

## Research Article

## Open Access

## Relationship between Grade Ii, Iii and IV Obesity, Disc Degeneration and Chronic Low Back Pain under Minimally Invasive Lumbar Surgery

Arrotegui I

Senior Consultant in Neurosurgery, Hospital General Universitario de Valencia. Dept. of Neurosurgery

**\*Corresponding author**

Arrotegui I., M.D, Ph. D, Senior Consultant in Neurosurgery, Hospital General Universitario de Valencia. Dept. of Neurosurgery. Avda Tres Cruces S/n .46014 – Valencia-Spain E-mail: athbio@yahoo.es

**Received:** October 24, 2019; **Accepted:** October 28, 2019, **Published:** October 31, 2019**Introduction**

Obesity (BMI>30Kg/m<sup>2</sup>) is a pandemic with severe medical and financial implications. There is growing evidence that relates certain metabolic processes within the adipose tissue, preferentially abdominal fat, with a low-intensity chronic inflammatory state mediated by adipokines and other substances that favor disk disease and chronic low back pain. Obesity greatly conditions both the preoperative evaluation and the spinal surgical technique itself. Some meta-analyses have confirmed an increase of complications following lumbar spine surgery (mainly infections and venous thrombosis) in obese subjects. However, functional outcomes after lumbar spine surgery are favorable although inferior to the non-obese population, acknowledging that obese patients present with worse baseline function levels and the prognosis of conservatively treated obese cohorts is much worse. The impact of preoperative weight loss in spine surgery has not been prospectively studied in these patients. It is known that overweight / obesity is a factor of demonstrated risk for many diseases metabolic, cardiovascular, and visceral and osteoarticular [1]. There is also a growing bibliographic evidence that relates obesity to an acceleration of the degeneration process lumbar disc and is with the presence of low back pain chronic [2-3]. The specific pathophysiological mechanisms that they relate the metabolic alteration of fatty tissue with the intervertebral disc degeneration are still in process of study [4]. Among them, certain cellular processes stand out induced by biochemical mediators (adipokines and others) that favor the development and maintenance of a state chronic low-intensity inflammatory that seems to contribute to the disc lesion in the obese patient [5]. Longitudinal studies and meta-analysis have found that the obese population experiences a complication rate, related to lumbar spine surgery, significantly higher than that of the overweight population, mainly in relation to infections of surgical wound and the incidence of venous thrombosis deep [6]. Also, the interventions that are performed in patients with overweight / obesity present difficulties specific ones related to the surgical technique itself, in general, due to the need for broader approaches, for the longest surgical time employed and longer blood loss or worse healing of the wounds [7]. However, the functional results are medium and long term of obese patients

intervened for lumbar spine lesions are comparable to those of the subjects with normal weight [8].

**Material**

We included E.D.D patients undergoing surgical management with lumbar instrumentation, they were divided into two groups according to their body mass index in obese (MISS) and obese (standard). Clinical variables were evaluated (such as return to work, hospital stay and postoperative pain) and variables measured intraoperatively (surgery time and loss of surgery). Intraoperative blood)

**Results**

Patients treated with minimally invasive surgery techniques obtained better results compared with open surgery techniques and conservative methods, with a shorter hospital stay and faster recovery of their work and social life. As for the other variables studied (especially the radiological ones, such as the height of the vertebral body or the kyphotic angle), there are no significant differences between the different therapeutic techniques.

