Osteoarthritis and obesity

Author: Dr.Mir Amir Aghdashi
Address: Urmia University of Medical Sciences
aghdashia@gmail.com

Obesity is one of the most significant, and potentially most preventable risk factors for the development of osteoarthritis . Numerous studies have shown a strong association between body mass index and osteoarthritis of the hip, knee, foot and hand. The WHO estimates from 2008 indicate that more than 1.4 billion adults are overweight and, of these, more than 200 million men and 300 million women are obese. It was estimated that the economic burden of OA in the USA was second only to diabetes in obesity.OA is a clinical syndrome of joint pain and dysfunction caused by joint degeneration, and affects more people than any other joint disease.80 per cent of people with osteoarthritis have compromised movement, and 25 per cent are unable to perform normal acts of daily living. The risk of knee osteoarthritis increases progressively throughout the Body Mass Index (BMI) categories. At the most extreme, very obese individuals with a BMI of 36 or more have a 14-fold higher risk of knee osteoarthritis compared to those within the healthy BMI range. Obesity is associated with the incidence and progression of OA of both weight-bearing and non weight-bearing joints -Non-weight bearing joints such as those in the hand are also marginally affected by obesity. This suggests that excess weight causes additional damage through a separate pathway, probably by raising blood glucose and insulin levels, thereby increasing inflammation of
the joint.. The pathophysiology of obesity-related OA is likely to be multi-factorial. Structural joint damage is thought to result from both mechanical factors, including increased forces about the joint, decreased muscle strength and altered biomechanics during everyday activities, and metabolic factors as being obese also increases the risk of OA in non weight-bearing joints such as the hands. Poor posture and unhealthy gait are more common in obese people, further predisposing
the joints to osteoarthritis. Leptin and adiponectin are the most abundantly produced adipokines and their receptors are expressed on the surface of chondrocytes, synoviocytes and subchrondral osteoblasts. Leptin has been found to increase levels of degradative enzymes, such as matrix metalloproteinases (MMPs) and nitric oxide, and production of pro-inflammatory cytokines. Chondrocytes from obese OA patients have been shown to exhibit a response pattern to leptin different from normal or overweight patients. Obesity leads to increased loading of the weight-bearing joint, which may be the most important mechanical contribution.
There is increasing evidence that exercise is most effective in reducing both pain and mobility when combined with weight loss. Studies show that even modest weight loss, when combined with exercise, reduces pain in obese patients with knee osteoarthritis, as well as improving mobility and physical functioning. Medical treatments can improve symptoms but on their own, without lifestyle change, may have

little benefit. Research shows that nearly 50 per cent of obese patients have a poor outcome from joint replacement surgery compared to less than ten per cent of those with a healthy weight.