ORTHOPAEDICS

MONITORING CREATINE KINASE IN SPINE SURGERY AND ITS RELATION TO ACUTE RENAL FAILURE

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ABSTRACT • Serum creatine kinase (CK) is the gold standard marker for muscle injury. Any muscle damage, if severe enough, can lead to rhabdomyolysis (RM) and subsequent renal failure if not treated properly. This is usually correlated with high levels of creatine kinase. Spine interventions in orthopaedic surgery are associated with surgical "injury" to the back muscles and abnormal levels of creatine kinase. Rhabdomyolysis associated with spine surgery is in fact multifactorial, and most commonly due to prolonged surgery time and certain operative positions, correlated with high risk of developing compartment syndrome (CS). However, the final complication of acute renal failure is exceptional in these procedures. This paper reviews all reported cases of rhabdomyolysis and acute renal failure following spinal interventions, and discusses the role of creatine kinase in the evaluation of muscle injury as a predictor of rhabdomyolysis and acute renal failure. Most spine surgeries are in fact not correlated with rhabdomyolysis and acute renal failure as this correlates with very high levels of creatine kinase. As a result, monitoring creatine kinase is only advised to follow in certain circumstances, and will only lead to unnecessary expenses.

Keywords : creatine kinase ; rhabdomyolysis ; acute renal failure ; spine surgery ; posterior instrumentation and fusion

RÉSUMÉ • La créatine kinase sérique est le marqueur de choix d'une lésion musculaire. Toute insulte musculaire, suffisamment grave, peut entraîner une rhabdomyolyse et même une insuffisance rénale si les précautions nécessaires n'ont pas été prises. Ceci est souvent corrélé avec des chiffres élevés de créatine kinase. La chirurgie du rachis en orthopédie est associée a un haut stress chirurgical des muscles du dos et par conséquent à un taux élevé de créatine kinase. La rhabdomyolyse associée à ce type de chirurgie est multifactorielle, le plus souvent due à un temps opératoire prolongé et à certaines positions à haut risque de syndrome de loges. Toutefois, l'insuffisance rénale aiguë reste exceptionnelle dans de telles procédures. Ce papier révise tous les cas publiés dans la littérature concernant la rhabdomyolyse et l'insuffisance rénale aiguë dans les chirurgies du rachis, et évalue le rôle de la créatine kinase considérée comme indicateur de lésion musculaire, ainsi que son rôle prédicteur de rhabdomyolyse et d'insuffisance rénale aiguë. En fait, la majorité des chirurgies du rachis ne sont pas corrélées à la rhabdomyolyse, ni à l'insuffisance rénale aiguë vu que ces complications sont associées à des chiffres très élevés de créatine kinase. Par conséquent, la surveillance du chiffre de la créatine kinase devrait être limitée à certaines circonstances, et n'aboutira qu'à des dépenses inutiles en cas de dosage systématique.

Mots-clés : créatine kinase ; rhabdomyolyse ; insuffisance rénale aiguë ; chirurgie du rachis ; instrumentation postérieure et arthrodèse

INTRODUCTION

Orthopedic surgery, and particularly spine surgery, is known for its invasive surgical approaches. Muscle damage can be extensile despite all precautions that can be taken, not only from direct trauma during the operative procedure, but also from prolonged immobilization required [1].

Rhabdomyolysis involves the rapid dissolution of injured skeletal muscle [2]. Any form of muscle damage can initiate RM. Diagnosis is based on plasma creatine kinase [3]. The fearful complication is RM-induced kidney failure. It occurs in one third of RM cases [4].

However, there are very few cases of acute renal failure (ARF) following an orthopedic intervention reported in the literature.

The purpose of this review is to evaluate the value of creatine kinase (CK) measurement as a predictor of ARF following spine surgery.

METHODS

An extensive literature search was undertaken by exploring Pubmed using the following keywords: creatine kinase, creatine phosphokinase, rhabdomyolysis, acute renal failure, spine surgery, posterior instrumentation and fusion. Fifty-six papers dealing partially or specifically with this review's subject were selected.

DEFINITION OF RM – ROLE OF CK

Bywaters and Beall were the first to describe rhabdomyolysis in 1941 after reporting pigmented casts in renal tubules during autopsies performed after crush injuries during World War II [5]. Rhabdomyolysis is a complex medical condition involving the disruption of striated muscle integrity. RM ranges from an asymptomatic condition to a life threatening one associated with ARF. Mortality

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rate can be as high as 8% [6]. It is clinically identified by a triad of symptoms: myalgia, weakness, and myoglobinuria. However, this triad is only observed in less than 10% of patients [2]).

An elevated CK level is the most sensitive laboratory test for evaluating an injury to muscles, with a half-life of 1.5 days. It is currently the gold standard for determining RM. Although a cutoff threshold has not been determined, a CK plasma concentration 5 times the upper limit of the normal range is commonly used [6]. A concentration > 16000 U/L is correlated to development of acute tubular necrosis [7].

