

Rhabdomyolysis Following Bariatric Surgery: a Retrospective Analysis

Francesco Forfori¹, Anna Sidoti^{1,*}, Riccardo Taddei¹, Nora Terrasini¹, Erik Arbeid¹, Dario Gregori² and Francesco Giunta¹

¹Department of Anaesthesia, University of Pisa, Italy

²Unit of Biostatistics, Public Health and Epidemiology, Dept. Cardiology, Thoracic and Vascular Sciences, University of Padua, Italy

Abstract: *Background:* Rhabdomyolysis (RML) indicates a skeletal muscle necrosis which results in an emission of intracellular contents from myocytes into the circulatory system. It has been recognized to be a complication of bariatric surgery. A high BMI and a prolonged operative time are the main risk factors associated to the development of RML. The aim of this study is to define the incidence and the main features of RML in a cohort of obese patients undergoing bariatric surgery.

Materials and Methods: a retrospective observational analysis was carried out on 100 patients undergone bariatric surgery. The bariatric operations were open or laparoscopic Roux-en-Y gastric bypass (RYGB) and laparoscopic sleeve gastrectomy (SG); they were performed at the university hospital Nuovo S. Chiara in 2011.

Results: 6 of 100 patients enrolled developed RML. Three of these also showed acute kidney injury due to RML (50%). A significant correlation between post-operative increased creatine phosphokinase (CPK) levels and BMI was found ($r=0.369$; $r^2=0.137$; $p=0.005$) as well as a significant correlation between the increased levels of creatine phosphokinase, myoglobin and operative time (increased operative time - highest CPK: $r=0.550$; $r^2=0.302$; $p<0.0001$; increased operative time - highest myoglobin: $r=0.553$; $r^2=0.305$; $p<0.0001$). Moreover, hypertension and prolonged operative time were found to be variables associated with RML in bivariate analysis.

Conclusions: The incidence of RML following bariatric surgery was 6%. Since rhabdomyolysis is the most important complication of bariatric surgery, appropriate precautions should be taken during surgery. Post-operative monitoring of CPK and myoglobin levels is essential for an early diagnosis of RML.

Keywords: Acute Kidney Injury, Bariatric surgery, Body Mass Index, Creatine Phosphokinase, Operative Time, Rhabdomyolysis.

INTRODUCTION

Obesity is an epidemic world health problem which affects over 200 million of men and nearly 300 million of women, in both developed and developing countries [1]. Obesity and overweight are related to several complications such as dyslipidemia, diabetes mellitus, risk of coronary diseases, arterial hypertension, sleep apnea syndrome, musculoskeletal disorders, psychological disorders and some forms of cancer [1]. Obesity treatments involve changes of lifestyle, diet, regular physical exercise, psychological support, medications and in the case of severe obesity, bariatric surgery (BS) may be indicated. Surgery is proposed to treat obesity when body mass index (BMI) is more than 40 kg/m² (morbid obesity) or more than 35 kg/m² in the presence of co-morbidities [2]. After bariatric surgery, several complications such as gastrointestinal leaks, local infections, thromboembolism and atelectasis may occur [3].

Rhabdomyolysis (RML) is a potentially life threatening disorder characterized by the necrosis of skeletal muscle resulting in the release of intra-cellular muscle components into the circulation and interstitial space; it has been recognized to be a complication of bariatric surgery. RML ranges from an asymptomatic state with increased CPK, lactate dehydrogenase (LDH) and myoglobin levels to a potentially life threatening condition associated with acute renal failure (ARF), electrolyte imbalance, disseminated intravascular coagulation (DIC) and cardiac arrest [4]. RML has several causes: muscle trauma is the most common, instead the use of drugs (statins, for instance), prolonged immobilization, excessive muscular activity, electrolyte imbalances, infection, toxins, seizure and genetic disorders are less common causes [5]. The pathogenesis follows a common final pathway based on myocyte destruction which originates from membrane Na⁺/K⁺ ATPase and Ca²⁺ ATPase pumps dysfunction caused by ATP depletion. The accumulation of sodium in the cytoplasm leads to an increase in the intracellular calcium concentration, which increases the activity of intracellular proteolytic enzymes degrading muscle cells; this condition promotes the release

*Address correspondence to this author at the Department of Anaesthesia, University of Pisa, Italy; Tel: 0039 050997608; Fax: 0039 050997607; E-mail: anna.sidoti1@gmail.com

