The Role of Obesity and Bariatric Surgery in the Management of Knee and Hip Osteoarthritis

60

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Contents

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Abstract

The prevalence of both obesity and osteoarthritis is increasing. Obesity is an independent risk factor for osteoarthritis. Both mechanical and inflammatory factors contribute to this risk. The gold-standard treatment for end-stage knee and hip osteoarthritis is total joint arthroplasty. Patients with obesity have worse outcomes than patients with normal weight following total joint arthroplasty. There is a dearth of information on the effects of bariatric surgery on the incidence of total joint arthroplasty, and the impact on outcomes is limited with mixed results. Further study on total joint arthroplasty in patients with obesity is needed to determine optimal risk stratification, bariatric procedure selection, and timing of bariatric surgery relative to total joint arthroplasty.

Keywords

Bariatric surgery · Lower extremity osteoarthritis · Arthroplasty · Outcomes

Introduction

In 2016, the prevalence of obesity in America was 39.8% among adults (Hales et al. 2017), with an estimated 7% reaching morbid obesity, defined as a body mass index (BMI) >40 kg/m^2 (Sturm and Hattori 2013). Osteoarthritis (OA) is also highly prevalent. It is estimated that 25.9% of the projected total adult population will have doctor-diagnosed arthritis by 2040 (Hootman et al. 2016). The lifetime risk of end-stage knee osteoarthritis alone is 13.3% for men and 18.8% for women (Ogden et al. 2014). Knee OA is the second most prevalent cause of disability in the world (GBD 2016 Disease and Injury Incidence and Prevalence Collaborators 2017). The risk of OA-associated disability is as elevated as of cardiac conditions (Wang et al. 2016) and is the most prevalent of all diseases in the elderly (Yokota et al. 2015). In terms of hospital charges, osteoarthritis is more expensive than pneumonia, stroke, or complications from diabetes (Teichtahl et al. 2009).

Obesity is an independent risk factor for osteoarthritis (Gu et al. 2019). Patients with grade II obesity have 4.7 times the likelihood of developing knee osteoarthritis compared to those with normal weights (Reyes et al. 2016). Research suggests this risk is due to both biomechanical and systemic inflammatory effects on joints (Koonce and Bravman 2013). These effects eventually lead to total joint arthroplasty (TJA) in an increasing number of patients with obesity (Belmont et al. 2014; Fehring et al. 2007; Kremers et al. 2014). In the United States, over one million total knee and total hip replacement procedures are performed yearly.

Patients with obesity are known to have a higher risk of complications for total joint arthroplasty (Salih and Sutton 2013; Belmont et al. 2014). The American Association of Hip and Knee Surgeons suggests weight loss prior to TJA for patients with BMI >40 kg/m^2 (Workgroup of the American Association of Hip and Knee Surgeons Evidence-Based Committee 2013). While diet and exercise are important for a healthy lifestyle, these methods will not achieve a significant sustained weight loss. Bariatric surgery has been shown to provide sustained weight loss, as well as resolution of many obesity-related comorbidities, including type II diabetes, gastroesophageal reflux, and hyperlipidemia (Sharples and Mahawar 2019). Data investigating the effect of prior bariatric surgery on postoperative TJA outcomes is mixed.

Impact of Obesity on Lower Limb Osteoarthritis

In the United States, 31% of those with obesity were diagnosed with arthritis, while only 16% of normal weight individuals carried the diagnosis (Center for Disease Control and Prevention 2018). Obesity has been shown to be an independent risk factor for osteoarthritis (Gu et al. 2019). In a Swedish study by Jarvholm et al.,

15–67 year-old male construction workers were found to be more likely to undergo knee or hip replacement as BMI increased, even when controlling for age and tobacco use (Jarvholm et al. 2008). Data from the Nurses' Health Study, which included over 121,000 female nurses, revealed those with a BMI >35 kg/m^2 were twice as likely to undergo total hip arthroplasty (THA) compared to those with BMI <22 kg/m^2. Interestingly, those with a higher BMI at age 18 years had the highest risk of undergoing THA (Karlson et al. 2003).

