Effect of Bariatric Surgery on Hypertension

1Department of Surgery, Korea University College of Medicine, Seoul, Korea, 2Department of Surgery, Al sabah Hospital, Kuwait City, Kuwait
Hana Alhomound1,2, Sungsoo Park1,2

High blood pressure is a serious health problem and a primary risk factor for both stroke and heart disease. Many studies have strongly assessed short-term effects as well as long-term effects of bariatric surgery on type2 diabetes mellitus, whereas little attention has been paid to the effect on hypertension. This review study was designed to evaluate the impact of Bariatric Surgery and weight loss on obese patients with hypertension, and whether the hypertension improved or resolved post-surgery. Relevant papers were searched using MEDLINE, Science Citation Index, Pub Med, and Clinical Evidence, by using the searched terms (Hypertension, Bariatric, Obesity, and Surgery). The association between weight reduction and Hypertension control is hard to analyze. And well-organized studies with a long term follow up are required to determine the effect of Bariatric Surgery and Hypertension control.

Key Words: Hypertension, Bariatric surgery, Weight loss

INTRODUCTION

As a result of unhealthy life-styles and genetic predisposition, the prevalence of obesity has nearly doubled since 1980 in developed countries [1].

The World Health Organization (WHO) has recently estimated that there are 700 million obese adults worldwide in 2015 [2].

Obesity is defined as a body mass index (BMI) of >30 kg/m². It is associated with development of comorbid conditions such as hypertension, diabetes mellitus, sleep apnea and congestive heart failure [3].

Obesity can be treated non-surgically or by means of metabolic/bariatric surgery [4,5], which has been widely demonstrated to lead to sustained weight loss and a reduction in comorbidities [6-9].

Until now, there are relatively strong evidences that metabolic/bariatric surgery can treat type 2 diabetes mellitus (T2DM) as well as obesity [10-13].

One of major medical co-morbid condition for obesity is hypertension but there is a few reports on how it went on with obesity and metabolic and bariatric surgeries.

The etiology of obesity-related hypertension is likely multifactorial, with certain mechanisms such as hyperinsulinemia, hyperlipidemia, and enhanced sympathetic and renin-angiotensin activities playing a potential role [14,15]. Hypertension must be known constituent of obesity-related (metabolic syndrome) of insulin resistance, dyslipidemia, glucose intolerance, and centripetal obesity. Adipose tissue is also a known source of neuroendocrine hormones [16]. Weight loss will therefore eliminate the adipose tissue related causes of hypertension.
This short review will deal with the impact of metabolic and bariatric surgery on hypertension incorporated with evaluating published data.

MATERIALS AND METHODS

An electronic search method using search-terms (Bariatric surgery, Hypertension and Comorbidities) was primarily used for the identification of the studies.

Comprehensive search of all electronic data bases (MEDLINE, Pub Med, Clinical Evidence, Web of Science, and Clinical Trials) was performed.

Studies of any design including adult obese patients undergoing metabolic and bariatric surgery were considered. A pre-screening was performed to identify the clearly irrelevant papers based on the title, abstract and key words.

1. Data extraction and management

The data extracted from the papers contains patients demographic (age, gender), Type of study (Randomized controlled trial, retrospective, prospective, case series) and baseline and post-intervention outcomes (Incidence of Hypertension, resolving and improvement of hypertension).

RESULTS

A total of 320 abstracts were scanned, and 279 full articles were obtained and critically appraised, of which 9 studies met the inclusion criteria and investigated the long-term effects of weight loss on blood pressure. Table 1 shows the basic characteristics of these 9 studies. These included one randomized controlled trial, one control clinical trial, one retrospective study, and six prospective studies [17-25]. The nine studies involved a total of 2756 patients (3 studies were from Sweden, 2 from United States.

