NURS 6501

Knowledge Check: Module 7
Student Response

This Knowledge Check reviews the topics in Module 7 and is formative in nature. It is worth 20 points where each question is worth 1 point. You are required to submit a sufficient response of at least 2-4 sentences in length for each question.

Scenario 1: Polycystic Ovarian Syndrome (PCOS)

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?

PCOS is a condition characterized by excessive ovarian or adrenal androgen secretion. The pathogenesis of PCOS is not clear, but several mechanisms are involved that play a vital role in its pathogenesis. These mechanisms incorporate hormonal imbalance, insulin resistance, and genetic inheritance. Any alteration in these mechanisms can cause the ovaries to get enlarged on both sides and form a thick, smooth, avascular capsule. For instance, a change to steroidogenesis and other factors external to the ovary like hyperinsulinemia results in excessive ovarian androgen secretion. These changes cause symptoms such as irregular periods or no periods at all, weight gain, oily skin or acne, difficulty getting pregnant and excessive hair growth (hirsutism)

or thinning of hair and hair loss from the head. For instance, in the case scenario, the patient had complaints of hirsutism and irregular periods as well as weight gain, indicating an excessive ovarian androgen secretion.

Question 2 of 2:

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

Even though having PCOS does not mean that a woman cannot get pregnant, it has a significant effect on the fertility of a woman. PCOS affects fertility in women because the hormonal imbalance interferes with the growth and release of eggs from the woman's ovaries or simply interferes with ovulation. If a woman cannot ovulate, then she cannot get pregnant, causing infertility issues. For instance, the case study patient had irregular menses, and she described irregular and infrequent menses to occur five or six per year, meaning that it is hard for the patient to determine when she may ovulate so that she can try getting pregnant.

Scenario 2: Pelvic Inflammatory Disease (PID)

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?

Pelvic inflammatory disease (PID) is an infectious disease and inflammatory disorder of the upper female genital tract incorporating the uterus, fallopian tubes, and adjacent pelvic structures. The pathophysiology of PID involves an ascending infection of cervicovaginal organisms, of which the most common pathogens are Neisseria gonorrhea and Chlamydia trachomatis. In its pathophysiology, PID mostly occurs in two stages. The initial stage involves the acquisition of a vaginal or cervical infection. The infection is mostly acquired through sexual intercourse and maybe asymptomatic. The second stage is the direct ascent of the microorganisms from the vagina or cervix to the upper genital tract, with infection and inflammation of the affected genital structures. The symptoms include pain in the lower and upper abdomen, painful sex; painful urination, irregular bleeding, and foul smell virginal discharge. For instance, the patient in the case study had a chief complaint of abdominal pain, foul-smelling vaginal discharge, and fever and chills for the past 4 days, indicating an infection in the genital tract.

Scenario 3: Syphilis

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

The four stages of syphilis include primary, secondary, latent, and tertiary. Primary is the first stage, and it causes one or more small, painless sores in or around the genitals, anus, or mouth. If one does not get treatment in the primary stage of the disease, PID may progress to the

second stage, which is secondary syphilis. The secondary stage is characterized by rough red or reddish-brown rash on palms of hands and soles of feet, swollen lymph nodes, fever, and sore throat. The secondary stage is curable, but if not treated, the disease may enter the latent stage then to the tertiary stage of syphilis, which is not curable. In The latent stage, the syphilis bacteria are still alive in the body, but one does not show symptoms or sign so infection. One is not contagious during this stage, but syphilis cause damage to the body organs, as well as lead to dementia, paralysis, or even death. This stage can last for years. The last stage is the tertiary or late syphilis stage that starts when the symptoms from the secondary stage disappear. Syphilis is not contagious in this stage, but it can affect the lungs. In the case scenario patient, he suffers from secondary syphilis because of the sore on his penis.

Scenario 4: Genital Herpes

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?

The pathophysiology of HSV-2 is associated with orolabial, lesions, stromal keratitis, and sometimes encephalitis. The pathophysiology of HSV-2 involves a viral infection that induces ballooning of cells with condensed chromatic within the nuclei of cells, followed by nuclear degeneration, generally within parabasal and intermediate cells of the epithelium. The cells lose intact plasma membranes and form multinucleated giant cells. With cell lysis, a clear fluid with a

large amount of virus appears between the epidermis and the dermal layer. In the dermal substructure, an intense inflammatory response occurs, usually in the corium of the skin, especially with primary infection than with recurrent infection. Symptoms include swollen glands in the pelvic region, throat, and under the arms; chills, headache, feeling achy and tired. For instance, the physical examination on the pelvic region of the patient in the case scenario revealed multiple fluids filled lesions on the vulva and introitus, as well as positive lymph nodes in inguinal areas, indicating infection with HSV-2.