**Figure 1**

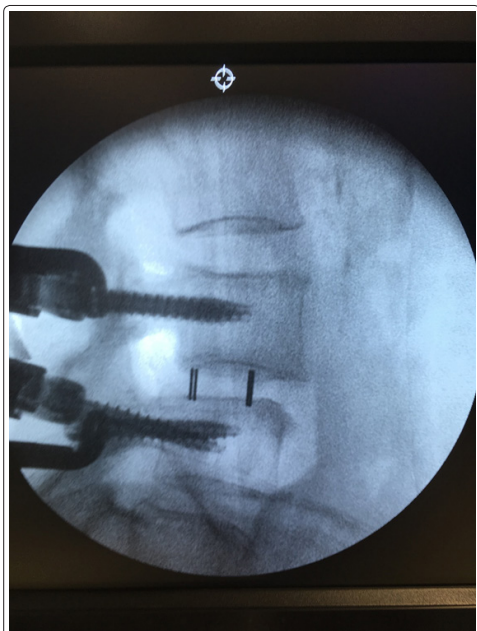


Figure 2

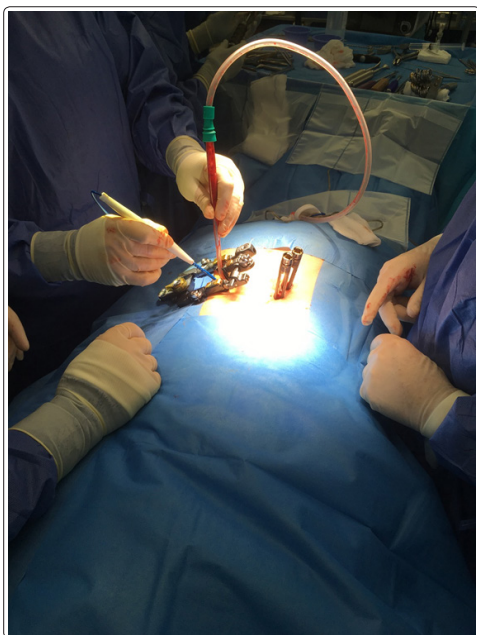


Figure 3

Obese patients: BMI > 30% with the MIS technique presented a more favorable clinical evolution compared to the group of obese patients with Standard surgery. After this study, it is the technique of choice in our unit.

### Discussion

Currently, obesity is defined based on BMI, calculable through a simple formula that relates the weight of individual in kilograms with its height in meters raised to square ( $BMI = Kg / m^2$ ) [1]. This concept became popular from of the 70s following the publication of Keys' work et al. (republished in 2014) [2]. In which various methods of measuring the relative weight of individuals at the level Population In reality, these authors recovered the concept BMI initially described by the Belgian statistician of 19th century Adolphe Quetelet, long forgotten [3]. Although there are other methods of estimating obesity such as the abdominal perimeter or waist-to-hip ratio, the BMI presents the enormous advantage of its simplicity, objectivity and reproducibility. It is known that

low back pain affects 80% of the population sometime in life [4]. Various studies and meta-analysis prove the association between disc disease and low back pain Chronic [5]. According to several authors, the development of the lesion Disc can be attributed to genetic, environmental conditions and lifestyle [6]. Of these, a very important one is overweight / obesity or, more generically, the amount of body fat and its anatomical distribution [7]. In the process of disc degeneration a degradation is verified enzymatic proteoglycans that make up the nucleus pulposus, while disorganization and fibrosis of the collagen sheets that form the fibrous ring. Likewise, these disc changes are accompanied by edema subchondral bone of adjacent vertebral plates that, from a radiological point of view, it translates into the so-called Modic [8]. Changes.

The degree of disc degeneration it has been classically evaluated according to radiological criteria (resonance) of Pfirrmann [9]. And, previously, of Schneider man [10]. It is known that the signal changes in the disk evaluated using these classifications do not have a direct correlation with degree of low back pain and, in fact, many asymptomatic subjects present them. But nevertheless, recent population studies confirm an association statistical and increasing between the degree of disc degeneration and that of chronic low back pain [11]. On the other hand, the association between body weight and degeneration disc has not been studied prospectively in well-designed epidemiological studies, so this The relationship is not established quantitatively [12]. Without However, since there is an association between obesity and pain chronic lumbar, and in turn, disc degeneration is a cause known from low back pain, it is reasonable to think that a BMI elevated be a causal agent of disc degeneration, which Once it favors the presence of low back pain. Adipose tissue is a specialized loose connective tissue composed of adipocytes, blood vessels, fibroblasts and macrophages, which serves as an energetic lipid reservoir and thermal insulator, and whose body distribution is determined for sex hormones.