Weight-Bearing Forces

Joint "wear-and-tear" secondary to mechanical and structural factors has classically been suggested as the mechanism for osteoarthritis. While these factors surely contribute, recent studies suggest they cannot solely account for the relationship between obesity and osteoarthritis (Sowers and Karvonen-Gutierrez 2010). For example, in a systematic review and meta-analysis by Long et al., both lean and fat mass in subjects with knee osteoarthritis were higher than in those without osteoarthritis. While fat mass percentage was positively associated with knee osteoarthritis, lean mass percentage was negatively associated with knee osteoarthritis. Increased fat mass was also associated with hand joint osteoarthritis (Long et al. 2019). The increased incidence of non-weight-bearing joint osteoarthritis in individuals with obesity has led to investigation into inflammatory factors.

Mechanical Factors

Abnormal loading of the knee and hip joint can lead to changes in the composition, structure, and mechanical characteristics of articular cartilage (Maly et al. 2005; Mundermann et al. 2005).

Joint load during walking is directly related to body weight. Peak *in vivo* hip contact forces during normal walking have been noted to be 2–3 times body weight (Heywood et al. 2019). So, as body weight increases, so do joint contact forces. The knee joint is especially vulnerable in the population with obesity where there is a high fat:lean mass ratio. The quadriceps muscle may fail to adequately absorb forces on the knee joint, leading to an increased load on the articular cartilage and subsequent progressive degeneration (Maly et al. 2005). Knee joint loads during walking and stair climbing have been shown to be higher than loads across the hip joint (Taylor et al. 2004).

Gait Changes

Gait disturbances are also common in patients with obesity. In a systematic review, Runhaar et al. found that patients with obesity had altered lower extremity biomechanics for everyday movements including walking, standing, and rising from a sit-to-stand position. When walking, patients with obesity took shorter, wider steps and chose a slower pace. During standing, individuals with obesity had greater toe-out angles. When transitioning from sitting to standing, those with obesity had less hip flexion and greater foot displacement. These adjustments lead to alterations in the regions of the articular cartilage within the joint that bears the load and higher loads across the hip and knee joints (Runhaar et al. 2011). While gait alterations are likely adaptations adopted as a means to temporarily offload the hip and knee joints, studies show that joint loads in excess of 4 times body weight can occur regularly. Furthermore, stumbling can result in loads over 8 times body weight (Bergmann et al. 2001).

Inflammatory Factors

Obesity has been shown to be associated with a chronic inflammatory state. Specifically, where adipocytes were previously seen as solely storage vesicles, they are now known to be metabolically active cells that secrete a multitude of factors, termed adipocytokines. Many of these adipocytokines are thought to play a role in cartilage homeostasis. Because obese patients have a

higher adipocyte mass, the secondary changes in the adipocytokine milieu are thought to contribute to the degradation of cartilage, and therefore, the development of osteoarthritis (Francisco et al. 2018b).

Research on adipocytes and adipocytokines aims to determine why obesity is associated with a range of comorbidities, including osteoarthritis. Adipocytokines of interest include leptin, adiponectin, resistin, visfatin, lipocalin-2, chemerin, and apelin (Fig. 60.1) (Sowers and Karvonen-Gutierrez 2010; Francisco et al. 2018a).

It is unknown why some patients with obesity develop osteoarthritis while others do not, despite all these patients experiencing increased joint loads and most having altered gait characteristics. However, the conglomerate of comorbidities associated with metabolic syndrome may have an interrelated etiology. Karvonen-Gutierrez et al. showed that cardiometabolic biomarkers are associated with knee osteoarthritis regardless of BMI. In this report, mid-aged women with both obesity and two or more cardiovascular risk factors were more than 6 times more likely to have knee osteoarthritis when compared to women without obesity or cardiometabolic risk factors (Karvonen-Gutierrez et al. 2012).

Association of Inflammation with Mechanical Stress

There is evidence that increased load on the knee joint experienced by individuals with obesity may lead to the release of cytokines, growth factors, and metalloproteinases triggered by mechanoreceptors on the surface of chondrocytes (Sowers and Karvonen-Gutierrez 2010), which may contribute to the inflammatory milieu in addition to the increased adipocytokine production by adipose tissue.

