Aesth Plast Surg (2011) 35:995–999
DOI 10.1007/s00266-011-9719-7

ORIGINAL ARTICLE

Effects of Reduction Mammoplasty on Metabolic Profile and Body Weight

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Received: 28 December 2010 / Accepted: 25 March 2011 / Published online: 13 April 2011
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Abstract
Background Advanced studies on adipose tissue have established that subcutaneous adipose tissue acts as an endocrine organ to help maintain homeostasis. Based on this information, many plastic surgeons have evaluated the metabolic effects of liposuction because liposuction is the most common surgical procedure in plastic surgery. Liposuction removes a substantial amount of subcutaneous fat from a specific area of the body. Mammoplasty is another procedure that removes a large amount of subcutaneous fat. In this study, the metabolic effects of reduction mammoplasty were evaluated with hemogram, blood glucose, lipid profile, insulin, and insulin resistance tests before and after surgery.

Methods The study involved 35 patients who underwent reduction mammoplasty between January 2006 and June 2009. All the patients were evaluated with physical examination and their history, height, and weight were obtained. Venous blood samples were collected before, 4 h after, and 3 months after the surgical procedure to evaluate hemogram, blood glucose, insulin, and lipid profiles. The HOMA scores of the patients were calculated. Inferiorly based dermal pedicle, central pedicle, and free nipple graft techniques were used in the operations and all excision materials were sent for histopathological examination.
Results The mean age of the patients was 39.6 ± 11.6 years. The mean excision volume was 2249 ± 1001 g. Body mass indexes were not significantly different before and 3 months after the surgery ($p > 0.05$). Blood glucose, LDL, HDL, triglyceride, total cholesterol, hemoglobin, and hematocrit values before and 4 h after the surgery were also different ($p < 0.05$). However, comparisons with the 3-month postoperative test results revealed no statistically significant differences ($p > 0.05$) and comparisons of all the measurements showed that insulin levels and HOMA scores were not significantly different ($p > 0.05$).
Conclusion The results of the present study showed that reduction mammoplasty operations do not have any positive effects on blood insulin, glucose profile, lipid profile, and body mass index 3 months after the surgery.

Keywords Reduction mammoplasty · Insulin · Lipid profile · Body mass index

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Reduction mammoplasty has been a common procedure in plastic surgery practice since it was first performed by William Durston in 1870 [1]. Since then, studies in

mammarian vascular anatomy and the practice of flap surgery have been highly advanced; thus, plastic surgeons have better and more choices of techniques in performing mammoplasty. The advanced techniques make it possible for surgeons to remove considerable amounts of mammary tissue without complications.

The excision material of reduction mammoplasty includes much more subcutaneous adipose tissue than glandular tissue, and the ratio of adipose to glandular tissue varies with patient body mass index [2]. Studies of adipose tissue have established that subcutaneous adipose tissue acts as an endocrine organ that produces numerous proteins called adipocytokines to help maintain homeostasis [3].

In light of this information, many plastic surgeons have evaluated the metabolic effects of liposuction, the most common surgical procedure in plastic surgery practice, on reducing subcutaneous fat [4–9]. Another popular procedure to remove a large amount of subcutaneous fat is reduction mammoplasty. The physical and aesthetic benefits of reduction mammoplasty are known; nevertheless, the literature does not present any studies on its metabolic effects. In this study we evaluated the metabolic effects of reduction mammoplasty on patients by testing hemogram, blood glucose, lipid profile, insulin, and insulin resistance before and after the surgery.

Patients and Methods

Sixty-two patients who underwent reduction mammoplasty operations between January 2006 and June 2009 were enrolled into the study. All the operations were performed at Ankara Ataturk Education and Research Hospital by the same team.

Patients who had no history of any known disease, had no history of any antihyperlipidemic and antidiabetic medication use, sustained the same weight (±1 kg) for at least the last 2 months before surgery, and had a sedentary lifestyle for at least 6 months were included in the study. Exclusion criteria were pregnancy just after the operation, malignancy, hepatic or renal failure, or having any other surgery during the study. The patients who had any complications that could affect metabolic values were also excluded (Table 1). According to these criteria, 35 patients were included in this study. Informed written consent was obtained from each subject, and the hospital-based Ethics Committee approved this study.

Before the patients were included, a detailed personal history and a physical examination of each patient were obtained. The weights and heights of the patients were also measured, and the body mass indexes (BMI, kg/m^2) of the patients were calculated as weight (kg) divided by the square of the height (m^2). Venous blood samples were obtained preoperatively and 4 h and 3 months postoperatively to evaluate hemogram, blood glucose, insulin, and lipid profiles. Homeostatic model assessment (HOMA) scores were calculated for insulin resistance state as fasting plasma glucose (mmol/l) multiplied by fasting serum insulin (mU/ml) divided by 25 [10]. However, postoperative 4-h HOMA scores were not calculated because of potential miscalculation of exact insulin resistance due to early postoperative metabolic changes.

