and tenesmus.
- Prolonged inflammation and scarring in the rectum can leave it so rigid and nondistensible that the patient is incontinent.
- In disease confined to the small intestine, stools are of larger volume and not associated with urgency or tenesmus.
- Patients with severe involvement of the terminal ileum and those who have undergone surgical resection of the terminal ileum may have bile salt diarrhea or steatorrhea.

Feature	Crohn's Colitis	Ulcerative Colitis
Mucosal lesions	<u>Aphthous ulcers</u> are common in early disease; late disease is notable for stellate, rake, bear-claw, linear, or serpiginous ulcers and <u>cobblestoning</u>	Micro-ulcers are more common, but larger ulcers are seen <u>Pseudopolyps</u> are more common
Distribution	Often discontinuous and asymmetric, with <u>skipped segments</u> of normal intervening mucosa, especially in early disease	<u>Continuous, symmetric, and diffuse</u> , with granularity or ulceration found in the entirety of involved segments; however, peri-appendiceal inflammation (cecal patch) is common, even when the cecum is not involved
Rectum	Complete, or <u>more often relative, rectal sparing</u>	<u>Typically involved</u> with variable proximal distribution
Ileum	<u>Often involved</u> (=75% of cases)	<u>Not involved</u> , except as backwash ileitis in panulcerative colitis
Depth of inflammation	<u>Mucosal, submucosal, and transmural</u>	<u>Mucosal</u> ; transmural only in fulminant disease
Serosal findings	Marked erythema and creeping fat (the latter is virtually pathognomonic)	Absent except in severe colitis or toxic megacolon
Perianal complications	<u>Often prominent, including large anal skin tags, deep fissures, perianal fistulas, that are often complex</u>	<u>Not prominent</u> (fissure or fistula if present, should be uncomplicated)
Strictures	<u>Often present</u>	<u>Rarely present</u> ; when present, suggests adenocarcinoma
Fistulas	<u>Perianal, enterocutaneous, rectovaginal, enterovesicular, and other fistulas may be present</u>	<u>Absent</u> , except for the rare occurrence of rectovaginal or perianal fistula
Histopathology	<u>Granulomas are present in 15%-60% of patients</u> (higher frequency in surgical specimens than in mucosal pinch biopsies) Crypt abscesses may be present Focally enhanced inflammation, often on a normal background, is the hallmark	Granulomas should not be present (microgranulomas may be associated with ruptured crypt abscess) <u>Crypt abscesses and ulcers are the defining lesion</u> Ulceration on a background of inflamed mucosa
Serology	pANCA in 20%-25%, <u>ASCA in 41%-76%</u>	<u>pANCA in 60%-65%</u> , ASCA in 5%

Extra-intestinal Manifestations

COMPLICATIONS	CROHN DISEASE	ULCERATIVE COLITIS
Ocular disorders (uveitis, episcleritis)	+	+
Arthropathy	+	+
Oral ulcers	+	-
Skin disorders (pyoderma gangrenosum, erythema nodosum)	+	+
Nephrolithiasis	+	+
Primary sclerosing cholangitis	+	+
Bone disorders (osteoporosis, osteomalacia)	+	-
Thromboembolic disease	+	+
B ₁₂ deficiency	+	-

Diagnosis

- Laboratory Studies

A complete blood count (CBC), urinalysis, stool analysis and culture and serum chemistry tests are appropriate during the initial evaluation. Serologic examination of blood for specific antibodies, perinuclear antineutrophil cytoplasmic antibodies (pANCA) [frequent in UC], antibodies to *Saccharomyces cerevisiae* (ASCA) [frequent in CD].

A newly recognized and clinically useful inflammatory marker for UC disease activity is fecal calprotectin, which is a protein secreted by neutrophils in the feces and is therefore a marker of intestinal inflammation.

- Barium enema and barium follow through
- Endoscopy

Differential diagnosis

Diagnosis	Clinical Features	Radiologic and Colonoscopic Features	Histologic Features
UC	<u>Bloody diarrhea</u>	Extends proximally from rectum; fine mucosal ulceration	Distortion of crypts; acute and chronic diffuse inflammatory cell infiltrate; goblet cell depletion; crypt abscesses; lymphoid aggregates
Crohn's colitis	<u>Perianal lesions are common; may be associated with ileitis; frank bleeding is less common than in UC</u>	Segmental disease; rectal sparing; strictures, fissures, ulcers, fistulas; small bowel involvement	Focal inflammation; submucosal involvement; granulomas; goblet cell preservation; transmural inflammation; fissuring
Ischemic colitis	<u>Occurs in older adults; sudden onset, often painful; usually resolves spontaneously in several days</u>	Segmental splenic flexure and sigmoid involvement are most common, with thumbprinting early and ulceration after 24-72 hr; rectal involvement is rare	Mucosal necrosis with ghost cells; congestion with red blood cells; hemosiderin-laden macrophages and fibrosis (when disease is chronic)
Microscopic colitis	<u>Watery diarrhea; normal-appearing mucosa at colonoscopy</u>	Usually normal	Chronic inflammatory infiltrate; increased intraepithelial lymphocytes (lymphocytic colitis) and/or subepithelial collagen band (collagenous colitis)
Infectious colitis	<u>Sudden onset; identifiable source in some cases (e.g., <i>Salmonella</i> spp.); pain may predominate (e.g., <i>Campylobacter</i> spp.); pathogens are present in stool</u>	Nonspecific findings	Crypt architecture is usually normal; edema, superficial neutrophilic infiltrate, crypt abscesses
Amebic colitis	History of travel to endemic area; amebae may be detected in a fresh stool specimen but ELISA for amebic lectin antigen is the preferable diagnostic test	Discrete ulcers; ameboma or strictures	Similar to UC; amebae present in lamina propria or in flask-shaped ulcers, identified by periodic acid-Schiff stain
Gonococcal proctitis	Rectal pain; pus	Granular changes in rectum	Intense polymorphonuclear neutrophil infiltration; purulent exudate; Gram-negative diplococci
Pseudomembranous colitis	<u>Often a history of antibiotic use; characteristic pseudomembranes may be seen on sigmoidoscopy; <i>Clostridium difficile</i> toxin is detectable in stools</u>	Edematous; shaggy outline of colon; pseudomembranes may be identified radiologically or seen at colonoscopy	May resemble acute ischemic colitis; summit lesions of fibrinopurulent exudate

