Arthritis essay

Science, Genetics

Rheumatoid Arthritis, which is the most common type of chronic inflammatory arthritis, can affect any organ system. What causes Rheumatoid Arthritis is still unknown. Genetic susceptibility combined with an external trigger, such as infection, smoking, and trauma, is theorized to cause the disease expression. It is believed that these external triggers cause an autoimmune reaction, resulting in chronic joint inflammation and synovial hypertrophy along with other manifestations (Hammer and McPhee, 2014). It has been observed that the patients of Rheumatoid Arthritis have higher levels of the Epstein-Barr virus (EBV) antibody titers and virus-infected B cells than the general people. The ability of the EBV to activate B cells for the production of rheumatoid factor is the primary reason for this virus to be identified as a potential trigger. Rheumatoid factor is detected in nearly 70% of patients afflicted with Rheumatoid Arthritis. There are enough evidences suggests its connection with the disease pathogenesis. Other viruses that garner interest regarding the cause of Rheumatoid Arthritis are parvovirus B19 and the retroviruses (Hammer and McPhee, 2014). However, there is no conclusive data available determining the viral pathogen as the primary agent for causing the disease.

Compared to Rheumatoid Arthritis, which is a chronic inflammatory disease of joints, Osteoarthritis is a disease of cartilage. Hyaline cartilage is 95% water and 5% chondrocytes (McCance and Huether, 1997). Its viscoelastic and compressive properties are caused by extracellular cartilage matrix, which is constituted of proteoglycans and type II collagen. In normal conditions, the extracellular matrix goes through a dynamic remodeling process that balances the degradative and synthetic enzyme activities,

which helps maintain the volume of cartilage. In Osteoarthritis cartilage, however, these degradative and synthetic enzymes are overactive, because of which the normal balance is lost, resulting in the loss of collagen and proteoglycans from the extracellular matrix (McCance and Huether, 1997). Like Rheumatoid Arthritis, what causes Osteoarthritis is also unknown. However, it has been observed that the trigger of Osteoarthritis begins when a tissue gets damaged from an injury and defects form in cartilage metabolism. The tissue damage incites chondrocytes to make reparable attempts, increasing the production of collagen and proteoglycans. However, the reparable attempts stimulate the cartilage degradative enzymes and inflammatory cytokines, which trigger an inflammatory cycle that eventually breaks down the cartilage, giving rise to the problem of Osteoarthritis (McCance and Huether, 1997).

Both gender and genetic factors influence Rheumatoid Arthritis. Numerous studies show the influence of genetic factors on an individual's susceptibility to Rheumatoid Arthritis. Like Rheumatoid Arthritis, genetic factors influence the development of Osteoarthritis too. The structure and shape of bones and joints are dependent on genetic factors, and in the case of an abnormality, the joint will more susceptible to this disease. For instance, the shape of hip joint has been identified as the most crucial predictor whether or not Osteoarthritis will develop in that joint (Hammer and McPhee, 2014). Several studies have provided evidence for the connection between genetic factors and Osteoarthritis.

In terms of gender, though Rheumatoid Arthritis affects both male and female, it has been observed that women are more susceptible to the

disease than men (Hammer and McPhee, 2014). Women are more likely to be afflicted with this disease after menopause or around perimenopause years due to hormonal abnormalities. Unlike Rheumatoid Arthritis that occurs more in women than men, Osteoarthritis occurs more in men than women. Especially, men below the age of 40 get afflicted with this disease most due to injury, whereas men rarely suffer from Rheumatoid Arthritis below the age of 45 years. However, women develop this disease most from the age of 40-70, and after this threshold, both men and women get equally affected (McCance and Huether, 1997).

References

McCance, K. L., & Huether, S. E. (1997). Pathophysiology: the biological basis for disease in adults and children. 3rd ed. St. Louis: Mosby Elsevier.

Hammer, G. D. and McPhee, S. J. (2014). Pathophysiology of Disease: An Introduction to Clinical Medicine. McGraw-Hill Medical. 7 edition.