Leptin

One of the most studied adipokines that likely plays a role in the development of osteoarthritis is leptin. Leptin is classically known as a metabolic adipocytokine that acts to reduce food intake and increase energy expenditure. It is known to be present in both plasma and synovial fluid in higher concentrations in individuals with obesity (Dumond et al. 2003). Griffin et al. showed that leptin-deficient mice could develop an obese phenotype without increasing the incidence of osteoarthritis, suggesting a key role for leptin in the development of osteoarthritis (Griffin

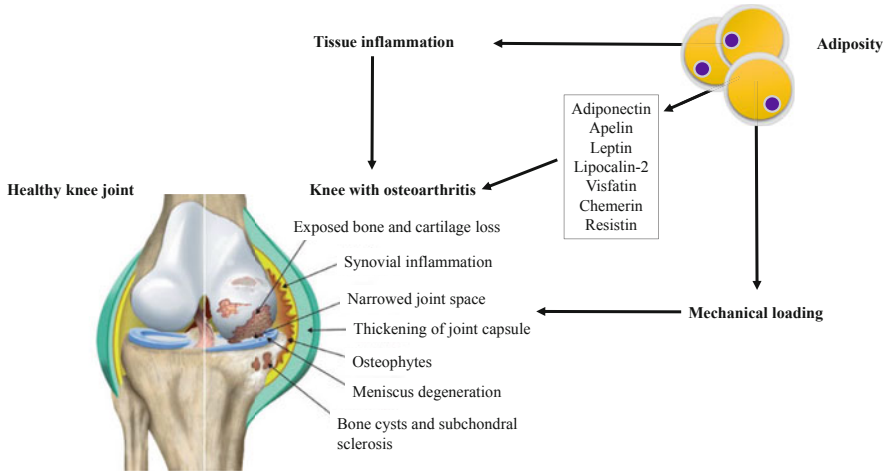

Fig. 60.1 Illustration depicting the effects of increased fat mass and dysregulation in cartilage degradation (Adapted from Uhalte E, Wilkinson JM, Southam L, Zeggini E. Pathways to understanding the genomic aetiology of osteoarthritis. Hum Mol Genet. 2017;26(R2):R193-R201)

et al. 2009). Leptin has a synergistic relationship with pro-inflammatory cytokines which leads to increased production of several inflammatory factors, growth factors, and matrix metalloproteinases by chondrocytes (Francisco et al. 2018a). Leptin also has been shown to affect chondrogenic progenitor cells by reducing their ability to migrate, changing their differentiation pathway, and modifying their cell cycle, ultimately altering their ability to maintain cartilage homeostasis and replace damaged tissue (Francisco et al. 2018b). Leptin likely has a dose-dependent relationship with manifestations of osteoarthritis. Leptin expression in cartilage, subchondral bone, synovial tissues, and osteophytes has been associated with the degree of cartilage degeneration and radiologic severity of osteoarthritis (Fig. 60.2) (Simopoulou et al. 2007; Francisco et al. 2018b).

Adiponectin

Adiponectin is a protein structurally similar to collagen that is synthesized by adipose tissue, although circulating levels are inversely related to weight. Using knockout mice, adiponectin has been associated with preventing insulin resistance and lipid accumulation in muscles when the mice are placed on a high fat/sucrose diet (Francisco et al. 2018a). Adiponectin receptors have been found in cartilage, bone, and synovial tissues (Sowers and Karvonen-Gutierrez 2010). Conflicting data exists regarding the role of adiponectin in the development of osteoarthritis with some studies suggesting a protective effect (such as inhibition of pro-inflammatory factors, stimulation of osteoclast proliferation, and mineralization), while others suggest a negative effect (such as increased production of

