Effect of Mild Hypothermia on Morbidity and Mortality in Post AMI Cardiogenic Shock

Abstract

Background: Acute myocardial infarction (AMI) complicated with cardiogenic shock is still associated with a significant death rate. Other interventions, including intra-aortic balloon counter pulsation and medical therapy, failed to improve prognosis in large-scale randomised studies, with the exception of early revascularization. Recently, mild therapeutic hypothermia, in which patients are lowered to 33°C over the course of 24 hours, has been proposed as a therapy option for cardiogenic shock patients. The purpose of this study is to determine the impact of mild hypothermia on morbidity and mortality associated with post-AMI cardiogenic shock.

Methods: This randomized, controlled, unblinded trial was conducted on 50 patients with AMI complicated by CS. Patients were randomly allocated into two equal groups; group I received MTH to 33°C for 24-36 h and group II (control group) did not receive MTH. Patients were subjected to full history taking, general and clinical examination, laboratory examination, echo, chest ultrasound (US), coronary angiography data and mild therapeutic hypothermia protocol.

Results: Stroke until day 30, duration of mechanical ventilation, length of ICU stay, duration of inotropic support, mortality and pulmonary congestion by US were insignificantly different between both groups. Arterial lactate and mean arterial blood pressure (MAP) at 4h, 6h, 8h, 10h, 12h, 14h, 16h, 18h, 20h were significantly increased in group I than Group II (P value<0.05). and were insignificantly different between both groups at 0h, 2h, 22h, 24h, 26h, 28h, 30h. Serum creatinine at 24h, 48h was significantly increased in group I than Group II (p value <0.05) and was insignificantly different between both groups at 0h.

Conclusions: Therapeutic hypothermia (TH) didn’t improve short term outcomes in patients with post AMI cardiogenic shock.

Keywords: Mild Hypothermia, Morbidity,Mortality,Post AMI, Cardiogenic Shock.

Introduction:

Acute myocardial infarction (AMI) complicated with cardiogenic shock is still associated with a significant death rate. Other interventions, including intra-aortic balloon counter pulsation and medical therapy, failed to improve prognosis in large-scale randomised studies, with the exception of early revascularization.Recently, mild therapeutic hypothermia, in which patients are lowered to 33°C over the course of 24 hours, has been proposed as a therapy option for cardiogenic shock patients[1].

Increases in myocardial contractility, cardiac output, and stroke volume may be a potential hemodynamic advantage of modest therapeutic hypothermia in cardiogenic shock [2].

Possible effects of mild therapeutic hypothermia on the heart in cardiogenic shock include a decrease in the total metabolic rate, a decrease in the myocardial metabolic rate that positively influences reperfusion injury, and an increase in cardiac contractility without an increase in oxygen consumption [3].

To investigate the hemodynamic effects of mild therapeutic hypothermia versus control on the need for vasopressors and inotropes, serum lactate level, and acute kidney injury in patients with cardiogenic shock complicating AMI who lacked classic indications for mild therapeutic hypothermia, we conducted a small, randomised trial.

Patients and Methods:

This randomized, controlled, unblinded trial was conducted on50 patients with AMI complicated by CS at the department of cardiology, Aswan Heart Centre over a period of one year from October 2020 to September 2021 after being approved from the institution of ethical committee Tanta university. Written informed consent was obtained from allpatients' guardian.

The inclusion criteria: Post-AMI cardiogenic shock is characterised by systolic blood pressure 90 mm Hg for >30 minutes or motropes required to maintain a systolic blood pressure >90 mm Hg in the absence of hypovolemia with signs of pulmonary congestion and signs of impaired organ perfusion characterised by at least 1 of the following: Intubated and sedated patients with altered mental status, cold, clammy skin, urine output 30 mL/h, or arterial lactate >2 mmol/L.

The exclusion criteria: Self-ventilated, Indication for targeted temperature management according to current standards for "out-of-hospital cardiac arrest with ROSC and no neurological recovery."

The patients were randomly classified into two groups; 25 patients in each group.Randomization had been done by computer generated random numbers. The random number was placed in an opaque envelope.

Patients were assigned to: Group I received MTH to 33°C for 24-36 h and group II (control group): did not receive MTH.

All participants in this study underwent the following: Taking a personal history, risk factors such as hypertension, diabetes, obesity, drug use, smokingprior stroke, prior myocardial infarction, prior PCI and concomitant conditions, including renal or collagen problems), General evaluation and clinical evaluation (Vital signs as heart rate, blood pressure and respiratory rate), local cardiac examination (abnormal pulsation, Heart sounds and murmurs), Echo: All studies were performed using (a GE vivid 7 Dimension Cardiac ultrasound phased array system) equipped with a 2.5 MHz variable frequency transducer. Standard views according to American Society of Echocardiography. It was used to assess the structure heart disease, presence of resting segmental wall motion and assessment of the ejection fraction. Mechanical complication as ventricular septal rupture (VSR) and acute

mitral regurgitation (AMR) and Chest U/S: pulmonary congestion by Ultrasound (US) was assessed.

Laboratory investigation: Arterial lactate, serum creatinine, mean arterial blood pressure, and norepinephrine and dobutamine dose were assessed at 0h, 2h, 4h, 6h, 8h, 10h, 12h, 14h, 16h, 18h, 20h, 22h, 24h, 26h, 28h, 30h, ALT and AST at 0h, 24h, 48h were assessed.

Coronary angiography data: Type of MI, Extent, left main stenosis, drug eluting stent (DES) used, TIMI flow before PCI, TIMI flow after PCI, ECMO, intra-aortic, sepsis and pneumonia were assessed for both groups. Outcome represented in stroke until day 30, duration of mechanical ventilation and Catecholamine support, length of Intensive care unit (ICU) stay, mortality. By comparing morbidity and mortality in both groups, morbidity (duration of mechanical ventilation, length of ICU stay and duration of inotropic support) and mortality were insignificantly different between the two groups.

