C-Reactive Protein Decrease After Postbariatric Abdominoplasty

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31.1 Introduction

An inflammatory reaction is a response that occurs after aggressive stimuli and is associated to vasodilation, increase in vascular permeability, inflammatory cell recruitment, and the release of inflammatory mediators by such cells, including cytokines, among others [1]. These cytokines are responsible for acute-phase inflammatory mediation, interfering in the production of several plasma proteins by hepatocytes, including CRP [2].

Considering that inflammatory states are associated with the development of several pathologic conditions such as cardiovascular events, the monitoring of plasma inflammatory markers is used as an auxiliary method in the prognosis of such events [1].

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Thus, CRP is a protein present in the acute phase of the event and is used as an early marker of inflammation and infection [3]. Its synthesis occurs predominantly in the liver developing a rapid increase in infections or inflammatory states [4]. CRP has this denomination due to its reaction to *Streptococcus pneumoniae* C-polysaccharide. In the presence of calcium, CRP binds to the polysaccharides present on the surface of pathogens. Such binding activates the classic complement chain that will culminate in phagocytosis [1]. CRP levels increase within 6 h after an acute inflammatory stimuli with the possibility of doubling in plasma levels at every succeeding 8 h period and peaking within approximately 50 h [5].

Although the elevation of plasma CRP levels are not specific to any condition, it works as a highly sensitive marker to inflammation (>90 %), becoming valuable in the clinical evolution of such processes allowing one to correlate the CRP levels to disease activity and treatment efficacy [1]. Besides being an inflammatory marker, plasma CRP levels serve as predictors of coronary disease [6–8] having as an indication its quantification associated to lipid profiles to improve cardiac risk assessment. Some studies speculate that there is also a direct action of CRP as causal factor of cardiovascular disease [9]. Elevated levels of CRP also occur in chronic renal failure and obesity context, demonstrating its correlation to chronic inflammatory states [10].

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Obesity is associated to many metabolic and hormonal dysfunctions that increase patient morbidity and mortality through increase in risk of type II diabetes mellitus and cardiovascular disease mainly due to development of insulin resistance [11–13]. There is also a correlation between obesity and subclinical systemic inflammatory states, when CRP levels were observed in obese men and women [14]. This occurs with greater relevance when adipose tissue concentrates in the abdomen, as in those patients with a high waist-to-hip ratio [15, 16].

Accordingly, the CRP reduction occurs in proportion to weight loss, through diet correction and exercise and, intensively, after a massive weight loss due to bariatric surgery, reducing the morbidity and mortality of these patients. In such context, patients submitted to surgical treatment of morbid obesity through bariatric surgery evolve with massive weight loss, and consequently, body dysmorphia ensues [17]. Then, the need for body contour reconstruction arises, demanding plastic surgery. This stage of treatment remains offered as multidiscipline action given the peculiarities of these patients that underwent a weight loss period under hypocaloric and nutritionally deficient pressures [18–21].

In general, the areas affected by development of skin and adipose folds after massive weight loss are the abdomen, flanks, breasts, arms, and thighs. Out of these regions, abdominoplasty is the most frequent procedure made in such patients, followed by mastopexy [22–24]. Although this procedure deals with subcutaneous adipose tissue and not with visceral fat, the effects of these plastic surgeries, under inflammatory and metabolic perspective, have been established. There is evidence that abdominoplasties in postbariatric surgery present metabolic and inflammatory advantages [25], mainly on long-term follow-up studies. However, the opposite has also been documented on a study that showed lipid profile deterioration involving total and low-density cholesterol levels [26].

Comparable conflict exists in liposuction procedure studies, without greater evidence, but with divergences far from dissolution. Some studies show systemic inflammatory and insulin resistance reduction, while in other studies the improvement of lipid profiles was confirmed [27–31]. In contrast, other studies showed no evidence of alteration in insulin activity or cardiovascular risk factors [32, 33]. In addition, a long-term follow-up protocol identified in a cohort study with seven obese women did not present any significant tendencies [34].

Considering the exponential increase in number of bariatric surgeries performed globally and the amplitude of population reached [35–37], long-term follow-up studies became necessary to determine the effects of body contour reconstruction surgery on inflammatory and metabolic markers.