Scenario 5: Epididymitis

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.

Bacteria from a urinary tract infection often spread from the infected site to the epididymis, thus causing epididymitis. For instance, individuals with a history of prostate or urinary tract infection are at high risk of suffering from the disease. Another risk factor for accumulation of urinary tract bacteria is due to the uncircumcised penis or anatomical abnormality of the urinary tract that may provide a sit for bacteria to accumulate. Besides, prostate enlargement increases the risk of bladder infections that may result in epididymitis. Symptoms of epididymitis include a swollen, red or warm scrotum, testicle pain and tenderness,

painful urination or urgent or frequent need to urinate, pain or discomfort in the lower abdomen or pelvic area. The patient in the case scenario experienced all these symptoms, indicating a bacterial infection in the epididymis.

Scenario 6: Prostatitis

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

Acute bacterial prostatitis occurs when there is a bacterial infection that affects the prostate gland, thus causing inflammation. The symptoms of acute bacterial prostatitis are sudden and can sometimes be painful. They include fever, chills, very painful burning while urinating, and difficulties draining the bladder. This form of prostatitis requires a urine test to detect if there are any bacteria. On the other hand, nonbacterial prostatitis is linked to other factors other than bacteria such as stress, nerve inflammation or irritation, injuries, or prior urinary tract infections. This form of prostatitis has no signs o bacteria in the urine or seminal fluid. From the symptoms described in the case scenario, the patient likely has acute bacterial prostatitis. The urine test showed a positive test for bacteria.

Scenario 7: Endometriosis

A 32-year-old woman presents to the clinic with a chief complaint of pelvic pain, excessive menstrual bleeding, dyspareunia, and inability to become pregnant after 18 months of unprotected sex with her husband. She states she was told she had endometrioses after a high school physical exam, but no doctor or nurse practitioner ever mentioned it again, so she thought it had gone away. She has no other complaints and says she wants to have a family. Past medical history noncontributory except for possible endometriosis as a teenager. Social history negative for tobacco, drugs or alcohol. The physical exam is negative except for the pelvic exam which demonstrated pain on light and deep palpation of the uterus. The APRN believes that the patient does have endometriosis and orders appropriate laboratory and radiological tests. The diagnostics come back highly suggestive of endometriosis.

Question:

Explain how endometriosis may affect female fertility.

Endometriosis may affect female fertility because it causes inflammation and irritation. For instance, inflammation of the fimbria, which picks up the egg and transports it into the fallopian tube, results in swelling and scarring, and as a result, the egg may not reach its required destination. This action makes it harder for a woman to get pregnant. Symptoms include painful sexual intercourse, pain that often occurs just before menstrual and disappears slowly after menstruation, cramping during sexual intercourse, infertility issues, pain with pelvic examination, and pain during bowel movement or urination. For instance, the patient in the above scenario had a chief complaint of pelvic pain, excessive menstrual bleeding, dyspareunia, and inability to become pregnant after 18 months of unprotected sex with her husband, indicating that she has endometriosis.

Scenario 8: Platelets

An APRN working in an anticoagulation clinic has been asked by the local college to present a lecture on platelets and their role in blood clotting to the graduate pathophysiology nursing students.

Question:

What key concepts should the APRN include in the presentation?

In the presentation, the APRN may include several key points. For instance, the APRN needs to define platelets to the pathophysiology graduate students before explaining the role it plays in blood clotting. Platelets are tiny blood cells that assist the body o form clots that help in the stoppage of bleeding. The plates play an important role in the blood clot. For instance, if one gets blood vessel damage, signals are sent to the platelets, which then rush to the site of damage to plug and form a clot to fix the damage site.

