

Serum C-Reactive Protein and White Blood Cell Count in Morbidly Obese Surgical Patients

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Abstract

Background Obesity has been widely recognized as a chronic inflammatory condition and associated with elevated inflammatory indicators including C-reactive protein (CRP) and white blood cell count (WBC). Recent studies have shown elevated CRP or WBC is a significant risk factor for cardiac events and stroke but the clinical significance of CRP and WBC has not been clearly studied in morbidly obese patients. This study is aimed at the clinical significance of WBC and CRP in morbidly obese patients and the change after bariatric surgery.

Methods The study was a prospectively controlled clinical study. From December 1, 2001 to January 31, 2006, of 640 (442 females and 198 males) consecutive morbid obese patients enrolled in a surgically supervised weight loss program with at least 1 year's follow-up were examined.

Results Of the patients, 476 (74.4%) had elevated CRP and 100 (15.6%) had elevated WBC at preoperative study. CRP and WBC were significantly related and both increased with increasing body mass index (BMI). CRP is also increased with increasing waist, glucose level, hemoglobin, albumin,

Ca, insulin, C-peptide, and metabolic syndrome while WBC is increased with metabolic syndrome but decreased with increasing age. Multivariate analysis confirmed fasting glucose level and hemoglobin are independent predictors of the elevation of CRP while age is the only independent predictor for elevated WBC. Both WBC and CRP levels decreased rapidly after obesity surgery. These improvements resulted in a 69.8% reduction of CRP and 26.4% reduction of WBC 1 year after surgery. Although individuals who underwent laparoscopic gastric bypass lost significantly more weight (36.8 ± 11.7 kg vs. 17.3 ± 10.8 kg; $p=0.000$) and achieved a lower BMI (27.8 ± 4.6 vs. 35.0 ± 5.5 ; $p=0.000$) than individuals who underwent laparoscopic gastric banding, there was no difference in the resolution of elevated CRP 1 year after surgery (95.9% vs. 84.5%; $p=0.169$) and WBC (99.4% vs. 98.3%; $p=0.323$).

Conclusions Both baseline WBC and CRP are elevated in morbid obese patients but CRP has a better clinical significance. Significant weight reduction 1 year after surgery markedly reduced CRP and WBC with a resolution rate of 93.9% and 98.2% separately. Obesity surgery performed by laparoscopic surgery is recommended for obese patients with elevated CRP or WBC.

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Introduction

Obesity and its associated comorbidities are an ongoing health-care problem worldwide [1, 2]. There is increasing evidence that obesity is a chronic inflammatory condition because some obesity comorbidities may be associated with inflammatory aspects of the disease [3, 4]. Both white blood cell (WBC) and C-reactive protein (CRP) are

commonly used systematic markers of inflammation and found to be elevated in obesity [5–7]. More importantly, WBC has been shown to be a predictor of coronary heart disease [8, 9] and CRP a strong independent predictor of risk for myocardial infarction and stroke [10]. However, the clinical significance and relative value of CRP and WBC has not been clearly studied in morbidly obese patients.

Weight loss is recommended for CRP reduction and benefits of moderate weight loss with conservative treatment have been described [11–13]. Marked reduction of CRP or WBC was also found after surgically induced weight loss in some preliminary studies [14–16], but not been adequately explored. The aim of this study was to explore, in a substantial number of severely obese patients enrolling in a surgically supervised weight loss program, the relationship between serum CRP and WBC, degree of obesity, basic anthropometry, and clinical and biochemical markers of the metabolic syndrome at baseline. Furthermore, we examined the efficacy of surgically induced weight loss on CRP and WBC count.

Materials and Methods

Patients

We studied 640 morbidly obese patients requiring surgical treatment (198 men, 442 women; median age, 31.3 (range, 18–64 years)), with a mean body mass index (BMI) of 41.5 (range, 32.0–77.0). The inclusion criteria for these patients were BMI higher than 32 and associated diabetes or other high-risk comorbidities for which nonsurgical treatment failed [17].