31.2 Method

In a prospective controlled study, which included retrospective information, it considered three distinct periods for data acquisition: prebariatric, pre-abdominoplasty, and post-abdominoplasty [38]. Two groups were determined after massive weight loss. A 20-patient group that was submitted to circumferential abdominoplasty [22] and a control group with 20 patients submitted to mastopexy with breast implants [39]. Clinical information and laboratory exams from prebariatric period were obtained from the patients' medical history files. The remaining data were obtained prospectively before and after the plastic surgeries. The following exams were tabulated: leukocyte count, C-reactive protein, hemoglobin, cholesterol levels and its fractions, triglycerides, blood glucose, and HbA1c tests [22].

31.3 Results

The variables between the two groups that did not present differences were age ($p = 0.703$), the time interval between bariatric and plastic procedures ($p = 0.360$), and the follow-up period after plastic surgery ($p = 0.135$).

In the control group, there was not any significant alteration in the body mass index (BMI), and nearly all metabolic markers analyzed remained stable. The exceptions were CRP reduction levels and increase in high density cholesterol levels according to Table 31.1.

The profile after mastopexy with breast implants revealed some unexpected alterations. These patients presented a lower BMI previous to bariatric and plastic surgeries, besides the sustained anemia in the years following breast

Table 31.1 Nutritional, metabolic, and inflammatory variables in abdominoplasty subjects

Variable	Prebariatric	Pre-abdominoplasty	Post-abdominoplasty	Significance[a]	
BMI (kg/m^2)	55.7 ± 13.0	31.9 ± 4.6	30.2 ± 3.7	$p < 0.001$	$p = 0.424$
Hemoglobin (g/dL)	12.5 ± 2.2	11.7 ± 1.7	11.8 ± 2.4	$p = 0.161$	$p = 0.662$
WBC (x10^3/mm^3)	6.6 ± 1.6	5.0 ± 1.1	5.2 ± 1.4	$p = 0.006$	$p = 0.277$
Total cholesterol (mg/dL)	176 ± 41	183 ± 12	187 ± 35	$p = 0.227$	$p = 0.246$
HDL cholesterol (mg/dL)	51.4 ± 8.4	61.8 ± 6.7	72.6 ± 13.2	$p = 0.041$	$p = 0.049$
LDL cholesterol (mg/dL)	110 ± 38	107 ± 14	96.3 ± 14.8	$p = 0.817$	$p = 0.256$
VLDL cholesterol (mg/dL)	16.3 ± 3.5	12.3 ± 1.5	13.6 ± 3.7	$p = 0.144$	$p = 0.257$
Triglycerides (mg/dL)	80.8 ± 13.0	60.8 ± 6.7	73.8 ± 17.2	$p = 0.041$	$p = 0.095$
Glucose (mg/dL)	99.6 ± 26.6	83.5 ± 5.8	86.4 ± 6.5	$p = 0.133$	$p = 0.555$
HbA1c (%)	8.5 ± 2.8	5.7 ± 1.6	5.4 ± 1.1	$p = 0.038$	$p = 0.223$
C-reactive protein (mg/dL)	6.6 ± 3.3	3.9 ± 2.3	2.2 ± 1.6	$p = 0.024$	$p = 0.008$

[a]First significance: prebariatric vs pre-abdominoplasty; second significance: pre-abdominoplasty vs postabdominoplasty

Table 31.2 Outcome variables in control patients (mastopexy with implants)