Treatment

Diet and nutrition

- Patients with mild symptomatic IBD usually are able to take food orally.
- The diet should be nutritious.
- Patients with Crohn's disease who have terminal ileal involvement and steatorrhea may require supplemental fat-soluble vitamins, medium-chain triglycerides, and parenteral vitamin B12.
- Replacement iron may be indicated in patients who are iron-deficient.

Corticosteroids

- Historically, corticosteroids have been used in patients with severe UC or Crohn's disease to induce a remission.
- Intravenous (IV) steroids (i.e., hydrocortisone 100 mg IV q6h or methylprednisolone 10-30 mg IV q6-8h) are usually used in such patients.
- When patients can take oral medications, prednisone at doses of 40 to 60 mg per day is usually given.

Antibiotics

- Bacteria is known to play an important role in the pathogenesis of Crohn's disease and it may play a role in UC.
- In Crohn's diseases, the indications for the use of antibiotics include perianal disease, localized peritonitis due to microperforation, bacterial

overgrowth secondary to a chronic stricture, and as an adjunct to drainage therapy for abscesses and fistulas.

- Metronidazole ,Ciprofloxacin and Rifaximin can be used.

Immunomodulators and biologic therapy

Immunomodulator drugs act by blocking lymphocyte proliferation, activation, or effector mechanisms

Azathioprine Methotrexate Cyclosporin can be used

Anti-Tumor Necrosis Factor Antibody (biologic therapy)

TNF is a key inflammatory cytokine and mediator of intestinal inflammation. The expression of TNF is increased in IBD.

Infliximab is a chimeric mouse-human monoclonal antibody against TNF that is effective in CD.

Anti-Adhesion Molecules

Adhesion molecules are important in cellular trafficking in IBD and other diseases, in which immune and inflammatory cells from the periphery are recruited into sites of inflammation.

Gut-selective anti-adhesion molecule, vedolizumab shows promising results.

Kinase Inhibitors

The Janus kinase (JAK) family of kinases, which include JAK1 and JAK3 which are critical for lymphocyte proliferation, function, and activation.

Tofacitinib, a novel oral inhibitor of JAK1 and JAK3, was recently shown to be safe and effective for the treatment of moderate to severe active UC.

Indications for surgical intervention

Ulcerative Colitis	Crohn's Disease
Intractable disease	Small Intestine
Fulminant disease	Stricture and obstruction unresponsive to medical therapy
Toxic megacolon	Massive hemorrhage
Colonic perforation	Refractory fistula
Massive colonic hemorrhage	Abscess
Extracolonic disease	Colon and Rectum
Colonic obstruction	Intractable disease
Colon cancer prophylaxis	Fulminant disease
Colon dysplasia or cancer	Perianal disease unresponsive to medical therapy
	Refractory fistula
	Colonic obstruction
	Cancer prophylaxis
	Colon dysplasia or cancer

Complications

Toxic megacolon

- Toxic megacolon is a condition in which the colon becomes atonic and dilated because of transmural inflammation.
- It is characteristically associated with severe ulcerative colitis, but it may complicate any severe inflammatory condition of the bowel, including Crohn's disease, bacterial colitis, amebiasis, pseudomembranous colitis, and ischemic colitis
- Worsening of the patient's clinical condition and the development of fever, tachycardia, and leukocytosis.

- Medical therapy is designed to reduce the likelihood of perforation.
- If the patient does not begin to show signs of clinical improvement during the first 24 to 48 hours of medical therapy, the risk for perforation increases markedly, and surgical intervention is indicated.