However, a recent retrospective cohort analysis of 522 patients diagnosed with RM showed that initial CK levels (median of 3541 U/L) are not predictive of mortality or renal dysfunction [8]. Another study done on 50 patients admitted for RM after trauma or electrical burns showed a CK level > 1250 U/L in 93.33% of 15 patients who developed ARF [9]. Rodriguez *et al.* assessed risk factors for ARF in 126 patients with severe RM (CK > 5000 U/L); 58% developed ARF and the following variables were independently associated: peak CK, hypoalbuminemia, metabolic acidosis, and decreased prothrombin time [10]. Sharp *et al.* also reviewed 148 patients with post-traumatic RM [11]. The authors found that a serum creatinine ≥ 1.5 mg/dL, base deficit ≤ -4, serum CK ≥ 5000 U/L, and myoglobinuria increase the risk of dialysis-requiring ARF. Serum CK level had a positive predictive value of 80%, and base deficit had a negative predictive value of 100%. They propose an algorithm for managing RM in traumatic and surgical patients; every patient with RM is qualified as low, intermediate or high risk for developing ARF based on his creatinine and base deficit serum levels. They conclude that CK monitoring is necessary only in the intermediate and high-risk group.

Another survey on 26 patients admitted for RM showed a correlation between onset of ARF (65%) and CK level on admission (mean level of 38351 U/L) [12].

In an hospital population group (n = 93) with severe RM (CK > 5000 U/L), incidence of ARF (51%) was higher in patients with CK levels exceeding 15000 U/L. Mortality in this group was 32% [13].

The causes of RM are many, but one that would interest us in our review is the compartment syndrome (CS).

Post-traumatic and/or ischemic muscle damage to muscle groups sheathed in noncompliant fascia leads to an elevation in intracompartmental pressure from engorged muscles [2]. This can lead to further tissue ischemia when pressure is > 30 mmHg. Whitesides *et al.* estimated that muscle ischemia develops when tissue pressures within a closed compartment are within 10-30 mmHg of the diastolic pressure [14]. Ihedioha *et al.* suggested that although elevated CK levels are not diagnostic of CS, they are a useful adjunct in making the diagnosis and therefore measuring CK should be done in all patients with high index of suspicion [15]. Fasciotomy is

the treatment of choice. They also found that early fasciotomy (< 12 hours) can significantly lower CK levels, and lessen muscle damage [15]. On 39 patients with CS, Valdez *et al.* found that CK level > 4000 U/L is associated with this entity, and when combining this finding to chloride level > 104 mg/dL and minimal blood urea nitrogen level < 10 mg/dL, the association is 100% [16].

Tsai *et al.* showed a high percentage of RM (44.2%) and subsequent ARF (39.1%) in a series of 52 patients admitted with traumatic limb compartment syndrome [17]. Compartment syndrome is an unusual but morbid postoperative complication that the orthopaedic surgeon should be aware of. The rare cases of compartment syndromes and subsequent RM and ARF after an orthopaedic intervention are mostly described in lumbar surgery. (See Table I)

POSTOPERATIVE RM

Acute postoperative renal failure is thought to arise secondary to acute tubular necrosis from volume depletion, reduction in glomerular filtration rate, hypotension, and nephrotoxic drugs [18]. Mechanical blocking of renal tubules by the degradation products of myoglobin secondary to RM also contributes to acute tubular necrosis. These products also seem to be toxic in an acidic medium [19].

In their recent review on surgical RM, De Tommasi and Cusimano found that patients undergoing bariatric, urologic and gynecologic surgery have the highest reported cases of postoperative RM [20]. Most cases are described in the dorsal lithotomy and knee chest position. Several cases are also reported in the lateral position. In their paper, they attributed RM to muscle damage in the hip grid muscles from prolonged surgical position, as they presented three cases of RM occurring after lateral position for a neurosurgery procedure with postoperative pain and hyperintensity on magnetic resonance imaging in this region. They also identified several risk factors associated with RM, including length of surgery, high BMI (> 28 kg/m²), large muscle mass, peripheral vascular diseases, flexion of the table at the iliac crest, the use of kidney bolster (likely increases pressure over the dependent flank and hip area), tourniquets, tight dressings, and casts, particularly in patients with episodes of hypotension during the surgical procedure.

Woernle *et al.* analyzed 150 patients undergoing elective neurosurgery intervention to determine risk factors for CK elevation postoperatively [21]. Fifty were in lateral position, and 100 were in prone or supine position. In 55 cases, intraoperative neurophysiological monitoring was performed. They found a significant association between serum CK level and the lateral position, and also between CK levels and the neurophysiological monitoring, where muscles were not pharmacologically relaxed. Another similar study on 96 patients found a correlation with the lateral position, duration of surgery, and BMI, but not with motor evoked potentials [22].