In addition, fatty tissue is a very important endocrine and paracrine organ that produces hormones and various proteins involved in development of several obesity related diseases such as they are type 2 diabetes or cardiovascular pathology. As weight is gained, the proportion of macrophages that infiltrate the fatty tissue and, as a result of the biochemical interaction between adipocytes and macrophages, cascades of production of proinflammatory cytokines are activated called adipokines [13]. Currently they know more of 50 adipokines, among which the most studied are leptin, adiponectin, resistin and the so-called RBP-45 These substances have multiple metabolic effects, inflammatory and on the immune system. Specific, leptin seems to be involved in the reorganization of cytoskeleton of the nucleus pulposus cells and in the alteration structural of the disc [14]. Thus, the presence of receptors for leptin in disks has been related to development of disc degeneration [15].

It also seems that the risk of Disc degeneration is higher in obese men than to women, probably because of the type of fat distribution of the male, predominantly trunk and per abdominal [7]. Would this preferential accumulation of fat in the abdominal area which would cause an increase in adipokine secretion proinflammatory and metabolic mediators that produce chronic inflammatory disc changes the intervertebral disc has an avascular structure relatively large that is very sensitive to ischemia. Since the disc is nourished indirectly by diffusion from the terminal vascularization that reaches the vertebral plate, any vascular defect (atherosclerotic or other etiology) that affects this, favors disc ischemia. Disc injury



Ischemic eventuality occurs due to a decrease in the synthesis of proteoglycans of the nucleus pulposus and by the accumulation of products such as lactate or so-called (Advanced Glycation End Products (AGE), among others. The causes of pain in patients with degenerated discs They are not fully elucidated, it is plausible that the cytokines pro-inflammatory secreted by macrophages favor degradation of the extracellular matrix of the disc and affect to the growth of nerve endings, which seems contribute to the generation and maintenance of pain [12].

Finally, certain mutations of various genes with disc involvement in the obese population. Significant with the presence of said pain. At the moment, most of these associations have been able to determine with a moderate level of evidence or weak. A detailed description of these alterations goes beyond the objective of this work, so we refer to reader to the systematic review of Eskola, et al. [6]. Obesity has been associated with longer surgical times, greater intraoperative blood loss, and more infections of surgical wound, higher incidence of thromboembolism postoperative, greater probability of peripheral nerve injury, higher overall mortality and worse healing of the surgical wound. The published works in which it is addressed this issue is all retrospective case series [16]. Or meta-analysis of these series [17]. It is true that the comorbidity present in patients obese is partly responsible for this increase in complications and acts as a confounding factor (or as a cause intermediate) in this subpopulation. Obese patients are more prone to type 2 diabetes and coronary heart disease, which in turn they are independent factors of postoperative morbidity. However, in the studies in which it is controlled due to medical comorbidities, only patients with obesity Type III (BMI > 40) seems to present a high and significant risk of complications and readmissions cannot be explained by comorbidity per se. In the Seicean retrospective study et al. on 49,314 patients undergoing spinal surgery are concluded that comorbidity contributes to the increased risk of postoperative complications and that morbid obesity is an independent risk factor for these complications [18]. Also, the number of comorbidities of a patient specifically it also correlates with the complication rate postoperative. According to Benotti et al. [19].

The prevalence of multiple comorbidities and the total number of them correlate strongly with an advanced age, an older BMI and male sex. An increase in surgical time means more time anesthetic and may be associated with a higher complication rate due to tissue ischemia that involves maintaining soft tissue separators or retractors during times more prolonged. In addition, it can increase the probability of contamination of the surgical field as it increases surgical exposure time. A surgical field of greater size it can suppose an increase of the blood loss and increased chance of hematoma of surgical bed and need for transfusion. According to the systematic review of Jackson et al. [20]. That compiles studies until 2015, the rate of surgical wound infections in the obese (BMI > 30) at least double and often triple, the non-obese rate. This data has been corroborated. In the meta-analysis of Jiang et al where 93,183 are collected subjects and a global infection risk of 2.33 is reported times higher in the obese (95% CI: 1.97-2.79) [21]. This same study confirmed the relationship between obesity and incidence of thromboembolism.

The study by Márquez-Lara et al on 24,196 patients, confirmed an increase in infections as the degree of obesity increases (1.3% in BMI > 25 compared to a 0.7% in BMI < 25, with a significant relative risk of 3.8 to from BMI > 40). It also found a risk of thrombosis venous of individuals with BMI > 25 that doubles that of individuals with BMI < 25 and a relative risk for embolism 1.9

lung statistically significant. Its interesting review the work of Mehta, et al. retrospective series on 298 patients, where a double infection rate is confirmed in obese people with BMI > 30 [22].

