Maturitas 89 (2016) 22–28

Contents lists available at ScienceDirect

Maturitas

journal homepage: www.elsevier.com/locate/maturitas

Review article

Obesity and osteoarthritis

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ARTICLE INFO

Article history:
Received 4 April 2016
Accepted 8 April 2016

Keywords:
Obesity
Osteoarthritis
Arthroplasty
Knee
Replacement
Outcomes

ABSTRACT

This paper provides an up-to-date review of obesity and lower limb osteoarthritis (OA). OA is a major global cause of disability, with the knee being the most frequently affected joint. There is a proven association between obesity and knee OA, and obesity is suggested to be the main modifiable risk factor. Obese patients (Body Mass Index, BMI, over 30 kg/m^2) are more likely to require total knee arthroplasty (TKA). The global prevalence of obesity has doubled since 1980; by 2025, 47% of UK men and 36% of women are forecast to be obese. This rising global burden is a key factor in the growing rise in the use of TKA. It is therefore important to appreciate the outcomes of surgery in patients with end-stage OA and a high BMI.

This review found that while OA is felt to contribute to weight gain, it is unclear whether TKA facilitates weight reduction. Surgery in obese patients is more technically challenging. This is reflected in the evidence, which suggests higher rates of short- to medium-term complications following TKA, including wound infection and medical complications, resulting in longer hospital stay, and potentially higher rates of malalignment, dislocation, and early revision. However, despite slower initial recovery and possibly lower functional scores and implant survival in the longer term, obese patients can still benefit from TKA in terms of improved function, quality of life and satisfaction.

In conclusion, despite higher risks and more uncertain outcomes of surgery, higher BMI in itself should not be a contraindication to TKA; instead, each patient's individual circumstances should be considered.

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http://dx.doi.org/10.1016/j.maturitas.2016.04.006
0378-5122/

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1. Introduction

This paper provides an up-to-date review on obesity and lower limb osteoarthritis (OA). It provides an evidence-based opinion regarding the burden of obesity, its contribution to the aetiopathogenesis of osteoarthritis, with specific reference to knee OA, and the impact of obesity on the outcome of treatments offered to treat end-stage osteoarthritis.

1.1. Osteoarthritis

Osteoarthritis (OA) is the clinical syndrome of joint pain accompanied by varying degrees of functional limitation and reduced quality of life. Pathologically, it is characterised by localised loss of cartilage, remodelling of adjacent bone, and associated inflammation [1].

Globally, hip and knee OA are the 11th highest contributors to global disability, with ageing populations expected to result in a jump to 4th by 2020 [1–3]. This significant disease burden results in 88,763 hip replacements and 96,986 knee replacements performed in the UK each year [4].

The knee is the most frequently affected joint. Knee OA affects 8.5 million people in the UK [5]. Females are affected more commonly than males (global prevalence 4.8% versus 2.8% for men) [6]. As women live longer, the majority of patients undergoing any total joint arthroplasty (TJA) are women, comprising 55% to 70% of most studies [7–13]. Opinion remains divided on whether total knee arthroplasty (TKA) should be performed in patients with higher BMI, given suggested poorer outcomes and higher risk of complications. The higher additional costs of undertaking TKA in obese patients (approximately £2135 per patient) places a higher burden on healthcare services, so the benefits in terms of functional and symptomatic improvement should be significant enough to warrant the additional risks [14].

1.2. Obesity

The global prevalence of obesity has doubled since 1980 [15,16]. Between 1993 and 2012 in the UK, the proportion of overweight and obese adults increased from 57.6% to 66.6% (men) and 48.6% to 57.2% (women). The precise factors causing obesity remain under debate. The Foresight Report concluded a "complex web of societal and biological factors that have, in recent decades, exposed our inherent human vulnerability to weight gain" [17].

The World Health Organisation (WHO) has defined several classifications of weight, based on Body Mass Index (BMI), including 'overweight' (BMI >25 kg/m^2) and 'obese' (BMI >30 kg/m^2) [15,16]. Obesity is further sub-classified as class I (BMI 30–34.99 kg/m^2), Class II (35–39.99 kg/m^2) and class III ('morbidly obese' $\geq$40 kg/m^2). A more recent category is 'super obese' (BMI >50 kg/m^2).

By 2025, it predicted 47% of UK men and 36% of women (aged between 21 and 60 years) would be obese. By 2050 the predictions are worse, with 60% adult men, 50% adult women, and 25% children (under 16 years) forecasted to be obese. Alongside the additional health risks to the individual – including chronic diseases such as heart disease, diabetes, hypertension, and elevated cholesterol – obesity is associated with higher healthcare costs; obese patients are estimated to incur 46% higher inpatient and 27% more outpatient costs [18,19].

1.3. Link between obesity and knee OA

Cross-sectional and longitudinal studies have consistently demonstrated an association between obesity, usually assessed by BMI, and the prevalence and incidence of knee OA, with obesity proposed to be the main modifiable risk factor [20–24].

Several large cohort studies have corroborated these findings using radiographic assessment of OA, including diagnostic changes of OA and bone volume [23,25]. Pattern of weight gain may be relevant, with a shift from normal to overweight in adult life carrying a higher risk for knee OA requiring TKA than being constantly overweight [26].

While radiographic markers of OA usually correlate with patient symptoms, this is not always the case, with studies showing radiographic OA changes to not always be associated with knee pain [27]. Aiming to show a similar association between obesity and OA using patient symptoms, rather than radiographic changes alone, some studies have found BMI to be a predictor of knee pain, independent of radiographic features [28,29].

1.4. Pathogenesis of obesity and OA

A variable combination of mechanical, humeral, metabolic and genetic factors are thought to play a role in the pathogenesis of OA [30–32]. With obesity, excess weight increases joint loading, resulting in deleterious effects on weight-bearing joints. The additional mass can stress articular cartilage beyond biological capabilities, therefore causing degenerative changes [23,24]. Gait analysis has demonstrated weight loss to reduce load across the knee joint, with approximately 0.5 kg weight loss resulting in a two- to four-fold reduction per step [33,34].

OA develops when cartilage breaks down faster than it is produced. Fat mass, rather than skeletal muscle mass, is a risk factor for cartilage defects; for every 1 kg increase in total body fat there is an increased risk of cartilage defects – a feature of early knee OA [35]. Metabolic and humeral factors may account for why a higher incidence of OA in individuals with higher BMI and body fat is found in non-weight bearing joints such as the hands [36–38]. Similarly, metabolic syndrome increases the risk of knee OA but not hip OA, suggesting mechanical factors may not be solely responsible [39].

1.5. Does weight loss improve OA?

Despite strong evidence linking weight gain to the development of knee OA, there is a relative paucity of evidence examining the impact of weight reduction, and the subsequent reduction in load across the joint, on improvements in either the symptoms or radiographic features of established OA [23,40,41].

Some studies have shown a reduction in BMI to reduce the risk of development of radiographic knee OA, with a large population study finding a reduction in BMI of $\geq$2 kg/m^2 over 10 years decreased the odds for developing knee OA by over 50% [42,43]. Using symptoms as a marker, significant bariatric surgery-induced weight loss can improve both hip and knee pain associated with OA [40]. Non-operative weight loss of at least 5.1%, including through dietary intervention, also yields symptomatic improvement [41]. Whether weight change modifies both knee joint cartilage volume and symptoms in the same cohort has not been widely assessed, but surgically-induced weight loss appears to an effective, rapid and dependable means of increasing medial joint space while also