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Research Article

Continuous infusion of magnesium–lidocaine mixture for prevention of ventricular arrhythmias during on-pump coronary artery bypass grafting surgery

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KEYWORDS

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Abstract *Background:* During on-pump coronary artery bypass grafting (CABG) surgery, the incidence of reperfusion ventricular fibrillation (VF) is high and post-bypass ventricular arrhythmias are common. Both reperfusion VF and ventricular arrhythmias can cause additional myocardial injury to the already ischemic myocardium. This trial aimed to test the assumption that continuous combined magnesium and lidocaine infusion would be efficient and long lasting for the prevention of post-myocardial vascularization ventricular arrhythmias including VF.
Methods: Eighty ASA III patients, who were candidates for CABG surgery, were randomly assigned into two groups: Group I (control group, $n = 40$) and Group II (Group ML, $n = 40$). After endotracheal intubation, patients of control group were infused with plain normal saline in a volume equivalent to study drugs' mixture volume. Patients of Group ML were infused with magnesium–lidocaine mixture to achieve a bolus of magnesium sulfate 2 g and lidocaine 100 mg followed by continuous infusion of Mg sulfate 500 mg/h and lidocaine 1 mg/min. The initial cardiac rhythm after aortic cross clamp (ACC) release and the occurrence of post-CPB significant ventric-

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ular arrhythmias were recorded.

Results: The incidences of reperfusion VF and post-CPB ventricular arrhythmias in Group ML were significantly lower than that in control group (22.5% vs. 72.5%) ($P < 0.001$) and (7.5% vs. 25%) ($P < 0.05$), respectively. However, in Group ML, this beneficial effect was associated with higher incidence of sinus bradycardia (72.5% vs. 17.5%) and hence pacing needs (22.5% vs. 0.0%) when compared with control group.

Conclusion: Our study concluded that, during on-pump CABG surgery, the combined administration of magnesium and lidocaine as a bolus dose starting after intubation followed by continuous infusion reduced the incidence of reperfusion VF by 62% and post-CPB ventricular arrhythmias by 70% on expense of increased the incidence of sinus bradycardia and pacing.

1. Introduction

Reperfusion ventricular fibrillation (VF) occurs after removal of aortic cross clamp (ACC) during on-pump coronary artery bypass surgery (CABG) in a very high incidence (74–94%). Reperfusion VF aggravates the myocardial damage and electrical defibrillation may cause additional injury for the already ischemic myocardium [1–5]. Also, ventricular arrhythmias are common during and after CABG with reported incidence of 34% [6]. Fortunately, fatal ventricular arrhythmias have a very low incidence (0.41–1.4%), but if an episode of either sustained ventricular tachycardia (VT) or VF occurs, the patient will become at high risk of recurrence and poor cardiac outcome. So prevention of both reperfusion VF and post-CPB ventricular arrhythmias is a crucial part of effective myocardial protection during CABG surgery [4,7,8].

Various interventions have been taken to prevent either reperfusion VF or ventricular arrhythmias after cardiopulmonary bypass (CPB) [1–10]. Some evidence has been developed that there is high risk of magnesium (Mg) depletion during CABG surgery with CPB. This hypomagnesaemia precipitates both cardiac arrhythmias and/or vasoconstriction of either coronary arteries or the used mammary artery graft which in turn aggravates the arrhythmias [6,9,11,12]. So Mg supplementation can stabilize the myocardial cell membrane and provide some cardio protective effect against ventricular arrhythmias [6,11].

Another approach to prevent the occurrence of VF or ventricular arrhythmias during CABG is the use of lidocaine which is a local anesthetic and antiarrhythmic agent. Prophylactic lidocaine reduced the incidence of reperfusion VF and defibrillation demand during CABG surgery [1–3].

