



Relationship between Osteoarthritis and Non-Alcoholic Fatty Liver Disease

Albert Chan *

Department of Infection and Liver Diseases, Hospital of Wenzhou Medical College, Wenzhou, China

DESCRIPTION

Osteoarthritis (OA) is currently the third most common illness in the senior population worldwide, and as life expectancy rises, it is predicted that by 2030, it will affect one-third of middle-aged adults. OA is seen as a chronic illness that affects different tissues in the joints and causes bone spurs, which leads to joint pain, stiffness, and restricted movement. These symptoms all lower a patient's physical activity and, in turn, their quality of life. When fat deposits are seen in the hepatocytes on imaging or histology in patients who have no history of significant alcohol or continuous drug use, or when liver disease is caused by other factors, such as genetic diseases that increase fat deposition, Non-Alcoholic Fatty Liver Disease (NAFLD) is diagnosed. NAFLD covers a wide range of illnesses, including non-alcoholic steatohepatitis and NAFLD linked to cirrhosis. According to recent data, the prevalence of NAFLD and non-alcoholic steatohepatitis is, respectively, 6%-51% and 3%-5%. Due to variations in the study population, diagnostic standards, and diagnosis of NAFLD utilized, there are variations in the reported prevalence of NAFLD. Nevertheless, NAFLD is becoming more commonplace in every country. Obesity has been linked to OA in the knees and hips, according to numerous researches. Furthermore, a number of studies have linked the metabolic syndrome to knee OA. There aren't many extensive researches examining a connection between NAFLD and knee OA.

OA is a metabolic disease in which biochemical factors are involved, and these factors may alter both the onset and the course of the disease. It is not simply a condition brought on by ageing or physical factors. According to reports, the pathogenic mechanisms causing osteopenia include those connected to inflammation, obesity, and the metabolic syndrome; as a result, a possible link between NAFLD and ankle Joints might be suggested. The gold standard for the diagnosis of NAFLD is a liver biopsy. Its routine application is challenging for all patients

since it is an intrusive technique with a chance for sample mistake and because it is pricey. To make NAFLD diagnosis easier, a number of predictive indications have recently been proposed. Such indication is the Hepatic Steatosis Index (HSI), which simply considers patient sex, ALT and AST levels, Body Mass Index (BMI), and diabetes status for predicting NAFLD. Comparatively to extrapolating the extensive data available from the National Health and Nutrition Survey, HSI is a valuable and straightforward technique for identifying NAFLD. OA has long been thought to as a special result of the cartilage breakdown caused by tearing and abrasion. Osteophytes are thought to form as a response of the bone to protect and maintain the changed joint. Cartilage loss happens when the joint is subjected to extreme mechanical stress. OA is a complicated condition that affects a variety of tissues in addition to the cartilage, including the synovium, subchondral bone, capsule, meniscus, muscle, and tendon. It is not just mechanical or only brought on by inflammation. Particularly, new studies on the functions of inflammatory hormones and cytokines have shown that, in addition to these processes, metabolic pathways also play a significant role in the development of OA. Along with age and injury, obesity is a major risk factor for OA, and the fact that it can affect hand joints in addition to weight-bearing joints suggests that obesity-related mechanisms may be involved. Inflammatory mediators released by cartilage, bones, and synovia generate the complicated illness OA.

Lipid mediators may contribute to the pathogenesis of OA by potentially causing cartilage breakdown. An elevated BMI was linked to a higher incidence of knee OA in persons with metabolic symptoms in a prospective population-based investigation. It has been suggested that a complex interplay between hereditary and environmental variables related to obesity contributes to the occurrence and severity of OA by examining the direct and indirect obesity-related factors linked to the development of OA in mice and other animal models.

Correspondence to: Albert Chan, Department of Infection and Liver Diseases, Hospital of Wenzhou Medical College, Wenzhou, China, E-mail: Chan@gmail.com

Received: 06-Sep-2022, Manuscript No. JLR-22-18303; **Editor assigned:** 09-Sep-2022, Pre QC No. JLR-22-18303 (PQ); **Reviewed:** 23-Sep-2022, QC No JLR-22-18303; **Revised:** 30-Sep-2022, Manuscript No. JLR-22-18303 (R); **Published:** 10-Oct-2022, DOI: 10.35248/2167-0889.22.11.147.

Citation: Chan A (2022) Relationship between Osteoarthritis and Non-Alcoholic Fatty Liver Disease. J Liver. 11:147.

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