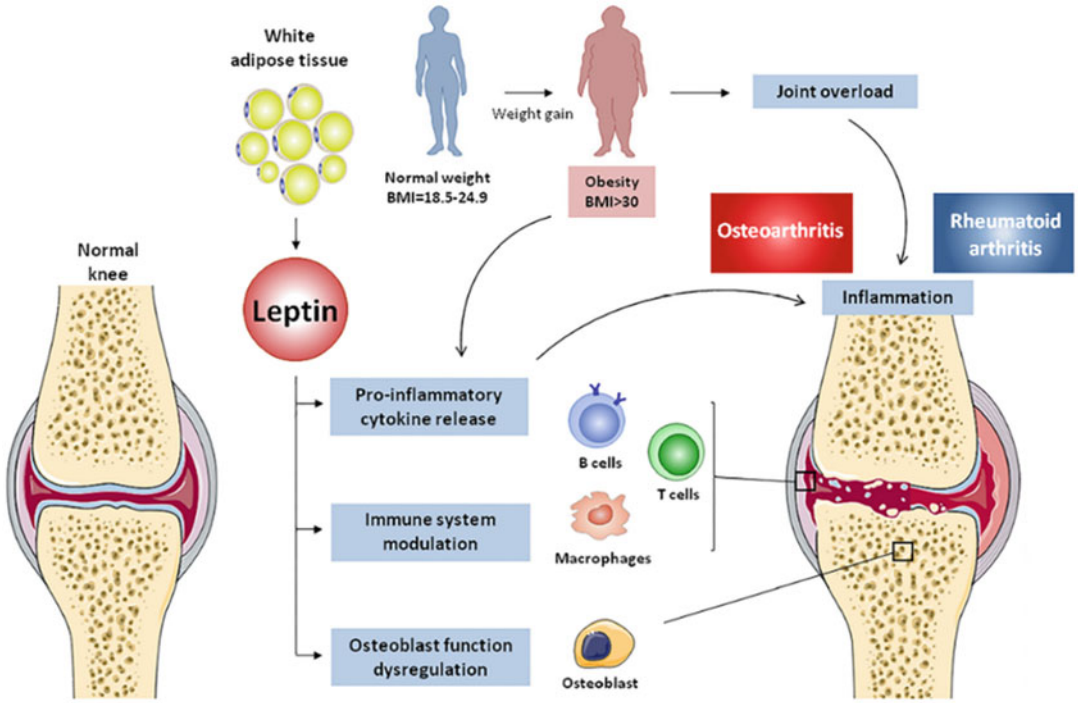

Fig. 60.2 Effects of adipose tissue-derived leptin on osteoarthritis and rheumatoid arthritis. Body weight gain, accompanied by white adipose tissue expansion, leads to obesity and subsequent increase of mechanical load, resulting in cartilage degradation and osteoarthritis onset. Adipose tissue-derived leptin causes osteoblast dysregulation in subchondral bone, thus promoting joint destruction. Additionally, leptin induces pro-inflammatory cytokine release from innate and adaptive immune cells, generating an inflammatory environment that prompts cartilage damage and rheumatoid arthritis (Reprinted from Francisco V, Pino J, Campos-Cabaleiro V, et al. Obesity, Fat Mass and Immune System: Role for Leptin. Front Physiol. 2018;9:640)

pro-inflammatory factors, inhibition of osteoclast differentiation, promotion of apoptosis) (Francisco et al. 2018a).

Studies have shown an increase in adiponectin in patients with osteoarthritis compared to heathy controls, as well as in patients with the radiologically most severe osteoarthritis disease. Both exercise and mechanical loading have been shown to increase adiponectin levels and its receptors in skeletal muscle, suggesting that adiponectin may play a protective role in preventing bone loss (Francisco et al. 2018a).

Resistin

Serum resistin levels have been shown to be increased in obesity and associated with adipose tissue inflammation. While in rodents the main source of resistin is adipocytes, in humans it is macrophages. Both serum and synovial fluid levels of resistin have been found in higher concentrations in patients with osteoarthritis compared to healthy controls; however, its correlation to radiographic damage is unclear, with conflicting evidence. Resistin is a pro-inflammatory cytokine that has been associated with increased levels of other cytokines and chemokines and with increased osteoblast proliferation (Francisco et al. 2018a).

Other Adipocytokines

Other adipokines that may contribute to the development of osteoarthritis in the population with obesity include visfatin, lipocalin-2, chemerin, and apelin. In patients with osteoarthritis, visfatin production was noted in several joint structures including the infrapatellar fat pad, synovium, and osteophytes. Visfatin is more prevalent in the serum and synovial fluid of osteoarthritis patients, where it increases both the production of pro-inflammatory factors and the degradation of joint connective tissue and extracellular matrix (Francisco et al. 2018a). Lipocalin-2 is a glycoprotein that circulates in a covalent complex with matrix metalloproteinase-9, whose main source is adipose tissue. Lipocalin-2 is expressed in joint tissues and found in elevated levels in patients with obesity as well as in patients with osteoarthritis. It has been shown to stimulate pro-osteoclastogenetic factors, inhibit anti-osteoclastogenic factors, and reduce chondrocyte proliferation. Levels have been correlated with fracture risk in the geriatric population (Francisco et al. 2018a).