So the aim of this prospective double blinded randomized trial is to test the assumption that combined Mg and lidocaine administration before ACC application in bolus doses followed by continuous infusions till the end of surgery would be efficient and long lasting for the prevention of post-myocardial vascularization ventricular arrhythmias including VF. The primary end points were the incidence, refractoriness of reperfusion VF, and the frequency of direct current (DC) counter shocks needed for defibrillation. The secondary end points were the incidence and type of post-CPB ventricular arrhythmias till the end of surgery.

2. Patients and methods

The study was conducted at Zagazig university hospital from March 2010 to October 2011 after approval of local ethics committee and obtaining patients' informed consent. The study included 80 ASA III patients, aged 40–70 years old, undergoing elective CABG surgery using CPB. Exclusion criteria included emergency operations, bradycardia (HR < 50/min), any preoperative cardiac rhythm other than sinus rhythm, preoperative antiarrhythmic drug usage other than β blockers, concomitant valvular lesion, compromised left ventricular function (EF < 50%), either acute or chronic renal failure (serum creatinine < 2 mg/dl), known allergy to any of the study medications, and inability to complete the study either due to intra-aortic balloon pump counter pulsation (IABP) use or intraoperative death.

All patients were premedicated using intravenous midazolam (0.05–0.1 mg/kg) before admission to the operating room (OR). On arrival to the OR, basic monitoring started with continuous five leads ECG monitoring, pulse oximetry, and non-invasive blood pressure monitoring (Siemens SC 7000). After arterial line insertion and establishment of continuous invasive blood pressure monitoring, all patients were induced using sodium thiopental (2–3 mg/kg) and fentanyl (5–8 ug/kg). Endotracheal intubation was facilitated with pancuronium (0.15 mg/kg) and then controlled lung ventilation (volume mode) was started using 100% oxygen. Central venous line was inserted and then central venous pressure (CVP), esophageal temperature and urine output (UOP) were continuously monitored. Hypotension, defined as a drop of MAP < 20% of baseline, was treated with fluids and vasopressors. Bradycardia defined as HR < 50/min was treated with atropine, adrenaline, and/or pacing in addition to stopping the study's drug infusion. Tachycardia defined as HR > 100/min was treated with increasing the anesthetic depth, infusing fluid bolus, and optimizing acid–base and electrolytes. Hypertension defined as an increase in MAP < 20% of baseline was treated with increasing anesthetic depth and dilators. Anesthesia was maintained using isoflurane 1.2–2% in oxygen–air mixture (replaced on CPB by propofol infusion) and intravenous infusion of fentanyl (5 ug/kg/h) and midazolam (0.05 mg/kg/h). Pancuronium (0.05 mg/kg) was given as intermittent time based boluses. Using randomization software generator, patients were randomly assigned to one of the two studied groups (40 patients each). After endotracheal intubation, patients of control group were infused with plain normal saline in a volume equivalent to study drugs' mixture volume. Patients of group II [group Mg–lidocaine (ML)] were infused with a mixture of Mg sulfate (2 g) and lidocaine (100 mg) diluted in 50 ml normal saline 0.9% over 20 min followed by continuous infusion of 20 ml/h normal saline solution con-