Table 1 Exclusion criteria of this study

Exclusion criteria	No. of patients
Antidiabetic usage	9
Antihyperlipidemic usage	12 (6 were either diabetic)
Weight fluctuation (last 2 months >2 kg)	4
Nonsedentary lifestyle	5
Postoperative reoperation due to hematoma and suture dehiscence	2
Postoperative operation due to acute cholecystitis	1

An inferior dermoglandular pedicle technique was performed on 22 patients, the free nipple graft technique on 11 participants, and the central pedicle technique on 2 participants. All the excision materials were sent for histopathological examination.

The results were analyzed with SPSS (Statistical Package for Social Sciences) for Windows version 16.0 (SPSS, Inc., Chicago, IL, USA). To evaluate the values, descriptive analyses and Friedman tests were used; $p < 0.05$ was considered statistically significant.

Results

All patients tolerated surgery without any life-threatening complications, and each patient returned to her daily life 15 days after the operation on condition of light work. The patients had a mean (±SD) age of 39.6 ± 11.6 years. All the preoperative anthropometric measurements were compared with the results obtained in the third postoperative month. Accordingly, the mean weights were 73.2 ± 12.3 and 71.8 ± 12.2 kg ($p > 0.05$), respectively. All the body mass indexes were calculated and the mean BMIs were 28 ± 5.6 and 27.6 ± 5.5 kg/m^2 ($p > 0.05$), respectively (Table 2), and the mean amount of excised breast tissue was 2249 ± 1001 g (min: 820, max: 5537).

All the preoperative and postoperative fourth-hour and third-month metabolic measurements are given in Table 3. The serum lipid profiles, including total cholesterol, high-density lipoprotein (HDL), low-density lipoprotein (LDL), and triglyceride, decreased 4 h after the surgery ($p < 0.01$).

Table 2 Anthropometric measurements

	Minimum	Maximum	Mean
Age	20	58	39.6
Height (m)	1.49	1.72	1.61
Preoperative weight (kg)	58	116	73.2
Postoperative weight (kg)	56	112	71.8
Preoperative BMI (kg/m^2)	22	46.5	28
Postoperative BMI (kg/m^2)	20.3	44.5	27.6

Table 3 Effects of reduction mammoplasty on metabolic parameters

	Preoperative	4 h postoperative	3 months postoperative
Glucose (mg/dl)	93.0 ± 9.9	100 ± 17.9	94 ± 8.8
Insulin (mU/ml)	11.5 ± 5.3	11.2 ± 6.5	12.1 ± 6.3
Total cholesterol (mg/dl)	192 ± 38	163 ± 39.1	190 ± 36.5
LDL (mg/dl)	117.3 ± 31.5	102.9 ± 33.3	116.7 ± 31.1
HDL (mg/dl)	48.5 ± 11.3	43.1 ± 10.5	48.9 ± 9.6
Triglyceride (mg/dl)	126.8 ± 76.8	88.4 ± 68.4	124.3 ± 54.2
Hemoglobin (mg/dl)	13.3 ± 1.3	11.7 ± 1.2	13.2 ± 1.2
Hematocrit (%)	39.5 ± 3.7	34.8 ± 3.5	39.5 ± 3.4

Values are mean ± SD

The levels in the third month after surgery were also lower than the preoperative levels, but the differences were not statistically significant ($p > 0.05$). A similar rate of change was observed in the insulin levels and in the serum lipids. Fasting glucose levels 4 h after surgery were higher than the preoperative levels ($p < 0.01$); however, in the third postoperative month, the difference was not statistically significant ($p > 0.05$). No statistically significant differences were found between preoperative and postoperative third-month calculated HOMA scores ($p > 0.05$) (1.5 ± 0.68 vs. 1.58 ± 0.8). Hemoglobin and hematocrit levels first decreased ($p < 0.01$) but the differences were not statistically significant in the third postoperative month ($p > 0.05$)

Discussion

The primary function of a woman's breasts is lactation, but they are also a symbol of female sexuality. Therefore, a patient's expectations are not only to be relieved from physical complaints but also to have aesthetically pleasing breasts. Patients' major physical complaints are poor body posture, shoulder pain, dermatitis at the inframammary fold, back pain, painful shoulder grooves due to bra straps, and exercise intolerance [11–16]. Some authors have also reported neurological symptoms such as ulnar nerve paresthesias, carpal tunnel syndrome, and hand numbness [17, 18]. Reducing breast size resolves these symptoms and provides a better aesthetic image, but one must ask about the potential metabolic effects of breast reduction surgery on homeostasis.