Variable	Prebariatric	Preimplant	Postimplant	Significance[a]	
BMI (kg/m^2)	43.7 ± 3.6	25.5 ± 2.2	24.6 ± 2.6	$p < 0.001$	$p = 0.233$
Hemoglobin (g/dL)	13.2 ± 0.9	12.1 ± 1.0	11.0 ± 1.8	$p = 0.017$	$p = 0.039$
WBC (x10^3/mm^3)	7.3 ± 2.2	5.5 ± 1.3	5.5 ± 1.5	$p = 0.015$	$p = 0.558$
Total cholesterol (mg/dL)	187 ± 31	151 ± 31	168 ± 35	$p = 0.004$	$p = 0.167$
HDL cholesterol (mg/dL)	47.0 ± 9.9	69.4 ± 19.5	66.0 ± 4.1	$p = 0.040$	$p = 0.869$
LDL cholesterol (mg/dL)	113 ± 23	70.8 ± 20.8	79.5 ± 25.9	$p = 0.006$	$p = 0.161$
VLDL cholesterol (mg/dL)	21.1 ± 14.1	13.6 ± 6.1	15.0 ± 8.4	$p < 0.001$	$p = 0.073$
Triglycerides (mg/dL)	159 ± 62	66.7 ± 21.3	75.3 ± 33.1	$p = 0.027$	$p = 0.088$
Glucose (mg/dL)	90.4 ± 9.7	83.9 ± 9.4	88.3 ± 3.4	$p = 0.027$	$p = 0.937$
HbA1c (%)	5.9 ± 0.3	5.9 ± 0.5	6.0 ± 0.6	$p = 0.874$	$p = 0.667$
C-reactive protein (mg/dL)	6.8 ± 2.1	4.6 ± 5.7	3.2 ± 1.0	$p = 0.143$	$p = 0.301$

[a]First significance: prebariatric vs preimplant; second significance: preimplant vs postimplant

Table 31.3 Metabolic evaluation after liposuction and abdominoplasty

Liposuction	Type	Follow-up	Response
14 overweight obese [27]	Large	4 months	Insulin resistance improved, lipids unchanged
45 obese [28]	Large	6 months	Lipids, inflammation improved
123 obese [29]	Large	3 months	Insulin and inflammation improved
15 obese [30]	Large	6 months	Insulin resistance improved
19 normal + obese [31]	Conv.	6 months	Total cholesterol decreased, LDL increased
15 overweight + obese [33]	Conv.	1 month	No inflammatory change, insulin resistance (?)
7 obese [34]	Large	4 years	No change
Abdominoplasty			
20 obese [25]	Conv.	40 days	Insulin resistance and inflammation diminished
9 normal [26]	Conv.	1 month	LDL and total cholesterol increased
20 obese [40]	Conv.	2 months	Reduced inflammation
Current study [38]	Circ.	2 years	C-reactive protein decreased

Author references are indicated in the first column
Conv. conventional technique, *Circ.* circumferential abdominoplasty

reconstruction. On the other hand, a pattern of nutritional stability was presented during the observation period (Table. 31.2).

On Table 31.3, literature data are presented regarding the impact of liposuction and abdominoplasty in several metabolic variables.

31.4 Discussion

In this investigation, the benefits of peripheral adipose tissue removal observed through lipid and glucose homeostasis markers have not been demonstrated, except for HDL cholesterol improvement. However, CRP also has diminished after abdominoplasty. Many mechanisms may be related to the conflicting results found in literature. The quantity of fat removed is generally higher in liposuction when compared to abdominoplasty. This allows for the conjecture of the existence of a factor that impedes the effects of liposuction fat removal on alteration of systemic markers. Table 31.3 shows that only one out of five high-volume liposuction series failed metabolically, with questionable results on both cohorts with more conservative procedures. On a similar manner, disappointing results were shown after omentectomy with the removal of 800 g of visceral tissue [40].

Another peculiarity is the simultaneous resection of fat involving patient's flanks and dorsum. This increase in surgical manipulation is not mandatory in abdominoplasty or liposuction, but it allows the attainment of a more adequate contour with higher esthetic results and may have as consequence a better relation among protective and deleterious adipocytes. There are no anatomical references easily identifiable that allow differentiation of a territory from another or precise limits for the region to be resected, with exception for genetic expression, immunoassays, and other cellular markers which are not available intraoperatively [41]. A third confusion variable is the weight variations after plastic surgery. Although, in this series, this bias was suppressed and both groups maintained a stable BMI during the observation period.

Thus, it was demonstrated in a study with a long-term follow-up period that postbariatric abdominoplasty reduces CRP levels, contributing for the reduction of this inflammatory marker with weight loss after bariatric surgery. Additional studies must be designed involving cytokines and other inflammatory markers, correlating particularly the inflammatory behavior after the removal of fat tissue. Also, prospective studies to compare the different abdominoplasty techniques (classic, anchor cut, circumferential) may be required.

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