				TABLE I					
			CASE REPORTS OF RHABDOMYOLYSIS AND ACUTE RENAL FAILURE FOLLOWING SPINE SURGERY						
Author	Patient(s) Gender	Age	BMI	Surgery	Position	Length of surgery (h)	CK peak (U/L)	ARF	CS
Gordon *et al.* 1953 [26]	M	35	NR	Lumbar hemilaminectomy	KPC	> 3	NR	Yes	NR
Keim *et al.* 1970 [27]	F	11.9	NR	Thoracic idiopathic scoliosis	KCP	4	11880	Yes	Yes
Cruette *et al.* 1986 [28]	-	-	-	Spondylolisthesis	KCP	5.33	-	Yes	-
	-	-	-	Spondylolisthesis	KCP	4.5	-	Yes	-
Aschoff *et al.* 1990 [29]	-	-	-	Lumbar discectomy	KCP	> 3	-	-	Yes
	-	-	-	Lumbar discectomy	KCP	> 3	-	-	Yes
Targa *et al.* 1991 [54] 13 patients	NR	Mean 47	NR	Hemilaminectomy	LDP	Mean 3.4	NR	Yes	NR
Foster 2003 [30]	M	40	NR	L2-L4 revision instrumented fusion	Prone	6	20480	No	NR
Sancineto & Godoy Monzon 2004 [31]	M	56	NR	Lumbar spine decompression	KCP	3.33	23800	Yes	Yes
Papadikas *et al.* 2008 [32]	M	52	125 kg	Posterior decompression L2-5 fusion & L4-5 IF	Prone	7	17741	Yes	NR
Gupta *et al.* 2008 [19]	M	35	NR	Lumbar discectomy	KCP	6	NR	Yes	Yes
Magagi *et al.* 2010 [35]	F	38	NR	L5-S1 disc replacement	Supine	NR	NR	NR	Yes
Rudolph *et al.* 2011 [55]	M	65	-	Lumbar spine decompression	KCP	-	-	Yes	Yes
Dakwar *et al.* 2011 [34]	M	60	25	T10-L4 IF	LDP	10	24704	Yes	-
	F	63	25	L1-4 IF	LDP	6.95	26800	Yes	-
	M	68	40	L2-5 IF	LDP	5.48	56000	Yes	-
	M	67	36	L2-5 IF	LDP	5.25	16800	Yes	-
	M	71	28	T11-L3 IF	LDP	7.3	5000	Yes	-
Ahmad *et al.* 2012 [36]	M	55	31	L2-L4 disc replacement + L2-5 posterior fusion	Lareral + Prone	> 6h	NR	NR	Yes
	M	49	NR	L3-S1 decompression + fusion	Prone	NR	NR	Yes	Yes
Dahab *et al.* 2012 [56]	F	47	34	L4-S1 fusion + L5 laminectomy	Prone	5	-	-	Yes
BMI: body mass index **CK:** creatine kinase **ARF:** acute renal failure **CS:** compartment syndrome **LDP:** lateral decubitus position **KCP:** knee chest position **NR:** not reported **IF:** interbody fusion **–:** not accessible									

One population at risk of developing postoperative RM that needs to be mentioned is the elderly obese patient. About 2 billion people are overweight and one third of them obese [23]. More than 30% of patients older than 60 years are in fact obese. Kelz *et al*. did a matched case-control study on 1208 elderly surgical patients aged between 65 and 80 years old (514 with ARF, with 35% following an orthopaedic procedure) [24]. They found that obesity is an independent risk factor for postoperative ARF (OR = 1.68, *p* = 0.01).

Another interesting finding reported by Nielson *et al*. is the higher incidence of postoperative ARF in patients taking an angiotensin axis blockade therapy after an elective major orthopaedic surgery [25]. In 922 patients who underwent spinal fusion, total knee arthroplasty (TKA), or total hip arthroplasty (THA), postoperative ARF was significantly higher in patients receiving this therapy (8.3% vs 1.7%; OR = 5.4; p < 0.001). It is also

important to note that postinduction hypotension was also significantly higher in these patients, along with prolonged hospital stay.

CK, RM & ARF IN SPINE SURGERY

The knee-chest position (KCP) is commonly used for lumber disc surgery because of easy access to the intervertebral space and reduced bleeding by decreasing epidural venous pressure [19]. However, it can lead to disturbance of arterial microcirculation and the capillary network, with subsequent edema and decrease in muscle perfusion, leading to cell death. An increase in the tissue pressure, exceeding 35 to 40 mmHg, over a prolonged period in a closed compartment can lead to compartment syndrome in the lower limbs. If unrecognized, it can lead to RM and subsequent ARF as discussed above.