Malignancy

- Patients with extensive ulcerative colitis have a markedly increased risk for colon cancer in comparison to the general population beginning 8 to 10 years after diagnosis and increasing with time.
 - There should be surveillance colonoscopy with random biopsies in patients with long-standing ulcerative colitis beginning 8 to 10 years after the onset of disease and repeated every 1 to 2 years.
 - If the specimens show dysplasia the patient is sent for colectomy.
-

Case2 Non-Alcoholic Fatty Liver Disease. Read week 12 and discuss week 13

A 37-year-old morbidly obese, previously diagnosed diabetic male was discovered to have an echogenic liver during an abdominal ultrasound for hematuria. He has no discomfort in the right upper quadrant. He consumes alcohol infrequently and does not take his diabetes medications regularly. In addition to hepatomegaly, his physical examination revealed pathological obesity with a body mass index of 42 kg/m² and morbid obesity. The results of a blood test reveal slightly elevated aminotransferase levels, significantly elevated blood sugar levels, and HbA1c levels of 14.

- a) What is the provisional diagnosis?
- b) What are the usual clinical features of such a condition?
- c) What are the different causes for this condition?
- d) What are the diagnostic tools for this condition?
- e) What are the treatment options for this condition?

Objectives;

- 1) The student will understand the nature of non-alcoholic fatty liver disease
- 2) The student will know the various causes of non-alcoholic fatty liver disease
- 3) The student will know the clinical presentation of non-alcoholic liver disease
- 4) The student will know the various diagnostic tools for non-alcoholic fatty liver disease
- 5) The student will understand the treatment options for non-alcoholic fatty liver disease

Tutor guide:

Non-Alcoholic Fatty Liver Disease

- Alcoholic liver disease and nonalcoholic fatty liver disease (NAFLD), which represent two of the most common forms of liver disease, can lead to cirrhosis, liver failure, and death.
- Although these two conditions have different risk factors and natural histories, in both conditions the hepatocytes are characterized by macrovesicular steatosis, which is the accumulation of triglycerides as one large cytoplasmic globule that displaces the nucleus.
- In microvesicular steatosis, cytoplasmic accumulation of fat occurs as multiple small globules with a central nucleus.
- On histologic examination, NAFLD resembles alcoholic liver disease, but it occurs in individuals without significant alcohol consumption.
- In addition, the definition of NAFLD excludes patients with a history of exposure to steatogenic medications, such as amiodarone, methotrexate, and tamoxifen.
- NAFLD encompasses a spectrum of abnormal liver histologic features, ranging from simple steatosis to nonalcoholic steatohepatitis (NASH) and cirrhosis.
- Nonalcoholic fatty liver disease (NAFLD) is a Clinicopathologic syndrome that encompasses several clinical entities that range from simple steatosis to steatohepatitis, fibrosis, and ESLD in the absence of significant alcohol consumption.
- Nonalcoholic steatohepatitis (NASH) is part of the spectrum of NAFLD and is defined as steatosis with hepatocellular ballooning plus lobular inflammation.
- **Etiology**
- Etiologies include metabolic syndrome, drug-induced liver injury (amiodarone, nifedipine, estrogens), surgical procedures (jejuno-ileal bypass, extensive small-bowel resection, biliary and pancreatic diversions), and miscellaneous conditions (total parenteral nutrition, hypobetalipoproteinemia, environmental toxins, etc.).
- **Causes**

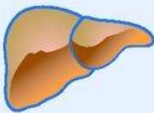
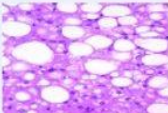
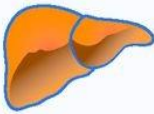
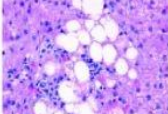
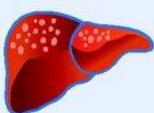
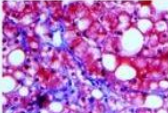

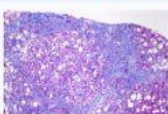
Acquired Metabolic Disorders
Diabetes mellitus
Dyslipidemia
Kwashiorkor and marasmus
Obesity
Rapid weight loss
Starvation
Cytotoxic and Cytostatic Drugs
Azacitidine
Bleomycin

Cisplatin

Pathogenesis

- Two fundamental defects in NAFLD are insulin resistance/ hyperinsulinemia and excessive levels of nonesterified fatty acids within the hepatocytes.
- An excessive influx of nonesterified fatty acids into the hepatocytes results in macrovesicular steatosis.
- Free fatty acids within the hepatocytes are considered the primary mediators of cell injury (lipotoxicity).
- Adipocytokines that play an important role in the pathogenesis of NAFLD include adiponectin and TNF- α .

