Question 1:

A 28-year-old woman presents to the clinic with a chief complaint of hirsutism and irregular menses. She describes irregular and infrequent menses (five or six per year) since menarche at 12 years of age. She began to develop dark, coarse facial hair when she was 14 years of age, but her parents did not seek treatment or medical opinion at that time. The symptoms worsened after she gained weight in college. She got married 3 years ago and has been trying to get pregnant for the last 2 years without success. Height 66 inches and weight 198. BMI 32 $kg.m^2$. Moderate hirsutism without virilization noted. Laboratory data reveal CMP within normal limits (WNL), CBC with manual differential (WNL), TSH 0.9 IU/L SI units (normal 0.4-4.0 IU/L SI units), a total testosterone of 65 ng/dl (normal 2.4-47 ng/dl), and glycated hemoglobin level of 6.1% (normal value ≤5.6%). Based on this information, the APRN diagnoses the patient with polycystic ovarian syndrome (PCOS) and refers her to the Women's Health APRN for further workup and management.

Question 1 of 2:

What is the pathogenesis of PCOS?

Answer:

Polycystic ovary syndrome (PCOS) is a heterogeneous disorder characterized by hyperandrogenism and chronic anovulation. Depending on diagnostic criteria, 6% to 20% of reproductive aged women are affected. PCOS presents as a phenotype reflecting a self-perpetuating vicious cycle involving neuroendocrine, metabolic, and ovarian dysfunction. PCOS reflects the interactions among multiple proteins and genes influenced by epigenetic and environmental factors. Functional ovarian hyperandrogenism due to ovarian steroidal dysregulation is at the center of the pathogenesis of polycystic ovary syndrome. Functional ovarian hyperandrogenism due to ovarian steroidal dysregulation is at the center of the pathogenesis of polycystic ovary syndrome. This has both genetic and environmental factors. The genetic factors are polycystic ovary morphology, insulin resistance, hyperandrogenemia, defects in insulin secretion. The steroidal dysregulation may lead to anovulation, irregular menses, virilization, hirsutism and infertility. Insulin resistance may also occur.

How does PCOS affect a woman's fertility or infertility?

Answer:

Women with PCOS have hormonal imbalances showing increased levels of testosterone. This hormonal imbalance prevents development and release of mature eggs. This prevents ovulation thus preventing pregnancy to occur.

Question 2:

A 20-year-old female college student presents to the Student Health Clinic with a chief complaint of abdominal pain, foul smelling vaginal discharge, and fever and chills for the past 4 days. She denies nausea, vomiting, or difficulties with defecation. Last bowel movement this morning and was normal for her. Nothing has helped with the pain despite taking ibuprofen 200 mg orally several times a day. She describes the pain as sharp and localizes the pain to her lower abdomen. Past medical history noncontributory. GYN/Social history + for having had unprotected sex while at a fraternity party. Physical exam: thin, Ill appearing anxious looking white female who is moving around on the exam table and unable to find a comfortable position. Temperature 101.6F orally, pulse 120, respirations 22 and regular. Review of systems negative except for chief complaint. Focused assessment of abdomen demonstrated moderate pain to palpation left and right lower quadrants. Upper quadrants soft and non-tender. Bowel sounds diminished in bilateral lower quadrants. Pelvic exam demonstrated + adnexal tenderness, + cervical motion tenderness and copious amounts of greenish thick secretions. The APRN diagnoses the patient as having pelvic inflammatory disease (PID).

Question:

What is the pathophysiology of PID?

Answer:

Pelvic Inflammatory Disease is mainly caused due to Neisseria gonorrhea (gonococci) or Chlamydia trachomatis. Neisseria gonorrhea use host Sialic acid to evade the complement attack and releases surface fragments that destroy the epithelial cells in cervix, endometrium, or fallopian tubes. Chlamydia has auto-transported polymorphic membrane proteins which helps the organism to penetrate the host cells and trigger a immune response.