taining Mg sulfate (25 mg/ml) and lidocaine (3 mg/ml) to achieve 500 mg/h. of Mg sulfate and 1 mg/min of lidocaine until skin closure. The infusions were running as long as UOP was accepted (< 1 ml/kg/h). The anesthetist and surgeon who managed the patients were unaware of the treatment group. Standard CPB was established using systemic cooling down to 28–30 °C. All patients were perfused by stocker roller pump at flow rate 2–2.5 L/min/m^2. The MAP was maintained at 70–80 mmHg. After ACC application, topical cooling was applied and ante grade cold crystalloid cardioplegic solution was infused through the aortic root with induction dose of 20 ml/kg. Cardioplegic solution maintenance dose was 10 ml/kg every 20–25 min. The constituents per 1000 ml saline 0.9% of cardioplegic solution were potassium (20 mEq), Mg (12 mEq), calcium (0.7 mEq), $NaHCO_3$ (20 mEq), and lidocaine (100 mg). In all patients, the left internal mammary artery was anastomozed as a bypass conduit to the left anterior descending artery. For free grafts after completion of the distal anastomosis, the aorta was declamped and the proximal anastomoses were performed with aortic side occlusion clamp. Before the aorta was declamped, the blood is rewarmed to 32 °C, ventricular deaireation was performed, and blood chemistry was optimized with serum K^+ maintained at 5–5.5 mEq/L. The initial cardiac rhythm after removal of ACC was analyzed. If the rhythm is VF, electrical defibrillation using 30–50 J internal shocks was performed. If VF was refractory, amiodarone (300 mg) was used and this was accompanied with optimization of MAP, correction of blood chemistry parameters, and prompt concern about coronary blood flow impairment (S–T segment changes). If myocardial ischemia was suspected, the surgeon revised the performed grafts and nitroglycerine was added when MAP allowed. Ventricular arrhythmias were treated with serum electrolytes' correction, amiodarone, and/or electrical cardioversion as appropriate. If the rhythm was AF, synchronized cardioversion using 10–20 J was tried. Electrical pacing was indicated for high degree atrio-ventricular conduction (A-V) block or sinus bradycardia HR < 60 mmHg. Weaning from CPB was started when the following criteria were met: finishing of bypass procedures, a manageable level of surgical bleeding, complete rewarming to 36.5 °C, and stable cardiac rate and rhythm. Fluid/blood boluses, inotropes, vasodilators, and/or vasopressors were used as indicated for hemodynamic stability and were recorded. Hemodynamic, ventilation, and blood chemistry parameters were optimized throughout the post-bypass period. In cardiac surgical ICU, all patients were ventilated and monitored till extubation criteria were met.

Preoperative collected patients' data included age, sex, weight, Euro SCORE II, β blockers, and diuretic usage, which coronary vessel was occluded, and serum Mg level. Intraoperative data included baseline patient's MAP, HR, CPB and ACC times, operative time (starting from anesthetic induction until skin closure), and the number of grafts done.

After release of ACC, the time to appearance of the first cardiac electrical activity and the initial cardiac rhythm were registered. Data recorded included the occurrence of refractory VF as well as the number of DC shocks needed for defibrillation and the use of further antiarrhythmic drugs, and also, the presence of sinus bradycardia (HR < 60/min) or A-V block was registered. After weaning from CPB, the needs for inotropes and/or vasoactive drugs as well as the need for electrical pacing were considered for analysis. If post-CPB ventricular arrhythmias occurred, their type and therapeutics needed were also recorded. In this study, significant ventricular arrhythmias included multiple (< 5 beats/min), multifocal, or bigeminal premature ventricular contractions (PVCs), non-sustained ventricular tachycardia (6 beats – 29 s), sustained VT (> 30 s duration or requiring intervention for termination), and VF [7,8,13]. Postoperative data included serum Mg level (Cobas Integra 400 + by Roche Diagnostics, Switzerland) on ICU admission and time to tracheal extubation (h) as measured from the end of CPB.

In a small pilot study with confidence interval 95% and power of the study 80%, the overall reduction in ventricular arrhythmias during CABG was 33%; so the sample size was estimated to be 40 patients in each group.

Statistical analysis: Qualitative variables were expressed as number and percentage and comparison between two groups was carried out using Chi-square test and Fisher Exact test when expected cells were less than 5. Quantitative variables were expressed as mean and standard deviation when data were parametric and comparison between groups was done by T test. Nonparametric data were expressed by median and compared by Mann–Whitney U test. P value less than 0.05 was considered significant.

3. Results

There were no significant differences between patients of the two groups as regard age, sex, body weight, Euro SCORE, diuretics, or β blockers usage. The patients did not differ either as regard basal hemodynamic profile, number of affected coronary arteries (either planned for vascularization or already vascularized), or CPB, ACC, operative, and extubation times. All patients in both groups had left anterior descending artery lesions and did not differ statistically as regard the other affected coronary arteries (Tables 1 and 2).