Chemerin and its receptor are both expressed in adipose tissue, and levels are correlated with BMI in humans. The adipokine has been shown to be upregulated in adipose tissue of obese rats with type 2 diabetes mellitus. It is suspected to serve as a bridge between innate and adaptive immunity and possibly plays a role in osteoblast differentiation (Francisco et al. 2018a). Apelin is thought to be a pro-inflammatory adipokine that is found in higher levels in synovial fluid in individuals with osteoarthritis and has been positively correlated with severity of disease. It increases expression of catabolic factors in chondrocytes and decreases proteoglycan in articular cartilage (Francisco et al. 2018a).

Impact of Obesity on Arthroplasty

The risk of complications after total joint arthroplasty is particularly high once BMI reaches 40 kg/m^2. In a cohort followed for 10 years, mortality was higher in both genders, whereas risk of revision and dislocation increased in men only, contrasting with risk of reoperation, elevated in women (Tohidi et al. 2019). Short-term postoperative complications are likely related to the increased incidence of comorbidities in patients with obesity, as well as increased intraoperative difficulty, such as technical errors, surgeon reported difficulty, and problems that occur during surgery (Jarvenpaa et al. 2010; Nunez et al. 2011). Type II diabetes and obstructive sleep apnea, common comorbidities in the population with obesity, have both been associated with an increase in serious postoperative complications after TJA (Jamsen et al. 2012).

Severe obesity is noted to be associated with an increased risk of surgical site infections, respiratory complications, thromboembolic events, and hospital length of stay (Zusmanovich et al. 2018).

In the most comprehensive systematic review and meta-analysis on the effect of obesity on postoperative outcomes of TJA for osteoarthritis, 31 studies from 18 different countries were included. Patients without obesity were found to have fewer postoperative infections and fewer deep venous thromboses after either total hip arthroplasty (THA) or total knee arthroplasty (TKA) when compared to patients with morbid obesity (Pozzobon et al. 2018).

Late Postoperative Abnormalities

Long-term complications are likely associated with the mechanical and structural effects of obesity on artificial joints over time. The differences in joint load and daily movements between individuals with obesity versus normal weight, mentioned in the prior section, lead to increased risk for accelerated bare surface wear, early prosthesis failure, implant loosening, need for revisional joint surgery, and component malposition in the population with obesity after TJA. Likely due to the increased load in the knee compared to the hip joint, these effects are particularly evident after TKA (Abdel et al. 2015; Kerkhoffs et al. 2012).

Revisions after TJA have been shown to be higher in individuals with obesity versus those without obesity, with more discrepancies after TKA than THA (Chee et al. 2010; Foran et al. 2004). Patients with obesity are also at increased risk of prosthetic dislocation after THA (Watts et al. 2016). In a systematic review by Barrett et al., THA outcomes were compared between 66,238 patients who were morbidly obese and 705,619 patients with a BMI <30 kg/m^2. The overall revision rate was 7.99% in patients with morbid obesity versus 2.75% in patients without obesity (Barrett et al. 2018).

Functional Outcome

Poorer functional results are likely due to the conglomerate effect of short-term and long-term complications experienced. In the same comprehensive systematic review and meta-analysis mentioned above, participants without obesity reported less knee pain at both short-term (<6 months) and long-term (>6 months) follow-up after TKA, and less hip pain at short-term follow-up after THA. Patients without obesity also reported less disability at long-term follow-up for both TKA and THA (Pozzobon et al. 2018). The functional outcome, quantified using the Harris Hip Score, was comparable between patients with and without obesity. However, the individual studies that looked at quality of life did report lower Short-Form scores in patients with obesity postoperatively (Barrett et al. 2018).

Impact of Weight Loss and Bariatric Surgery on Osteoarthritis

A 5–10% weight loss significantly improves pain, self-reported disability, and quality of life in adults with both obesity (BMI 33–36 kg/m^2) and mild to moderate knee osteoarthritis (Chu et al. 2018).

