**Example of a Small Group Case Study Response**

**Group 6 Case study 80, Questions 3, 4, 5, 8, and 13 (Rheumatoid arthritis)**

Patient Case Question 3. Identify two abnormal findings from the physical exam above that are consistent with rheumatoid arthritis.

Two abnormal findings identified during exam consistent with rheumatoid arthritis include an elevated temperature and lymphadenopathy. An elevated temperature (100.5 degrees F) would indicate general systemic inflammation that occurs in rheumatoid arthritis. According to Corwin, “onset of RA is characterized by general symptoms of inflammation including fever, body aches and joint swelling” (Corwin, 2008). The patient also presents with mild lymphadenopathy bilaterally. McCance, Huether, Brashers & Rote state “Lymphadenopathy of the nodes close to the affected joints may develop” (McCance, Huether, Brashers & Rote, 2010). Lymphadenopathy in RA is frequently related to Sjögren’s syndrome which occurs in 10-15% of patients (mostly women) that have rheumatoid arthritis (http://www.hopkins-arthritis.org). Sjögren’s syndrome is a “chronic inflammatory disorder characterized by lymphocytic infiltration of lacrimal and salivary glands” which has a “polyclonal lymphoproliferative reaction characterized by lymphadenopathy” (http://www.hopkins-arthritis.org). This is likely the cause of the lymphadenopathy exhibited by our patient.

Patient Case Question 4. What is the association between the “fixed nodule(s) at pressure point(s)” on the left wrist/elbow and a diagnosis of rheumatoid arthritis?

Rheumatoid nodules are commonly found over pressure areas of the hands, and elbows in the subcutaneous tissue. These nodules occur in 20% of individuals with rheumatoid arthritis, and are actually aggregates of inflammatory cells surrounding a core of fibrinoid, cellular debris, which is sometimes necrotic. T lymphocytes are the predominant leukocyte and B lymphocytes , plasma cells and phagocytes are found around the periphery of the necrotic core of debris (McCance, Huether, Brashers & Rote, 2010). The nodules are representative of the inflammatory process of rheumatoid arthritis and indicate advancing disease.

Classic rheumatoid nodules commonly occur in genetically predisposed patients with severe, sero-positive arthritis (Garcia-Pathos, 2007). In addition, the presence of rheumatoid nodules at extra-articular pressure points indicate advanced extent of joint involvement (usually bilateral), and disease severity. A diagnosis of rheumatoid arthritis with extra-articular involvement with rheumatoid nodules has a worse prognosis. The incidence of nodules also is an index of rating the disease as mild, moderate and severe (Fye, 2002).

Over time, rheumatoid nodules in subcutaneous tissue in pressure points could spread to other areas such as cardiac, pulmonary, renal, and scleral tissue where they can cause serious complications such as cardiac disease, renal involvement and severe glaucoma with subsequant blindness. Fixed rheumatoid nodules usually do not pose any health complications if left intact. In the case that they would rupture there is the risk of infection (McCance, Huether, Brashers & Rote, 2010). However, in a small percentage of patients, fixed rheumatoid nodules can be the source of pain and neuropathy, and inhibit function (Uptodate.com, 2011).

Patient Case Question 5. Why is it reasonable that this patient has no stiffness, pain, or swelling in the DIP joints of the fingers?

The most likely reason is that the proximal, not the distal interphalangeal joints that are most often affected by RA (Bruyere, 2009).

However, there are also several nonspecific reasons ranging from the pain in RA does not always affect all the small joints of the hands (Medicine.net, 2007a) to the progressive damage to M.J.’s joints is not correlating with the degree of pain, stiffness, or swelling present that is actually there (Shiel, 2011) to an extremely wide and varying range of RA symptoms (Medicine.net, 2007b).

Or, perhaps M.L. does not have RA. An interesting study was presented by Lonardo, Neri, Pietrangelo (2001) who found that sometimes an early erroneous diagnosis of RA is common in patients with arthropathy due to hereditary hemochromatosis.

Adding to the hemochromatosis possibility is that M.J. has pain in the 4th and 5th MCP joints bilaterally but not in the DIP joints, which loosely correlates with a case study presented by Tarver (2001) who found involvement of the 4th and 5th MCP joints but not the interphalyngeal joints in a 68 year old man with hemochromatosis.

Further adding to the possibility of hemochromatosis is M.L.’s hand X-ray findings in which there were no erosions which is consistent with hemochromatosis and not RA (Radiology Image Bank, n. d.).