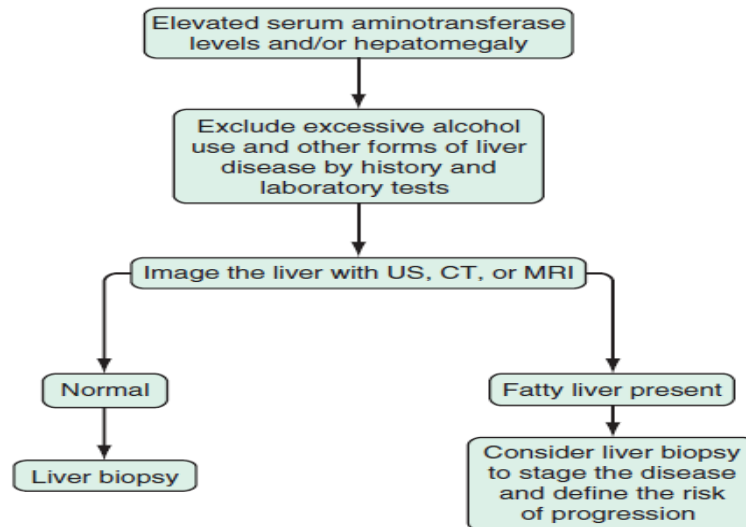
NAFLD: Spectrum of disease

	Image	Histopathology	Pathophysiology
Non-alcoholic fatty liver (hepatic steatosis)			Accumulation of fat in liver (when excessive alcohol consumption is ruled out).*
Non-alcoholic steatohepatitis (NASH)			Accumulation of fat in liver is combined with inflammation and cell damage.
Fibrosis			Scarring (excess fibrous tissue) in an inflamed liver. Categorized into stages 0 to 4 (or mild, moderate and advanced) based on extent and distribution of scarring.
Cirrhosis			Late stage of chronic liver disease marked by nodules of damaged liver cells surrounded by scarring.

Clinical presentation

Symptoms	Signs	Laboratory Features
Common None (48%-100% of patients)	Hepatomegaly	2- to 4-fold elevation of serum ALT and AST levels AST/ALT ratio < 1 in most patients Serum alkaline phosphatase level slightly elevated in one third of patients Normal serum bilirubin, serum albumin, and prothrombin time Elevated serum ferritin level
Uncommon Vague right upper quadrant pain Fatigue Malaise	Splenomegaly Spider telangiectasias Palmar erythema Ascites	Low-titer (<1 : 320) antinuclear antibodies

Diagnosis of NAFLD



- US and CT had sensitivity rates of 100% and 93%, respectively, for detecting hepatic fat involving greater than 33% of the liver.
- Role of liver biopsy:

In practice, most patients with NAFLD do not undergo a liver biopsy. The ability to distinguish NASH from IFL (isolated fatty liver) is critical because patients with NASH are at risk of progression to cirrhosis. The ideal approach would be to select patients for whom liver biopsy might influence management through more aggressive treatment.

- Imaging to Detect Fibrosis
 - Newer imaging techniques have been successful in identifying hepatic fibrosis.
 - The most studied and widely available has been transient elastography (Fibroscan) which uses a low- amplitude shear wave that propagates through the liver parenchyma.
 - The speed at which the wave moves is correlated with liver stiffness, measured in kilopascals.

Treatment

- 1) Diet
- 2) Exercise
- 3) Drug therapy
 - Vitamin E 800-1000 IU daily
 - Pioglitazone 30-45 mg daily
 - Pentoxifylline 400 mg three times daily
 - Statins
- 4) Bariatric surgery

Obstetrics module :

Case 1: GIT disorder (liver)

Acute Fatty liver in pregnancy

- ✓ A 23-year-old G1P0 presents with a 3-day history of malaise, anorexia, nausea, and vomiting. Her first trimester sonography reveals she is 34 weeks pregnant. Her pregnancy has thus far been uncomplicated. She denies a past of medical issues and operations.
- ✓
- ✓ On physical examination, the patient has jaundice symptoms. Temperature: 98.9 degrees Fahrenheit; blood pressure: 120/78 mm Hg; pulse: 105 beats per minute; respiratory rate: 18 breaths per minute; lungs: clear; heart rhythm: regular. HR: 99 beats per minute with a systolic ejection murmur of grade 2/4. The fundal height is 33 cm, and there is no guarding or recurrence in the epigastric tenderness. The extremities lack edema and tenderness. The foetal heart rate trace demonstrates a baseline of 150 seconds, moderate variability, positive accelerations, and no decelerations. On the tocodynamometer, irregular contractions are detected every 10 to 25 minutes, although the patient is unaware of them. On digital examination, her cervix appears closed and long. The ultrasound performed at the bedside reveals foetal biometry consistent with 34 weeks, an anterior placenta, and normal amniotic fluid.
- ✓
- ✓ During the evaluation, the patient vomited three times. Antiemetics and intravenous fluids with potassium are administered. The results of the laboratory are as follows: Hct 33%, WBC 19 x 10³/L, platelet count 127,000/mm³, AST 482 IU/L, ALT 402 IU/L, conjugated bilirubin 5.2 mg/dL, total bilirubin 6.0 mg/dL, LDH 302, serum creatinine 1.1 mg/dL, serum glucose 51 mg/dL, K⁺ 3.0 mEq/L. Amylase, lipase, ammonia, uric acid, and coagulation tests are normal. Urine analysis is notable only for specific gravity of 1.03 and large ketones, but is negative otherwise. What is the differential diagnosis?
- ✓ What is the most likely diagnosis?
- ✓ What are the maternal risks associated with this diagnosis?
- ✓ What are the foetal risks associated with this diagnosis?

Objectives

1. Recognize the clinical presentation of AFLP.

2. Become familiar with the evaluation and management.
3. Understand genetic implications associated with AFLP.

Tutor guide :

ANSWERS TO CASE 14;

Acute Fatty Liver of Pregnancy

Summary: This is a 23-year-old G1 at 34⁰/₇ weeks' gestation with malaise, nausea, vomiting, abdominal tenderness as well as clinical and laboratory evidence of liver dysfunction.