Scenario 9: Iron Deficient Anemia (IDA)

A 36-year-old woman presents to the clinic with complaints of dyspnea on exertion, fatigue, leg cramps on climbing stairs, craving ice to suck or chew and cold intolerance. The symptoms have come on gradually over the past 4 months. The only thing that make the symptoms better is for her to sit or lie down and stop the activity. She denies bruising or bleeding and states this is the first time this has happened. Past medical history noncontributory except for a new diagnosis of benign uterine fibroids 6 months ago after experiencing heavy menstrual bleeding every month. Social history noncontributory and she denies alcohol, tobacco, or drug use. Physical exam: pale, thin, Caucasian female who appears older than stated age. Physical exam remarkable for a soft I/IV systolic murmur, pallor of the mucous membranes, spoon-shaped nails (koilonychia), glossy tongue, with atrophy of the lingual papillae, and fissures at the corners of the mouth. The APRN suspects the patient has iron deficient anemia (IDA) secondary to excessive blood loss from uterine fibroids. The appropriate laboratory tests confirmed the diagnosis.

Question:

Discuss iron deficiency anemia and how the patient's menstrual bleeding contributed to the diagnosis.

Iron deficiency anemia is a common type of anemia, a condition in which blood lacks enough health red blood cells. Notably, red blood cells carry oxygen to the body's tissues; as the name suggests, iron deficiency refers to insufficient iron in the blood. The patient's menstrual bleeding may have contributed to the diagnosis because of the heavy menstrual bleeding every month. Heavy menstrual leads to too much blood loss, which consequently results in the loss of

iron in the blood. For the four months, the patient has lost too much blood, replace about a half of it, and again loose too much the following month, thus depleting the iron in the blood.

Scenario 10: Pernicious Anemia

A 67-year-old woman presents to the clinic with complaints of weakness, fatigue, paresthesias of the feet and fingers, difficulty walking, loss of appetite, and a sore tongue. These symptoms have been present for several months but the patient thought they were due to her recent retirement and geographic move from the Midwest to New England. The symptoms have gotten worse over the past few weeks and she has noticed that she is much more forgetful. This is of great concern as she worries she might have the beginning stages of Alzheimer's Disease. Past medical history significant for Hashimoto thyroiditis that she developed in her early 20s. The rest of PMH and social history non- contributory. Physical exam reveals an average sized female whose skin has a sallow appearance. BP 128/74, Pulse 120, respirations 18 and temperature 99.0F orally. Examination of the head and neck reveals a smooth and beefy red tongue. Abdominal exam negative for hepatomegaly or splenomegaly.

The APRN recognizes these symptoms and physical exam indicate the patient has pernicious anemia. After appropriate laboratory data received, the definitive diagnosis of pernicious anemia was made.

Question 1 of 2:

How does pernicious anemia develop?

Pernicious anemia is a deficiency in red blood cells that is caused by the lack of vitamin B12 in the blood. The condition often occurs when there is impaired uptake of vitamin B12 due to the lack of a substance known as an intrinsic factor (IF) that is secreted by the stomach lining. Symptoms include fatigue or light-headedness, pale skin, the feeling of pins and needles, shortness of breath, gastritis, or weight loss. For instance, the case scenario patient has several of the symptoms such as fatigue, paresthesias of the feet and fingers, difficulty walking as well as sallow skin appearance.

Question 2 of 2:

How does pernicious anemia cause the neurological manifestations that are often seen in patients with PA?

Pernicious anemia causes the neurological manifestations that are often seen in patients with PA due to myelin degeneration and loss of nerve fibers in the dorsal and lateral columns of the spinal cord and cerebral cortex. The neurological manifestations of the patient include paresthesias and weakness.

Scenario 11: Anemia of Chronic Disease (ACD)

A 49-year-old man with a 22-year history of severe rheumatoid arthritis (RA) presents to clinic for his preadmission testing (PAT) and medical clearance for a planned right total hip arthroplasty. The patient had been severely limited in ambulation due to the RA. Current medications include prednisone 20 mg po qd and methotrexate 7.5 mg Thursdays, 5mg Fridays, and 7.5 mg Saturdays. The patient had a complete blood count (CBC) with manual differentiation and red blood cell indices, complete metabolic panel (CMP) and coagulation studies (prothrombin time [PT], international normalized ratio [INR] and activated partial thromboplastin time [aPTT]). All the laboratory studies come back within normal limits except for the red blood cell indices. The hemoglobin and hematocrit were low along with mean corpuscle volume, plasma iron and total iron binding capacity, and transferrin also being low. There was a normal reticulocyte count, normal ferritin, serum B_{12}, folate and bilirubin.

The APRN in the PAT clinic recognizes that the patient has anemia of chronic disease (ACD).

Question 1 of 2:

What is ACD and how does it develop?