Excision material of reduction mammoplasty contains much more adipose tissue than glandular tissue, contrary to what was thought before, and that proportion is parallel to the patient's body mass index [2]. In 2002, Cruz-Korchin et al. [2] studied reduction mammoplasty specimens from 25 patients and found that enlarged breasts consisted primarily of fat rather than glandular tissue. They also observed that the lateral side of the breast contains more adipose tissue than the central side.

Previous studies on subcutaneous adipose tissue showed that it is an endocrine organ that affects all of the body by its production of adipocytokines (leptin, tumor necrosis factor α, interleukin-6, plasminogen activator inhibitor-1, resistin, adipicin, adiponectin, and acylation-stimulating protein) [3]. Liposuction is the most popular procedure in plastic surgery practice that reduces adipose tissue [4]. The literature presents several studies about its metabolic effects [4–9]. Guigliano et al. [5] evaluated BMI, blood glucose, insulin, IL-6, IL-18, TNF-α, CRP, adiponectin, and lipid profiles in the sixth month after liposuction and determined that there were decreased levels of insulin resistance, TNF-α, IL-6, IL-18, and CRP and positive metabolic effects of liposuction. In 2002, Gonzalez-Ortiz et al. [7] reported that the surgical removal of subcutaneous fat did not change the patient's lipid profile but increased insulin sensitivity by decreasing insulin and glucose levels. Another study on abdominal liposuction found that insulin levels did not change but cholesterol and glucose levels decreased after the procedure.

All of these studies emphasized the positive metabolic effects of liposuction due to reduction of subcutaneous adipose tissue. In their 2004 study, Klein et al. [9] did not find any positive metabolic effects of liposuction, unlike the other studies. They conducted their study on 15 obese patients who underwent abdominal liposuction. They evaluated blood glucose, insulin, lipid profile, leptin, adiponectin, TNF-α, IL-6, and CRP levels before and 3 months after the surgery but did not find any significant differences. The findings of our study are compatible with their findings since in our study, no statistically significant differences were found between the preoperative and postoperative third-month findings for the metabolic criteria measured after the mean reduction of 2249 ± 1001 g of breast tissue. Insulin and glucose levels did not decrease and the lipid profile (total cholesterol, LDL, HDL, and triglyceride) results showed no statistically significant differences.

Our findings for postoperative fourth hour were significantly different from our preoperative findings.

Considering that the results could be affected by surgical stress, intravenous liquids given during or after the surgery, and some other factors, we thought that they were not as important as the results for the postoperative third month. Nevertheless, we believe that it is important to know that there is a period with metabolic changes after reduction mammoplasty.

In 2008, however, Rinomhota et al. [19] determined significant postoperative changes because their participants gained weight after surgical subcutaneous fat reduction. Their study was on patients who had undergone both abdominoplasty and reduction mammoplasty. In that study, they did not evaluate the metabolic functional changes, but the adverse effects of regained weight on metabolism can be predicted.

Before the report by Rinomhota et al. many researchers had published experimental studies on animals that underwent partial lipectomy with similar results [20–23]. They found a significant compensatory growth of adipose tissue after lipectomy. This increase was due to adipocyte hyperplasia and/or hypertrophy in different parts of the body other than the surgical area, and there were no changes in the animals' food intake before or after the surgery. Even though the authors observed this increase, they could not explain the exact reason for the increase or why the increase occurred at different parts of the body other than the lipectomized area [23].

In our study, the amount of excised adipose tissue was not more than what could be lost by diet, pharmacotherapy, or physical exercise. Weight loss by behavioral changes and pharmacotherapy or diet can positively change the metabolic status, but we did not observe any positive metabolic effects on insulin resistance or lipid profile after surgical adipose tissue reduction with mammoplasty.

It has been shown that weight loss is a homogeneous decrease of fat characterized by visceral, intrahepatic, and intracellular adipose tissue reduction. However, a sudden reduction of fat by surgery cannot decrease the adipose tissue in these areas [9]. Moreover, there are experimental and clinical studies with results of compensatory adipose tissue growth after sudden adipose tissue removal [19–23]. In our study, there were no statistically significant differences between the mean BMIs measured before and after the surgery (28 ± 5.6 vs. 27.6 ± 5.5 kg/m^2). We also did not observe an increase in patient breast sizes; thus, weight gain may have occurred in other parts of the body.

Conclusions

Even though reduction mammoplasty decreases adipose tissue, it does not provide any positive effects on metabolic profile or BMI in the early postoperative period without supportive programs such as diet and/or exercise.

Conflict of interest The authors declare that they have no conflicts of interest to disclose.

