The Effects of Obesity on Spine Surgery: A Systematic Review of the Literature

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Abstract

Study Design   Literature review.

Objective   The aim of this literature review is to examine the effects of obesity on postoperative complications and functional outcomes after spine surgery.

Methods   A review of the relevant literature examining the effects of obesity and spine surgery was conducted using PubMed, Google Scholar, and Cochrane databases.

Results   Obesity contributes to disk degeneration and low back pain and potentially increases the risk of developing operative pathology. Obese patients undergoing spine surgery have a higher risk of developing postoperative complications, particularly surgical site infection and venous thromboembolism. Though functional outcomes in this population may not mirror the general population, the treatment effect associated with surgery is at least equivalent if not better in obese individuals. This reduction is primarily due to worse outcomes associated with nonoperative treatment in the obese population.

Conclusion   Obese individuals represent a unique patient population with respect to nonoperative treatment, postoperative complication rates, and functional outcomes. However, given the equivalent or greater treatment effect of surgery, this comorbidity should not prohibit obese patients from undergoing operative intervention. Future investigations in this area should attempt to develop strategies to minimize complications and improve outcomes in obese individuals and also examine the role of controlled weight loss preoperatively to mitigate these risks.

Introduction

Obesity represents a growing public health challenge, with an estimated prevalence of 34.9% of U.S. adults, or 78.6 million people. On an economic level, Americans spend 147 billion dollars annually to combat the health-related medical problems attributed to obesity. The World Health Organization (WHO) defines obesity by calculating the body mass index (BMI). This measure is derived from dividing weight in kilograms by height in meters squared. Individuals with a BMI between 25 and 29.9 kg/m² are classified as overweight. Those with a BMI between 30 and 40 kg/m² are obese, and those with BMIs >40 kg/m² are considered morbidly obese. Obesity is associated with a litany of medical comorbidities including diabetes mellitus, hypertension, osteoarthritis, obstructive sleep apnea, and depression. Though the effects on overall health are well documented, a growing body of literature suggests that obesity may contribute to increased rates of disk degeneration, spinal arthritis, and low back pain. As the incidence of obesity continues to rise in the general population, spine surgeons will undoubtedly perform more surgical procedures on these individuals. The purpose of this article...
is to detail the effects obesity has on the adult spine as well as the effects on postoperative complication rates and functional outcomes in individuals undergoing spine surgery through a review of the existing literature.

**Effects on the Spinal Column**

Obesity has been linked with increased rates of disk degeneration, low back pain, sciatica, and spine surgery.\(^9\)\(^\text{-}\)\(^13\) In a review of magnetic resonance imaging of the spine in 975 individuals, Teraguchi et al reported a significantly higher incidence of disk degeneration in the cervical (odds ratio [OR]: 1.60; \(p < 0.05\)), thoracic (OR: 3.12; \(p < 0.0001\)), and lumbar spine (OR: 2.56; \(p < 0.05\)) in obese subjects.\(^11\) In a separate meta-analyses, Shiri and colleagues reported a significant association between obesity and low back pain, sciatica, and surgery for disk herniation.\(^9\)\(^,\)\(^10\) Although intuitively it seems that the most obvious cause of obesity-related disk degeneration would be the greater mechanical loads imparted on the spine in heavier individuals, more recent research suggests a multifactorial origin. Additional explanations for the increased incidence of disk degeneration in obese individuals include obesity-related chronic inflammation, gene–environment interaction, and decreased blood supply to the disk leading to ischemic changes.