The individual components of MS (waist circumference; levels of serum glucose, high density lipoprotein cholesterol (HDL-C), and triglyceride; and blood pressure) as well as levels of total cholesterol, uric acid, aspartate aminotransferase (AST), alanine aminotransferase (ALT), albumin, complete blood cell count, and CRP were assessed at 1 day before surgery and at 1, 3, 6, 12 months postoperatively then once a year for routine postoperative outpatient visit. Body weight was measured in light clothing without shoes to the nearest 0.1 kg and body height to the nearest 0.1 cm. Body mass index was calculated as weight in kilograms divided by the height in meters squared. The waist circumference was measured at the level midway between the lateral lower rib margin and the superior anterior iliac crest. Criteria used to define metabolic syndrome comorbidities were considered according to ATP III definition with modification for Asia [18]. Patients were considered as affected by metabolic syndrome meeting three or more of the following criteria: (1) abdominal obesity, waist >90 cm in men and >80 cm in

women, (2) hypertriglyceride >149 mg/dl, (3) low-HDL cholesterol <40 mg/dl in men and <50 mg/dl in women, (4) high blood pressure >130/85 mmHg, and (5) high fasting glucose >109 mg/dl.

Blood was drawn for laboratory examination from an antecubital vein following an overnight fast. The test was measured on the Hitachi 7170 and J&J nephelometer assay (DEDE BEHRING, USA). Serum insulin and C-peptide level were measured on DPC IMMULITE analyzer. All specimens were stored at –30°C and processed within 24 h of collection.

Descriptive data are expressed as mean±SD. Variables were assessed for correlation using Pearson parametric analysis. Variables were assessed within their groupings as described above after controlling for age and sex. Then, variables from groups were modeled together to assess for independent variables that were associated with variance in the CRP level. Group characteristics were compared by unpaired *t*-tests, and differences between proportions were tested by χ^2 tests. The statistic analysis was performed using SPSS 8.0 version for Windows (SPSS Inc., Chicago, IL, USA). Statistical significance was inferred at a two-tailed *p* value of less than 0.05.

Interventions

All the patients in the study group received laparoscopic weight reduction surgery at our center. The surgical procedures performed were laparoscopic adjustable gastric banding (LAGB; using Lap Ban from Bioenterics Corp., Carpinteria, CA, USA) and laparoscopic minigastric bypass (LMGBP). The techniques of LAGB and LMGBP are described in detail elsewhere [19, 20]. We performed LAGB in 118 patients (18.4%) and LMGBP in 522 (81.6%). Patients could choose either procedure by themselves. There was no significant difference between the two different procedure groups except more male patients choose the banding procedure (Table 1). There was no operation mortality in this series. Nine patients (1.4%) developed major complications and 21 (3.3%) minor complications.

Table 1 Compare gastric bypass and gastric banding patients on clinical factors

	Bypass (<i>n</i> =522)	Banding (<i>n</i> =118)	<i>p</i> value
Age, years	31.2±8.9	31.81±9.1	0.525
Sex, M/F	141/381	57/61	0.000*
BMI (kg/m ²)	41.3±6.6	42.0±7.1	0.310
WBC (10 ³ /μl)	8.8±3.6	8.4±2.3	0.318
CRP (mg/dl)	0.9±1.1	0.8±1.1	0.538

BMI Body mass index, *SBP* systolic blood pressure, *DBP* diastolic blood

**p*<0.05

Results

Baseline Analysis

Of 640 consecutive individuals enrolled in the obesity surgery program, 476 (74.4%) had elevated CRP levels (>0.3 mg/dl) and 100 (15.6%) had elevated WBC (>10.8×10³/μl). The average preoperative CRP level was 0.84±1.1 mg/dl (range 0.0148–13 mg/dl) and WBC count was 8.7±3.4 (range 2.7–16.3×10³/μl). Both WBC and CRP increased continually with an escalating of BMI (Fig. 1). The prevalence of CRP elevation increased from 49.5% in those BMI above 32% to 93.1% in those BMI >50. The prevalence of leukocytosis increases from 6.5% to 22.1%. In correlation study, baseline CRP was significantly associated with ten clinical variables, including waist, BMI, glucose, albumin, Ca, hemoglobin, insulin, C-peptide, metabolic syndrome, and WBC as shown in (Table 2). WBC was associated with four clinical variables, including age, BMI, metabolic syndrome, and CRP. A significant positive linear correlation of WBC and CRP is shown in Fig. 2. Multivariate analysis confirmed fasting glucose level and hemoglobin are independent factors associated with CRP while age is the only independent factor associated with WBC (Table 3).

