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Review

Central blood pressure in morbid obesity and after bariatric surgery☆

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ABSTRACT

Various mechanisms are related to arterial hypertension in obesity. Central blood pressure (BP) seems to correlate more than peripheral BP with future cardiovascular risk. Bariatric surgery is an effective method to reduce BP along with weight loss in patients with morbid obesity.

The study of the relationship between weight modification after bariatric surgery and ambulatory BP measurement, not only peripheral BP, but also central BP, could provide information regarding the mechanisms of organic damage associated with elevated BP in obesity.

In this review we analyze the available evidence regarding the association between central BP with obesity and its modifications after bariatric surgery.

Presión arterial central en la obesidad mórbida y tras la cirugía bariátrica

Palabras clave:
Presión arterial central
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RESUMEN

La obesidad está asociada con la hipertensión arterial por mecanismos diversos. La presión arterial (PA) central parece estar más correlacionada que la PA periférica con el riesgo cardiovascular futuro. La cirugía bariátrica constituye un método eficaz para disminuir la PA paralelamente a una pérdida de peso significativa en pacientes con obesidad severa. El estudio de la relación entre la modificación de peso tras cirugía bariátrica y la medición ambulatoria de PA, no solo a nivel periférico, sino también a nivel central, podría aportar información respecto a los mecanismos del daño orgánico asociado a la PA elevada en la obesidad.

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En esta revisión analizamos la evidencia disponible respecto a la asociación entre la PA central con la obesidad y sus modificaciones tras la cirugía bariátrica.

Peripheral blood pressure (BP) is an important independent predictor of cardiovascular risk; rBP reduction reduces the risk of major vascular events, particularly stroke.[1] However, recent evidence suggests that aortic (central) BP has a greater association with future cardiovascular events than peripheral BP. This is because the heart, kidneys, and cerebral vasculature are more exposed to aortic pressure than to brachial pressure.[1–3] Thus, central BP appears to be more closely related to cardiovascular risk markers such as intima–media thickness in carotid and left ventricular mass.[4]

Systolic BP varies throughout the vascular tree; the central systolic BP may be up to 40 mmHg lower than peripheral systolic BP; this difference is variable between individuals, while diastolic BP remains relatively constant.[2,5] A drawback for the measurement of central BP was that the measurement methods were invasive, requiring the use of a catheter not applicable in routine clinical practice. However, today we have several non-invasive methods to indirectly measure central BP, including tonometry using arterial flattening[6] which obtain pulse waves that are almost identical to those obtained by intra-arterial catheterization.[7] This technique is applicable in radial, carotid or femoral artery.[8] Our group received a grant from the Spanish Society of Nephrology for Nephrology Research in 2014 to evaluate, among other things, hemodynamic changes in relation to obesity and bariatric surgery.

Obesity and hypertension

Obesity is a chronic multifactorial disease that has become a worldwide epidemic. The World Health Organization (WHO) defines obesity if the body mass index (BMI) is $\geq$30 kg/m^2, and morbid obesity if the BMI is $\geq$40 kg/m^2. According to the WHO report in 2008 the prevalence of obesity in adults had reached 35%.[10,11] Furthermore, 2.3% of men and 5% of women have morbid obesity. If this trend continues, it is calculated that in 2025 the global prevalence of obesity will reach 18% in men and 21% in women, while morbid obesity will reach 6% in men and 9% in women.[11]

It has been shown that obesity is associated with type 2 diabetes mellitus, dyslipidemia, osteoarthritis, sleep apnea–hypopnea syndrome, some types of cancer and high blood pressure (HTN)[12]; in addition there is higher frequency of appearance of subclinical target organ injury in patients with HTN. This increase in subclinical injury suggests that obesity is a relevant cause of cardiovascular morbidity and mortality, that is observed at earlier ages, and in addition it decreases life expectancy between 4 and 10 years.[13,14] Globally, 3 million deaths per year have been attributed to obesity,[15] so it is essential to develop treatment strategies to reduce, among other risks, the hypertension associated with obesity and its possible complications, as well as to effectively control blood pressure in obese patients.[16]

The prevalence of HTN in obese patients is high, 40%, compared to the population with normal weight, in which HTN occurs is approximately 15%.[17] Some studies have reported that up to 80% of hypertensive patients are overweight or obese,[18] and that the presence of HTN increases proportionally with the increase in BMI. It is estimated that for each 5% increase in body weight the risk of HTN increases by 20–30%.[19,20] Furthermore, obesity increases the risk of resistant HTN.[21,22]

The mechanisms whereby obesity is associated to HTN are multiple and complex, and are related to genetic, epigenetic, dietary, and environmental factors.[20,22,23] Adipocyte dysfunction in obese patients contributes to insulin resistance and dysfunction of the sympathetic nervous system and the renin-angiotensin–aldosterone system through the production of leptin. Functional and structural changes at the renal, cardiac and vascular levels, including activation of intrarenal angiotensin II, are also important for the development of hypertension associated with obesity.[24] Recently, Aroor et al. in a mouse study demonstrated that obesity promotes stiffness in the aortic artery through activation of the endothelial mineralocorticoid receptor.[25] In addition, some researchers suggest that hypertension in obese patients is mediated, in part, by sodium reabsorption by the proximal tubule, increase in intravascular volume and in cardiac output.[26,27] Additionally, there is evidence suggesting that hyperuricemia can also affect adipocyte function, affecting vascular remodeling, and causing kidney damage.[28]