In-hospital complications after hip and knee arthroplasty, and 90-day troubles after knee arthroplasty, diminish in patients who underwent prior bariatric surgery (McLawhorn et al. 2018a, 2018b). As many as 91% of patients undergoing gastric bypass or sleeve gastrectomy experienced a resolution of arthropathy by 21 months after surgery (Nelson et al. 2006). All patients undergoing sleeve gastrectomy had resolution of their joint pain 1 year after surgery, in another experience (Moon Han et al. 2005).

Studies assessing the specific effect of bariatric surgery on osteoarthritis progression suggest several benefits including: radiographic improvement of disease, decreased frequency and intensity of joint pain, improved physical function, and improved range of motion (Groen et al. 2015).

Improvements have also been noted in postural stability and sway during walking, which may decrease the magnitude and frequency of abnormal joint loads experienced by patients with obesity (Ponta et al. 2014).

Improved gait parameters including decreases in step width in the frontal plane, increases in step length, decreased torque around the hip and knee joint, and increased self-directed walking speed were noted after bariatric surgery. However, other studies suggest weight loss leads to increased torque across joints due to the increased stride length and gait velocity that accompany weight loss (Vartiainen et al. 2012; Vincent et al. 2012).

Impact of Bariatric Surgery on Arthroplasty

Because of the increased short-term and long-term complications, poorer functional outcomes, and increased cost of TJA in patients with morbid obesity compared to patients with normal BMI, the American Association of Hip and Knee Surgeons suggested to strongly recommend consideration for weight loss prior to TJA in patients with BMI > 40 kg/m^2 (Workgroup of the American Association of Hip and Knee Surgeons Evidence Based Committee 2013). Bariatric surgery has been shown to be the only reliable method of weight loss and comorbidity resolution for patients in this BMI range, though the effect of bariatric surgery on TJA outcomes shows mixed results.

The three most recent systematic reviews evaluating the effect of bariatric surgery prior to TJA include the same 13 studies in different combinations. Gu et al. reported conflicting evidence on the impact of bariatric surgery prior to TJA on in-hospital complications, 30-day complications, 90-day complications, revision rates, and hospital length of stay (Gu et al. 2019). Similarly, Stavrakis et al. reviewed 7 studies, 6 of which were included in the meta-analysis by Gu et al., to describe the conflicting evidence regarding the effect of bariatric surgery on TJA outcomes (Stavrakis et al. 2018). A third review and meta-analysis by Smith et al. which was included in the review by Stavrakis, included 5 of the studies used by Gu et al. No significant differences in the incidence of superficial wound infection, deep wound infection, deep vein thrombosis, pulmonary embolism, reoperation, joint revision, or mortality were found between the two groups in this meta-analysis. There were, however, more medical complications in patients who did not undergo bariatric surgery prior to TJA (Smith et al. 2016).

There are several individual studies reporting advantages to undergoing bariatric surgery prior to TJA. In-hospital complications after hip and knee arthroplasty, and 90-day troubles after knee arthroplasty, diminish in patients who underwent prior bariatric surgery (McLawhorn et al. 2018a, 2018b). A large study by Werner et al using a Medicare database with 78,036 patients compared the 90-day complication rate in three groups: (1) patients without obesity, (2) patients with morbid obesity who did not undergo bariatric surgery, and (3) patients with morbid obesity who underwent bariatric surgery prior to TKA. They found a decreased rate of both major and minor complications in patients with morbid obesity who underwent bariatric surgery when compared to patients with morbid obesity who did not undergo bariatric surgery prior to TKA (Werner et al. 2015). Watts et al compared the outcome of THA in patients with morbid obesity who did and did not previously undergo bariatric surgery in a matched cohort study. They found a decrease in reoperations and revisions for patients who underwent bariatric surgery prior to THA (Watts et al. 2016b). Kulkarni et al found that patients with obesity who underwent bariatric surgery prior to TJA had 3.5 times lower likelihood of wound infection and 7 times lower likelihood of hospital readmission compared to those who underwent TJA alone (Kulkarni et al. 2011). Nearing et al found operative time and length of hospital stay were decreased for patients who had arthroplasty performed after bariatric surgery. Early complications and late reinterventions were similar (Nearing et al. 2017).