of myoglobin, CPK, potassium, LDH and aspartate transaminase into the blood. When the levels of circulating myoglobin exceed the protein-binding capacity of the blood, myoglobin can precipitate into renal glomeruli causing acute kidney injury (AKI), an important and frequent complication of RML [4-8], which occurs in 33 % of patients with rhabdomyolysis, associated with a mortality of 20% [5]. Several mechanisms are involved in kidney damage during RML: i) hypoperfusion due to hypovolemia caused by a shift of circulating fluids into damaged muscle; the fluid shift causes an intravascular hypovolemia that leads to renal vasoconstriction and pre-renal failure [5]; ii) tubular necrosis, caused either by precipitation of myoglobin in the tubuli or to the direct nephrotoxic effect of ferriheme which is released from myoglobin when urinary pH falls below 5.6 [5, 6]; heme proteins induce a synergic effect on renal vasoconstriction along with the hypovolemia and the activation of the cytokine cascade [9]. That being so, an aggressive intravenous hydration is essential to preserve renal function in the treatment of RML; the administration of mannitol (in order to maintain urine output > 200 ml/h) and sodium bicarbonate (in order to maintain urinary pH = 6.5), despite their common use, remains unable to prove benefit [4]. RML has been recognized as a complication of bariatric surgery. Recently, several retrospective and prospective studies described such complication in obese patients undergoing bariatric or general surgery [3, 10-12]. Probably RML following bariatric surgery is caused by a muscle pressure occurring during surgery which results in the creation of a compartmental syndrome [12]. The high BMI, the duration of operations, the lithotomy position, the ASA (American Society of Anesthesiologists) physical status III-IV, the presence of comorbidities such as hepatic steatosis, hypertension and diabetes mellitus [3, 11-14], are risk factors associated with the development of RML. The incidence of post-operative RML has been estimated from a minimum of 1.4% [15] to a maximum of 75% [10]. The CK levels to diagnose RML vary among authors, some of them accept CPK > 1000 UI/l (five times the normal value); other ones accept CPK > 5000 UI/l and even > 10000. Lower values are safer for an early diagnosis in order to avoid RML complications [16]. Surely, RML prevention is very important and it may avoid negative patient outcome. For this reason, during all phases of bariatric surgery it is essential to actualize some precautions which can help to preserve muscle integrity: padding of pressure areas, use of pneumatic beds during operations, optimal position of the surgical table, reduction of surgical time, change of patient position intra- and postoperatively and encouraging patients to an early ambulation [14]. Furthermore, it is possible to early diagnose RML after surgery by monitoring CPK and myoglobin levels in order to prevent and promptly treat its complications (hypovolemia, acidosis, hyperkalemia, AKI, DIC and hepatic dysfunction) [11, 14].

The aim of this study is to determine the incidence and the main characteristics of RML in a cohort of obese patients undergoing bariatric surgery.

MATERIALS AND METHODS

A retrospective observational analysis was carried out on 100 obese patients undergone bariatric surgery. The

operations took place at the third-level teaching hospital Nuovo S. Chiara, situated in Pisa, in 2011. After surgery, all the enrolled patients were admitted to intensive care unit (ICU) for postoperative monitoring. The length of stay was of 24 hours for the patients who did not develop any complication, and more for those who developed RML (48, 72 or more hours according to cases). Appropriate permission was obtained by the Local Ethical Committee. Standardized data of all patients were collected and inserted in a database. The variables recorded were: age, gender, weight, BMI, co-morbidities (i.e., hypertension, diabetes mellitus), therapies (e.g., statins), type of operation, operative time, intra-operative fluid balance, pre-operative and post-operative serum CPK, post-operative serum myoglobin, postoperative serum creatinine. AKI was defined according to the AKIN classification [17]; the diagnosis was performed within a 48-hour period through the comparison of serum creatinine levels at ICU admission to their values 48 hours later.

STUDY SAMPLE

The research encompassed male and female patients from 24 to 72 years, undergone either RYGBP or SG. All patients were monitored in ICU for at least 24 hours after surgery. The patients with renal failure were excluded. The established upper limit of normal CPK was 190 IU/l and that of myoglobin was 58 ng/ml. RML was defined in the presence of postoperative values > 950 IU/l (five times the upper normal limit). All data of the patients undergone bariatric surgery are presented in Table 1.

STATISTICAL ANALYSIS

Data have been reported using median or percentage. Comparisons between groups were performed using the Mann-Whitney test for continuous variables and the Chi-square test for categorical variables. Pearson's correlations were calculated to quantify associations between continuous variables and a bivariate analysis was performed in order to identify risk factors associated to RML. Chi-square test was also used to compare RML group *versus* no-RML group. A 2-sided p value < 0.05 was considered statistically significant. For statistical analysis, we used XL stat program (version 7.5).