Changes after Surgery

After surgical therapy, a decrease in CRP level and WBC count occurred at 1 month and persisted to 2 years (Fig. 3). The decreasing of CRP is more rapid than the decreasing of WBC. Six months after surgery, the mean CRP level has been lower than 0.3 mg/dl. One year after surgery, the mean CRP level was 0.2±0.4 mg/dl and WBC count was 6.4±1.6 (×10³/μl). These resulted in a 69.8% reduction of CRP and 26.4% reduction of WBC. One year after surgery, only 30

Table 2 Correlation study of CRP and WBC with various clinical factors

	Mean±SD	R (CRP)	R (WBC)
Age, years	31.3±8.9	0.007	-0.167**
Sex, M/F	198/442	-0.078	0.049
Waist (cm)	119.1±16.2	0.135**	0.057
BMI (kg/m ²)	41.5±6.7	0.159***	0.100*
SBP (mmHg)	133.1±16.9	0.060	-0.010
DBP (mmHg)	83.3±12.9	0.061	-0.037
Glucose (mmol/l)	6.1±2.3	0.111**	0.005
T-cholesterol (mmol/l)	196.0±36.7	0.014	0.031
HDL-C (mmol/dl)	44.8±13.9	-0.091	-0.066
Triglyceride (mg/dl)	175.8±156.9	0.014	-0.006
Uric acid (mg/dl)	8.1±16.4	0.014	0.034
AST (IU/l)	35.8±28.2	0.072	-0.007
ALT (IU/l)	52.6±44.6	0.022	-0.001
GGT (IU/l)	46.3±35.4	0.088	0.048
Albumin (gm/dl)	5.5±20.9	0.078*	0.068
ALP (IU/l)	73.5±21.5	0.077	-0.013
Ca (mg/dl)	9.1±1.5	0.307***	0.061
Hemoglobin (gm/dl)	14.0±3.3	0.154***	0.043
Insulin (pmol/l)	26.3±35.4	0.087*	0.076
C-peptide (mmol/l)	5.1±8.0	0.088*	0.048
HbA1C (IU/l)	6.1±2.4	0.060	0.026
Metabolic syndrome ^a	1.8±1.1	0.121**	0.091*
WBC (10 ³ /μl)	8.7±3.4	0.100*	1.000
CRP	0.8±1.1	1.000	0.100*

BMI Body mass index, SBP systolic blood pressure, DBP diastolic blood pressure, HDL high density lipoprotein cholesterol, MCV mean cellular volume of red blood cell, ALP alkaline phosphatase, GGT gamma-glutamyl transferase

*p<0.05, **p<0.01, ***p<0.001

^aNumbers of involved components of metabolic syndrome

(6.1%) patients had elevated CRP levels (>0.3 mg/dl) and four (1.7%) patients had leukocytosis. The accumulated percentages of resolution of CRP elevation were 12.7%, 56.7%, 78.4%, 93.9%, 98.5% at 1,3, 6, 12, and 24 months after surgery, respectively. The change of WBC after

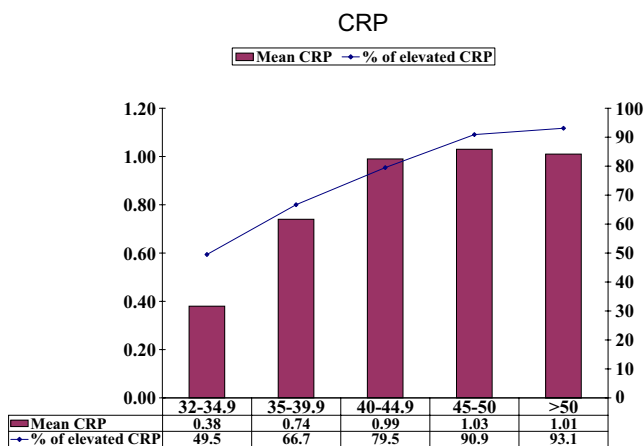


Fig. 1 Mean CRP, WBC, and prevalence (%) of elevated CRP in 640 severely obese patients across quintiles of BMI