| Author        | Country | Year | Number of patients | Study design | Surgical technique | Follow-up period |
|---------------|---------|------|--------------------|--------------|--------------------|------------------|
| Sjöström et al. [17] | Sweden  | 1999 | 845                | Prospective  | VBG/LRGBP         | 2 Years          |
| Sjöström et al. [18] | Sweden  | 2000 | 346                | Prospective  | VBG/GB/LRGBP      | 8 Years          |
| Sjöström et al. [19] | Sweden  | 2001 | 1157               | Prospective  | VBG/GB/LRGBP      | 8 Years          |
| Ahmed et al. [20] | USA     | 2009 | 100                | Prospective  | LRGBP             | 1 Year           |
| Nordstrand et al. [21] | Norway  | 2012 | 90                 | Control Clinical Trial | LRGBP       | 1 Year           |
| Fenske et al. [22] | United Kingdom | 2013 | 34                 | Prospective  | GB/LSG/LRGBP      | 1 Year           |
| Liang et al. [23] | China   | 2013 | 108                | Randomized Controlled Trial | LRGBP     | 1 Year           |
| Dogan et al. [24] | USA     | 2014 | 52                 | Retrospective | LRGBP            | 8 Years          |
| Bonfils et al. [25] | Denmark | 2015 | 24                 | Prospective  | LRGBP             | 1 Year           |

VGB = Vertical banded gastroplasty, GB = gastric band, LRGBP = laparoscopic Roux-en-Y Gastric Bypass, LSG = laparoscopic sleeve gastrectomy.

| Study          | Type of surgery | Weight difference (kg) | SBP difference (mmHg) | DBP difference (mmHg) |
|----------------|-----------------|------------------------|------------------------|------------------------|
| Sjöström et al. [17] | VBG/LRGBP      | 28±15 kg               | 7±18                   | 6±11                   |
| Sjöström et al. [18] | VBG/GB/LRGBP   | 20±15.7 kg             | 2.9±22                 | 1.9±14                 |
| Sjöström et al. [19] | VBG/GB/LRGBP   | 33.8±18.1 kg           | −20                    | −11                    |
| Ahmed et al. [20] | LRGBP           | 45 kg                  | −15                    | −9                     |
| Nordstrand et al. [21] | LRGBP         | 41 kg                  | −23                    | −13                    |
| Fenske et al. [22] | LRGBP/GB/LSG   | 45 kg                  | 18±1.6                 | 12±1.0                 |
| Liang et al. [23] | LRGBP           | 24% reduction          | 34±4.9                 | −10                    |
| Dogan et al. [24] | LRGBP           | 39.4±17.1 kg           | 28% reduction          | 28% reduction          |
| Bonfils et al. [25] | LRGBP           | 38.5±21.8 kg           | 5.9±5.4                | 2.4±3.6                |
of America, and one each from United Kingdom, Norway, China, and Denmark).

There is a variable difference between the systolic blood pressure and diastolic blood pressure compared to weight loss during the follow-up period within the studies. Table 2 shows the weight differences with systolic and diastolic blood pressure difference after surgery.

Surgery resulted in rapid weight loss over the first 6 months in all studies and more weight loss was noted in LRGBP compared to GB, LSG and VGB. Sjöström et al. [17] showed, at 2 years, gastric bypass resulted in $33 \pm 10\%$ weight reduction corresponding to VBG and GB were $23 \pm 10\%$ and $21 \pm 12\%$ respectively. In the long term study which was done by the Swedish obese subjects [19] showed that there was a rapid weight loss in the first 6 months and, on average, nadir weights at 1 year. At that time, the weight reductions were $-25.8 \pm 12.9\, kg$, $-30.7 \pm 11.8\, kg$, and $-44.0 \pm 15.0\, kg$ in patients treated with GB, VBG and LRGBP respectively. From 1 year and onward, the three groups relapsed slowly. Compared with the start of intervention, the weight losses were $20.7 \pm 16.6\, kg$, $20.8 \pm 13.1\, kg$, and $33.8 \pm 18.1\, kg$ respectively. Fenske et al. [22] who included LSG for weight loss showed that the excess weight loss for the patients who underwent LRGBP, LSG and GB was $48.7\% \pm 2.6\%$, $47.8\% \pm 4.5\%$, and $45.0\% \pm 2.4\%$ respectively. All papers were in favor of LRGBP.