RESULTS

A population of 100 patients who underwent bariatric surgery (open or laparoscopic RYGB and laparoscopic SG) was retrospectively studied. The overall intra- and postoperative data are shown in Table 1. Six of them (6%) developed RML, 3 of this group developed consequently AKI (two patients in stage 1 and one patient in stage 3). Data of the patients with RML are shown in Table 2. Patients classification was established according to creatinine-based AKIN classification. Table 3 shows a comparison between patients who developed post-operative RML and patients who did not. There were substantial differences between the two groups; in fact, patients who developed RML had a higher BMI ($60.3 \pm 14.6 \text{ kg/m}^2$ *versus* $48 \pm 7 \text{ kg/m}^2$), a higher incidence of diabetes mellitus (67% *versus* 43%) and a higher incidence of arterial hypertension (100% *versus* 57.8%)

Table 1. Intra - and Postoperative Data of the Patients Undergoing Bariatric Surgery

Sample Characteristics	Mean or Percentage	SD
Age (years)	24/ 47/ 72	11
BMI (kg/m ²)	30/ 48,7/ 85	8.26
Diabetes Mellitus	44%	
Arterial Hypertension	61%	
Preoperative CPK (IU/ml)	21/ 130.5/ 410	90
Undergoing Laparoscopic RYGB	75%	
Undergoing Laparothomic RYGB	7%	
Undergoing Laparoscopic Sleeve Gastrectomy	18%	
Operative time (hours)	1/ 2,78/ 7	0,87
Intraoperative Fluid Balance (ml)	-3600/ -579/ +1900	996
Post-operative Fluid balance: first 24 hours after surgery (ml)	-1525/ 858/ +3490	1139
CPK ICU admission (IU/l)	60/ 197/ 886	134
CPK 1st day after surgery (IU/l)	62/ 852/ 1854	3013
Myoglobin ICU admission (ng/ml)	41/ 160/ 2471	252
Creatinine ICU admission (mg/dl)	0.51/ 0,8/ 1.33	0,2
Creatinine 1st day after surgery (mg/dl)	0,43/ 0,75/ 1,53	0,29
Theraphy with Statins	0,1	

Table 2. Data of the Patients who Developed Post-Operative RML

Sample Characteristics	Mean or Percentage	SD
Age (years)	44/ 50/ 56	5
BMI (kg/m ²)	45/ 60.3/ 85	14.6
Diabetes Mellitus	0,67	
Arterial Hypertension	1	
Preoperative CPK (IU/ml)	82/ 97/ 113	16
Undergoing Laparoscopic RYGB	1 (16.6%)	
Undergoing Laparothomic RYGB	4 (67%)	
Undergoing Laparoscopic Sleeve Gastrectomy	1 (16.6%)	
Operative time (hours)	2/ 5/ 7	2
Intraoperative Fluid Balance (ml)	-2500/ -287/ +1546	1297
Post-operative Fluid balance: first 24 hours after surgery (ml)	212/ 1232/ 3490	1371
Post-operative Fluid Balance: 1st post-operative day (ml)	925/ 1688/ 2275	692
CPK ICU admission (IU/l)	86/ 341/ 758	266
CPK 1st day after surgery (IU/l)	374/ 6738/ 18544	7473
CPK 2° day after surgery IU/l)	1222/ 7180/ 13616	5925
Myoglobin ICU admission (ng/ml)	150/ 703/ 2471	997
Myoglobin 1st day after surgery(IU/l)	534/ 5631/ 17967	8293

Table 2. contd...

Sample Characteristics	Mean or Percentage	SD
Myoglobin 2° day after surgery (IU/l)	561/ 1677/ 3455	1556
Creatinine ICU admission (mg/dl)	0.58/ 0.89/ 1.16	0.19
Creatinine 1st day after surgery (mg/dl)	0.65/ 1.12/ 1.53	0.33
Creatinine 2° day after surgery(mg/dl)	0.71/ 1.38/ 1.92	0.52
Therapy with Statins	0	

Table 3. Comparison Between Patients without Post-Operative RML *Versus* Patients with Post-Operative RML

Sample Characteristics	Without RML– Mean±SD or Percentage	With RML– Mean±SD or Percentage
N	94	6
Age (years)	47±11	50±5
BMI (kg/m ²)	48±7	60.3±14.6
Diabetes Mellitus	41(43%)	4 (67%)
Arterial Hypertension	55 (57.8%)	6(100%)
Preoperative CPK (IU/ml)	132±92	97±16
Undergoing Laparoscopic RYGB	68 (72.3%)	1(16.6%)
Undergoing Laparothomic RYGB	3 (3.19%)	4 (67%)
Undergoing Sleeve Gastrectomy	23(24.4%)	1 (16.6%)
Operative time (hours)	3±1	5±2
Intraoperative Fluid Balance (ml)	-602±976	-287±1297
Post-operative Fluid balance: first 24 hours after surgery (ml)	+783±1105	+1232±1371
Post-operative Fluid Balance: 1st post-operative day (ml)		+1688±692
CPK ICU admission (IU/l)	187±116	341±266
CPK 1st day after surgery (IU/l)	153±94	6738±7473
CPK 2° day after surgery IU/l)		7180±5925
Myoglobin ICU admission (ng/ml)	130 ± 69	703±997
Myoglobin 1st day after surgery(IU/l)	66±52	5631±8293
Myoglobin 2° day after surgery (IU/l)		1677±1556
Creatinine ICU admission (mg/dl)	0.78 ± 0.2	0.89±0.19
Creatinine 1st day after surgery (mg/dl)	0.68±0.22	1.12±0.33
Creatinine 2° day after surgery(mg/dl)		1.38±0.52
Therapy with Statins	10%	0%

Table 4. Results of Bivariate Analysis

	N	Without RML	With RML	Odds ratio	CI 95%	P value
Without DM	56	53 (94.6%)	3 (5.3%)			
With DM	44	41 (93%)	3 (6.8%)	0,774	0,418–4,034	0,7
Without Hypertension	39	39 (100%)	0			

Table 4. contd...