After the release of ACC, the incidence of reperfusion VF in Group ML was highly significantly lower than that in control group (22.5% vs. 72.5%) ($P < 0.001$), but the incidence of sinus bradycardia and the associated pacing needs were significantly higher in group ML compared with control group ($P < 0.001$ and 0.05, respectively). However, no patient in both groups was presented by either atrial fibrillation or high degree of heart block after the release of ACC (Fig. 1).

As regard the time to appearance of electrical activity after the ACC release, it was highly prolonged in group ML (about 9 min) compared with control group (about 3 min) ($P < 0.001$). The number of DCs needed for defibrillation was highly significantly lower in group ML compared with control group ($P < 0.001$) (Table 3).

There was no significant difference between patients of both groups in their needs for either vasopressors or inotropic support after CPB (Table 4).

The number of patients with post-CPB ventricular arrhythmias was significantly lower in group ML compared with control group. The difference was mainly due to the significantly lower incidence of multiple PVCs in the control one. All cases of post-CPB ventricular arrhythmias responded to increased anesthetic depth and optimization of potassium and acid base, but the two cases who develop sustained VT and VF (both in control group) mandated electrical cardio version/defibrillation and amiodarone infusion (Fig. 2).

Table 1 Patients' characteristics, basal hemodynamics, operative times, and number of the affected coronary arteries (either planned for vascularization or already vascularized).

	Control (N. 40)	Group ML (N. 40)	Test of significance	*P* value
Age (Ys)	58.05 ± 10.89	57.10 ± 10.08	0.41	0.68
Body weight (kg)	96.32 ± 14.69	93.0 ± 13.77	1.04	0.3
Euro score	1.07 ± 0.37	1.06 ± 0.4	0.14	0.88
MAP_0 (mmHg)	103.7 ± 8.13	103.2 ± 8.83	0.26	0.79
HR_0 (beat/min)	73.87 ± 6.55	75.05 ± 6.43	0.81	0.42
CPB time (min)	111.75 ± 38.42	110.62 ± 34.79	0.14	0.89
ACC time (min)	85.12 ± 27.9	84.25 ± 29.1	0.141	0.89
Operative time (min)	267.5 ± 50.56	271.87 ± 45.92	0.41	0.68
Extubation time (h)	8.4 ± 3.67	9.1 ± 3.74	0.84	0.4
Number of grafts planned to be done	1.95 ± 0.68	2.05 ± 0.68	0.65	0.51
Number of grafts already done	1.7 ± 0.57	1.75 ± 0.64	0.37	0.71

MAP_0: baseline mean arterial pressure, HR_0: baseline heart rate, CPB: cardiopulmonary bypass, ACC: aortic cross clamp.
Group ML: magnesium–lidocaine group. Data are expressed as mean ± SD. No significant differences between the studied groups.

Table 2 Patients' sex, preoperative drug therapy, and type of diseased coronary arteries.

	Control (N. 40)		Group ML (N. 40)		Test of significance	*P* value
	N.	%	*N.*	%		
Sex female	16	40	13	32.5	$\chi^2 = 0.49$	0.48
Male	24	60	27	67.5		
Patients on diuretics	9	22.5	7	17.5	$\chi^2 = 0.31$	0.57
Patients on B-blockers	34	85	32	80	$\chi^2 = 0.34$	0.55
Right coronary artery (RC)	22	55%	21	52.5%	0.05	0.82
Circumflex artery	10	25%	13	32.5%	0.55	0.45
Oblique marginal artery (OM)	7	17.5%	6	15%	0.09	0.76

Group ML: magnesium–lidocaine group. Data are expressed as number and percentage. No significant differences between the studied groups.