An Infection of the upper genital tract occurs (mainly by Gonococci and/ or Chlamydia or other bacteria such as Bacteroides, Gardnerella vaginalis, Hemophilus influenza, Mycoplasma, Escherichia coli) leading to pathological changes in the columnar epithelium of the genital tract and produces inflammation in cervix, endometrium of uterus or fallopian tubes. Microbial invasion, disruption of normal flora, alteration of cervical mucus barrier host immune defense mechanisms against infectious agent leads to inflammatory response, edema, and local tissue damage. If Gonococci, it enters the fallopian tubes, it will cause inflammation and damage whereas Chlamydial infection leads to permanent scarring of the fallopian tubes. The infectious agents then gain access to the abdominal cavity via fallopian or uterine tubes leading to Pelvic inflammatory Disease (PID).

QUESTION 4

1. A 27-year-old male comes to the clinic with a chief complaint of a “sore on my penis” that has been there for 3 days. He says it burns and leaked a little fluid. He denies

any other symptoms. Past medical history noncontributory. Social history: works as a bartender and he states he often “hooks up” with some of the patrons, both male and female after work. He does not always use condoms. Physical exam within normal limits except for a lesion on the lateral side of the penis adjacent to the glans. The area is indurated with a small round raised lesion. The APRN orders laboratory tests, but feels the patient has syphilis.

Question:

Describe the 4 stages of syphilis.

Answer:

Primary syphilis begins at the site of bacterial invasion. Where T. Palladium multiplies and the epithelium and produces a granulomatosis tissue reaction called a chancre. some microorganism strain with lymph into adjacent lymph nodes. Within the nodes in at the site of the canker, the cell mediated, and humoral immune responses are stimulated.

Secondary syphilis is systemic. During this stage, blood borne bacteria spread to all major organ systems. The secondary stage is followed by a period during which the immune system can suppress the infection. Even without treatment, spontaneous resolution of the skin lesions occurs, and the individual enters the latent stage of infection.

Latent syphilis may be subdivided into early and late stages however, no specific criteria delineates one from the other period medical history and serologic studies can show that syphilis is present, but the individual has no clinical manifestations. Transmission remains possible during this phase.

Tertiary syphilis is the most severe stage involving significant morbidity and mortality. The pathogenesis of syphilitic manifestations at the stage remains unclear. The destructive skin, bone, and soft tissue lesions also called gummas of tertiary syphilis probably are caused by a severe hypersensitivity reaction to the microorganism. Within the cardiovascular system, infection with T. palladium may cause aneurysms, heart valve insufficiency's, in heart failure.

QUESTION 5

1. A 19-year-old female presents to the clinic with a chief complaint of “fluid filled bumps” and intense pruritis of her vulva. She states these symptoms have been present for about 10 days, but she thought she had a yeast infection. She self-medicated with over the counter (OTC) metronidazole (Flagyl™) intravaginally but the symptoms got worse. No other complaints except for fatigue out of proportion to her activity level. Past medical history noncontributory. Social history: sexually active with several men and did forget to use a condom during one sexual encounter. Physical exam negative except for pelvic exam which revealed multiple fluid filled (vesicular) lesions on the vulva and introitus. Positive lymph nodes in inguinal areas. The APRN diagnoses the patient with herpes simplex virus-type 2 known as genital herpes.

Question:

What is the pathophysiology of HSV-2?

Answer:

Once initial exposure two HSV 2 takes place, the virus enters mucocutaneous sites or abraded skin, the virus undergoes replication locally in the dermis and epidermis. This leads to cell destruction transportation and vesicle formation. The virus spreads to contiguous cells and eventually into sensory nerves this process often occurs and causes a systemic, inflammatory, immune response, especially with HSV 2 infection that includes fever and malaise. In rare cases the herpes can cause CNS manifestations. Painful lesions can last from days to weeks. Eventually the virus is transported intraxonally to the dorsal root where it remains in the latent stage until it becomes reactivated. The viral envelope is secured by HSV glycoprotein C and assists in the viral entry. Recognition of HSV DNA by toll like receptors results in the activation and the development of interferon gene products by the innate an adaptive immune systems via a dynamic interplay between HSV virion protein products and the immune system, viral suppression of whole sales responses and subsequent evasion of the immune system is achieved. In the early stages of the infection, the HSV protein virion host shutoff or VHS is developed to suppressed host-cell responses. In addition, glycoprotein C binds to complement C3B, inhibits immunity mediated by complement and plays a role in inhibiting neutralization of antibodies.