- ✓ **Differential diagnosis:** In the second and third trimester, acute fatty liver of pregnancy (AFLP), intrahepatic cholestasis of pregnancy (IHCP), and severe preeclampsia with HELLP (hemolysis, elevated liver enzymes, low platelets) should be considered in a patient with evidence of liver dysfunction. Other conditions that may occur at any gestational age include viral hepatitis, pancreatitis, drug toxicity, cholelithiasis or rarely, malignancy. Conditions associated with nausea, vomiting, and abdominal pain that should also be considered in the differential include pyelonephritis, appendicitis, and hyperemesis gravidarum (HEG), however, these are less likely in this case.
- ✓ **Most likely diagnosis:** The most likely diagnosis is AFLP given this patient's symptoms, evidence of liver dysfunction, and hypoglycemia.
- ✓ **Maternal risks associated with this diagnosis:** Maternal complications include pulmonary edema, coagulopathy, acute renal failure, infection, pancreatitis, diabetes insipidus (DI), hepatic encephalopathy, coma, liver transplantation, and maternal death.
- ✓ **Fetal risks associated with this diagnosis:** Fetal demise is a potential complication with AFLP if the diagnosis is delayed and delivery is not expedited. Prematurity complications are increased due to risk of both spontaneous and iatrogenic preterm birth. The fetus may also be affected with a fatty acid oxidation disorder.

ANALYSIS

Considerations

It is not uncommon for pregnant women to present with nonspecific symptoms of nausea, vomiting, and malaise. In most instances, these symptoms may be attributed to normal pregnancy or hyperemesis gravidarum (HEG), especially during first trimester. Other times symptoms are secondary to a benign, self-resolving process such as a viral syndrome. On rare occasions such as this, nonspecific symptoms may represent a serious and potentially life-threatening condition.

Acute fatty liver of pregnancy (AFLP) should always be in the differential for any patient who presents in the third trimester with nausea, vomiting, and abdominal pain. Although the incidence of AFLP is reportedly 1 in 7000 to 1 in 16,000 pregnancies, this may very well be an overestimation from published case series derived from tertiary referral centers.

Often times, there is a delay in diagnosis as there are overlapping clinical and laboratory findings of AFLP with other conditions. The major diagnosis that must be excluded is preeclampsia (HELLP syndrome). Although the frequency of hypoglycemia is variable with AFLP, its presence may help to distinguish it from HELLP syndrome. Another laboratory abnormality seen with AFLP that is not seen with HELLP syndrome is **a markedly reduced antithrombin III levels**. However, routine testing for antithrombin III is not practical since it may take several days to obtain results. In the absence of hypertension and proteinuria, the diagnosis of HELLP syndrome is also less likely; however, it is important to note that approximately 15% and 10% of HELLP syndrome occurs in the absence of hypertension and proteinuria, respectively. Hemolysis occurs in both conditions but is more common with HELLP syndrome than with AFLP. On the other hand, jaundice, coagulopathy, and impaired renal function are more common with AFLP than with HELLP syndrome. Liver biopsies from cases of HELLP syndrome reveal periportal hemorrhage and fibrin deposition which is in contrast to the microvesicular fatty infiltration seen with AFLP! Fortunately, the management of both conditions is similar in that delivery is indicated when either is suspected.

Another pregnancy-specific condition associated with liver dysfunction that needs to be considered in the third trimester is intrahepatic cholestasis of pregnancy (IHCP). The predominant symptom with IHCP is pruritus (without a rash) which helps to distinguish it from AFLP. Other conditions that need to be considered that are coincidental to pregnancy and can occur at any gestational age include viral hepatitis, cholelithiasis, pancreatitis, drug induced toxicity, and, rarely, thrombotic thrombocytopenic purpura (TTP) and hemolytic uremic syndrome (HUS). It is also important to note that pyelonephritis and acute appendicitis must be considered with any patient that presents with nausea, vomiting, and abdominal pain. Because management strategies and outcomes differ among these conditions, obtaining the correct diagnosis is of the utmost importance.

The diagnosis of AFLP is most commonly made by clinical symptoms and laboratory findings. In this ill-appearing patient with emesis, abdominal tenderness, jaundice, liver impairment, and hypoglycemia, the most likely diagnosis is AFLP. Renal insufficiency, hemolysis, hemoconcentration, and leukocytosis are also evident in this patient and are not uncommon with AFLP. Hospitalization is indicated as this condition is progressive and sudden deterioration of both mother and fetus may occur at any time. The ultimate treatment is delivery after maternal stabilization. As long as maternal and fetal status is stable and reassuring, this patient is a good candidate to undergo induction of labor. Cesarean delivery should be reserved for the usual

obstetrical indications or if delivery is not affected within a reasonable time and maternal and/or fetal status deteriorates.

