Obesity in neurosurgery

- Association between body mass index and intracranial procedural complications in patients undergoing endovascular treatment for unruptured aneurysms
- Risk factors for surgical site infection in patients after hysterectomy: A systematic review and meta-analysis
- Weight change patterns following surgery for cervical spondylosis in overweight and obese individuals: a nationwide longitudinal study
- Affordable Irrigation-Drainage System for Early Postoperative Adhesion and Surgical Site Infection Prophylaxis in Posterior Lumbar Surgery: A Technical Note
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- A Retrospective Study of Lumbar Disk Herniation: An Analysis of Clinical Cases and Treatment Plans
- Therapeutic Potential of Infrared and Related Light Therapies in Metabolic Diseases
- Current Concepts on Imaging and Artificial Intelligence of Osteosarcopenia in the Aging Spine - A Review for Spinal Surgeons by the SRS Adult Spinal Deformity Task Force on Senescence

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Concern exists for increased complications due to surgical challenges posed by obese patients and their often-prevalent comorbidities.

Compared with nonobese children with epilepsy, obese children with epilepsy had a higher intraoperative blood loss. It is necessary to conduct early weight management of children with epilepsy as long as possible [1)]

Obesity is a major risk factor globally and it is associated with an increased risk of severe vision loss due to idiopathic intracranial hypertension (IIH). There has been an increase in obesity prevalence in the Middle East countries mainly affecting the Gulf Council Countries (GCC), which parallels increased industrial development. This rise may be contributing to the increasing incidence of IIH in these countries. Other risk factors may also be contributing to IIH in Middle East countries and the differences and similarities to Western IIH merit further study [2)].

A study aimed to investigate the relationship between obesity and mortality in patients with community-acquired pneumonia (CAP) in China.

In total, 909 patients with CAP were recruited for this study from January 2010 to June 2015. All patients were selected and divided into 4 groups according to their body mass index (BMI) values. All patients' clinical information was recorded. The associations among mortality; BMI; the 30-day, 6-month and 1-year survival rates for different BMI classes; the etiology of pneumonia in each BMI group; and the risk factors for 1-year mortality in CAP patients were analyzed.

With the exception of the level of C-reactive protein (CRP), no other clinical indexes showed significant differences among the different BMI groups. No significant differences were observed among all groups in terms of the 30-d and 6-month mortality rates ($p>0.05$). There was a significantly lower risk of 1-year mortality in the obese group than in the nonobese group, ($p<0.05$). Logistic regression analysis showed that there were seven independent risk factors for 1-year mortality in CAP patients, namely, age, cardiovascular disease, cerebrovascular disease, obesity, APACHE II score, level of CRP and CAP severity.

Compared with nonobese patients with CAP, obese CAP patients may have a lower mortality rate, especially with regard to 1-year mortality, and CRP may be associated with the lower mortality rate in obese individuals than in nonobese individuals [3)].

Li et al., from Beijing aimed to examine the relationship between metabolically healthy obese (MHO) and risk of cardiovascular diseases (CVD) among the Chinese population.

The China Health and Retirement Longitudinal Study is a prospective cohort study of 7849 participants aged ≥45 years without CVD at baseline. Metabolic health status was assessed based on blood pressure, triglycerides, high-density lipoprotein cholesterol, glycated hemoglobin, fasting glucose, and C reactive protein. A cutoff point of body mass index of 24.0 kg/m2 was used to define over-weight/obesity (≥24.0 kg/m2) or normal weight (<24.0 kg/m2). CVD was based on self-reported doctor's diagnosis of heart problems and stroke. Incidence rate ratio (IRR) with 95% confidence interval (CI) was deduced from modified Poisson regression.

During a mean 3.6 years of follow-up, 880 incident CVD events were recorded. 789 (10.05%) were identified MHO among 3321 (42.3%) obese individuals. Compared with metabolically healthy normal weight individuals, the multivariable adjusted IRR of CVD was 1.33 (95%CI: 1.19-1.49) for MHO, 1.29 (95%CI: 1.22-1.38) for metabolically unhealthy normal weight, and 1.61 (95%CI: 1.51-1.75) for metabolically unhealthy obese in the full adjusted model.

MHO individuals are associated with the increased risk of cardiovascular diseases among the Chinese population [4)].

Obesity in aneurysmal subarachnoid hemorrhage

see Obesity in aneurysmal subarachnoid hemorrhage.

Obesity in spinal surgery

see Obesity in spinal surgery.

Obesity in traumatic brain injury

see Obesity in traumatic brain injury

Deep brain stimulation for obesity

Sixteen Sprague-Dawley rats were maintained on a high-fat diet. Daily food intake and weight gain were measured for 7 days, at which time the animals underwent stereotactic placement of 0.25-mm-diameter bipolar stimulating electrodes bilaterally in the LH. On postoperative Day 7, eight animals began to receive continuous stimulation of the LH. The remaining eight animals were left unstimulated as the control group. Individual animal weight, food intake, and water intake were monitored daily and continuously throughout the experiment until postoperative Day 24.