This same author relates skin-sheet distance and thickness of subcutaneous tissue with an increased rate of infections [23]. Obesity also seems to predispose to nerve injury peripheral related to the position on the surgical table, possibly by direct compression of the tissues and times elongated surgical. Although there are no studies that specifically address this complication, they have communicated some cases that occur, in general, in patients with morbid obesity. Finally, obesity is associated with complicated healing [24]. Of the surgical wound and more prolonged in time. In the interesting review published by Pierpont et al. they propose various causal mechanisms: anatomical issues of fatty tissue, vascular insufficiency of the adipose lobe and relative hypoxia, oxidative stress, maintenance of a chronic inflammatory state of low intensity, alterations in immunological mediators, nutritional deficiencies and, perhaps, certain genetic alterations present in patients obese. It is likely that a reduction in weight that brings the obese towards overweight or normal weight should result in postoperative clinical outcomes comparable to those of said population groups [25].

The recent Meta-analysis by Zhang et al. confirms that overweight and Obesity are risk factors for the appearance of low back pain and suggests that maintaining a correct weight (what which they call healthy body weight) can be one of the factors that prevent such pain [26].

On the other hand, a higher recurrence rate has been reported. Discs after lumbar spine surgery in obese (about 10%), attributed to an axial repetitive load on the operated disc. However, there are no studies that evaluate the effect of a weight reduction on the incidence recurrence Disc.

Finally, obese patients undergoing surgery minimally invasive lumbar seem to get results functional comparable to non-obese patients with complication rates and similar hospital stays [27]. The results from the study of Goldin and Alander corroborate these findings, so minimally invasive surgery not only does not it is contraindicated in obese but it can be a good alternative. Conclusions Obesity is a medical and social problem with implications Huge economic. In many industrialized countries its prevalence is close to one third of the population. Is known that obesity is related and is a causative agent from a multitude of metabolic, cardiovascular diseases, visceral, oncological and osteoarticular [28].

There is evidence solid epidemiology that associates obesity and disease degenerative spinal with chronic low back pain. However, the association between obesity and disease Lumbar disc is worse established. At present, said ratio is attributed not only to chronic axial disc overload but to a complex metabolic interaction between adipocytes and macrophages of adipose tissue (especially fatty tissue of central-abdominal location) that trigger a cascade of proinflammatory mediators that damage the intervertebral disc. Other mechanisms involved in disc degeneration are the relative ischemia of the disc and genetic determinants, currently under study.

## References

1. (1998) Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults—The evidence report National Institutes of Health. 2: 51S-209S.

