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Allen, Pamela, "Joint Manifestations of Rheumatoid Arthritis" (2016). *Nursing Student Class Projects (Formerly MSN)*. 186.

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Joint Manifestations of Rheumatoid Arthritis

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Introduction

Rheumatoid arthritis (RA) is a common autoimmune disorder, affecting approximately 1% of the population worldwide. The interaction of genetic and environmental factors results in a cascade of immune reactions, which eventually lead to the development of structural bone damage, joint damage, and synovitis (Gibofsky, 2012). We understand more about the pathophysiology of this crippling disease. Early detection of RA is encouraged so that effective management can be started before pathological changes become irreversible (Gibofsky, 2014). It is the goal of this poster to outline the underlying pathophysiology, signs, and symptoms, and treatment options available to patients who suffer from joint manifestations of RA.

I chose RA as I am interested in how this disease process correlates with the need for joint replacements. Interestingly, total joint replacement surgery is needed less often as active treatment strategies combined with the availability of new medications has led to more effective RA control (Cooles & Isaacs, 2011).

Signs and Symptoms

The signs and symptoms of rheumatoid arthritis include the following:

- Fatigue
- Joint inflammation
- Joint pain
- Warm joints
- Morning stiffness
- Rheumatoid arthritis is most prevalent in middle-aged women and is most typically a persistent, progressive disease that affects multiple small and large synovial joints (McPhee & Hammer, 2010).

Pathophysiology

Rheumatoid arthritis usually manifests itself as a symmetrical arthritis affecting multiple small and large synovial joints. (McPhee & Hammer, 2010). Involvement of the small joints of the hands, wrists, and feet as well as the larger, peripheral joints, including the hips, knees, shoulders, and elbows, is typical (McPhee & Hammer, 2010). Exacerbations and remissions are common. In highly active cases, extra-articular manifestations can occur such as lung nodules and ocular inflammation (McPhee & Hammer, 2010).

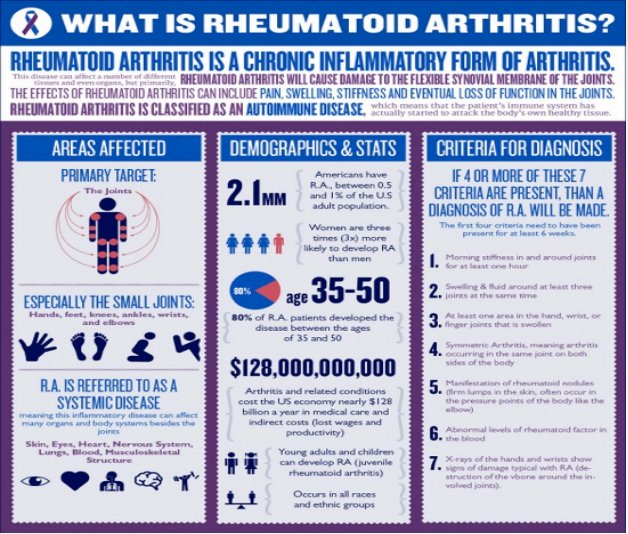
RA is an autoimmune disease. The major distinguishing characteristics of RA are that it is autoimmune, inflammatory, chronic, and systemic. (Dewing et al., 2012). The initial triggers of RA are unclear; hormones, genetics, and environmental factors may all play a role. Once the initial immune response is triggered, cells of the immune system produce autoantibodies and inflammatory cytokines, creating a cascade of inflammation resulting in the formation of pannus: the pannus invades and destroys cartilage and bone (Dewing et al., 2012). Additional joint damage and systemic complications ensue, resulting from a complex process of inflammatory mediators being released in the affected joint (Dewing et al., 2012).

Much of the pathologic damage that characterizes rheumatoid arthritis is centered around the synovial linings of joints (McPhee & Hammer, 2010). Normal synovium is composed of a thin cellular lining (one to three cell layers thick) and an underlying interstitium, which contains blood vessels but few cells (McPhee & Hammer, 2010). The synovium normally provides nutrients and lubrication to adjacent articular cartilage. Rheumatoid arthritis synovium, in contrast, is markedly abnormal, with a greatly expanded lining layer 8-10 cells thick) composed of activated cells and a highly inflammatory interstitium replete with B cells, T cells, and macrophages and vascular changes (including thrombosis and neovascularization) (McPhee & Hammer, 2010). At sites where synovium and articular cartilage are contiguous, rheumatoid arthritis synovial tissue (called pannus) invades and destroys adjacent cartilage and bone (McPhee & Hammer, 2010).