APPROACH TO

Acute Fatty Liver of Pregnancy

AFLP affects women of all ages, race, and geographic areas. It is more common in nulliparas with a male fetus and in multiple gestations. It usually manifests in the third trimester with onset between 28 to 38 weeks' gestation. Although rare, earlier cases have been reported in the second trimester. Less often, symptoms may not develop until the first few days of the postpartum period.

The typical patient appears ill with nonspecific symptoms for 1 to 2 weeks. The most common symptoms are **nausea and vomiting** (75%), **abdominal pain** (50%), jaundice (37%), and malaise (31%). **Half of women with AFLP may also have preeclampsia** at presentation or at some point during their disease course. Patients occasionally present with altered mental status due to hepatic encephalopathy. On physical examination, the patient may have hypertension, tachycardia (secondary to dehydration), low-grade fever, mild jaundice, and/or epigastric tenderness. In cases of severe coagulopathy, bleeding may be evident from multiple sites. Although less common (15%-20% cases), patients may actually be asymptomatic at the time of presentation or they may present for other reasons. Some patients may present with preterm contractions/labor or with decreased fetal movement (secondary to maternal acidosis).

As the disease progresses, additional complications may arise. Marked hypoglycemia may occur with variable frequency, developing in 17% to 100% of women due to impaired gluconeogenesis from liver dysfunction. A recent review of 16 cases of AFLP from three tertiary centers showed a 50% incidence of hypoglycemia.

Hypoglycemia has also been found to be more common in the postpartum period. Additional maternal complications are hepatic encephalopathy (60%), acute renal failure (50%), disseminated intravascular coagulopathy (DIC) (55%), pulmonary edema/acute respiratory distress syndrome (ARDS) (25%), and infection.

Acute pancreatitis may complicate 15% of cases and confers a poor prognosis. This complication may occur later in the disease process and is associated with

Hyperglycemia.

Transient central diabetes insipidus in first week after delivery has also been reported. The mechanism for this is unknown but it is presumably from elevated vasopressinase levels which results in prodigious urine output.

Laboratory evaluation usually reveals parameters consistent with liver dysfunction. Prolonged clotting times, reduced fibrinogen as well as antithrombin are associated with coagulopathy. Hyperbilirubinemia is usually of the conjugated type and has been reported to be in the range of 5 to 10 mg/dL. Liver enzymes (aspartate transaminase, alanine transaminase) are mildly elevated, rarely exceeding 1000 U/L. Endothelial

exudation activation may cause hemoconcentration, leukocytosis, and thrombocytopenia. Hemolysis is likely from impaired cholesterol synthesis which contributes to erythrocyte membrane damage. LDH levels may vary from 250 to 4000 IU/L. As renal failure progresses, metabolic acidosis with elevated creatinine and uric acid levels is common. Either hypoglycemia or hyperglycemia may be present with the latter occurring in association with pancreatitis. In 12 cases of AFLP complicated by pancreatitis, the average peak for amylase was 552 U/L (range 26-113 U/L) and lipase 1866 U/L (range 100-5869 U/L). Elevated ammonia levels are more common with progressive liver failure. Table 14-1 shows a summary of laboratory findings of 169 women with AFLP taken from six published studies.

Computed tomography (CT), magnetic resonance imaging (MRI), and ultrasound imaging of the liver have limited usefulness and therefore the diagnosis of AFLP is made by clinical and laboratory evaluation. Hepatic imaging is helpful in excluding other disorders that may be confused with AFLP when the diagnosis is unclear. It is useful in detecting suspected complications of AFLP such as a pancreatic pseudocyst or hemorrhagic pancreatitis. The finding of fat on CT or ultrasound has been reported in cases of AFLP. This finding is nonspecific. The gold standard for diagnosis is the presence of microvesicular fatty infiltration of hepatocytes on liver biopsy. In patients without this diagnostic feature, special staining with oil red O on frozen section or electron microscopy can help to confirm the diagnosis of AFLP. Liver biopsy may be considered after coagulopathy has been corrected in those in whom the diagnosis remains in question; however, it is rarely used in clinical practice.

Once the diagnosis of AFLP is made, delivery is recommended regardless of gestational age. Antenatal corticosteroids to induce lung maturity can be administered as soon as preterm birth is anticipated if gestational age < 34 weeks. Delivery should not be delayed to complete a full course of antenatal steroids once the diagnosis is made as maternal and fetal status may deteriorate trying to accomplish this. Consultation with maternal-fetal medicine and neonatology is advised. Although there is no definitive treatment, maternal stabilization with supportive care is a mainstay of management.