The chronic inflammatory state associated with obesity likely plays a role in the development of disk degeneration. Adipocytes are the body’s basic fat-building cells. Although serving as a reservoir for energy storage, adipocytes also have an endocrine function through the release of cytokines, termed adipokines. The most heavily researched of these adipokines is leptin. Higher levels of systemic adipokines have been associated with the development and progression of osteoarthritis in multiple joints. In a study of patients undergoing knee arthroplasty and arthroscopy, Dumond et al noted that higher concentrations of leptin in arthritic joints correlated with BMI.\(^14\) Furthermore, leptin has also been associated with the progression of osteoarthritis in the hand.\(^15\) In the vertebral column, adipokines contribute to cell clustering and matrix degradation within the disk tissue.\(^12\)\(^,\)\(^16\)\(^,\)\(^17\) These findings suggest that adipokines play a role in the development of joint degeneration in both the axial and appendicular skeleton.

A gene–environment interaction may also contribute to the increased incidence of disk degeneration in the obese population. An arginine-to-tryptophan change in the COL9A3 gene (Trp 3 allele) has been independently associated with disk degeneration and sciatica.\(^18\)\(^,\)\(^19\) In a cross-sectional study of 135 middle-aged men, Solovieva et al reported a synergistic relationship between obesity and this Trp 3 allele for the development of decreased disk height, posterior disk bulge, and disk signal changes on magnetic resonance imaging.\(^18\) This finding suggests that a genetic predisposition in a portion of the obese population may increase their risk of disk degenerative changes and potential back-related disability.

Obese individuals may experience higher rates of spine pathology secondary to atherosclerotic and lipid-related disk ischemia. The blood supply to the intraosseous portion of the vertebral body is derived from the nutrient and metaphyseal arteries.\(^20\) Each of these has centrifugal branches that terminate at the end plates and arise from the lumbar arteries, which branch directly from the aorta.\(^20\) Having no direct blood supply, the intervertebral disk relies on diffusion from the adjacent vertebral bodies as its only source of nutrition and to expel cellular waste such as lactate. A compromise in this supply of nutrients, oxygen and waste expulsion at any point along the vascular tree reduces cellular viability and proteoglycan production, which leads to disk degeneration.\(^21\) In two postmortem studies, calcification of the abdominal aorta and atherosclerotic stenosis of the lumbar arteries were correlated with an increased risk of disk degeneration and low back pain.\(^22\)\(^,\)\(^23\) Higher plasma levels of apolipoprotein E have also been associated with chronic lumbar pain.\(^24\) As obesity has been linked with both apolipoprotein E and peripheral vascular disease,\(^25\) the ischemia-related disk degeneration produced by these two risk factors may also contribute to the higher incidence of spine-related conditions in this population.

Though the exact contribution each of these factors plays in the development of spine-related pain is not fully understood, the existing evidence suggests a multifactorial problem. Developing a more complete understanding of this phenomenon may improve existing theories on the development of disk degeneration and help identify novel therapies aimed at prevention of disk degeneration and neurologic compression in this population.

**Effects on Postoperative Complications**

Obesity has been associated with longer operative times, increased blood loss, increased treatment cost, higher risk of mortality, and higher rates of both surgical site infection and venous thromboembolism.\(^6\)\(^,\)\(^26\)\(^-\)\(^36\) The complex relationship between increased rates of postoperative complications and obesity is likely at least partially dependent on confounding variables within this population. Obese patients undergoing spine surgery have higher rates of medical comorbidities, such as diabetes mellitus and coronary arterial disease, which serve as independent risk factors for postoperative adverse events.\(^5\)\(^,\)\(^6\)\(^,\)\(^35\)\(^,\)\(^37\) To this end, in a retrospective cohort study of 49,314 patients undergoing elective lumbar procedures, Seicean et al reported higher rates of both complications and readmission in individuals with BMI greater than 30 kg/m\(^2\).\(^38\) However, on matched propensity scoring controlling for medical comorbidities, only individuals with BMI > 40 kg/m\(^2\) demonstrated a significantly higher rate of complications, readmission, or nonroutine discharge.\(^39\) The results of this study suggest that comorbid medical conditions contribute to the increased rate of postoperative complications. However, in the morbidly obese population, obesity alone is an independent risk factor for complications.