	N	Without RML	With RML	Odds ratio	CI 95%	P value
With Hypertension	61	55 (90%)	6 (9.8%)	0		0,043
BMI						
35-40	14	14 (100%)	0			
41-50	48	46 (96%)	2 (4%)			
≥ 51	38	34 (89.4%)	4 (10.6%)	2,706	0,468-15,638	0,25
Operative time						
≤ 2	34	33 (97%)	1 (3%)			
3-4	58	57 (98%)	1 (2%)			
≥5	7	3 (43%)	4 (57%)	76	6,365-907,409	<0,0001
Intra-operative fluid balance						
Positive	23	21 (91%)	2 (9%)			
Negative	77	73 (95%)	4 (5%)	0,575	0,098-3,362	0,53

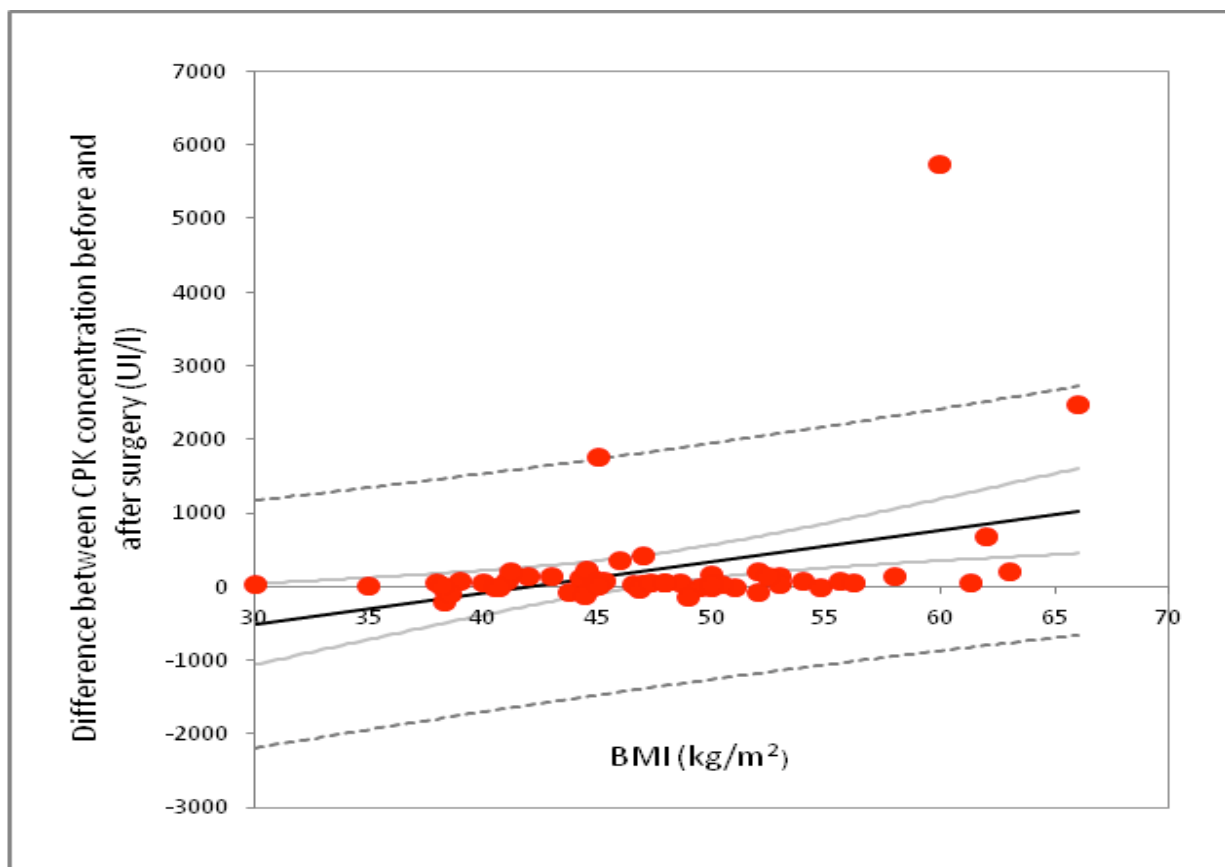


Fig. (1). Correlation between BMI and difference between pre-operative and post-operative CPK. $r=0,369$; $r^2=0,137$; $p=0,005$. The post-operative increase of CPK was positively correlated with BMI.

compared to no-RML group. Furthermore, operative time in RML group was higher compared to no-RML group (5 ± 2 hours *versus* 3 ± 1 hours). The majority of patients who developed RML underwent laparotomic RYGB (67%). A significant correlation between post-operative increase of