TABLE 14-1 LABORATORY FINDINGS IN 169 WOMEN WITH AFLP

STUDY	N	WBC (X10 ³ /μL)	FIBRINOGEN (MG/DL)	PLATELET (X10 ³ /μL)	AST (IU/L)	ALT (IU/L)	T.BILIRUBIN (MG/DL)	D. T.BILIRUBIN (MG/DL)	GLUCOSE (MG/DL)	CR (MG/DL)	AMMONIA (μMOL/L)	LDH (IU/L)
Reyes ^a	12	18.5 (8.5-34.2)	Na	Na	Na	315 (28-1200)	9.1 (1.8-17.3)	6.4 (0.64-12)	Na	Na	Na	Na
Usta ^a	14	Na	139 (37-110)	126	1067 (200-3670)	Na	Na	Na	Na	2.4 (1.1-3.6)	Na	Na
Castro ^a	28	Na	125 (32-446)	113 (11.186)	210 (45-1200)	Na	Na	Na	Na	2.5 (1.1-5.2)	Na	Na
Pereira ^b	32	Na	Na	123 (25-262)	99 (25-911)	Na	8.3 (3.7-37.5)	Na	Na	2.7 (1.1-8.6)	Na	Na
Vigil-De Gracia ^a	10	27.4 (8.5-34.2)	136 (15-345)	76 (21-179)	444 (85-1025)	392 (107-900)	11 (1.5-27)	Na	37 (6-59)	Na	Na	993 (379-1503)
Fesenmeier ^a	16	Na	Na	151 (33-303)	523 (120-2371)	423 (43-1504)	Na	5.8 (0.9-11.9)	81 (11-159)	2.4 (0.5-4.4)	69.7 (15-150)	1438 (244-3922)
Knight ^b	57	20.7 (8.5-46.5)	Na	122 (14-436)	310 (37-3198)	300 (21-1156)	Na	5.9 (1.0-39.6)	56 (18-148)	1.9 (0.7-4.5)	73 (22-121)	Na

It is important to monitor vital signs and repeat laboratory tests every 6 to 8 hours or more frequent as needed. Transfusion of blood products may be necessary for correction of clinical coagulopathy and anemia. Serial glucose monitoring is also recommended with intravenous glucose infusion in order to maintain blood glucose levels > 60 mg/dL. It also is important to correct any electrolyte abnormalities and reduce high ammonia levels with lactulose when necessary. Continuous fetal monitoring to assess fetal well-being is also recommended. It is important to note that the fetal status may deteriorate rapidly, likely secondary to maternal acidosis or uteroplacental insufficiency or both.

Induction of labor is very reasonable in the absence of other indications that may preclude vaginal delivery. Vaginal delivery is optimal in a patient with AFLP who is at very high risk of hemorrhage and postoperative complications from AFLP. Cesarean delivery should be considered for the usual obstetrical indications or if maternal/fetal status deteriorates necessitating expeditious delivery. Anesthesia should be based on coexisting coagulopathy. If preeclampsia develops, magnesium sulfate should be administered for seizure prophylaxis.

Intensive care unit admission may be necessary intrapartum or postpartum for several reasons including maternal stabilization as well as close monitoring of laboratory abnormalities and complications that may develop in the postpartum period. Although clinical improvement is expected 2 to 3 days after delivery, laboratory abnormalities may continue for 7 to 10 days. Fulminant liver failure and need for liver transplant is very rare but has been reported.

The maternal mortality rate in older studies was reportedly in the order of 60% to 70%. In more recent case series, the maternal mortality rate has been found to be lower, in the range of 1.8% to 20%. Prompt diagnosis and aggressive treatment of associated complications may contribute to improved maternal outcome. Perinatal mortality is approximately 13% based on the most recent literature; however, neonatal morbidity remains high due to prematurity complications.

Molecular advances have improved our understanding of the pathogenesis of AFLP. Several reports have provided evidence to support an association between AFLP and an inherited fatty acid oxidation disorder of **long-chain 3-hydroxyacyl-CoA dehydrogenase (LCHAD)** deficiency in the fetus. This enzyme catalyzes the third step in the β -oxidation of fatty acids in mitochondria by facilitating the formation of 3-ketoacyl-CoA from 3-hydroxyacyl-CoA. With deficiency of the enzyme, the latter metabolite accumulates in the fetus and enters the maternal circulation causing hepatic fat deposition and impaired liver function.

The most common mutation of LCHAD deficiency associated with AFLP that has been studied is **E474Q** (or GC1528C). In a study of 24 families, Ibdah and colleagues examined the association between LCHAD deficiency in children and severe liver disease in their mothers. Of the 19 children that were found to be homozygous for E474Q or compound heterozygous (E474Q plus a different mutation on the other allele), the majority of these mothers developed AFLP, HELLP syndrome, or both during their pregnancy involving these children. Thus based on this study, carrying a fetus with LCHAD deficiency is associated with a 79% risk of developing AFLP or HELLP syndrome with the former having a stronger association than the latter. A subsequent prospective study confirmed this association and it is suggested that 1 in 5 women who develop AFLP may carry an LCHAD deficient fetus. Whether the pathogenesis of AFLP and HELLP syndrome is related remains unclear. At

present, 17 mutations of B-oxidation have been identified raising the possibility that other defects may be associated with ALFP but are less well studied to date.

Molecular testing for the most common mutation, E474Q, and known genetic variants are available for women who develop AFLP, their partner, or their infants. This information is important for counseling a couple regarding their risk of having an affected fetus with LCHAD deficiency. With an autosomal recessive pattern of inheritance, a heterozygous couple has a 25% chance of having an affected fetus. Prenatal diagnosis by enzyme assay of amniocytes or chorionic villus samples may be available for an at-risk couple. Infants or children with LCHAD deficiency are at risk of developing fatal nonketotic hypoglycemia, defects in urea cycle formation, cardiomyopathy, or progressive neuromyopathy. The recurrence risk of AFLP in subsequent pregnancies (whether or not there is an associated inherited fatty acid oxidation defect) is likely to be increased but difficult to quantify based on the available literature.