Multiple investigators have also documented longer operative times and increased blood loss in obese patients.\(^6\)\(^,\)\(^27\)\(^,\)\(^32\)\(^,\)\(^35\)\(^,\)\(^37\)\(^,\)\(^39\) Each of these factors have been correlated with an increased incidence of postoperative complications.\(^30\)\(^,\)\(^40\) Longer operative times contribute to higher complication rates by increasing the amount of soft tissue ischemia from retraction, exposing sterile instrumentation to a higher likelihood of contamination, and increasing
the total anesthetic and positioning time. Larger volumes of blood loss can lead to both higher rates of postoperative transfusion and hematoma formation, which have both been identified as risk factors for postoperative complications.41–43

The presence of obesity has been correlated with higher rates of postoperative infection in multiple investigations (Table 1).6,26,27,30,35,38,44,45 In a retrospective analysis of the American College of Surgeons National Surgeons Quality Improvement Program (NSQIP) database, Lim et al noted a significantly higher risk of surgical site infection in the obese population (OR: 1.628, 95% CI 1.94 to 2.34).30 However, in a meta-analysis reviewing 24 studies, Jiang et al reported a significant relationship between obesity and surgical site infection (OR: 2.33, 95% CI 1.94 to 2.79).31

Though each of these investigations associated risk of infection with obesity (BMI > 30 kg/m²), Mehta and colleagues suggested that the distribution of adipose tissue plays a significant role in the development of surgical site infection following spine surgery.44,46 To this end, the authors found significant associations between infection and the skin-to-lamina distance as well as the thickness of the subcutaneous tissue as measured on magnetic resonance imaging in cervical and lumbar procedures.44,46 These studies introduce the idea that body habitus, rather than BMI, may be a better predictor of surgical site infection, which may be particularly true in more muscular individuals with relatively high BMIs but little adipose tissue. Understanding this potential limitation in the use of BMI alone may help practicing spine surgeons better understand this elevated risk of postoperative infection.

Another commonly feared complication associated with obesity following spine surgery is venous thromboembolism. In the general population, obesity is an independent risk factor for both deep venous thrombosis and pulmonary embolism.47,48 This strong association is the result of (1) obesity-related chronic inflammation, (2) impairment of normal fibrinolysis, (3) increased thrombin generation and increased platelet activity.49,50 A higher risk of venous thromboembolism has also been noted in obese individuals undergoing elective spine surgery by multiple investigators (Table 2).27,29,31 In a retrospective analysis of 24,196 patients undergoing lumbar spine surgery from the NSQIP database, Marquez-Lara reported significantly higher rates of thromboembolic events in individuals with a BMI greater than 25.29 In a meta-analysis analyzing six studies, Jiang et al noted a significantly higher risk of venous thromboembolic complications in obese individuals undergoing spine surgery (OR: 3.15, 95% CI 1.92 to 5.17).27 The increased incidence of this potentially fatal complication in obese patients should be considered in the formulation of a postoperative anticoagulation strategy, particularly those with mobilization precautions or comorbid conditions that would further predispose them to thromboembolic events.

Obesity is also widely believed to be a risk factor for perioperative peripheral nerve injury.33,51,52 The causes of this phenomenon are likely related to higher compressive forces in contact areas and potentially longer operative times. To date, no large studies validating obesity as a risk for positional nerve injury in patients undergoing spine surgery have been performed. However, in a small retrospective case study.