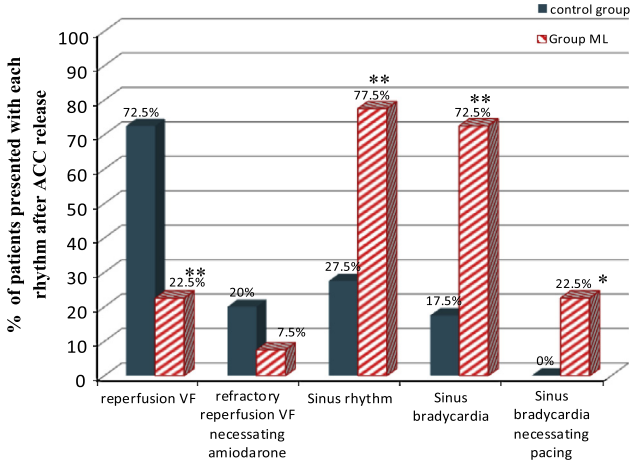

Figure 1 Initial cardiac rhythm after aortic cross clamp (ACC) release: Percentage of different types of ventricular arrhythmia in both groups. Group ML: magnesium–lidocaine group. VF: ventricular fibrillation. ACC: aortic cross clamp. Data were expressed as percentage. *Significant difference ($P < 0.05$). **Highly significant difference ($P < 0.001$).

Basal serum Mg levels did not differ between the two groups. However, postoperative Mg level was highly increased in group ML, and in contrast, it was highly reduced in the control group when compared with basal levels (Fig. 3).

4. Discussion

The current study revealed that the continuous infusion of magnesium–lidocaine (ML) mixture resulted in a delay in the reappearance of spontaneous electrical activity and reduction in the incidence of both spontaneous VF after release of ACC and post-CPB ventricular arrhythmias compared with control group during CABG surgery. The combination also lowered the number of DC shocks needed for defibrillation. These effects were at the expense of the increased incidence of sinus bradycardia and pacing needs with no additional need for inotropes or vasopressors. Postoperative serum Mg level was seen to be lower in non-Mg treated patients.

Multiple randomized controlled trials (RCTs) demonstrated the ability of Mg either as a bolus or as a continuous infusion to reduce postoperative arrhythmias after open heart surgery by different percentages [8,9,14–17]. However, other studies found no significant beneficial effect of using Mg, either before or during CPB on the incidence of either reperfusion VF or post-CPB ventricular arrhythmias during CABG surgery [6,18].

The use of lidocaine in multiple RCTs, either as IV bolus only or IV bolus followed by maintenance infusion before ACC release, showed conflicting results. In some of the trials, lidocaine can reduce the incidence of reperfusion VF by up to 84% [1–4,19]; in others, no reduction in the incidence of VF was noticed [20,21].

Table 3 Time to appearance of electrical activity after the release of aortic cross clamp and the number of DCs needed for electrical defibrillation.

	Control (N. 40)	Group ML (N. 40)	*Z* value	*P* value
Time to appearance of electrical activity after the release of aortic cross clamp (min)	3 (0.5–4)	9 (4–12)**	7.67	**< 0.001**
The number of DCs needed for electrical cardio version	2 (0–12)	0 (0–8)**	4.15	**< 0.001**

DCs: direct current shocks.
Group ML: magnesium–lidocaine group. Data are expressed as median and range.
** Highly significant difference ($P < 0.001$).

Table 4 The need for vasopressors and inotropic support after CPB.

	Control (N. 40)		Group ML (N. 40)		χ^2	*P* value
	N.	%	*N.*	%		
The need for inotropic support	32	80	35	88	0.83	0.36
The need for vasopressors	8	20	10	25	0.29	0.59

Group ML: magnesium–lidocaine group. Data are expressed as number and percentage. No significant differences between the studied groups.