Table I is a summary of the reported cases of RM and ARF following orthopedic spinal surgery. The first case of ARF following a lumbar surgery in the KCP was described in 1953 [26]. Keim *et al.* in 1970 described a case of ARF following a spinal fusion in the KCP for idiopathic scoliosis in a 12-year-old girl [27]. Surgery took 4 hours. The authors attributed this unexpected complication to extreme hips and knees flexion that obstructed blood flow to lower limbs during the operation, which lead to CS and myositis. Serum CK peaked at day 1 postoperatively at 11880 mg/dL. Another 2 cases of RM with subsequent ARF were reported in 1986 following surgery for spondylolisthesis in the KCP that lasted around 5 hours [28]. The authors attributed this finding to prolonged muscle compression and advised the time for a KCP to be kept within 3 hours to avoid any similar problem. Aschoff *et al.* in 1990 reported 2 cases of CS following lumbar discectomy in the KCP [29]. The authors urge a fasciotomy within 6 hours to avoid serious muscular deficits. They recognize the compartment syndrome as a specific, but rare risk of prolonged KCP. Foster described a case of RM in an obese patient undergoing a revision lumbar surgery in the prone position on a Jackson table [30]. He attributed this complication to incomplete decompression of the abdominal girth after positioning the patient. The procedure lasted 6 hours, with episode of hypotension on transfer to the supine position at the end. Serum CK level at day 1 was 20480 U/L. ARF was prevented. Areas of skeletal muscle that may have been ischemic include the abdominal wall, the paraspinal muscles from continuous retraction, and the thighs, although the author used thigh pads and did not find any signs of external pressure on thighs postoperatively. Moreover, hips and legs were extended during the procedure. Sancineto and Godoy Monzo reported one case of RM and ARF following lumbar decompression in the KCP [31]. They urge a fasciotomy using 30 mmHg as the critical intracompartmental pressure in a normotensive patient to obtain an excellent recovery, because a pressure of about 30 mmHg seems to be the limit for obstruction of flow in muscle capillaries.

Another case of ARF followed spinal decompression and fusion on an obese patient in the prone position [32]. The authors used the Jackson frame to avoid excessive muscle pressure. However, they conclude that abdominal decompression is not always satisfactory if the patient is obese. In fact, Lagandre *et al.* reported in their prospective study that marked obesity (BMI > 40 kg/m^2), diabetes, and duration of surgery > 4 hours are associated with increased risk of RM [33]. Dakwar *et al.* recently reported the first case series of RM and ARF following minimally invasive spine surgery [34]. Five of 315 patients undergoing lateral transpsoas spine surgery developed ARF (See details in Table I). The authors advise to consider staging the procedure in high-risk patients.

Magaji *et al.* reported in 2010 the first and only case of CS of the leg following spine surgery through an anterior approach in the supine position [35]. However, one major factor in this case is the injury of the iliac vein. The authors specified that no pressure was actually applied on lower limbs muscle group and therefore the chance of having CS from direct pressure in the supine position is highly unlikely. Ahmad *et al.* were the last to report 2 cases of RM following lumbar surgery on a Jackson table in a prone position [36]. The authors related this complication to reversing the position of the iliac crest and hip pads on the Jackson table. Although they achieve better lumbar lordosis, the hips were slightly extended rather than slightly flexed, which could have generated more pressure in the region directly above the pads.

Serum CK level and especially its isoenzyme MM is significantly correlated to the severity of surgery-induced tissue damage. It increases gradually after incision [37].

One study compared CK levels in 3 groups postoperatively; group A included spine surgery in the knee-chest position, group B neurosurgery without any muscle stretching, and group C abdominal surgery with retractors [38]. Interestingly, CK levels were significantly higher in groups A and C.

Kawagushi *et al.* evaluated intraoperative factors for back muscle injury and found positive correlation with retraction pressure, time, and extent of exposure [39]. CK (and especially isoenzyme MM, 90% of total CK) is increased after surgery and reached a plateau by day 1, followed by return to normal within one week. CK elevations following spine surgery is significantly higher in men than in women, most probably due to muscle mass difference [40]. Although abdominal muscles and the iliopsoas muscle are involved in the anterior spine approaches, CK elevations are significantly lower than in posterior approaches that involve multifidus and erector spinae muscles. This is explained by the difference in muscle volume iatrogenically damaged.

Decreased muscle strength and atrophy is typical after back surgery, as was demonstrated by Mayer *et al.* through a CT scan evaluation of back muscles in 44 patients 3 months after lumbar surgery [41]. Suwa *et al.* also evaluated postoperative changes in paraspinal muscles in 89 patients (42 single interlaminar level proce-

dures, 13 multiple levels, and 34 posterolateral fusion procedures). They found that paraspinal muscle thickness significantly decreased in the third group [42]. However, patients in this group were older and the authors related this in part to natural aging.