CPK levels and BMI was found (Fig. 1). As the operative time increased the values of post-operative CPK increased (Fig. 2), reaching the highest values with the longest operative time (Fig. 3). A positive correlation between post-operative myoglobin levels and operative time was shown as

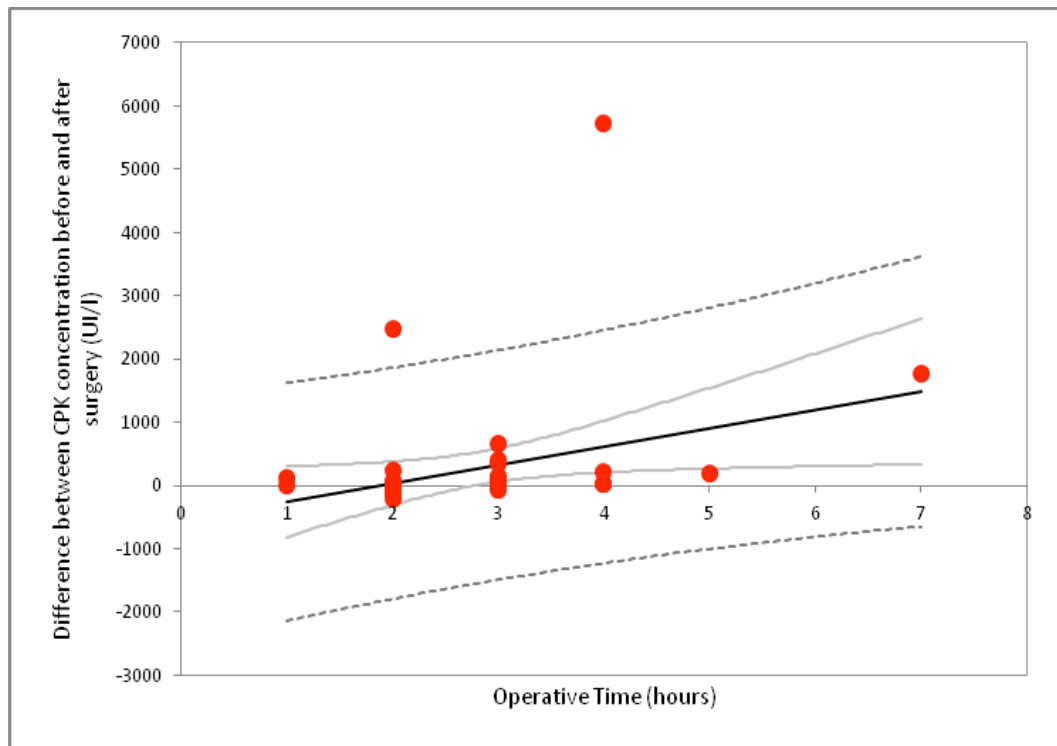


Fig. (2). Correlation's analysis between operative time and difference between pre-operative and post-operative CPK. $r=0,308$; $r^2=0,095$; $p=0,024$. The post-operative increase of CPK was positively correlated with operative time.