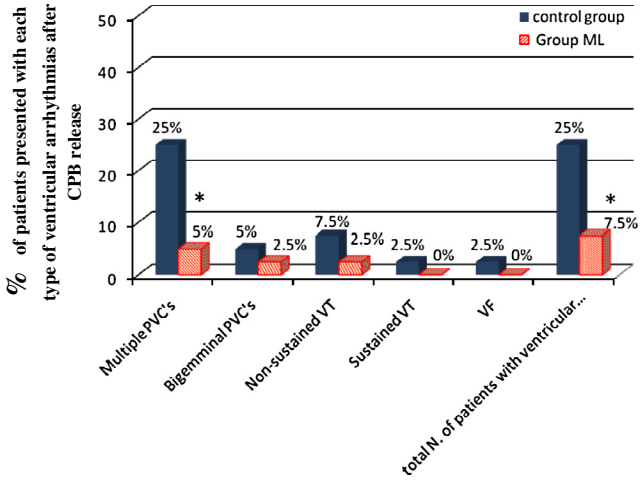

Figure 2 Percentage of different types of post-CPB ventricular arrhythmia in both groups. Group ML: magnesium–lidocaine group. CPB: cardiopulmonary bypass. VF: ventricular fibrillation. VT: ventricular tachycardia. PVC's: premature ventricular contractions. Data are expressed as percentage. * Significant difference ($P < 0.05$).

In this study, we tried to benefit from the potential synergistic effect of using both Mg and lidocaine in safe doses to avoid their side effects. As Mg administration before, during surgically induced myocardial ischemia, and at the time of myocardial reperfusion appears to improve post-ischemic myocardial mechanical recovery but if given after myocardial reperfusion has begun, it does not produce this beneficial effect [14]. We use the mixture as bolus (Mg sulfate 2 g and lidocaine 100 mg) before CPB followed by infusion of Mg sulfate

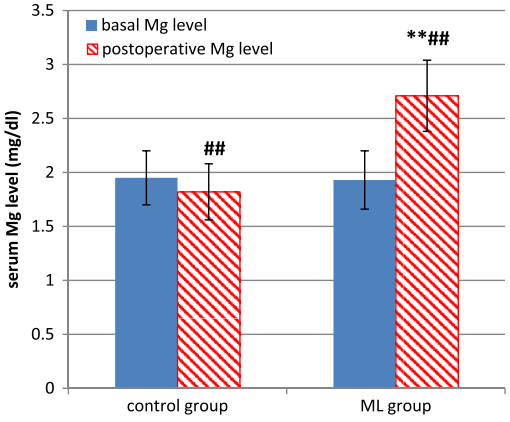

Figure 3 pre- and postoperative serum Mg level (mg/dl) in both groups. Group ML: magnesium–lidocaine group. Mg: magnesium. Data are expressed as mean ± SD. ** Highly significant difference from control group ($P < 0.001$). ## Highly significant difference from baseline ($P < 0.001$).

(500 mg/h) and lidocaine (1 mg/min) to allow long lasting prophylactic effect on both reperfusion VF and post-CPB ventricular arrhythmias.

Reappearance of spontaneous electrical activity, after the release of ACC, was delayed on using ML mixture in our study. This was considered beneficial to allow better perfusion to the myocardium without increase in myocardial oxygen consumption. A similar effect was revealed by Capsi and associates on the use of 4 g of Mg sulfate on the prebypass period [15].

Our results showed a reduction in the incidence of reperfusion VF by 62% in ML group. This is in agreement with Vaziriand associates who revealed a reduction of 72.7% or 38.9% in the incidence of reperfusion VF on using an IV bolus dose of Mg (30 mg/kg) or lidocaine (1.5 mg/kg) respectively before ACC release [10]. Similar findings were revealed on using lidocaine 100 mg, 200 mg or 2 mg/kg before ACC release [1–4].

The number of patients, who suffered refractory VF after ACC release in the present study, was lower in group ML but the difference did not reach statistical significance. However, the average number of DC shocks, needed for defibrillation, was statistically lower in group ML compared with control one and the difference reached high significance. Our finding was in agreement with Praeger and associates who revealed that lidocaine, given before ACC release, was associated with lower number of DC shocks per patient with sustained

fibrillation [3]. In contrast, Hecker and associates showed no significant beneficial effect of Mg on the incidence of reperfusion VF, when used either before (0.375 mEq/kg) or during CPB (25 mEq/kg), and found that the number and energy of DC shocks, needed for defibrillation, were greatest among the patients who received Mg before CPB [18]. The difference of our findings from that revealed by Hecker and associates may be due to different Mg infusion regimen used or the combined use of Mg and lidocaine in the current study.