Question 6:

A 27-year-old male presents to the clinic with a chief complaint of a gradual onset of scrotal pain and swelling of the left testicle that started 2 days ago. The pain has gotten progressively worse over the last 12 hours and he now complains of left flank pain. He complains of dysuria, frequency, and urgency with urination. He states his urine smells funny. He denies nausea, vomiting, but admits to urethral discharge just prior to the start of his severe symptoms. He denies any recent heavy lifting or straining for bowel movements. He says the only thing that makes the pain better is if he sits in his recliner and elevates his scrotum on a small pillow. Past medical history negative. Social history + for sexual activity only with his wife of 3 years. Physical exam reveals red, swollen left testicle that is very tender to touch. There is positive left inguinal adenopathy. Clean catch urinalysis in the clinic + for 3+ bacteria. The APRN diagnoses the patient with epididymitis.

Question:

Discuss how bacteria in the urine causes epididymitis.

Answer:

Bacteria in urine as evidenced by the urinalysis results bacteria 3+ can cause epididymitis. The bacteria demonstrate retrograde ascent if there is bladder outflow secondary to urethral stricture or prostate enlargement. Most cases of epididymitis are caused by an infection, usually by the bacteria Mycoplasma or Chlamydia. These infections often come by way of sexually transmitted diseases. The bacterium *E.*

coli can also cause the condition. Other infections, including with the mumps virus and, rarely, tuberculosis, can also cause epididymitis.

Sometimes epididymitis occurs when urine flows backward into the epididymis. This can happen because of heavy lifting. Other causes of epididymitis include: Blockage in the urethra (the tube that carries urine from the body); An enlarged or infected prostate gland (a muscular, walnut-sized gland that surrounds part of the urethra); Use of a catheter (a tube that drains the bladder); Traumatic groin injury

QUESTION 7

1. A 42-year-old male presents to the clinic with a chief complaint of fever, chills, malaise, arthralgias, dysuria, urinary frequency, low back pain, perineal, and suprapubic pain. He says he feels like he can't fully empty his bladder when he voids. He states these symptoms came on suddenly about 12 hours ago and have gotten worse. He noticed some blood in his urine the last time he voided. He tried to have a bowel movement several hours ago but could not empty his bowel due to pain. Past medical and social history noncontributory. Physical exam reveals an ill appearing male. Temperature 101.8 F, pulse 122, respirations 20, BP 108/68. Exam unremarkable apart from left costovertebral angle (CVA) tenderness. Rectal exam difficult due to enlarged and extremely painful prostate. Complete blood count revealed an elevated white blood cell count, elevated C-reactive protein and elevated sedimentation rate. Urine dip in the clinic + for 2+ bacteria.

   Question:

   Explain the differences between acute bacterial prostatitis and nonbacterial prostatitis.

Answer:

Acute bacterial prostatitis is caused bacterial infection in the prostate. The infection ascends from the urinary track and it tends to occur in men between the ages of 30 and 50 years but also is associated with BPH in older men infection stimulates an inflammatory response in which the prostate becomes enlarged tender firm embody the onset of prostatitis may be acute in unrelated to previous illnesses or it may follow catheterization or cystoscopy. The bacteria get in the prostate through infected urine flowing backwards from the urethra. Bacteria are found in the seminal fluids, urine, and the prostate fluid.
Nonbacterial prostatitis can be linked to stress, irritation or inflammation in the nerves, or infections in the urinary tract. Nonbacterial prostatitis does not have any signs of bacteria in the seminal fluid or urine as it is observed in bacterial prostatitis. It can be treated with antibiotics until a diagnosis of bacterial infection is detected through lab testing in order to get in front of the treatment.

QUESTION 8