While the decrease in joint loading and improvement in gait mechanics account for a portion of the improvement in joint pain and osteoarthritic progression, the changes in the hormonal milieu secondary to bariatric surgery are

also likely to contribute. There have been studies investigating the changes in leptin secondary to bariatric surgery. Chen et al. found that levels of several adipocytokines and related factors decreased over the course of the first year after bariatric surgery. Even more interestingly, leptin reduction and stabilization corresponded closely with the initial reduction and stabilization in osteoarthritic knee pain (Chen et al. 2018). This suggests that a decrease in leptin, initiated by bariatric surgery, is directly associated with an improvement in osteoarthritis symptoms and potentially joint damage.

There are also a few studies that suggested worse postoperative TJA outcomes in patients who had prior bariatric surgery. A New York statewide cooperative study over a 10-year period revealed that 90-day postoperative complications after total joint arthroplasty were increased in obese patients who previously underwent bariatric operations. Immediate hospital costs were also more substantial (Liu et al. 2019). In a previous article with the same global database, they found that bariatric surgery was not a risk factor for nonelective readmissions at 30-days, 90-days, or 1 year and did not change overall cost. Nevertheless, it did predict an increase in elective admission for up to 1 year (Liu et al. 2018).

Within a similar perspective, a controlled investigation using the Medicare database unveiled about twice the risk of dislocation, as well as revision surgery, in the bariatric population compared to no weight-reducing operation in obese or lean controls. However, the bariatric patients in this study were significantly more comorbid than the non-bariatric population, calling to question the real reason for these differences in outcome (Nickel et al. 2018).

Lee et al. used Medicare 5% part B data from 1999–2012 to identify patients who underwent THA or TKA. A history of prior bariatric surgery as well as the presence of comorbid conditions such as diabetes, hypothyroidism, impaired renal function, osteomalacia, and Cushing's syndrome was identified for all patients. Kaplan-Meier risk of revision of THA or TKA within 0.5, 1, 2, and 5 years was evaluated for each condition, including the presence of prior bariatric surgery. Bariatric surgery prior to THA was not associated with an increased overall risk for revision, but was associated with an increased risk for revision for periprosthetic infection. Patients undergoing TKA following bariatric surgery were at increased overall risk for revision, but not at increased risk for revision for periprosthetic infection (Lee et al. 2018).

Bariatric Surgery and Total Arthroplasty: Which Comes First, the Chicken or the Egg?

While it is possible that immobility secondary to osteoarthritis contributes to the development of obesity, evidence does not suggest that TJA will lead to postoperative weight loss. Despite increased mobility after TJA, most patients maintain and many actually gain weight postoperatively (Springer et al. 2017). While in one study, postoperative weight loss was most strongly associated with preoperative BMI (Inacio et al. 2014), this may just indicate better results of TJA when concurrent metabolic syndrome is earlier in its course. TJA is not suggested as a treatment for significant weight loss.

The timing of bariatric surgery relative to TJA may have an effect on TJA outcomes in patients after bariatric surgery.

Schwarzkopf et al. used the Healthcare Cost and Utilization Project California State Inpatient Database to identify patients who underwent TJA following bariatric surgery from 2007–2011. There were 330 patients who underwent bariatric surgery followed by THA and 1017 patients who underwent bariatric surgery followed by TKA with 19% of patients having THA within 6 months of their bariatric procedure and 10% of patients having TKA within 6 months of their bariatric procedure. There was no association found between time of bariatric surgery and THA or TKA and 90-day complications in multivariate logistic regression analysis. However, patients undergoing THA more than 6 months after bariatric surgery were significantly less likely to have a 90-day readmission compared to patients

undergoing THA within 6 months of bariatric surgery (Schwarzkopf et al. 2018).

Severson et al compared the outcomes of three groups of patients: (1) patients with TKA prior to bariatric surgery (n = 39) (2) patients who underwent TKA less than 2 years after bariatric surgery (n = 25) (3) patients who underwent TKA more than 2 years after bariatric surgery (n = 61). They found patients in group 3 had shorter anesthesia time, total operative time, and tourniquet time while 90-day complication rates and duration of hospital stay did not differ among groups (Severson et al. 2012).