Significant reduction in the incidence of postoperative ventricular arrhythmias was demonstrated by King and associates on using lidocaine (100 mg IV bolus followed by 2 mg/min infusion) and by England and associates on using Mg chloride (2 g IV bolus) after CPB [9,22]. In contrast, Juneja and associates showed that lidocaine administration cannot produce significant benefit for prophylaxis against ventricular arrhythmias in patients with poor left ventricular function [20] and also Hamid and associates failed to demonstrate significant beneficial effect on the prevention of ventricular arrhythmias after CPB when using 2 g of Mg sulfate after intubation [6]. Our results showed a 70% reduction in the incidence of post-CPB ventricular arrhythmias in group ML; this may be due to the use of Mg–lidocaine combination or the different study's patient population.

The current study supported the myocardial protective benefit of the Mg–lidocaine combination that can be explained by their mechanisms of action. Mg acts as a natural calcium channel blocker which can also block the formation of oxygen free radicals [14] and may also play a role as a free radical scavenger [11]. Mg, as a cofactor for many cellular ATPases, can prevent the intracellular sodium load and has a central role in restoration of oxidative metabolism and regulation of myocardial muscle function and tone [14]. Lidocaine also is an antiarrhythmic drug that acts via inhibition of Na channels, so it can increase the threshold for VF and preserve the energy away from electromechanical activity allowing better recovery of the heart. Lidocaine also can block the slow calcium channels [2,23–25].

In the current study, the reduction in the incidence of reperfusion VF was associated with increased incidence of sinus bradycardia and pacing needs in ML group. This finding was in agreement with that by Wistbacka and associates on using high dose of Mg chloride (>4 g prebypass and >12 g until the end of 1st postoperative day) [26]. Our patients who required pacing were weaned from it by the end of the 1st postoperative day. Tchervenkov and associates demonstrated a highly significant reduction in the incidence of spontaneous VF after ACC release, on using lidocaine (500 mg/L) containing crystalloid cardioplegia, at the expense of increased incidence of high degree A-V block [27]. In our study, no patient suffered high degree A-V block, and this may be due to the lower and continuous dose of lidocaine infusion and this in agreement with many RCTs which showed the beneficial effect of lidocaine (given as bolus either followed by continuous infusion or not) to prevent reperfusion VF without significant A-V block [1–4,19]; also no patient in our study suffered bradycardia <50/min that mandated stoppage of the study's drugs.

Hypotension is a possible side effect with Mg infusion [8]; however, in the present study, episodes of hypotension were transient and rapidly responding to fluid loading without significant increase in the need for inotropes or vasopressors in patients of group ML compared with control one. This can be explained that myocardial depressant effect of the Mg was compensated by reduction in systemic and coronary vascular resistance maintaining effective pumping function [15,28]. The reduction in the rate of reperfusion VF may also play an essential role in improvement of myocardial performance and cardiac output [4,9,23].

Post-cardiac surgery reduction in serum Mg, in non-Mg treated patients was revealed in many studies [6,16]. Postoperative serum Mg level, in current study, was significantly reduced in non-Mg treated patients. Although postoperative serum Mg remained within normal limit, intracellular hypomagnesaemia cannot be excluded [14].

Limitations of the current study were the small patients' number and the limited study period as postoperative course was not included. Further studies are needed to investigate the efficacy of this prophylactic approach and to relate reduction in both reperfusion VF and post-CPB arrhythmias to the cardiac morbidity and mortality of these patients.

This study concluded that, during on-pump CABG surgery, the combined administration of Mg and lidocaine as a bolus dose starting after intubation followed by continuous infusion reduced the incidence of reperfusion VF by 62% and post-CPB ventricular arrhythmias by 70% at the expense of increasing the incidence of sinus bradycardia and hence pacing needs without additional inotropic or vasopressor requirements.

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