Kumbhare *et al*. studied muscle injury in lumbar surgery. Through a progressive evaluation of serum CK in 12 patients, the authors found that time to CK peak ranges between 9 to 47 hours postoperatively [43]. CK levels returned to baseline within 6 to 7 days, as were demonstrated in other studies [39]. The authors suggested a pre-surgery CK baseline for each individual to follow instead of using a standard range for an accurate detection of CK elevation. Furthermore, they recommend a CK test prior to the day of surgery to eliminate any falsely elevated CK levels due to fasting protocols. In another study, the authors found a direct correlation between the surface area of retracted muscles and postoperative CK levels taken at different times postoperatively in a group of patients undergoing posterior lumbar decompression surgery [3].

Therefore, serum CK is a valid measurement of muscle injury in lumbar surgery. This validity is strongest at 12 hours (R = 0.57, $p < 0.05$) and 24 hours (R = 0.58, $p < 0.05$), but weaker at 6 hours (R = 0.45, $p < 0.05$) and nonsignificant at 48 hours (R = 0.28, $p > 0.1$).

On the other hand, rare are the studies that did not find the same results. Davidas *et al*. studied CK and myoglobin in patients with different surgeries [44]. They conclude that CK is not a good index of the release of haematic pigments (the dangerous ones in RM), and that myoglobinuria could prove to be more helpful.

With the evolution era of minimally invasive approaches, recent studies tried to evaluate the effect of minimally invasive procedures on muscle injury. In 2010, Fan *et al*. in a prospective study compared 28 patients undergoing minimally invasive (MI) one-level PLIF to 31 patients undergoing an open approach [45]. Subjects were followed for over a year, and 16 from each group were evaluated by MRI. The MI group had less back pain ($p < 0.001$), less multifidus muscle atrophy ($p < 0.001$) with mean reductions of cross sectional area of 12.2% compared to 36% in the conventional open group. These changes were also significantly correlated with postoperative CK levels. Another consecutive series compared 41 patients undergoing MI one level PLIF to 50 undergoing traditional open approach [46]. Serum CK was markedly less in the MI group. Twenty-one patients (11 from the MI group versus 10 from the open group) underwent MRI evaluation at the last follow-up (Over a year). Postoperative cross-sectional area of multifidus muscle was significantly smaller than the preoperative one in the open group. On the contrary, there was no difference in the MI group. However, there was more fatty infiltration postoperatively in both groups, being greater in the open group. In both studies, the authors always evaluated muscle changes (or fatty infiltration) after 10 months of surgery based on a previous study showing that

muscle edema can last as long as 10 months following lumbar surgery [42]. In 2011, a double-blinded RCT on 216 patients with lumbar disc herniation compared tubular discectomy with conventional microdiscectomy, both considered as MI techniques, but subperiosteal muscle dissection is in fact needed in the conventional technique [47]. Postoperative CK levels were evaluated in all patients, and cross-sectional area of multifidus muscle at one year along with muscle atrophy were analyzed in 140 patients. Tubular discectomy, a muscle splitting technique, was expected to reduce muscle damage. However, there was no statistical difference in CK elevations or in atrophy grade between both groups, although cross-sectional area ratio (post/preoperative) was significantly higher in conventional microdiscectomy. The authors conclude that tubular discectomy did not result in reduced muscle injury. Moreover, patients undergoing the latter technique showed higher postoperative pain scores. Tubular approach could disrupt muscle fibers even more than a sharp incision.

ROLE OF CK-MB IN SPINE SURGERY

CK-MB is known as a cardiac marker. Its specificity for cardiac muscle injury is superior to isoenzyme MM. However, it is not correlated with myocardial infarction after spine surgery [48]. Eleven of 30 patients in a prospective analysis had serum CK-MB without myocardial infarction. Moreover, CK-MB was found in paraspinal muscles of 93% of patients in this study. Lenke *et al*. also demonstrated elevations of CK-MB following 20 spinal procedures without any overt manifestations of myocardial injury [49].

However, Wukich *et al*. recommend determining CK-MB along with a ratio for lactate deshydrogenase (LD-1/LD-2) to evaluate a suspected myocardial infarction postoperatively [50]. CK-MB levels exceeding 50 IU/L combined with LD ratio exceeding 1 should not be attributed to muscle damage alone.