2. Keys A, Fidanza F, Karvonen MJ, Kimura N, Taylor HL (2014) Indices of relative weight and obesity. *Int J Epidemiol* 43: 655-65.
3. Ogden CL, Carroll MD, Kit BK, Flegal KM (2014) Prevalence of childhood and adult obesity in the United States, 2011-2012. *JAMA* 311: 806-14.
4. Vos T, Flaxman AD, Naghavi M, Lozano R, Michaud C et al. (2012) ( Years lived with disability (YLDs) for 1160 sequelae of 289 diseases and injuries 1990-2010: A systematic analysis for the Global Burden of Disease Study 2010 380: 2163-2196.
5. Xu X, Li X, Wu W (2015) Association between overweight or obesity and lumbar disk diseases: A meta-analysis. *J Spinal Disord Tech* 28: 370-376.
6. Eskola PJ, Lemmelä S, Kjaer P, Solovieva S, Männikkö M et al. (2012) Genetic association studies in lumbar disc degeneration: A systematic review. *PLoS One* 7: e49995.
7. Takatalo J, Karppinen J, Taimela S, Niinimäki J, Laitinen J et al. (2013) Association of abdominal obesity with lumbar disc degeneration—a magnetic resonance imaging study. *PLoS One* 8: e56-244.
8. Wang Y, Videman T, Battié MC (2012) ISSLS prize winner: Lumbar vertebral endplate lesions: Associations with disc degeneration and back pain history. *Spine (Phila Pa 1976)* 37: 1490-1496.
9. Pfirrmann CW, Metzdorf A, Zanetti M, Hodler J, Boos N (2001) Magnetic resonance classification of lumbar intervertebral disc degeneration. *Spine (Phila Pa 1976)* 26: 1873-1878.
10. Schneiderman G, Flannigan B, Kingston S, Thomas J, Dillin WH, et al. (1987) Magnetic resonance imaging in the diagnosis of disc degeneration: Correlation with discography. *Spine (Phila Pa 1976)*. 12: 276-281.
11. Heymsfield SB, Wadden TA (2017) Mechanisms, pathophysiology, and management of obesity. *N Engl J Med*. 376: 254-66.
12. Samartzis D, Karppinen J, Cheung JP, Lotz J (2013) Disk degeneration and low back pain: Are they fat-related conditions? *Global Spine J* 3: 133-144.
13. Balistreri CR, Caruso C, Candore G (2010) the role of adipose tissue and adipokines in obesity-related inflammatory diseases. *Mediators Inflamm* 2010: 802078.
14. Li ZI, Shen J, Wu WK, Yu X, Liang J et al. (2013) The role of leptin on the organization and expression of cytoskeleton elements in nucleus pulposus cells. *J Orthop Res* 31:847-857.
15. Zhao CQ, Liu D, Li H, Jiang LS, Dai LY (2008) Expression of leptin and its functional receptor on disc cells: Contribution to cell proliferation. *Spine (Phila Pa 1976)* 33: 858-864.
16. Marquez-Lara A, Nandyala SV, Sankaranarayanan S, Noureldin M, Singh K (2014) Body mass index as a predictor of complications and mortality after lumbar spine surgery. *Spine (Phila Pa 1976)*. 39: 798-804.
17. Jackson KL 2nd, Devine JG (2016) The effects of obesity on spine surgery: A systematic review of the literature. *Global Spine J*. 24: 394-400.
18. Seicean A, Alan N, Seicean S, Worwag M, Neuhauser D et al. (2014) Impact of increased body mass index on outcomes of elective spinal surgery. *Spine (Phila Pa 1976)* 39: 1520-1530.
19. Benotti P, Wood GC, Still C, Petrick A, Strodel W (2006) Obesity disease burden and surgical risk. *Surg Obes Relat Dis* 2: 600-606.
20. Jackson KL 2nd, Devine JG (2016) The effects of obesity on spine surgery: A systematic review of the literature. *Global Spine J* 24: 394-400.
21. Teng Y, Fan Z, Khan S, Xia Y (2013) Does obesity affect the surgical outcome and complication rates of spinal surgery? A meta-analysis. *Clin Orthop Relat Res*. 472: 968-975.
22. Marquez-Lara A, Nandyala SV, Sankaranarayanan S, Noureldin M, Singh K (2014) Body mass index as a predictor of complications and mortality after lumbar spine surgery. *Spine (Phila Pa 1976)*. 39: 798-804.
23. Mehta AI, Babu R, Karikari IO, Grunch B, Agarwal VJ et al. (2012) 2012 Young Investigator Award winner: The distribution of body mass as a significant risk factor for lumbar spinal fusion postoperative infections. *Spine (Phila Pa 1976)* 37: 1652-1666.
24. Patel N, Bagan B, Vadera S, Maltenfort MG, Deutsch H et al. (2007) Obesity and spine surgery: Relation to perioperative complications. *J Neurosurg Spine* 6: 291-297.
25. Pierpont YN, Dinh TP, Salas RE, Johnson EL, Wright TG et al. (2014) Obesity and surgical wound healing: A current review. *ISRN Obes* 2014: 638936.
26. Zhang TT, Liu Z, Liu YL, Zhao JJ, Liu DW, Tian QB (2018) Obesity as a risk factor for low back pain: A meta-analysis. *Clin Spine Surg* 31: 22-27.
27. Rosen DS, Ferguson SD, Ogden AT, Huo D, Fessler RG (2008) Obesity and self-reported outcome after minimally invasive lumbar spinal fusion surgery. *Neurosurgery* 63:956-960.
28. Goldin AN, Alander DH (2015) Effect of body mass index on early outcomes of minimally invasive degenerative lumbar surgery. *J Surg Orthop Adv*. Spring 24: 12-17.

**Copyright:** ©2019 Arrotegui I. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.