The relationship between obesity and elevated peripheral BP was first described in 1956 by Vague.[29] Later, in 1967, a prospective analysis of the Framingham *Heart Study* again showed this association,[30] although the nature of this association remained uncertain until the mid-1980s, when several clinical and population studies clarified many aspects of the association between obesity and HTN.[31,32] Later, numerous studies have shown that loss of 10% of weight significantly decreases peripheral BP, thereby reducing mortality from cardiovascular disease[33,34] (Table 1).

Twenty four hour BP monitoring is a better predictor of cardiovascular risk and the appearance of subclinical target organ injury than the measurement BP in the clinic.[35] There are not many published studies on the relationship between obesity and 24-h BP monitoring. A study of 3216 patients published in 2005 showed that BMI was significantly related to BP levels on 24-h monitoring, and that the obese also had higher BP levels during the sleep period.[36] The relationship between obesity and central BP has been studied in recent years showing nonuniform results.[10,37–40] In 1999, a first study showed that central BP was higher in obese adult individuals than in non-obese individuals.[39] Subsequently, in 2013 an

Table 1 – Obesity and HTN.

Authors	Type of study and population	Main findings
Lapidus et al., 1984[31]	Cross-sectional study, 1462 adult women	Obesity is associated with HTN and cardiovascular risk at 12 years of follow-up
Larsson et al., 1984[32]	Prospective study, 792 adult men	Obesity is associated with HTN and cardiovascular risk at 13 years of follow-up
Neter et al., 2003[33]	Meta-analysis, 4874 hypertensive and obese patients	Weight reduction is associated with decreased BP
Kotsis et al., 2005[36]	Cross-sectional study, 3126 HTN patients, 825 obese	Higher central BP in obese
Scuteri et al., 2012[37]	Cross-sectional study, 6148 adults, mean BMI: 25.4 kg/m^2	Higher BP in obese
Rodrigues et al., 2012[38]	Cross-sectional study, 1608 adults, mean BMI: 26 kg/m^2	No relationship between BMI and central BP
Westerbacka et al., 1999[39]	Cross-sectional study, 16 adults, 8 obese	Higher central BP in obese
Higashino et al., 2013[40]	Prospective study, 39 obese adults	Lower central BP after lifestyle modification
Harbin et al., 2018b[41]	Cross-sectional study, 348 young patients, 212 obese	Higher central BP in obese
Pichler et al., 2015[10]	Cross-sectional study, 351 patients, 274 overweight or obese	Lower central BP in overweight and obesity

study in overweight and obese patients showed that changes in lifestyle decreased central BP.[40] In 2015, another study with a total of 351 patients, of whom 78% were obese, showed that overweight or obese patients tended to have lower values of central BP than patients with normal weight, especially in women, both in normotensive patients and hypertensive patients.[10] Given the limited evidence, more studies will be needed to determine what is the impact of obesity on central BP.

Bariatric surgery and high blood pressure

Over the course of several decades, multiple investigations have shown that lifestyle interventions and pharmacotherapy in patients with severe obesity are generally insufficient to achieve weight reduction with long term substantial improvement in their health and quality of life.[41–45] However, there is increasing evidence that bariatric surgery can lead to significant reductions in weight, improving comorbidities such as diabetes mellitus, hypertension, dyslipidemia, and sleep apnea, and therefore prolonging survival.[46–55] The two main techniques in bariatric surgery are *Roux*-en-Y gastric *bypass* and tubular gastrectomy.[50]

Data about changes in peripheral BP after bariatric surgery are variable (Table 2) but most studies show a significant improvement in BP after this intervention.[57] In a meta-analysis by Wilhelm et al. A total of 57 prospective studies or randomized trials were identified, most of them with a follow-up period of less than 2 years, providing data of BP in relation to bariatric surgery. In 32 of these studies, an improvement in hypertension was observed in 64% of patients (32,628 patients out of 51,241), results comparable to the obtained in studies with more than one year of follow-up, and a remission of HTN in 50% of the patients of the 46 studies that reported this data.[58]

In another meta-analysis including 52 studies, Heneghan et al. showed a 68% reduction in HTN associated with obesity, with a mean follow-up time of 34 months. Twenty three percent of the studies included in this sample had a follow-up time of more than 48 months that confirmed the remission of hypertension associated with obesity.[59] Regarding the type of surgery performed, most studies indicate that *bypass* surgery and/or gastric resection have superior results to the purely restrictive techniques such as gastric banding.[60]