Table 1  Incidence of infection in the obese population undergoing spine surgery

| First author | Study design | Subjects (n) | Incidence of infection | Comments |
|--------------|--------------|-------------|------------------------|----------|
| De la Garza-Ramos6 | Retrospective cohort study | 732 | BMI < 29.9: 3.78%; BMI > 30: 12.86% (p = 0.001) | Review of one- to three-level lumbar posterolateral fusions |
| Djuracovic45 | Retrospective case series | 270 | BMI < 29.9: 0.6%; BMI > 30: 5.5% (p = 0.018) | Review of lumbar fusions for degenerative causes |
| Higgins35 | Retrospective case series | 801 | BMI < 29.9: 1.5%; BMI 30–40: 4.2% (p = 0.03); BMI > 40: 15.0% (p < 0.001) | Study includes all instrumented cases at a single institution from all regions of the spine |
| Jiang27 | Meta-analysis | 93,183 | OR BMI > 30 kg/m²: 2.33; 95% CI: 1.94–2.79 | Evidence graded as moderate |
| Lim30 | Retrospective multivariate analysis | 3,353 | OR BMI > 30 kg/m²: 1.63; 95% CI: 1.042–2.544 (p = 0.032) | Review of single-level lumbar fusion cases |
| Marquez-Lara29 | Retrospective database review | 24,196 | BMI < 29.9: 0.7%; BMI > 25: 1.3% (p < 0.001) | Relative risk of infection increased as BMI increased (BMI > 40: RR: 3.8, 95% CI: 2.5–5.9; p = 0.001) |
| Mehta44 | Retrospective case series | 298 | BMI < 29.9: 5.1%; BMI > 30: 12.3% (p = 0.025) | Skin-to-lamina distance and the thickness of the subcutaneous tissue were also associated with increased rates of infection |
| Soroceanu26 | Retrospective database review | 241 | OR BMI > 30 kg/m²: 4.88 (p = 0.02) | Review of adult spinal deformity cases |

Abbreviations: BMI, body mass index; CI, confidence interval; OR, odds ratio; RR, relative risk.
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## Table 2 Incidence of venous thromboembolism in the obese population undergoing spine surgery

| First author     | Study design                     | Subjects (n) | Incidence of infection                                                                 | Comments                                                                                      |
|------------------|----------------------------------|--------------|---------------------------------------------------------------------------------------|-----------------------------------------------------------------------------------------------|
| Jiang27          | Meta-analysis                    | 85,085       | OR BMI > 30 kg/m²: 3.15, 95% CI: 1.92–5.17                                             | Evidence graded as moderate                                                                  |
| Kalanithi31      | Retrospective database review     | 84,607       | OR BMI > 40 kg/m²: PE/DVT: 3.34, 95% CI: 2.01–5.54                                       | Review of morbidly obese patients undergoing cervical or lumbar fusion                        |
| Marquez-Lara29   | Retrospective database review     | 24,196       | RR for BMI > 25.0 kg/m²: DVT: 2.0, 95% CI: 1.2–3.5 (p = 0.009); PE: 1.9, 95% CI: 1.0–3.6 (p < 0.001) |Reviewed results after all lumbar spine surgeries                                               |

Abbreviations: CI, confidence interval; DVT, deep vein thrombosis; OR, odds ratio; PE, pulmonary embolism; RR, relative risk.

### Series, Patel et al noted nerve compression injuries only in individuals with morbid obesity (BMI > 40 kg/m²).33 Though the small sample size combined with the low number of nerve injuries observed in this study (n = 2) prevent drawing definitive conclusions, extra care should be dedicated to the positioning of obese patients prior to spine surgery.