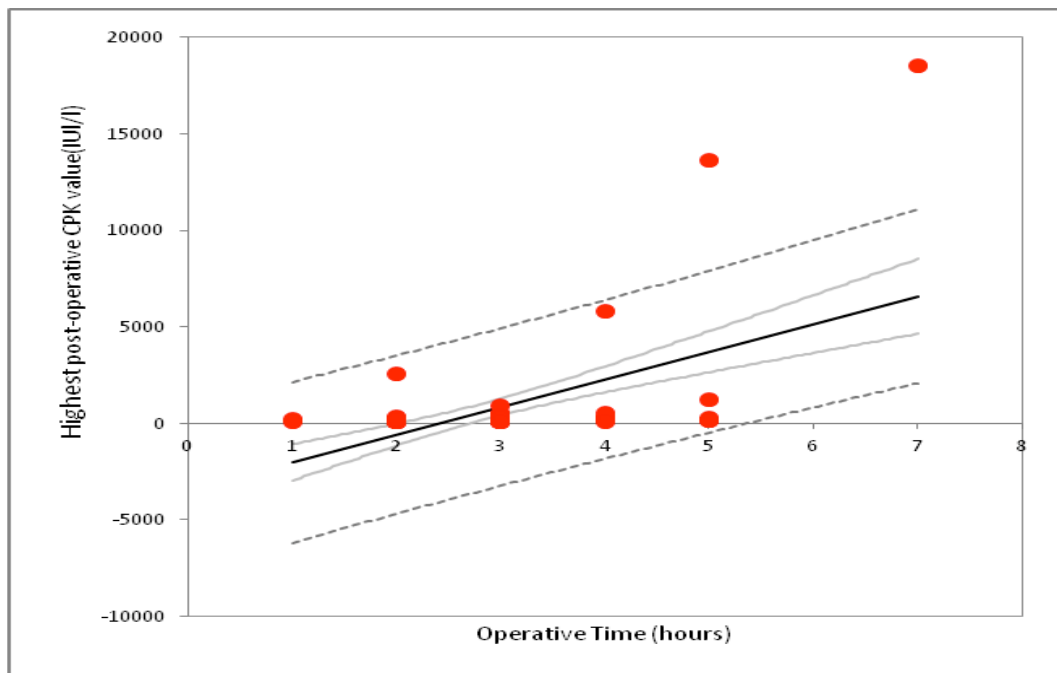


Fig. (3). Correlation between operative time and highest post-operative CPK value. $r=0,550$; $r^2=0,302$; $p<0,0001$. The highest post-operative CPK value was positively correlated with operative time.

well (Fig. 4). On the other hand, no significant association between intra-operative fluid balance and post-operative high CPK and myoglobin levels was found. The comparison of post-operative CPK and myoglobin concentrations between patients undergone RYGB *versus* SG by using the Mann–Whitney test, showed no significant differences. In bivariate analysis, the presence of arterial hypertension and a prolonged operative time were found to be associated with

RML (Table 4). In our study, 57% of patients whose surgical time was ≥ 5 hours developed RML.

DISCUSSION

The incidence of rhabdomyolysis among the patients involved in our study was 6%. This result is consistent with Ettinger *et.al* [11] who described an incidence of 7% and in

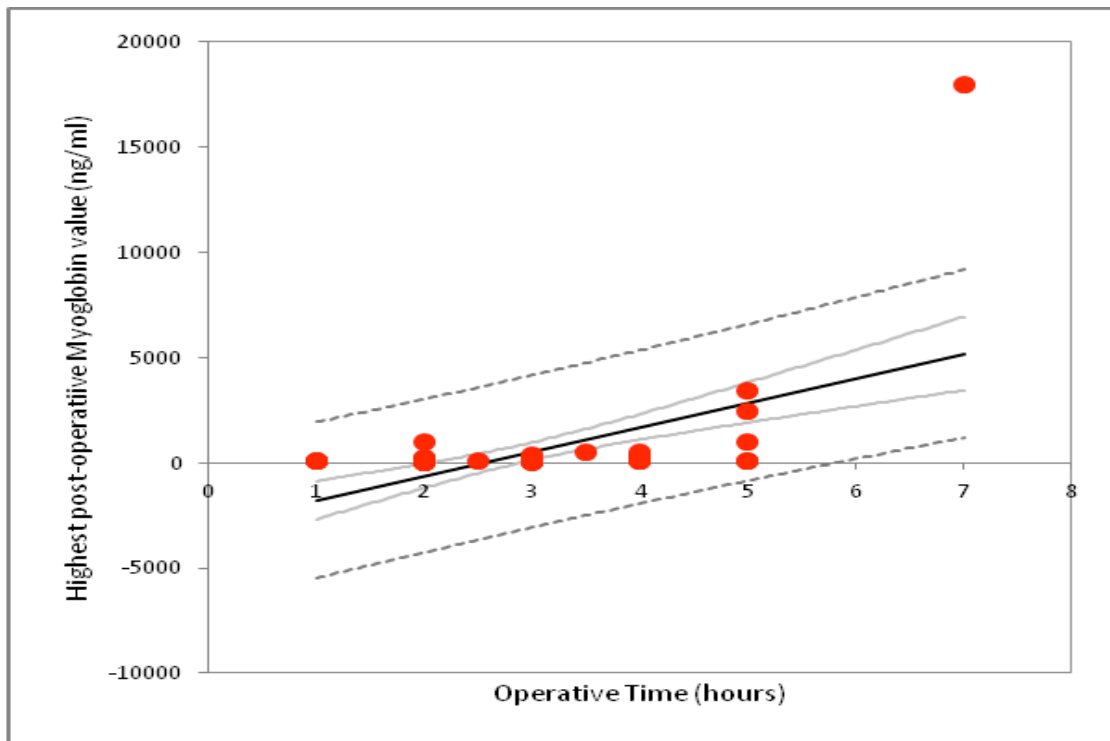


Fig. (4). Correlation between operative time and highest post-operative myoglobin value. $r=0,553$; $r^2=0,305$; $p<0,0001$. The highest post-operative myoglobin value was positively correlated with operative time.

contrast with Mognol *et al* [12] and Lagandré *et al.*[3], who reported an incidence of 22.7% and 26% respectively. A prolonged operative time is an important risk factor for the development of post-operative RML [3, 11, 12, 14], indeed in our study, almost all the patients who developed RML had operative time ≥ 4 hours (5 out of 6, 83%). High BMI and arterial hypertension are potential risk factors for the development of RML. These results are consistent with Ettinger *et al.* who found a BMI ≥ 51 kg/m² and a high arterial pressure to be risk factors for RML [11]. Obese patients may have peripheral vascular disease, which predisposes to the development of a compartment syndrome [12], and arterial hypertension increases the risk of RML due to the microvascular alterations associated [14]. Even though we did not identify any significant correlation between diabetes mellitus (DM) and RML, we cannot completely exclude a possible influence of DM in the displaying of RML since 67 % of the patients who developed RML had DM. Lagandré *et al.* [3] and Mognol *et al.* [12] identified DM as a risk factor for the development of RML.