Renal disorders (cases)

Pyelonephritis with pregnancy

- ✓ A 20-year-old G1 PO woman is hospitalised for acute pyelonephritis at 29 weeks of gestation. She does not have a history of pyelonephritis. Since 48 hours ago, she has been receiving ampicillin and gentamicin infusions. She is experiencing severe difficulty of breath. Her heart rate is 100 beats per minute (bpm), her respiratory rate is 45 beats per minute and laboured, and her blood pressure is 120/70 mm Hg. The right costovertebral angle is tender. The examination of her abdomen reveals no masses or tenderness. The foetal heart tones range between 140 and 150 beats per minute. The culture of urine revealed ampicillin-sensitive *Escherichia coli*. What is the most likely diagnosis?

Objectives

1. Understand the clinical presentation of pyelonephritis.
2. Know that the primary treatment of pyelonephritis is intravenous antibiotic therapy.
3. Understand that endotoxins can cause pulmonary damage, leading to acute respiratory distress syndrome (ARDS).

Tutor guide :

ANSWER TO:

Pyelonephritis, Unresponsive

Summary: A 20-year-old G1 PO woman at 29 weeks' gestation has received intravenous ampicillin and gentamicin for 48 hours for acute pyelonephritis. She complains of acute shortness of breath. On examination, her HR is 100 bpm, RR 45 breaths per minute, and BP is

120/70. Right costovertebral angle tenderness is noted. The urine culture revealed E coli sensitive to ampicillin.

> **Most likely diagnosis: Acute respiratory distress syndrome (ARDS).**

ANALYSIS

Considerations

This patient is 20 years old at 29 weeks' gestation. She presented with pyelonephritis. She had been treated with intravenous ampicillin and gentamicin. The diagnosis is confirmed since E coli has been cultured from the urine. She is now presenting with dyspnea and tachypnea. The most likely etiology for her respiratory symptoms is ARDS, with pulmonary injury due to the endotoxin release. This typically occurs after antibiotics have begun to lyse the bacteria, leading to endotoxemia. The endotoxins can induce damage to the myocardium, liver, and kidneys, as well as the lungs. The mechanism is leaky capillaries, which allows fluid from the intravascular space to permeate into the alveolar areas. A chest film may show patchy infiltrates; however, if the disease process is early, the chest radiograph may be normal. Treatment would include oxygen supplementation, careful monitoring of fluids (not to overload), and supportive measures. Occasionally, a patient may require intubation, but usually the condition stabilizes and improves.

APPROACH TO

Pyelonephritis in Pregnancy

DEFINITIONS

PYELONEPHRITIS: Kidney parenchymal infection most commonly caused by gram-negative aerobic bacteria, such as E coli.

ENDOTOXIN: A lipopolysaccharide that is released upon lysis of the cell wall of bacteria, especially gram-negative bacteria.

ACUTE RESPIRATORY DISTRESS SYNDROME (ARDS): Alveolar and endothelial injury leading to leaky pulmonary capillaries, clinically causing hypoxemia, large alveolar-arterial gradient, and loss of lung volume.

CLINICAL APPROACH

Pyelonephritis in pregnancy can be a very serious medical condition, with an incidence of 1% to 2% of all pregnancies. It is the most common cause of sepsis in pregnant women. The patient generally complains of dysuria, urgency, frequency, costovertebral tenderness, fever and chills, and nausea and vomiting. The urinalyses is usually will reveal pyuria and bacteriuria; a urine culture revealing greater than 100,000 colony-forming units/mL of a single uropathogen is diagnostic. The most common organism is E coli, seen in about 80% of cases. Klebsiella pneumoniae, Staphylococcus aureus, and Proteus mirabilis may also be isolated.

Pregnant women with acute pyelonephritis should be hospitalized and given intravenous antibiotics. Cephalosporins, such as cefotetan or ceftriaxone, or the combination of ampicillin and gentamicin are usually effective. The patient should be treated until the fever and flank tenderness have substantially improved and then switched to oral antimicrobial therapy, and

then suppressive therapy for the remainder of the pregnancy. Up to one-third of pregnant women with pyelonephritis will develop a recurrent UTI if suppressive therapy is not utilized. A repeat urine culture should be obtained to ensure eradication of the infection. If clinical improvement has not occurred after 48 to 72 hours of appropriate antibiotic therapy, a urinary tract obstruction (ie, ureterolithiasis) or a perinephric abscess should be suspected.

Approximately 2% to 5% of pregnant women with pyelonephritis will develop acute respiratory distress syndrome (ARDS), which is defined as pulmonary injury due to sepsis, usually endotoxin related. The endotoxins derived from the gram-negative bacterial cell wall enter the blood stream, especially after antibiotic therapy, and may induce transient elevation of the serum creatinine as well as liver enzymes. Also, the endotoxemia may cause uterine contractions and place a patient into preterm labor. Diffuse bilateral or interstitial infiltrates are typically seen on chest radiograph.