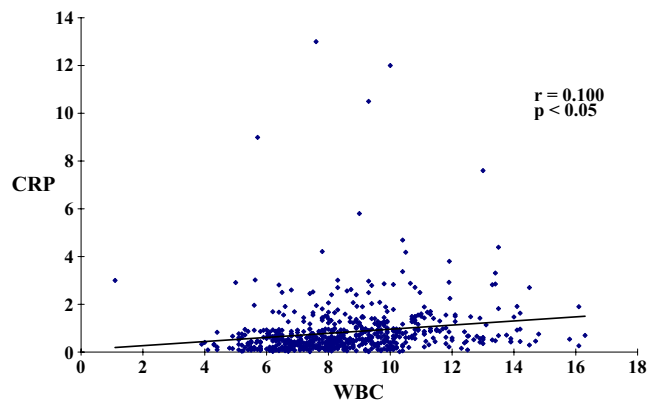


Fig. 2 CRP levels as a function of WBC

Table 3 Multivariate analysis of risk factors for CRP and WBC

Factor	95% CI	p value
CRP		
BMI	-0.008–0.038	0.195
Waist	-0.005–0.014	0.317
Glucose	0.001–0.006	0.005*
Alb	-0.036–0.018	0.511
WBC	-0.008–0.040	0.185
Insulin	-0.005–0.004	0.856
C-peptide	-0.008–0.015	0.504
Ca	-0.226–0.217	0.967
Hemoglobin	-0.162–0.032	0.003*
Metabolic syndrome	-0.021–0.176	0.123
WBC		
Age	-0.094–0.033	0.000*
BMI	-0.010–0.073	0.137
Metabolic syndrome	-0.094–0.398	0.225
CRP	-0.026–0.475	0.079

surgical therapy was similar to the CRP but less persisted. The percentage of reduction for CRP and WBC 1 year after surgery was 69.8% and 26.4%, separately. The resolution rate for elevated CRP and leukocytosis 1 year after surgery was 93.9% and 98.3%, separately.

The weight loss was significantly greater in the gastric bypass group than the gastric banding group at all follow-up intervals (Fig. 4). However, both groups had achieved a significant reduction of CRP and mean CRP level lower than 0.3 mg/dl (Fig. 5). The bypass group had a lower CRP level than the band group 1 year after surgery but without statistical significance (0.1 ± 0.5 vs. 0.2 ± 0.2 ; $p=0.169$). There was also no difference in the resolution of elevated CRP 1 year after surgery between the bypass and band groups (95.9% vs. 84.5%; $p=0.169$). The resolution of leukocytosis for the gastric bypass and gastric banding was 99.4% vs. 98.3%; $p=0.323$.