Patient Case Question 8. Provide a reasonable explanation for the serum uric acid test results shown in Table 80.1.

A normal serum uric acid level is 1.4-5.8 mg/dL for females (Bruyere, 2009). ML is within normal limits at 2.9 mg/dL.

The most likely reason for the testing of the uric acid is to help determine whether ML has gout or has rheumatoid arthritis (RA). Often people who are diagnosed with rheumatoid arthritis when they actually have gout and it goes unnoticed and undiagnosed (Casey, 2007).

There is no specific known cause for rheumatoid arthritis. It is believed that it is a combination of genetic, environmental and hormonal factors (Bruyere, 2009). On the other hand, gout is caused by an inflammatory response to increased uric acid production or excretion. This causes an increase in uric acid in the blood, also known as hyperuricemia (McCance, Huether, Brashers & Rote, 2010).

Symptoms in both RA and gout are very similar. Inflamed joints including pain, selling, redness, tenderness and warmth are possible in both types of arthritis. A characteristic manifestation of gout is the sudden onset of pain and swelling in the great toe, but other common sites include the ankle, wrist and knee. While in RA, inflammation in the fingers, hands, wrists, knees, ankles and toes are most common. So not only are the symptoms similar, but so are the sites. Fever, chills, leukocytosis and malaise can also be present in both diseases (Bruyere, 2009).

It is vital to determine exactly which type of arthritis ML has because the treatments between gout and RA are very different. Also, the education behind the diseases is very different. For example, someone with gout would be educated staying away from foods high in purines that produce uric acid such as beer, wine, red meat and shellfish. While someone with RA would be educated on the importance of exercise, the use of heat and cold to relieve symptoms and dietary modifications such as n-3 fatty acid and oral collagen supplements.

Patient Case Question 13. What causes limitation of joint motion that occurs late in the clinical course of rheumatoid arthritis?

In RA, several types of leukocytes are drawn out of the blood and deposited into the synovial membrane, which lines the joint cavity (McCance, Huether, Brashers & Rote, 2010). Neutrophils and macrophages then ingest the immune complexes and cause powerful lysosomal enzymes and reactive oxygen species to be released which in turn damages the synovial tissue and articular cartilage (McCance, Huether, Brashers & Rote). The dead cells then attract more white blood cells setting the scene for inflammation.

B and T lymphocytes are also activated. B lymphocytes produce rheumatoid factor (RF), which is protein that if present in someone with RA will have a more aggressive clinical course than someone without the protein (Bruyere, 2009). T lymphocytes cause enzymes to be released which increase the inflammatory response.

These immune and inflammatory processes cause leukocyte infiltration thus causing swelling (McCance, Huether, Brashers & Rote, 2010). Tissues in the surrounding areas undergo reactive hyperplasia, or increased cell division in response to a stimulus. Blood vessels dilate as production of cytokines, including histamine and prostaglandins are released which results in increased blood flow, warmth and redness (Bruyere, 2009).

As inflammation progresses, the cells of the synovial membrane proliferate and enlarge which causes hyperplastic thickening of the membrane (McCance, Huether, Brashers & Rote, 2010). The synovial inflammation then progresses to the surrounding blood vessels which causes small venules to become obstructed by hypertrophied endothelial cells, fibrin, platelets, and inflammatory cells. This ultimately causes decreased blood flow to the synovial tissue. This decreased blood flow in addition to the increased metabolic needs due to the hypertrophy and hyperplasia cause hypoxia and then metabolic acidosis, promoting further injury (McCance, Huether, Brashers & Rote, 2010).

Eventually, a new network of blood vessels develops in the synovial membranes. The inflammation causes hemorrhage, coagulation, and fibrin deposition in the synovial membrane, the intracellular matrix and the synovial fluid (McCance, Huether, Brashers & Rote, 2010). The fibrin then develops into granulation tissue called pannus. Pannus is only found in rheumatoid arthritis and no other types of arthritis. The pannus eventually develops in the joint space and leads to the formation of scar tissue, reduce joint motion and fibrous or bony fusion across a joint (Bruyere, 2009). This process is called joint immobilization. As the disease progresses, structural damage can lead to instability of the joint, muscle atrophy, stretching of ligaments, tendon contractures and deformity of the joint. Unfortunately, these changes are irreversible and depend on the degree and duration of the inflammation (Bruyere, 2009).

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