None of the patients who developed RML had statin therapy: 10 % of the patients enrolled used chronically statins, but none of them developed RML. As a result, therapy with statins does not seem to be a risk factor for the development of RML. This result is consistent with the study of Bostanjian *et al.* [18] who demonstrated that statin therapy is not related to the development of RML. On the other hand, it is in contrast with Ettinger *et al.* who recommended discontinuing statins therapy before bariatric surgery [17].

Three out of 6 patients with post-operative RML developed AKI. Serum creatinine reached the highest value in the second day after surgery in each patient. The increase of creatinine was preceded by the rise of CPK in such patients.

Therefore, we can conclude that post-operative monitoring of CPK is useful to recognize this complication and implement appropriate strategies in patient management. The patients who developed AKI due to RML had mild negative intra-operative fluid balance; we obtained a positive postoperative fluid balance by using a vigorous intra-venous hydration in order to maintain a urine output of more than 200 ml/h. Kidney function of such patients recovered and they were discharged from ICU in the 3th or 4th post-operative day.

Wool *et al.* compared liberal intra-operative fluid administration (40 ml/kg) to restrictive fluid administration (15 ml/kg): no change in the incidence of RML was shown in patients undergoing laparoscopic bariatric surgery [19].

We noted no difference in the intra-operative fluid balance between patient who developed post-operative RML and who did not. In fact, almost all the patients enrolled had a mild negative fluid balance at the time of ICU admission, and a positive balance was maintained in the following post-operative days. Fluid overload prevents dehydration which can cause pre-renal AKI; obese patients are more predisposed to its development than non-obese patients [20] because of the presence of underlying chronic kidney damage [21]; moreover, vigorous fluid administration can reduce myoglobin-mediated kidney damage in patients with RML [4].

Plasma CPK levels remain high until muscle damage persists; since this enzyme is not removed from plasma by the kidney and has a slow process of degradation, its levels are high for a time interval much longer than that of myoglobin, which is rapidly cleared from the plasma. As a consequence, CPK is more reliable than myoglobin in the assessment of the presence and the extent of muscular damage [16]. Thus, CPK is a significant marker of RML; it

is useful to perform the diagnosis of RML and follow the evolution over time.

CONCLUSIONS

RML is a common complication of bariatric surgery. In our study, the incidence was very low (6%) due to the high prevalence of laparoscopic operations (93%), performed as laparoscopic SG (18%) and laparoscopic RYGB (75%) which are characterized by a short operative time.

A prolonged operative time is the most important risk factor for the development of post-operative RML; in our study 5 out of 6 patients who developed RML had a surgical time ≥ 4 hours. Furthermore, all the patients who developed RML had arterial hypertension, showing that this is a risk factor for RML.

RML following bariatric surgery should be taken into account when patients have a high BMI (≥ 50 kg/m²) and/or in the case of a laparotomic operation with a prolonged operative time.

Because of an increase in the number of bariatric operations worldwide [22], an increasing interest in RML as a complication of bariatric surgery has been developed. The reduction of body weight before bariatric surgery may decrease the duration of the operation and the risk to develop RLM. Since rhabdomyolysis is the most important complication of bariatric surgery, the best precautions focus on the prevention of muscle damage; examples are: padding of all pressure-points during surgery, changes in patient position, decrease in the operative time through preoperative weight loss or staging, advise younger surgeons not to select patients who fall into the RML risk group, providing aggressive and reasonable fluid replacement.

Obese patients undergoing bariatric surgery would benefit from routine post-operative monitoring of CPK levels which is essential for an early diagnosis of RML and to follow its evolution when this complication occurs.

Considering the global increase of the obesity prevalence, the recourse to bariatric surgery for the reduction of body weight could involve a larger number of people over the next years. For this reason, it is important to work hardly in order to reduce and avoid the development of RML. Many surgeons have developed interest for the study and the prevention of RML so far, being aware of the complications which may occur in these patients. More research in regard to RML and prevention systems is necessary.

ABBREVIATIONS

AKI	=	Acute Kidney Injury
AKIN	=	Acute Kidney Injury Network
ASA	=	American Society of Anesthesiologists
BMI	=	Body Mass Index
CPK	=	Creatine Phospho Kinase
DM	=	Diabetes Mellitus
LDH	=	Lactate Dehydrogenase
ICU	=	Intensive Care Unit

OT	=	Operative Time
RML	=	Rhabdomyolysis
RYGB	=	Roux-en-Y Gastric By-pass
SG	=	Sleeve Gastrectomy

CONFLICT OF INTEREST

The authors confirm that this article content has no conflicts of interest.