ACD is anemia that is found in individuals with particular long-term or chronic medical conditions that involve inflammation. ACD is caused by the alterations triggered by chronic disease. The alterations include a problem with using an iron to make red blood cells, even when there is adequate iron in the body. Sometimes, the bone marrow fails to make red blood cells as it should be making, thus leading to ACD. For instance, in the case scenario patient, the contributory chronic disease is the history of severe rheumatoid arthritis, which affected the production of red blood cells as indicated in the low hemoglobin and hematocrit as well as a low plasma iron and total iron-binding capacity and transferring.

Question 2 of 2:

Why do patients with chronic kidney disease (CKD) develop ACD?

Patients with chronic kidney disease (CKD) often develop ACD because the exacerbations of glomerulonephritis, as well as the underlying systematic disease caused by CKD, lead to cytokine-mediated disorders of erythropoiesis. The cytokine-mediated disorders lead to metabolic disorders or iron metabolism that are associated with increased iron absorption and retention by reticuloendothelial system cells, followed by lowering of iron admission to the bone marrow.

Scenario 12: Immune Thrombocytopenia Purpura (ITP)

A 14-year-old female is brought to the Urgent Care by her mother who states that the girl has had an abnormal number of bruises and "funny looking red splotches" on her legs. These bruises were first noticed about 2 weeks ago and are not related to trauma. Past medical history not remarkable and she takes no medications. The mother does state the girl is recovering from a "bad case of mono" and was on bedrest at home for the past 3 weeks. The girl noticed that her gums were slightly bleeding when she brushed her teeth that morning.

Labs at Urgent Care demonstrated normal hemoglobin and hematocrit with normal white blood cell (WBC) differential. Platelet count of 100,000/mm3 was the only abnormal finding. The staff also noticed that the venipuncture site oozed for a few minutes after pressure was released. The doctor at Urgent Care referred the patient and her mother to the ED for a complete work up of the low platelet count including a peripheral blood smear for suspected immune thrombocytopenia purpura (ITP).

Question:

What is ITP and why do you think this patient has acute, rather than chronic, ITP?

ITP is an immune disorder in which the blood does not clot normally and can result in excessive bruising and bleeding. This condition often occurs due to a low level of platelets or thrombocytes in the blood. Besides, it can occur when the immune system mistakenly attacks the platelets, thus, deleting the number of platelets in the blood. The symptoms include easy bruising, bleeding, and pinpoint-sized reddish-purple spots on the lower legs. In the scenario

above, the patient had an abnormal number of bruises accompanied with low levels o platelets. The normal platelet count ranges from 50,000 to 450,000 platelets per microliter of blood.

Scenario 13: Heparin Induced Thrombocytopenia (HIT)

A 22-year-old male is in the Surgical Intensive Care Unit (SICU) following a motor vehicle crash (MVC) where he sustained multiple life-threatening injuries including a torn aorta, ruptured spleen, and bilateral femur fractures. He has had difficulty maintaining his mean arterial pressure (MAP) and has required various vasopressors. He has a triple lumen central venous catheter (CVC) for monitoring his central venous pressure, administration of medications and blood products, as well as total parenteral nutrition. Per hospital protocol, he is receiving an unfractionated heparin 1:1000 flush after administration of each of the triple antibiotics that have been ordered to maintain patency of the lumens. Seven days post injury, the APRN in the SICU is reviewing the patient's morning labs and notes that his platelet count has dropped precipitously to 50,000 /mm^3 from 148,000/mm^3 two days ago. The APRN suspects the patient is developing heparin induced thrombocytopenia (HIT).

Question 1 of 2:

What is underlying pathophysiology of heparin induced thrombocytopenia?

In its pathophysiology, the mechanism underlying the heparin-induced thrombocytopenia is mainly an immune response. The primary antigen is a complex of heparin and platelet factor 4 (PF4). In this case, the activation of platelets leads to the release of PF4 into the circulation, and some of it binds to the platelet surface. Due to opposite charges, heparin and other glycosaminoglycans bind to the PF4 molecules, thus exposing neoepitopes that act as immunogens resulting in antibody production. The Fc portion of the HIT antibody then binds to the platelet Fc receptor, and the interaction triggers activation and aggregation of the platelets. Activated platelets release PF4, thereby perpetuating the cycle of heparin-induced platelet activation. Additionally, the platelet activation results in the production of prothrombotic platelet microparticles, which encourage coagulation. Lastly, as a result of the presence of heparin-like molecules on the surface of endothelial cells, the HIT antibody-PF4-heparan sulfate complexes formed on the endothelial surface induces tissue factor expression with additional activation of

the coagulation cascade and thrombin generation, thus resulting in heparin-induced thrombocytopenia.