As far as factors influencing the remission of HTN, Flores et al. evaluated 526 patients who had undergone bariatric surgery and a 50% were hypertensive. Twelve months after surgery, 65% of these patients had remission of HTN. The duration and severity of previous HTN were predictors of absence of subsequent remission. Baseline BMI, presence of type 2 diabetes mellitus, vitamin D levels, type of surgery, and weight loss were not significantly correlated with remission of HTN.[61]

Studies dedicated to analyze changes in 24-h BP after bariatric surgery[62–64] have produced different results. In 2015, Careaga et al. carried out a prospective study analyzing 42 obese patients (14 hypertensive and 28 normotensive) who underwent bariatric surgery. At 12 months, it was observed a significant decrease in 24-h systolic and diastolic BP although the prevalence of the *non-dipper* pattern continued to be high.[62] Another study[63] analyzed a group of 8 obese patients with HTN with a *non-dipper* pattern that after 8 weeks of bariatric surgery, not only resulted in a decrease in BP levels but also all patients had normalization in the circadian pattern. However, these results should be interpreted with caution due to the low statistical power of such small sample size and the very short-term evaluation. In a very recent study, the variation in 24-h BP was analyzed at 10 days and 6 months after bariatric surgery. There were 90 obese patients analyzed, of which 64 (74.4%) were hypertensive; in these patients, at 10 days, there was a significant reduction in systolic and diastolic BP, and in the variability of 24-h BP. Again, no changes in the circadian pattern were observed. The remission or improvement of HTN was maintained in approximately 50% of patients after 6 months.[64]

An exhaustive review of the literature has not shown any study that had analyzed the modifications of the 24-h outpatient central BP after bariatric surgery at either medium or long term post-surgery. The only data available is a small study including 12 women that evaluated changes in central BP data by applanation tonometry at week 6 after bariatric surgery.[65]

Table 2 – Bariatric surgery and HT.

Authors	Type of study and population	Main findings
Wilhelm et al., 2014[58]	Meta-analysis. Includes 96,460 patients with bariatric surgery, 52,151 with HTN	BP improves in 64% of patients
Heneghan et al., 2011[59]	Systematic review. Includes 16,867 patients with bariatric surgery, 8264 with HTN	BP improves in 68% of patients, with a mean follow-up of 34 months
Vest et al., 2012[60]	Systematic review, 19,543 patients with bariatric surgery, 8598 with HTN	BP improves in 63% of patients
Flores et al., 2014[61]	Prospective study, 526 patients with bariatric surgery, 264 with HTN	BP improves in 65% of patients at 12 months
Careaga et al., 2015[62]	Prospective study, 42 patients with bariatric surgery, 14 with HTN	Significant decrease in of 24 h BP, although the prevalence of *non-dipper* pattern remained high
Głuszewska et al., 2018[64]	Prospective study, 8 patients with bariatric surgery with HTN	BP improves in 100% and normalization of circadian pattern after 8 weeks
Czupryniak et al., 2005[63]	Prospective study, 90 patients with bariatric surgery, 64 with HTN	24-h BP improves in 50% of patients at 6 months

Organic damage and bariatric surgery

There are studies evaluating changes in subclinical target organ injuries as cardiac remodeling after bariatric surgery, showing positive results.[66–69] Alpert et al. demonstrated that the longer the duration of morbid obesity, the greater the regression observed in left ventricular hypertrophy.[67] These favorable results were reproduced in subsequent studies with a 3-year follow-up.[68] Another study showed that these changes were related to the weight loss achieved, and took place basically at the level of the relative thickness of the interventricular septum and the mass of the left ventricle.[69] Additionally, weight loss reduces microalbuminuria, which is an independent cardiovascular risk factor.[70–72] A prospective multicenter study showed a decrease in the prevalence of microalbuminuria from 17 to 11% in 242 obese adolescents, 3 years after the intervention, although this change it was not related to the variation in body weight.[72] A meta-analysis in 2016 that included 30 studies concluded that bariatric surgery significantly reduced microalbuminuria and hyperfiltration in obese patients.[73]

Conclusions

Obesity is becoming a global epidemic, and it has been shown to be clearly associated with peripheral hypertension. Significant weight loss obtained by bariatric surgery decreases peripheral BP and cardiovascular risk. Central or aortic BP is a stronger predictor of cardiovascular risk than peripheral BP, and is more powerfully related to target organ injuries such as left ventricular hypertrophy and intima–media thickness. However, there are not enough studies evaluating the relationship between obesity and central BP and its modifications after bariatric surgery.

A prospective study is currently underway that analyzes 24-h changes in central BP in patients with morbid obesity undergoing bariatric surgery (clinicaltrials.gov identifier: NCT03115502), with funding from the Spanish Society of Nephrology, which is expected to provide data from interest in this regard.

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Conflict of interests

The authors declare that they have no conflict of interest.

Key concepts

- Obesity is strongly associated with HTN by various mechanisms.
- Central BP seems to correlate better than peripheral BP with cardiovascular risk.
- Bariatric surgery contributes to lowering BP in parallel with significant weight loss in patients with severe obesity.
- At the moment there are no studies evaluating the association of obesity with central BP and after bariatric surgery.

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