CONSIDERATIONS IN SCOLIOSIS SURGERY

The literature is very poor concerning this particular situation. Youngman and Edgar reviewed early and late complications on 319 operated adolescent idiopathic scoliosis [51]. In their series, no renal damage or RM was reported. However, the paper lacked any information concerning renal function and CK levels. Rylance *et al*. in 1988 did a prospective study to evaluate the effect of induced hypotension on renal function after a scoliosis intervention [52]. Following 52 operations in 43 patients, of which 38 were adolescent idiopathic scoliosis, no renal function was altered. Their hypothesis was that peroperative hypotension, blood loss, myoglobinuria from muscle damage, and fat embolism from bone trauma can compromise renal function. Forty-three were positioned prone for posterior fusion, and the rest were lateral decubitus for anterior fusion. Harrington rods were the implants used for instrumentation, and Zielke implants were used in

some patients operated by an anterior approach. Blood pressure was maintained at a systolic level of 60 to 70 mmHg. Blood loss ranged from 120 to 3200 mL (Mean: 725 mL). Marked elevations of CK were noted in the first 4 days, especially at day 1 postop (Range: 321 to 7090 UI/L) No rise of CK-MB was noted. The authors concluded that induced hypotension for reducing blood loss in scoliosis surgery is safe in the presence of a good intravenous infusion to protect renal perfusion. Another similar study comparing two groups of adolescent idiopathic scoliosis (one group with controlled hypotension versus one group without hypotension) showed the same result [53].

RECOMMENDATIONS FOR MONITORING CK

The literature is inconclusive concerning a cutoff threshold of CK levels correlated with ARF. What is sure is that not only the blood level of CK matters but also many factors play a role in developing RM-induced ARF.

This paper focuses on RM and ARF following spine surgery. It shows that theses complications are only described in certain situations, and are dependent mainly on the patient himself, the length of surgery, and the surgical position. This review can give a glimpse on situations where CK monitoring is really essential to prevent RM and subsequent ARF, and on situations where high numbers of CK only direct us towards more blood tests, useless hospital stay, and avoidable financial costs. Therefore, the following situations listed below in Table II are all propositions from the current literature where monitoring CK levels is essential, but more prospective trials are needed for a better conclusion.

CK is a valid measure of muscle injury following spinal surgery, and particularly CK-MM. However, CK-MB elevations constitute only a small proportion of total CK, and they are not associated with any cardiac muscle injury. Therefore, total CK remains the best cost-effective measurement to muscle damage following spine surgery.

TABLE II
CIRCUMSTANCES WHERE MONITORING CREATINE KINASE LEVELS IS ESSENTIAL
Surgery where muscle retraction is unavoidable
Obese patients, and particularly the morbid obese (BMI > 40 kg/m^2)
The elderly and the diabetic patient
Prolonged lateral decubitus and chest knee position (> 4h)
Patients receiving ARBs or ACEIs (high risk of hypotension preoperatively)
Postoperative metabolic acidosis (base deficit ≤ -4) or **Serum creatinine ≥ 1.5 mg/dL**

REFERENCES

1. Rocher L, Braham N, Miquel A, Petz W, Menu Y. Postoperative rhabdomyolysis of the erector spinae muscles. J Radiol 2010; 91(4) : 509-10.
2. Torres PA, Helmstetter JA, Kaye AM, Kaye AD. Rhabdomyolysis: pathogenesis, diagnosis, and treatment. Ochsner J 2015; 15 (1): 58-69.
3. Kumbhare D, Parkinson W, Dunlop B. Validity of serum creatine kinase as a measure of muscle injury produced by lumbar surgery. J Spinal Disord Tech 2008; 21 (1): 49-54.
4. Lima RSA, da Silva Junior GB, Liborio AB, Daher EDF. Acute kidney injury due to rhabdomyolysis. Saudi J Kidney Dis Transplant 2008; 19 (5): 721-9.
5. Bywaters EG, Beall D. Crush injuries with impairment of renal function. Br Med J 1941; 1 (4185): 427-32.
6. Cervellin G, Comelli I, Lippi G. Rhabdomyolysis: historical background, clinical, diagnostic and therapeutic features. Clin Chem Lab Med 2010; 48 (6): 749-56.
7. Zutt R, van der Kooi AJ, Linthorst GE, Wanders RJA, de Visser M. Rhabdomyolysis: review of the literature. Neuromuscul Disord 2014; 24 (8): 651-9.
8. Baeza-Trinidad R, Brea-Hernando A, Morera-Rodriguez S et al. Creatinine as predictor value of mortality and acute kidney injury in rhabdomyolysis. Intern Med J 2015; 45 (11): 1173-8.
9. Bhavsar P, Rathod KJ, Rathod D, Chamania CS. Utility of serum creatinine, creatine kinase and urinary myoglobin in detecting acute renal failure due to rhabdomyolysis in trauma and electrical burns patients. Indian J Surg 2013; 7 5(1): 17-21.
10. Rodríguez E, Soler MJ, Rap O, Barrios C, Orfila MA, Pascual J. Risk factors for acute kidney injury in severe rhabdomyolysis. PloS One 2013; 8 (12): e82992.
11. Sharp LS, Rozycki GS, Feliciano DV. Rhabdomyolysis and secondary renal failure in critically ill surgical patients. Am J Surg 2004; 188 (6): 801-6.
12. De Meijer AR, Fikkers BG, de Keijzer MH, van Engelen BGM, Drenth JPH. Serum creatine kinase as predictor of clinical course in rhabdomyolysis: a 5-year intensive care survey. Intensive Care Med 2003; 29 (7): 1121-5.
13. Veenstra J, Smit WM, Krediet RT, Arisz L. Relationship between elevated creatine phosphokinase and the clinical spectrum of rhabdomyolysis. Nephrol Dial Transplant 1994; 9 (6): 637-41.
14. Whitesides TE, Haney TC, Morimoto K, Harada H. Tissue pressure measurements as a determinant for the need of fasciotomy. Clin Orthop 1975; (113): 43-51.
15. Ihedioha U, Sinha S, Campbell AC. Do creatine kinase levels influence the diagnosis or outcome in patients with compartment syndrome? Scott Med J 2005; 50 (4): 158-9.
16. Valdez C, Schroeder E, Amdur R, Pascual J, Sarani B. Serum creatine kinase levels are associated with extremity compartment syndrome. J Trauma Acute Care Surg 2013; 74 (2): 441-5.
17. Tsai WH, Huang ST, Liu WC et al. High risk of rhabdomyolysis and acute kidney injury after traumatic limb compartment syndrome. Ann Plast Surg 2015; 74 (Suppl 2): S158-S161.
18. Mavrogenis A, Mitsiokapa EA, Romantini M et al. Acute renal failure in orthopaedic surgery. J Long Term Eff Med Implants 2011; 21 (2): 149-58.
19. Gupta R, Batra S, Chandra R, Sharma VK. Compartment syndrome with acute renal failure: a rare complication of spinal surgery in knee-chest position. Spine 2008; 33 (8) : E272-E273.
20. De Tommasi C, Cusimano MD. Rhabdomyolysis after

