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Rheumatoid Arthritis

Ana Vigil
vigil_ana@yahoo.com

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Ana Vigil RN,BSN,CCRN

Otterbein University, Westerville, Ohio

Introduction

- The topic of Rheumatoid arthritis (RA) was chosen as this disease process affects up to 1% of the U.S. population causing disability and early mortality (Mayo Clinic, 2017).
- Advanced practice providers (APPs) are able to detect and diagnosis RA early on before damage to patient's joints occur (Riley et al., 2017).
- As of 2017, there is an anticipated increase in demand over the next decade for health care professionals to be able to detect and develop treatment plans for patients with RA (Riley et al., 2017).
- Studies have shown that failing to recognize early or more insidious symptoms can hinder swift referral to a Rheumatologist and initiation of treatment (Riley et al., 2017).
- Over the past 20 years, more than 10 new medications for RA have been developed and approved by the FDA. These new drugs have improved outcomes for patients diagnosed with RA, but can cause decisions about treatment plans to be more complex (Mayo Clinic, 2017).
- As a future Nurse Practitioner (NP), there is a high likelihood of encountering and diagnosing a patient with this disease process.

About RA

- RA is an immune mediated inflammatory disease where your immune system attacks the lining of your joints causing swelling and inflammation (Barton et al., 2016).
- RA typically affects the small joints in your hands and feet first, causing pain and stiffness that can develop into permanent damage and deformity if left untreated (Barton et al., 2016).
- Researchers have yet to discover the exact trigger/cause of RA but it is likely that genetics and environmental causes play a role in the development of RA (Barton et al., 2016).
- There is no cure for RA, merely management of symptoms and prevention of joint deterioration (Barton et al., 2016).

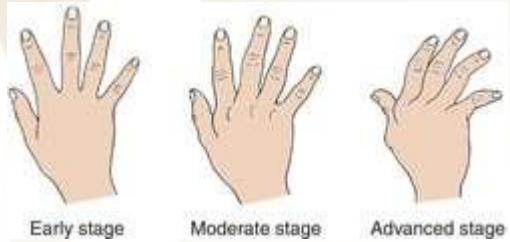
Signs & Symptoms

- RA usually develops slowly over a period of weeks to months before a diagnosis by your Primary Care Provider or NP is able to be made (*Rheumatoid Arthritis*, 2014).
- Classic symptoms that will present include: pain, swelling and stiffness of the peripheral joints and often occur in the morning, lasting longer than 6 weeks (*Rheumatoid Arthritis*, 2014).
- General systemic symptoms of inflammation occur which include:
  - Fever
  - Fatigue
  - Weakness
  - Anorexia
  - Weight loss
  - Generalized aching and stiffness (Richards & Edwards, 2014).
- Pain early on in the disease process occurs mainly from the pressure created from swelling (Huether & McCance, 2008).
- Later on in the disease process, pain occurs from abnormal hardening of body tissue (sclerosis) of the subchondral (layer of bone just below the cartilage in a joint) bone and new bone formation (Huether & McCance, 2008).
- The synovial joints affected are usually symmetric in presentation and when palpated are warm and feel boggy with a ruddy, cyanotic hue and thin shiny skin (*Rheumatoid Arthritis*, 2014).

Pathophysiology

- RA is a complex disease process that can be best characterized as an immune mediated inflammatory disease (Bingham, 2013).
- In patients with normal functioning immune systems, the body's self-antigens are in a state of tolerance with the host's immune system. When an autoimmunity response occurs, the patient's immune system begins to recognize the host's self-antigens as foreign (Huether & McCance, 2008).
- Although researchers, do not know the exact trigger that induces a RA autoimmune response, there is some data that suggests the gene HLA-DR4 is involved (Bingham, 2013).
- Even with some research that points to a genetic cause of RA, data has not been compiled that supports one specific trigger for the initiation of RA, pointing to other factors in the disease development (Barton et al., 2016).
- One of these other factors is a bacterial or viral infection that triggers an inflammatory response, or modifiable risk factors such as cigarette smoking that induces inflammation of the cells (Bingham, 2013).

Image 1. Shows the different stages of RA and their physical presentation

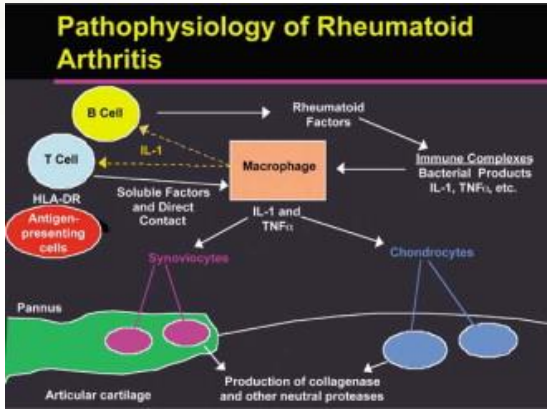


(Rheumatoid Arthritis, n.d.)