More study is needed in this to determine the optimal timing between bariatric surgery and TJA.

Mobility and Access to Care

There are potential advantages to performing bariatric surgery prior to TJA other than postoperative outcomes, such as potential increased access to care and decreased costs. Many rural facilities will refer patients with obesity to tertiary medical centers to undergo TJA. This requires increased transportation costs and difficulty with follow-up. If a patient with osteoarthritis was able to lose weight with bariatric surgery, the patient may be able to undergo TJA at an institution closer to home, improving access to care and eliminating some of the complexities of follow-up (Springer et al. 2017).

Financial Issues

McLawhorn et al. compared the cost of performing bariatric surgery prior to TKA in patients with morbid obesity and osteoarthritis to TKA alone in BMI-matched patients. Costs included costs of treatment, complications, and 90-day follow-up. Quality-adjusted-life-years (QALY) were also calculated for both groups. While performing bariatric surgery prior to TKA was more expensive than TKA alone, the QALY was also higher. Given this, the incremental cost-effectiveness ratio between the two groups was found to be $13,910 per QALY. This value was significantly less than the $100,000 per QALY used as the threshold willingness to pay, leading to the conclusion that performing bariatric surgery prior to TKA is cost-effective (McLawhorn et al. 2016). Kremers et al. found that, for every 5-unit increase in BMI over 30 kg/m^2, hospitalization cost increased $300 for TKA and $650 for revisional TKA, even when adjusting for comorbidities and complications (Kremers et al. 2014).

Culler et al. used the Medicare Provider Analysis Review file to show that any adverse event for TJA increased mean hospital cost by $3429 and increased hospital length of stay by 1 day. Given these findings, in the setting of bundled payment for TJA, these authors suggested undertaking all possible complication-reducing measures to decrease the risk of complications and therefore reduce hospital cost (Culler et al. 2016; Kremers et al. 2014).

Conversely, Giori et al. demonstrated that for patients with BMI >40 kg/m^2 who undergo TJA, there is only one complication for every 14 complication-free operations. These authors suggested not using a strict BMI cutoff for patients who otherwise qualify for TJA. Similarly, Springer et al. followed 289 patients with BMI >40 kg/m^2 and end-stage osteoarthritis for 2 years. They found that requiring weight loss prior to TJA in this population most often leads to these patients remaining morbidly obese and never undergoing TJA. Only 20% of patients underwent bariatric surgery. These authors suggested increasing the resources and coordinated care to facilitate patients' weight loss (Giori et al. 2018).

Ongoing Trials and Future Perspectives

There is currently no consensus on the utility of bariatric surgery for weight loss prior to TJA, and no prospective or randomized controlled trials on the topic. In addition, no prospective studies have evaluated whether reduced biomechanical strain on lower extremity joints after bariatric surgery eliminates the need for TJA in certain patients, or if, conversely, the increased mobility and exercise capability of patients who lose significant weight

from bariatric surgery lead to an increase in TJA in this population.

The Surgical Weight-Loss to Improve Functional Status Trajectories Following TKA (SWIFT) Trial (NCT02598531) is the first prospective, controlled, multicenter trial comparing postoperative outcomes in patients with severe obesity who undergo TKA with a well-matched cohort of patients who undergo weight loss surgery prior to elective TKA. This trial is currently recruiting patients (Surgical Weight-Loss to Improve Functional Status Trajectories Following Total Knee Arthroplasty/SWIFT Trial n.d.) (Benotti et al. 2018).

Future Directions

Important areas for study include: comparing the effects of surgical weight loss and medical weight loss on TJA necessity and outcome, comparing the effect of different surgical weight loss procedures on TJA necessity and outcome, investigating risk stratification protocols for patients with obesity and end-stage osteoarthritis, determining a safe and cost-effective BMI cutoff for TJA, and ultimately determining an appropriate weight management algorithm and multidisciplinary approach for obese patients with end-stage osteoarthritis. In addition to studies investigating the effect of bariatric surgery on osteoarthritis and TJA outcomes, further investigation into the mechanisms that regulate peripheral and central adipokine activity and their contribution to the development of osteoarthritis may lead to novel treatments for osteoarthritis in the morbidly obese population.

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