ACKNOWLEDGEMENTS

This research has been partially supported by an unrestricted grant of the Italian Ministry of Foreign Affairs about the program: "Programmi di alta rilevanza scientifica e tecnologica Italia-Messico".

REFERENCES

- [1] WHO. Obesity and overweight. Geneva: World Health Organization May 2012. Available from: <http://www.who.int/mediacentre/factsheets/fs311/en/>.
- [2] Steinbrook R. Surgery for severe obesity. *N Engl J Med* 2004; 350:1075-9.
- [3] Lagandré S, Arnalsteen L, Vallet B, *et al.* Predictive factors for rhabdomyolysis after bariatric surgery. *Obes Surg* 2006; 16(10): 1365-70.
- [4] Huerta-Alardín AL, Varon J, Marik PE. Bench-to bedside review: Rhabdomyolysis-an overview for clinicians. *Crit Care* 2005; 9(2): 58-69.
- [5] Khan FY. Rhabdomyolysis: a review of the literature. *Netherlands J Med.* 2009; 67: 272-80.
- [6] Boutaud O, Roberts LJ. Mechanism-based therapeutic approaches to rhabdomyolysis-induced renal failure. *Free Radic Biol Med* 2011; 51(5): 1062-7.
- [7] Lima RS, da Silva Junior GB, Liborio AB, *et al.* Acute kidney injury due to rhabdomyolysis. *Saudi J Kidney Dis Transpl* 2008; 19(5): 721-9.
- [8] Malik GH. Rhabdomyolysis and Myoglobin-induced Acute Renal Failure. *Saudi J Kidney Dis Transpl* 1998; 9: 273-84.
- [9] Beetham R. Biochemical investigation of suspected rhabdomyolysis. *Ann Clin Biochem* 2000; 37(5): 581-7.
- [10] Faintuch J, de Cleva R, Pajeccki D, *et al.* Rhabdomyolysis after gastric bypass: severity and outcome patterns. *Obes Surg* 2006; 16(9): 1209-13.
- [11] Ettinger JE, Marcílio de Souza CA, Azaro E, *et al.* Clinical features of rhabdomyolysis after open and laparoscopic Roux-en-Y gastric bypass. *Obes Surg* 2008; 18(6): 635-43.
- [12] Mognol P, Vignes S, Chosidow D, *et al.* Rhabdomyolysis after laparoscopic bariatric surgery. *Obes Surg* 2004; 14(1): 91-4.
- [13] de Oliveira LD, Diniz MT, de Fátima HS, *et al.* Rhabdomyolysis after bariatric surgery by Roux-en-Y gastric bypass: a prospective study. *Obes Surg* 2009; 19(8): 1102-7.
- [14] de Menezes Ettinger JE, dos Santos Filho PV, *et al.* Prevention of rhabdomyolysis in bariatric surgery. *Obes Surg* 2005; 15(6): 874-9.
- [15] Khurana RN, Baudendistel TE, Morgan EF, *et al.* Postoperative rhabdomyolysis following laparoscopic gastric bypass in the morbidly obese. *Arch Surg* 2004; 139(1): 73-6.
- [16] Ettinger JE, de Souza CA, Santos-Filho PV, *et al.* Rhabdomyolysis: diagnosis and treatment in bariatric surgery. *Obes Surg* 2007; 17(4): 525-32.
- [17] Cruz DN, Ricci Z, Ronco C. Clinical review: RIFLE and AKIN--time for reappraisal. *Crit Care* 2009; 13(3): 211.
- [18] Bostanjian D, Anthone GJ, Hamoui N, *et al.* Rhabdomyolysis of gluteal muscles leading to renal failure: a potentially fatal complication of surgery in the morbidly obese. *Obes Surg* 2003; 13(2): 302-5.
- [19] Wool DB, Lemmens HJ, Brodsky JB, *et al.* Intraoperative fluid replacement and postoperative creatine phosphokinase levels in laparoscopic bariatric patients. *Obes Surg* 2010; 20(6): 698-701.

- [20] Bucaloiu ID, Perkins RM, Difilippo W, *et al.* Acute kidney injury in critically ill, morbidly obese patient: diagnostic and therapeutic challenges in a unique patient population. *Crit Care Clin* 2010; 26: 607-24.
- [21] Kambham N, Markowitz GS, Valeri AM, *et al.* Obesity-related glomerulopathy: an emerging epidemic. *Kidney Int* 2001; 59(4): 1498-509.
- [22] Buchwald H, Williams SE. Bariatric surgery worldwide 2003. *Obes Surg* 2004; 14: 157-64.

Received: January 17, 2013

Revised: January 25, 2013

Accepted: March 04, 2013

© Forfori *et al.*; Licensee *Bentham Open*.

This is an open access article licensed under the terms of the Creative Commons Attribution Non-Commercial License (<http://creativecommons.org/licenses/by-nc/3.0/>) which permits unrestricted, non-commercial use, distribution and reproduction in any medium, provided the work is properly cited.