There was a decreased rate of weight gain after surgery in all animals, but the unstimulated group recovered and resumed a linear weight gain curve. The stimulated group, however, failed to show weight gain and remained below the mean baseline for body mass. There was a significant weight loss between the stimulated and unstimulated groups. On postoperative Day 24, compared with the day of surgery (Day 0), the unstimulated group had a mean weight gain of 13.8%, whereas the stimulated group had a 2.3% weight loss on average ($p = 0.001$), yielding a 16.1% weight difference between the two groups [5)].

The lateral hypothalamus and ventromedial hypothalamus are the appetite and satiety centers in the brain, respectively. Substantial data support targeting these regions with DBS for the purpose of appetite suppression and weight loss. However, reward sensation associated with highly caloric food has been implicated in overconsumption as well as obesity, and may in part explain the failure rates of conservative management and bariatric surgery. Thus, regions of the brain's reward circuitry, such as the nucleus accumbens, are promising alternatives for DBS in obesity control [6)].

Several studies have shown involvement of the nucleus accumbens in these and other addictive behaviors. In a case report, a patient who quit smoking and lost weight without any effort.

A 47-year-old woman presented with chronic treatment-refractory obsessive-compulsive disorder, nicotine dependence, and obesity.

The patient was treated with deep brain stimulation of the nucleus accumbens for obsessive-compulsive disorder. Unintended, effortless, and simultaneous smoking cessation and weight loss were observed.

This study supports the idea of compulsivity with common circuitry in the processing of diverse rewards and suggests that deep brain stimulation of the nucleus accumbens could be a possible treatment of patients with a dependency not responding to currently available treatments [7].

Deep brain stimulation must achieve a success rate of 83% to be equivalent to bariatric surgery. This high-threshold success rate is probably due to the reported success rate of LRYGB, despite its higher complication rate (33.4%) compared with DBS (19.4%). The results support further research into the role of DBS for the treatment of obesity [8].

Appetite modulation in conjunction with enhancing metabolic rate with hypothalamic lesions has been widely documented in animal and even in humans. It appears these effects can be reproduced by DBS, and the titratability and reversibility of this procedure, in addition to well established safety profile, make DBS an appealing option for obesity treatment. Targeting the hypothalamus with DBS has already been shown to be feasible and potentially effective in managing patients with intractable chronic cluster headache. The surgical risk however must be cautiously taken into account when targeting the hypothalamus, where some mortality cases have been reported when targeting the posterior part. The development of new surgical approach will probably reduce this surgical risk. Moreover, the role of functional neurosurgery in obesity is not a new idea. In fact, LH was targeted in obese humans with electrocoagulation more than 30 years ago, resulting in significant yet transient appetite suppression and slight weight reduction. All those elements have made possible the recent regain of interest in DBS for morbid obesity and open an exciting new area of research in neurosurgery and endocrinology [9].

Ho et al. present a review of the evidence of the neuroanatomical basis for obesity, the potential neural targets for deep brain stimulation (DBS), as well as a rationale for DBS and future trial design. Identification of an appropriate patient population that would most likely benefit from this type of therapy is essential. There are also significant cost and ethical considerations for such a neuromodulatory intervention designed to alter maladaptive behavior. Finally, the authors present a consolidated set of inclusion criteria and study end points that should serve as the basis for any trial of DBS for obesity [10].

Dupré et al. review the history of deep brain stimulation (DBS) in patients for treating obesity, describe current DBS targets in the brain, and discuss potential DBS targets and nontraditional stimulation parameters that may improve the effectiveness of DBS for ameliorating obesity. Deep brain stimulation for treating obesity has been performed both in animals and in humans with intriguing preliminary results. The brain is an attractive target for addressing obesity because modulating brain activity may permit influencing both sides of the energy equation-caloric intake and energy expenditure [11].

Tumor

Using a cross-sectional design, young adults (18-39 years) previously treated for pediatric central nervous system tumors and followed in a survivorship clinic during 2016-2021 were examined. Demographic, BMI, and diagnosis information were extracted from medical records of the most recent clinic visit. Data were assessed using a two-sample t-test, Fisher's exact test, and multivariable logistical regression. 198 survivors (53% female, 84.3% White) with a BMI status of underweight (4.0%), healthy weight (40.9%), overweight (26.8%), obesity (20.2%), and severe obesity (8.1%) were examined. Male sex (OR, 2.414; 95% CI, 1.321 to 4.414), older age at follow-up (OR, 1.103; 95% CI, 1.037 to 1.173), and craniopharyngioma diagnosis (OR, 5.764; 95% CI, 1.197 to 27.751) were identified as significant ($p < 0.05$) obesity-related (≥25.0 kg/m2) risk factors. The majority of patients were overweight or obese. As such, universal screening efforts with more precise determinants of body composition than BMI, risk stratification, and targeted lifestyle interventions are warranted during survivorship care [12)].

Findings highlight obesity as a risk factor for overall brain/CNS tumors, meningiomas and gliomas among females, as well as for meningiomas among males [13)].

For Niedermaier et al adiposity is related to enhanced risk for meningioma but is unassociated with risk for glioma. Based on a limited body of evidence, physical activity is related to decreased risk of meningioma but shows little association with risk of glioma [14)].

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