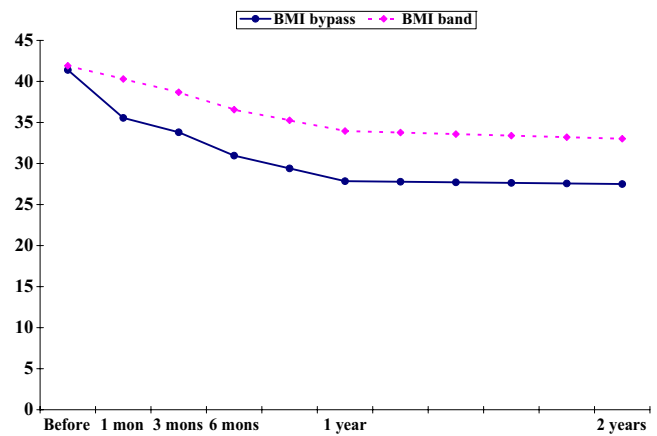


Fig. 4 Change in body mass index (BMI; kg/m²) after bypass and banding surgery

Comment

In this study, we found both chronic inflammatory indicators, WBC and CRP, are commonly elevated in morbid obese surgical patients. Although both are correlated and increasing with the escalating of BMI, CRP is more sensitive than WBC in predicting the comorbidities associated with obesity. There were ten significant clinical variables associated with CRP while only four associated with WBC. Multivariate analysis confirmed hyperglycemia and hemoglobin are independent predictors of elevated CRP. Although WBC in this study did increase with increasing BMI and associated with metabolic syndrome as shown in previous studies [8, 9, 21], multivariate analyses confirmed that age is the only independent predictors. This finding corroborated with a recent study from Dixon et al. [16]. Therefore, we recommended CRP as a useful clinical indicator of chronic inflammatory state of morbid surgical patients and might be included in preoperative assessment.

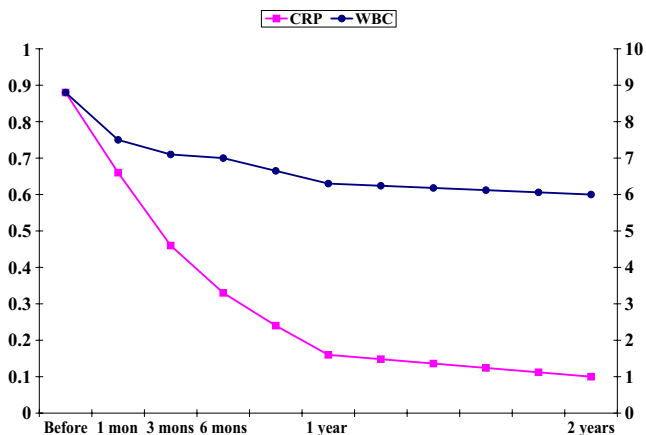


Fig. 3 Change in WBC and CRP level after surgery

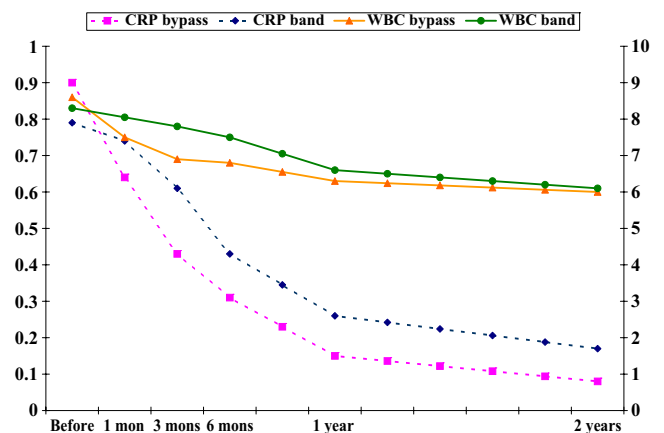


Fig. 5 Changes in WBC and CRP level after bypass and banding surgery

Recent findings suggest that adipous tissue is the underlying feature of elevated CRP by generating inflammatory signals and connect obesity with chronic inflammatory state [10, 21]. The CRP was found to be consistently elevated as the BMI increasing up in our study and others. In a large epidemiological study, Visser et al. have found elevated CRP levels in 20% of those with a BMI <25 kg/m² and elevated values in 35% of those with a BMI 25–29.9 kg/m² [6]. Our study disclosed elevated CRP levels in 49.5% in those with a BMI 32–34.9 kg/m² and escalated increasing to 93.1% in those with a BMI >50 kg/m² corroborated with the previous study. However, this study have shown fasting glucose level and hemoglobin is independent indicators of elevated CRP rather than BMI itself, implicating that obesity associated comorbidities probably are more important than obesity itself for elevated CRP in morbidly obese patients. This is an important finding and had not been reported previously.

