My topic for the RUA paper is "Cirrhosis of the Liver."

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Chamberlain University College of Nursing

NR283: Pathophysiology

Dr. Silva

[redacted]

Hyperuricemia and Gout: Pathophysiological Processes.

Hyperuricemia and gout are closely related disorders, with hyperuricemia as a clinical signal of gout's possible development. Hyperuricemia is defined by high amounts of uric acid in the blood, which can form urate crystals in the joints. While not everyone with hyperuricemia develops gout, it is still a significant risk factor (Kuo et al., 2024). Gout is a metabolic illness that causes severe inflammatory arthritis, characterized by acute pain, edema, and stiffness, particularly in the joints. The most typically affected joint is the big toe. According to recent research, gout affects about 3.9% of the adult population in the United States, with males having a higher frequency than women. While curable, frequent flare-ups can cause long-term joint damage if left untreated.

Etiology and Risk Factors

Hyperuricemia is caused by an imbalance between uric acid synthesis and excretion through the kidneys. This disorder can be caused by eating too much purine-rich foods such as red meat, seafood, and alcohol, which raises uric acid levels (FitzGerald et al., 2023). Other risk factors include genetic susceptibility, obesity, hypertension, chronic renal disease, and certain drugs like diuretics. Although hyperuricemia may not necessarily result in symptoms, it is an important precursor to gout (Punzi et al., 2024). Men are more likely to get gout, especially between the ages of 30 and 50, but women have a decreased risk until after menopause. Sedentary lifestyles paired with high-sodium diets contribute to an elevated risk. Obesity, alcohol consumption, and dehydration worsen the disease. Older people are more vulnerable since they have less kidney function and have been exposed to risk factors for a more extended period.