neurosurgery: a review and a framework for prevention. Neurosurg Rev 2013; 36 (2): 195-202.

21. Woernle CM, Sarnthein J, Foit NA, Krayenbühl N. Enhanced serum creatine kinase after neurosurgery in lateral position and intraoperative neurophysiological monitoring. Clin Neurol Neurosurg 2013; 115 (3): 266-9.

22. Neidert MC, Losa M, Regli L, Sarnthein J. Elevated serum creatine kinase after neurosurgeries in lateral position with intraoperative neurophysiological monitoring is associated with OP duration, BMI and age. Clin Neurophysiol 2015; 126 (10): 2026-32.

23. Seidell JC, Halberstadt J. The global burden of obesity and the challenges of prevention. Ann Nutr Metab 2015; 66 (Suppl 2): 7-12.

24. Kelz RR, Reinke CE, Zubizarreta JR et al. Acute kidney injury, renal function, and the elderly obese surgical patient: a matched case-control study. Ann Surg 2013; 258 (2): 359-63.

25. Nielson E, Hennrikus E, Lehman E, Mets B. Angiotensin axis blockade, hypotension, and acute kidney injury in elective major orthopedic surgery. J Hosp Med 2014; 9 (5): 283-8.

26. Gordon BS, Newman W. Lower nephron syndrome following prolonged knee-chest position. J Bone Joint Surg Am 1953; 35-A(3): 764-8.

27. Keim HA, Weinstein JD. Acute renal failure – a complication of spine fusion in the tuck position. A case report. J Bone Joint Surg Am 1970; 52 (6): 1248-50.

28. Cruette D, Navarre MC, Pinaquy C, Siméon F. Rhabdomyolysis after prolonged knee-chest position. Ann Fr Anesthésie Réanimation 1986; 5 (1): 67-9.

29. Aschoff A, Steiner-Milz H, Steiner HH. Lower limb compartment syndrome following lumbar discectomy in the knee-chest position. Neurosurg Rev 1990; 13 (2): 155-9.

30. Foster MR. Rhabdomyolysis in lumbar spine surgery: a case report. Spine 2003; 28 (14): E276-E278.

31. Sancineto C, Godoy Monzon D. Compartment syndrome of the thigh: an unusual complication after spinal surgery. J Spinal Disord Tech 2004; 17 (4): 336-8.

32. Papadakis M, Sapkas G, Tzoutzopoulos A. A rare case of rhabdomyolysis and acute renal failure following spinal surgery. J Neurosurg Spine 2008; 9 (4): 387-9.

33. Lagandré S, Arnalsteen L, Vallet B et al. Predictive factors for rhabdomyolysis after bariatric surgery. Obes Surg 2006; 16 (10): 1365-70.

34. Dakwar E, Rifkin SI, Volcan IJ, Goodrich JA, Uribe JS. Rhabdomyolysis and acute renal failure following minimally invasive spine surgery: report of 5 cases. J Neurosurg Spine 2011; 14 (6): 785-8.

35. Magaji SA, Debnath UK, Mehdian HS. Compartment syndrome of leg following total lumbar disc replacement via anterior retroperitoneal approach: a rare complication of anterior spinal surgery. Spine 2010; 35 (3): E74-E76.