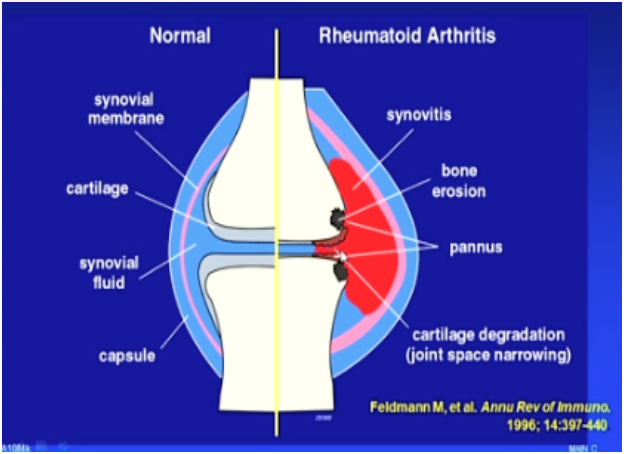
Advances in therapy have dramatically altered the long-term outlook for patients with rheumatoid arthritis. It is now clear that early diagnosis and subsequent treatment with combination drugs can minimize or even prevent the pain, joint destruction, and extra-articular complications associated with rheumatoid disease (Eleanor, 2003). Rheumatoid arthritis is treated with a comprehensive program of pharmacologic agents to decrease inflammation, physical therapy to maintain function, and surgery to prevent or correct deformities, should they occur despite aggressive medical therapy (Eleanor, 2003). Goals of treatment are to relieve pain, decrease inflammation, maintain function, prevent joint damage, and systemic illness. There is no cure, but disease-modifying anti-rheumatic drugs can help produce long-term remission of the disease (Eleanor, 2003).



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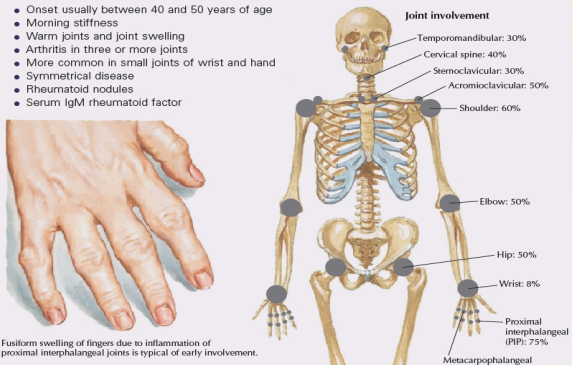


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Implications for Nursing Care

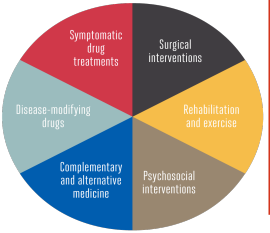
Nursing care centers on education, of the disease process of RA as well as encouraging compliance with physical therapy and medication regimens. Early treatment is key in preventing joint complications that may lead to lifelong disability. In addition, nursing care should include teaching to prevent osteoporosis, a common complication of RA. Patients should get enough calcium and Vitamin D, stop smoking, take preventative medications and continue to make the efforts to do some exercises. The tug of muscles on bones whenever you move (weight-bearing exercises) strengthens your bones and helps prevent osteoporosis (Arthritis, 2014).



Conclusion

Early diagnosis and immediate, aggressive treatment of RA are crucial, because only a brief window of opportunity exists to prevent permanent disability and lifelong complication. Diagnosis can be difficult as early symptoms mimic osteoarthritis, flu, and other viral illness. Rheumatologist consults can be beneficial as specialists can recognize subtle clues that are the clue to accurate early diagnosis and treatment (Arthritis, 2014). Significant advances have been made in the diagnosis and treatment of RA in recent years, and treatment effectiveness has greatly improved with the use of biologic medications (Salt & Crofford, 2011). However, the proportion of patients not reaching remission in RA remains high, necessitating further research (Salt & Crofford, 2012).

RA Management Strategies



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