Obesity, especially central obesity, is known to associate with insulin resistance, glucose intolerance, and type 2 diabetes. Therefore, it is not a surprise to find that fasting glucose level is independently associated with elevated CRP. However, the relationship between hemoglobin and CRP is more interesting and an important finding in this study. The reason for hemoglobin to associate with elevated CRP is probably the secondary effect of obesity hypoventilation syndrome. Obesity hypoventilation syndrome and associated sleep apnea is a common morbidity associated with severe obesity. Because obesity associated sleep apnea will bring morbid obese patient into a chronic hypoxia state, these patients may have induced erythropoiesis and secondary polycythemia. The elevated hemoglobin level probably reflected the severity of obesity hypoventilation syndrome and the risk of sudden death as well. Therefore, morbid obese patients with polycythemia may highlight the severity of sleep apnea and chronic hypoxia. Because previous study has shown that CRP is elevated in obstructive sleep apnea [22] and effective continuous positive airway pressure (CPAP) therapy can reduced the elevated CRP in severe obstructive sleep apnea [23], it is recommended that special attention should be paid to these patients including perioperative monitor of sleep apnea and using CPAP for the prevention of sudden death.

CRP is a sensitive acute phase reactant [24]. It may induce adhesion molecules and production of monocyte chemotactic factors in endothelial cells, thereby enhancing local inflammatory responses within atherosclerotic plaques with recruitment of monocytes and lymphocytes [25]. CRP also binds low-density lipoproteins (LDL), which are taken up by macrophages at the damaged endothelial sites to create foam cells or soft plaque. Elevated CRP has been found to be a strong independent predictor of risk for cardiac events and stroke, associated with a five-fold

increase in the risk of any vascular event and a seven-fold increase in the risk of myocardial infarction or stroke [5]. Because elevated CRP is a great threaten to the health, aggressive treatment in order to reduce CRP should be considered in severe obese patients associated with elevated CRP. Changes in body weight and in total body fat mass were positively associated with plasma CRP reductions. Several studies reported decreased CRP level with nonsurgical weight reduction [11–13]. Medical treatment, especially with statin to reduce the LDL, has also been proven to be effective in reducing the elevated CRP in patients with metabolic syndrome [26]. However, for severe obese patients, surgery has been proven to be more effective and long lasting than medical treatment [27, 28]. Recent studies have also reported significant and rapid reductions in BMI and CRP levels after obesity surgery [14, 15]. Our previous study also showed a high percentage of metabolic syndrome in severe obese Taiwanese [29] and a dramatic resolution of metabolic syndrome in severe obese patients after obesity surgery [30]. These studies support the finding that marked weight reduction after obesity surgery might reverse the proinflammatory state and reduce the risk for morbidity and mortality in these subjects [14–16].

With more safe obesity surgery, such as gastric banding, obesity surgery will be used more freely as an effective treatment for severe obese patients with comorbidities or elevated CRP. Although less effect in weight reducing than gastric bypass surgery, this study confirmed that gastric banding is equally effective in reducing CRP and WBC of morbidly obese patients. Current indications for obesity surgery were based on BMI, including BMI >40 kg/m² or >35 with comorbidities. The BMI is an estimate of total body fat mass and is probably the most useful scale to define obesity. The finding in this study that higher BMI is associated with higher CRP also supports the using of BMI as indication of obesity surgery. However, Asian patients tend to have similar incidence of obesity related metabolic abnormalities at lower BMI level comparing to Caucasians [31]. It is therefore that WHO consensus group had used a BMI above 30 to define obesity and the Asia-Pacific perspectives suggested a BMI >25 to define obesity in Asian people [32]. The Asia-Pacific Bariatric Surgery Consensus had also modified the criteria for obese surgery down to BMI 32 with comorbidities [17]. Because the safety and efficacy of laparoscopic obesity surgery, some reports have now advocated the indication can be lowered to 30 [28, 33]. Further study is indicated to confirm the advantage of using bariatric surgery at lower BMI patients with elevated CRP.

In conclusion, elevated CRP and WBC were commonly findings in severely obese individuals but CRP has a better clinical implication than WBC. The finding that glucose and hemoglobin level is independent predictor of elevated

CRP supports the theory that obesity associated comorbidities play a key role in CRP elevation. Significant weight loss 1 year after laparoscopic bariatric surgery shows that the procedure was an extremely effective treatment for reducing the body weight and CRP. Additional clinical research is needed to evaluate the cost effectiveness of obesity surgery in the treatment of individuals with moderate obesity and elevated CRP.

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