36. Ahmad FU, Madhavan K, Trombly R, Levi AD. Anterior thigh compartment syndrome and local myonecrosis after posterior spine surgery on a Jackson table. World Neurosurg 2012; 78 (5): 553. e5-8.

37. Tabatabai M, Segal R, Amidi M, Stremple JF, Caines M, Kirimli B. Serum creatine phosphokinase, lactic dehydrogenase, and their isoenzymes in the perioperative period. J Clin Anesth 1989; 1 (4): 277-83.

38. Jourdan C, Convert J, Terrier A, Tixier S, Bouchet C, Montarry M. A comparative study of CPK during spinal surgery in the knee-chest position. Apropos of 93 patients. Cah Anesth 1992; 40 (2): 87-90.

39. Kawaguchi Y, Matsui H, Tsuji H. Back muscle injury after posterior lumbar spine surgery. A histologic and enzymatic analysis. Spine 1996; 21 (8): 941-4.

40. Kawaguchi Y, Matsui H, Tsuji H. Changes in serum creatine phosphokinase MM isoenzyme after lumbar spine surgery. Spine 1997; 22 (9): 1018-23.

41. Mayer TG, Vanharanta H, Gatchel RJ et al. Comparison of CT scan muscle measurements and isokinetic trunk strength in postoperative patients. Spine 1989; 14 (1): 33-6.

42. Suwa H, Hanakita J, Ohshita N, Gotoh K, Matsuoka N, Morizane A. Postoperative changes in paraspinal muscle thickness after various lumbar back surgery procedures. Neurol Med Chir (Tokyo) 2000; 40 (3): 151-4.

43. Kumbhare D, Parkinson W, Dunlop B et al. Biochemical measurement of muscle injury created by lumbar surgery. Clin Investig Med 2007; 30 (1): 12-20.

44. Davidas JL, Roullit S, Dubost J et al. Creatine phosphokinases and serum and urinary myoglobin following a procedure in prolonged knee-chest position for the treatment of spondylolisthesis. Ann Fr Anesthésie Réanimation 1986; 5 (1): 31-4.

45. Fan S, Hu Z, Zhao F, Zhao X, Huang Y, Fang X. Multifidus muscle changes and clinical effects of one-level posterior lumbar interbody fusion: minimally invasive procedure versus conventional open approach. Eur Spine J 2010; 19 (2): 316-24.

46. Fan S, Hu Z, Fang X, Zhao F, Huang Y, Yu H. Comparison of paraspinal muscle injury in one-level lumbar posterior inter-body fusion: modified minimally invasive and traditional open approaches. Orthop Surg 2010; 2 (3): 194-200.

47. Arts M, Brand R, van der Kallen B, Lycklama à Nijeholt G, Peul W. Does minimally invasive lumbar disc surgery result in less muscle injury than conventional surgery? A randomized controlled trial. Eur Spine J 2011; 20 (1): 51-7.

48. Healey JH, Kagen LJ, Velis KP, Levine DB. Creatine kinase MB in skeletal muscle and serum of spine-fusion patients. Clin Orthop 1985; (195): 282-8.

49. Lenke LG, Bridwell KH, Jaffe AS. Increase in creatine kinase MB isoenzyme levels after spinal surgery. J Spinal Disord 1994; 7 (1): 70-6.

50. Wukich DK, Van Dam BE, Graeber GM, Martyak T. Serum creatine kinase and lactate dehydrogenase changes after anterior approaches to the thoracic and lumbar spine. Spine 1990; 15 (3): 187-90.

51. Youngman PM, Edgar MA. Posterior spinal fusion and instrumentation in the treatment of adolescent idiopathic scoliosis. Ann R Coll Surg Engl 1985; 67 (5): 313-7.

52. Rylance PB, Carli F, McArthur SE, Ransford AO, Mansell MA. The effect of induced hypotension and tissue trauma on renal function in scoliosis surgery. J Bone Joint Surg Br 1988; 70 (1): 127-9.

53. Dastych M, Cundrle I, Vlach O. The effect of controlled hypotension during spinal surgery on kidney function. Anaesthesist 1990; 39 (4): 231-5.

54. Targa L, Droghetti L, Caggese G, Zatelli R, Roccella P. Rhabdomyolysis and operating position. Anaesthesia 1991; 46 (2): 141-3.

55. Rudolph T, Lobeko JE, Andreassen L. Bilateral gluteal compartment syndrome and severe rhabdomyolysis after lumbar spine surgery. Eur Spine J 2011; 20 (Suppl 2): S180-S182.

56. Dahab R, Barrett C, Pillay R, De Matas M. Anterior thigh compartment syndrome after prone positioning for lumbosacral fixation. Eur Spine J 2012; 21 (Suppl 4): S554-S556.