### Effects on Functional Outcomes

Obesity’s influence on clinical outcomes is controversial. In a retrospective review of 271 patients undergoing lumbar fusion 2 years after surgery, Djurasovic et al reported no significant difference in mean improvement seen between obese and nonobese patients in respect to Short Form-36 (SF-36) physical composite summary and Oswetry Disability Index (ODI) (ΔSF-36 physical composite summary: 4.22 versus 6.17, p = 0.147; ΔODI: 15.35 versus 14.03, p = 0.602).45 Similarly, in a comparison of surgical outcomes in the obese and nonobese populations within the Spine Patient Outcomes Research Trial (SPORT) data, Rihn et al noted no significant difference in outcome measures at 4 years in both lumbar stenosis and degenerative spondylolisthesis, with the exception of SF-36 physical function scores in the degenerative spondylolisthesis population (22.1 versus 27.9, p = 0.022).28 To the contrary, in a review of 2,633 patients from the Swedish Spine Registry, Knutsson et al noted that obese patients (BMI > 30 kg/m²) had significantly worse ODI and EuroQol Group Index (EQ-5D) scores than normal weight patients (BMI < 25 kg/m²; EQ-5D 0.56 versus 0.64, p < 0.001; ODI 33 versus 25, p < 0.001) and less mean improvement 2 years after surgery.51 In this study, the obese population also experienced significantly greater risk for dissatisfaction with the results of surgery (OR: 1.73, 95% CI: 1.36 to 2.19).51 In a review of patients undergoing surgery for lumbar disk herniation from the SPORT data, Rihn et al noted significantly less improvement in ODI and all components of the SF-36 scores as well as the Sciatica Brothersomeness and Low Back Pain Brothersomeness Indices in the obese population.54

Although these investigations provide contradictory information on whether obese patients experience less improvement with surgery, they should be interpreted carefully as obesity has been associated with worse baseline functional scores and less improvement with nonoperative treatment.26,28,37,45,53,54 Differences in baseline functional measures were demonstrated in the investigation by Knutsson et al, where obese individuals had worse preoperative EQ-5D and ODI scores than the normal weight population (EQ-5D: 0.34 versus 0.38, p = 0.0026; ODI: 46 versus 42, p < 0.0001).53 Djurasovic et al also noted higher (worse) pretreatment ODI scores in the obese population (ODI: 55.83 versus 51.42, p = 0.017).45 The obese population also seems to receive less benefit from nonoperative treatment. In multiple examinations of the SPORT data, obese individuals with lumbar disk herniation, lumbar stenosis, or degenerative spondylolisthesis have demonstrated less improvement in functional outcome measures at up to 4-year follow-up.28,37,54 These differences in both baseline disability and response to nonoperative treatment limit the value of directly comparing long-term functional outcome measures in the obese and nonobese populations to determine the efficacy of surgery.

In this context, it is more important to consider the treatment effect of operative intervention as a measure of therapeutic success. The treatment effect is defined as the difference between mean improvement in a surgical population compared with mean improvement associated with nonoperative treatment in a similar population. When considering this measure of success, obese patients have similar or better responses to operative intervention than their nonobese counterparts.28,37,54 In examining this metric, the SPORT data likely provides the most complete insight due to the fact it represents a large prospective data set collected at multiple institutions (Tables 3, 4, and 5). In a subgroup analysis of these results, Rihn et al reported equivalent surgical treatment effects in the obese and nonobese populations for lumbar disk herniation using SF-36 and ODI scores.54 However, obese individuals had greater treatment effects with surgery in both the lumbar stenosis (SF-36 physical profile) and degenerative spondylolisthesis (ODI) populations.28 The authors attributed these differences in treatment effect to the worse outcomes observed with nonoperative management in the obese group. Though controversy may persist on whether obese patients experience the same magnitude of functional improvement after surgery, the greater treatment effects noted indicate that surgery,
compared with nonoperative treatment, is at least as advantageous in this group of patients.

**Conclusion**

With important differences in postoperative complication rates and response to both operative and nonoperative treatment, obese individuals represent a unique patient population. As the global incidence of obesity continues to rise, obese patients will likely constitute a larger portion of the spine surgeon’s practice. Though the current literature points to increased rates of postoperative complications, particularly infection and venous thromboembolic events, there is also a suggestion of potentially greater treatment effects with surgical intervention owing largely to worse outcomes with nonoperative treatment. Future research in this field should examine measures to minimize the complication rates in this population as well as the effect of controlled weight loss before surgery on complications and outcomes.

**Disclosures**

Keith L. Jackson II: none
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**Disclaimer**

The views expressed herein are those of the author(s) and do not reflect the official policy of the Department of the Army, Department of Defense, or the U.S. Government.

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