Pathophysiology continued

- RA is associated with class II major histocompatibility (MHC) antigens. When there is a deficiency of MHC class II molecules, a decrease in the recruitment of T- helper cells is seen, which affects the host's normal antibody response (Bingham, 2013).
- T-helper cell's responsibilities include the antigen- driven development of both B and T cells (Huether & McCance, 2008).
- Because the body is not able to properly form an attack against the antigen (whether foreign or self), the inflammatory cascade is initiated.
- As stated by Huether and McCance (2008), "The phagocytes of inflammation ingest the immune complexes and, in the process of doing so, release powerful enzymes that degrade synovial tissue and articular cartilage" (p. 1049).
- The damage occurs primarily from three functions:
  1. Macrophages, neutrophils and other phagocytes of inflammation in the synovial fluid become activated, degrading the cartilage (Bingham, 2013).
  2. Cytokines stimulate synthesis of proinflammatory compounds. Many cytokines are present including tumor necrosis factor (TNF), interleukin (IL)-1, IL-6 and IL-8. These cytokines effects include: upregulation of adhesions molecules, activation of osteoclasts, induction of other inflammatory mediators and activation of B-cells among other things. These proinflammatory compounds cause chondrocytes to attack the cartilage (Bingham, 2013).
  3. Activation of B-lymphocytes cause the production of more autoantibodies such as rheumatoid factor (RF) and anti-citrullinated peptide antibody (ACPA). While T-lymphocytes produce enzymes that amplify and perpetuate the inflammatory response, creating a never ending cycle of destruction (Bingham, 2013).

Image 2. Illustrates the complex cellular process that occurs in RA



(Arend, 2001)

Significance of Pathophysiology

- Understanding the pathophysiology of RA will help providers create a unique treatment plan that is tailored to the patient. Because RA is an inflammatory and an immune mediated disease there are different drugs that can be utilized to prevent symptoms of progression of cartilage and bone destruction.
- The 2015 American College of Rheumatology developed guidelines that address 6 major topics for treatment of RA which include
  1. Use of traditional disease-modifying antirheumatic drugs (DMARDs), biologic DMARDs, and tofacitinib.
  2. Utilizing glucocorticoids.
  3. Combination use of traditional and biologic DMARDs in patients with serious co-morbidities.
  4. Using vaccines in patients before they start treatment of traditional or biologic DMARDS.
  5. Screening for tuberculosis before starting treatment.
  6. Frequent laboratory monitoring for patients who are prescribed traditional DMARDs.

  (Singh et al., 2015).
- DMARDs work to suppress an overactive immune system (Palmer & Miedany, 2013). This can slow or stop the progression of joint damage seen from the inflammatory and autoimmune cascades outlined in the pathophysiology section.
- Biologic DMARDs are suggested for treatment of patients with RA who are not responding to traditional DMARD therapy. Biologic DMARDs are TNF inhibitors which have shown to be effective in preventing joint damage and halting the inflammatory response (Palmer & Miedany, 2013).
- As stated by Palmer and Miedany (2013), "Enhanced understanding of the pathophysiology of RA and the role of cytokines has enabled the development of innovative biological agents that target specific parts of the immune response" (p. 317).
- This is why understanding the pathophysiology of RA is so vital to NPs and APPs who will develop treatment plans for their patients.

Implications for Nursing

The role of NPs in managing and diagnosing patients with RA are expected to increase over the next 10-20 years (Riley et al., 2017). Yet a survey conducted by Riley et al. (2017), showed that NP's had a low to moderate level of confidence when diagnosing RA and a low level of confidence in managing and implementing treatment for patients with RA. This is concerning as NP's working in primary care may potentially be the first line for diagnosis of these patients.

This knowledge gap shows that increased training and education in RA could help improve NPs awareness of the signs and symptoms of RA, leading to an earlier initiation of treatment or a referral to a Rheumatologist (Riley et al., 2017).

As this disease process is incurable, it is also important for nurses to consider the emotional impact that RA takes on their patient's lives. RA, as outlined previously, causes pain, deformity, fatigue and decreased physical function. These symptoms can cause emotional burden to patients, and it is crucial for nurses to assess the health-related quality of life (HRQL) of their patients when assessing treatment efficacy (Strand, Wright, Bergman, Tambiah & Taylor, 2015).

Treatment of RA, from a patient's perspective, went beyond disease management. Patients wanted a targeted approach to disease management that incorporated goal setting (Strand et al., 2015). Patients found that setting goals allowed them to assess treatment success and made them feel more positive (Strand et al., 2015). Nurses have the ability to help patient's set these goals and encourage discussion with their APPs.

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Conclusion

Rheumatoid Arthritis is an immune mediated inflammatory disease of unclear etiology. The disease process primarily affects the synovial joints of the hands and feet and presents in a symmetric presentation (Rheumatoid Arthritis, 2014). The presence of autoantibodies perpetuates the inflammatory cascade which causes joint pain, swelling and if left untreated deformity. Studies have shown that early intervention is critical for patients receiving optimal outcomes (Rheumatoid Arthritis, 2017).

Nurse Practitioners, especially in the primary care setting, will potentially be first line access to a healthcare professional with patients who are newly diagnosed or have established RA (Riley et al., 2017). Education in RA and RA treatment is important for NPs in optimizing the management of RA.

Overall, RA is an insidious disease process, that can not be prevented but can be managed to prevent long-term disability and improved patient outcomes.

References