Question 2 of 2:

The APRN assesses the patient and notes there is a decreased right posterior tibial pulse with cyanosis of the entire foot. The APRN recognizes this probably represents arterial thrombus formation. How does someone who is receiving heparin develop arterial and venous thrombosis?

An individual receiving heparin may develop arterial and venous thrombosis because heparin may cause activation of platelet that leads to the release of procoagulant platelet microparticles, platelet consumption, and thrombocytopenia. Besides, heparin also causes a marked generation of thrombin, activation of monocytes and other inflammatory cells, and endothelial injury and activation follow, thus resulting in venous and arterial thromboses.

Scenario 14: Thrombotic Thrombocytopenic Purpura (TTP)

A 33-year-old female is brought to Urgent Care by her husband who states his wife has gotten suddenly confused and complains of a severe headache. He also noticed large bruises on her legs which were not there yesterday. Only significant past medical history is that the patient developed herpes zoster 2 weeks ago and was given acyclovir for treatment. Physical exam revealed well developed female who is only oriented to person. Large areas of ecchymosis noted on both arms and legs. Stat CBC revealed a platelet count of 18,000/$mm^{3,}$ hemoglobin of 8 g/dl and hematocrit of 24%. The patient was immediately transported to the Emergency Room by Emergency Medical Services (EMS) where further work up demonstrated idiopathic thrombotic thrombocytopenic purpura (TTP).

Question:

What is the pathophysiology of TTP?

Thrombotic thrombocytopenic purpura (TTP) is a disorder characterized by von Willebrand factor (VWF)-rich microthrombi that affects the arterioles and capillaries of several organs in the body. The pathophysiology of TTP is characterized by patients having unusually large multimers of von Willebrand factor (vWF) in their plasma, and they lack a plasma protease that is responsible for the breakdown of these ultra-large vWF multimers. Therefore, inherited or

acquired ADAMTS13 deficiency allows the unrestrained growth of microthrombi that are composed of von Willebrand factor and platelets, which account for the thrombocytopenia, hemolytic anemia, schistocytes, and tissue injury that characterize TTP. Symptoms include purplish bruises on the skin or mucous membranes' pinpoint-sized red or purple dots on the skin, paleness or jaundice, fever, fatigue, and a fast heart rate or shortness of breath. In the scenario above, the patient complained of severe headaches and large bruises on her legs, indicating that she experiences the symptoms of TTP.

Scenario 15: Heparin Induced Thrombocytopenia (HIT)

A 64-year man is recovering from a transurethral resection of the prostate for treatment of benign prostate hyperplasia. The patient is receiving intravenous antibiotics for the urinary tract infection that was found on the preoperative urine culture and sensitivity (C & S). The post-operative course has been smooth and the APRN is removing the 3-way Foley catheter when there is a sudden release of bright red blood with many blood clots in the Foley bag. The patient becomes hypotensive, tachycardic and the APRN notes new ecchymoses on the patient's arms and legs. The patient was immediately transferred to the surgical intensive care unit (SICU) and a stat hematology consult was conducted. Stat CBC, d-dimer, peripheral blood smear, partial thromboplastin time, Prothrombin time/international normalization ratio (INR), and fibrinogen labs were drawn. Results were:

CBC with markedly decreased platelet count, peripheral blood smear showed decreased number of platelets and presence of large platelets and fragmented red cells (schistocytes), prothrombin time prolonged as was the partial thromboplastin time. The d-dimer was markedly elevated, and fibrinogen level was low. The diagnosis of disseminated intravascular coagulation (DIC) was made based on clinical picture and laboratory data.

Question 1 of 2:

What is DIC and how does it develop?

DIC is a condition that affects the ability of an individual's blood to clot and stop bleeding. The condition develops due to sudden exposure of blood to procoagulants such as tissue factor (TF), or thromboplastin, which generated intravascular coagulation. Thereafter,

compensatory hemostatic mechanisms are quickly overwhelmed, and, as a result, a severe consumptive coagulopathy results, leading to hemorrhage.

Question 2 of 2:

What factors contribute to the development of DIC?

In the case scenario patient, the factors that contributed to the development of DIC include decreased platelet count, prolonged prothrombin time, a low fibrinogen level as well as the presence of large platelets explaining why the patient was not able to control bleeding. With less platelet, their clotting action is adversely affected.