The incidence of hypertension was reduced significantly in all of the studies in the short term up to one year, however, in long term follow-up study done in Sweden [18]: there was unremarkable change in blood pressure compared to weight loss. The study suggested that the course of blood pressure over the study period showed a different pattern, during the first 6 months of rapid weight loss, SBP was reduced by $11.4 \pm 19.0\, mmHg$ and DBP was reduced by $7.0 \pm 11.0\, mmHg$, in spite of a continuous weight loss during the following 6 months, the reduction in DBP ceased, whereas SBP seemed to increase. From 1 year, SBP and DBP increased gradually over the remaining 7 years. Where other investigators have concluded that even small net weight losses might be beneficial for the control of hypertension, Sjöström et al. [18] showed that over a long period, not even a maintained 16\% weight loss is sufficient to achieve a reduction of the 8-year incidence of hypertension in the severely obese. Blood pressure measurement was taken as early as one week post- surgery in all studies and compared with follow-up measurements at 5 weeks, 6 months, and 12 months in most of the studies, and two years and eight years in the other long term follow-up studies. The maximum benefit is soon after initial weight loss.

DISCUSSION

Over the last few years, the aim of bariatric surgery has changed from weight loss to controlling metabolic disease. This review was done to determine the effect of metabolic and bariatric surgery on Hypertension. However, with the 9 studies finally selected, the mechanism between weight loss and hypertension is complex and hardly to explain.

Ahmed et al. [20], showed that hypertension remission rate was 66\% in a Prospective study on 100 patients, suggesting a hormonal mechanism maybe involved for the changes observed, various neuroendocrine changes have been postulated to play a role in this. The gut peptide glucagon-like peptide 1 (GLP-1) has been implicated by some in the early improvement in glycemic control after LRYGBP [26]. GLP-1 may also have a role in blood pressure improvement via an effect on the anatomic nervous system [27] or through inducing natriuretic [28]. Another explanation is that the obese state is associated with raised levels of plasma renin activity, aldosterone, an angiotensin–converting enzyme leading to sodium retention and that after bariatric surgery these abnormal hormone levels tend to normalize [29], other theories to explain the early reduction in blood pressure include: (a) reduced food and salt intake after bariatric surgery, (b) dumping syndrome after LRGBP surgery causing avoidance of high-osmolarity substances such as salts, and (c) restoration of endothelial function [20].

More mechanisms involved in blood pressure reduction after weight reduction suggested by Sjöström et al. [18], include improved insulin resistance and endothelial function, a decrease in renin-angiotensin-aldosterone system activation, reduction in sympathetic nervous system activity, decreased serum leptin levels, and leptin resistance, and increased post-operative natriuresis. A reduc-
tion in visceral fat mass and intra-abdominal pressure might also reduce renin-angiotensin-aldosterone activity, increase natriuresis and, thus decrease arterial blood pressure [30].

Meta-analysis and Meta-Regression study with 5 Year follow-up on the long term effects of Bariatric surgery on type II Diabetes, Hypertension and Hyperlipidemia by Ricci et al. [31], found that the reduction in the risk of Hypertension reached a plateau about 20 months after surgery, whereas the reduction in the risk of type II Diabetes and Hyperlipidemia continued for a further year before leveling off. The biological mechanisms involved in rapid weight loss include paracrine and endocrine hormones, the excessive adipocyte hypertrophy of obese patients induces hypoxia and leads to adiposopathy; i.e. anatomical and functional abnormalities in adipocytes and adipose tissue. This is accompanied by a reduction in the production of adiponectin and omentin and the greater release of adipocytokines, which has been found to associate with cardiovascular disease [32].

Many theories may explain why that blood pressure does not decrease after continuous weight loss following Bariatric surgery.

CONCLUSION

The result of this review article indicate that weight loss results in blood pressure improvement in general, however, the long term outcome of weight loss on blood pressure is not well explained. A sufficiently long and appropriately sized, multicenter randomized controlled study is required to clarify the impact of long term Bariatric surgery on Hypertension.

CONFLICTS OF INTEREST

No potential conflict of interest relevant to this article was reported.

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