References

1. Psillakis JM, Cardoso de Oliveira M (1990) History of reduction mammoplasty. In: Goldwyn RM (ed) Reduction mammoplasty, 1st edn. Little, Brown, Boston, pp 1–15
2. Cruz-Korchin N, Korchin L, Gonzales-Kealan C, Climent C, Morales I (2002) Macromastia: how much of it is fat? Plast Reconstr Surg 109:64–68
3. Emral R (2006) Adiponectin and other cytokines. Türk Klin J Med Sci 26:409–420
4. Hong YG, Kim HT, Seo SW, Chang CH, Rhee EJ, Lee WY (2006) Impact of large volume liposuction on serum lipids in orientals: A pilot study. Aesthetic Plast Surg 30:327–332
5. Guigliano G, Nicoletti G, Grella E, Guigliano F, Esposito K, Scuderi N et al (2004) Effect of liposuction on insulin resistance and vascular inflammatory markers in obese woman. Br J Plast Surg 57:190–194
6. Robles Cervantes JA, Yanez-Diaz S, Cordenas Camarena L (2004) Modification of insulin, glucose and cholesterol in non-obese woman undergoing liposuction. Is liposuction metabolically safe? Ann Plast Surg 52:64–67
7. Gonzales-Ortiz M, Robles-Cervantes JA, Cordenas Camarena L, Bustos-Saldana R, Martinez-Abundis E (2002) The effects of surgically removing subcutaneous fat on the metabolic profile and insulin sensitivity in obese women after large volume liposuction treatment. Horm Metab Res 34:446–449
8. Rizzo MR, Paolisso G, Grella R, Barbieri M, Grella E et al (2005) Is dermolipectomy effective in improving insulin action and lowering inflammatory markers in obese woman? Clin Endocrinol 63:253–258
9. Klein S, Fontana L, Young L, Coggan A, Kilo C, Patterson BW et al (2004) Absence of an effect of liposuction on insulin action and risk factors for coronary heart disease. N Eng J Med 350:2549–2557
10. Matthews DR, Hosker JP, Rudenski AS et al (1985) Homeostasis model assessment: insulin resistance and β-cell function from fasting plasma glucose and insulin concentrations in man. Diabetologia 28:412–419
11. Blomqvist L, Eriksson A, Brandberg Y (2000) Reduction mammoplasty provides long term improvement in health status and quality of life. Plast Reconstr Surg 106(5):991–997
12. Chao JD, Memmel HC, Redding JF, Egan L, Odom LC, Casas LA (2002) Reduction mammoplasty is a functional operation, improving quality of life in symptomatic women: a prospective single center breast reduction outcome study. Plast Reconstr Surg 110(7):1644–1652
13. Jones SA, Bain JR (2001) Review of data describing outcomes that are used to assess changes in quality of life after reduction mammoplasty. Plast Reconstr Surg 108(1):62–67
14. Benditte-Klepetko H, Leisser V, Paternostro-Sluga T, Rakos M, Trattnig S, Helbich T, Schemper M, Deutinger M (2007) Hypertrophy of the breast: a problem of beauty or health? J Women Health (Larchmt) 16(7):1062–1069
15. Gonzalez F, Walton RL, Shafer B, Matory WE Jr, Barah GL (1993) Reduction mammoplasty improves symptoms of macromastia. Plast Reconstr Surg 91:1270–1276

16. Mizgala CL, Mackenzie KM (2000) Breast reduction outcome study. Ann Plast Surg 44(2):125–133 discussion 133-134
17. Brown DM, Young VL (1993) Reduction mammoplasty for macromastia. Aesthetic Plast Surg 17:211–223
18. Pernia LR, Ronel DN, Leeper JD, Miller HL (2000) Carpal tunnel syndrome in women undergoing reduction mammoplasty. Plast Reconstr Surg 105:1314–1319
19. Rinomhota AS, Bulugahapitiya DUS, French SJ, Caddy CM, Griffiths RW et al (2008) Women gain weight and fat mass despite lipectomy at abdominoplasty and breast reduction. Eur J Endocrinol 158:349–352
20. Bailey JW, Anderson DB (1974) Rate of fat compensation and growth efficiency of lipectomized Sprague-Dawley rats. J Nutr 110:1785–1792
21. Chlouverakis C, Hojnicki D (1974) Lipectomy in obese hyperglycemic mice (ob/ob). Metabolism 23(2):133–137
22. Forger NG, Dark J, Zucker I (1986) Recovery of white adipose tissue after lipectomy in female ground squirrels. Can J Zool 64:128–131
23. Hausman DB, Lu J, Ryan DH, Flatt WP, Harris Ruth BS (2004) Compensatory growth of adipose tissue after partial lipectomy: involvement of serum factors. Exp Biol Med 229:512–520