After percutaneous coronary intervention and transit to the intensive care unit, patients receiving MTH were cooled using a commercially available equipment (CoolGard, ZOLL Medical Corp) (group I). By procedure, the maximum rate of cooling to the goal temperature of 33°C was established. Using the automatic temperature management function of the CoolGard system and a central measurement of the urinary bladder's temperature, the target temperature was maintained for 24 hours after it was reached. After 24 hours, rewarming with a rate of 0.25°C/h was commenced to reach the desired 37.0°C. In order to prevent shivering in the MTH group, patients were administered a treatment that included profound sedation and optional muscle relaxation. No specific temperature control was applied to patients assigned to the control group.

Statistical analysis

SPSS v26 was used to perform statistical analysis (IBM Inc., Chicago, IL, USA). Comparing the two groups using an unpaired Student's t- test, quantitative variables were provided as mean and standard deviation (SD). When applicable, qualitative variables were given as frequency and percentage (percent) and analysed using the Chi-square test or Fisher's exact test. P value less than or equal to 0.05 was considered statistically significant.

Results:

Patient's characteristics (age, sex and BMI) were insignificantly different between both groups. Hypertension (HTN), Hypercholesterolemia, Smoking, Prior stroke, Prior myocardial infarction, Prior myocardial infarction and Prior PCI were insignificantly different between both groups, however group I included more diabetic patients than Group II (P value <0.05). **[Table 1]**

Table 1: Patient's characteristics between both groups

	Group I (N = 25)	**Group II (N = 25)**	**P value**
Age (years)	61.4 ± 5.27	59.72 ± 5.82	0.290
	52 – 70	50 - 69	
Gender	19 (76.0%)	17 (68.0%)	0.529
	6 (24.0%)	8 (32.0%)	
BMI (kg/m^2)	30.93 ± 3.81	29.32 ± 3.72	0.139
	24.3 - 39.4	24.2 - 36.9	
HTN	21 (84.0%)	22 (88.0%)	0.684
DM	18 (72.0%)	9 (36%)	**0.022***
Hypercholesterolemia	14 (56.0%)	15 (60.0%)	0.775
Smoking	13 (52.0%)	11 (44.0%)	0.571
Prior stroke	6 (24.0%)	4 (16.0%)	0.480
Prior myocardial infarction	7 (28.0%)	9 (36.0%)	0.544
Prior PCI	6 (24.0%)	7 (28.0%)	0.747

BMI: body mass index. *Statistically significant as p value<0.05, HTN: hypertension, DM: diabetes mellitus, PCI: percutaneous coronary intervention.

Type of MI, extent, left main stenosis, DES used, TIMI flow before PCI, TIMI flow after PCI, ECMO, intra-aortic balloon pumb counter pulsation, sepsis and pneumonia) were insignificantly different between both groups.**[Table 2]**

Table 2: Type of MI, Extent, left main stenosis, DES used, TIMI flow before PCI, TIMI flow after PCI, ECMO, Intra-aortic balloon pumb counter pulsation, Sepsis and Pneumonia between both groups

		Group I (N = 25)	Group II (N = 25)	P value
Type of MI	**STEMI**	15 (60.0%)	17 (68.0%)	0.556
	Non-STEMI	10 (40.0%)	8 (32.0%)	
Extent	**1 Vessel**	6 (24.0%)	3 (12.0%)	0.397
	2 Vessel	8 (32.0%)	12 (48.0%)	
	3 Vessel	11 (44.0%)	10 (40.0%)	
Left main stenosis	**Present**	6 (24.0%)	4 (16.0%)	0.725
	Not present	19 (76.0%)	21 (84.0%)	
DES used	**Used**	19 (76.0%)	21 (84.0%)	0.725
	Not used	6 (24.0%)	4 (16.0%)	
TIMI flow before PCI = 0		7 (28.0%)	9 (36.0%)	0.544
TIMI flow after PCI = 3		20 (80%)	21 (84.0%)	0.952
ECMO	**Used**	2 (8.0%)	3 (12.0%)	0.667
	Not used	23 (92.0%)	22 (88.0%)	
Intra-aortic balloon pumb counter pulsation	**Used**	13 (52.0%)	16 (64.0%)	0.390
	Not used	12 (48.0%)	9 (36.0%)	
Sepsis	**Developed**	3 (12.0%)	1 (4.0%)	0.334
	Not Developed	22 (88.0%)	24 (96.0%)	
Pneumonia	**Developed**	9 (36.0%)	11 (44.0%)	0.564
	Not Developed	16 (64.0%)	14 (56.0%)	

MI: acute myocardial infarction, DES: Drug eluting stent, TIMI: Thrombolysis in Myocardial Infarction, ECMO: extracorporeal membrane oxygenation.

Total leucocytic count and Baseline serum creatinine were insignificantly different between both groups before intervention. Heart rate, systolic blood pressure, diastolic blood pressure and EF were insignificantly different between both groups.**[Table 3]**

Table 3: Total leucocytic count, baseline serum creatinine, heart rate, systolic blood pressure, diastolic blood pressure and EF between both groups before intervention

	Group I (N = 25)	Group II (N = 25)	P value
Total leucocytic count ($*10^3$ cells/dl)	12.60 ± 1.89	12.89 ± 2.27	0.628
	9 - 15.8	9.3 - 16.8	
Baseline serum creatinine (mg/dL)	2.28 ± 0.39	2.17 ± 0.39	0.314
	1.5 - 2.8	1.5 - 2.8	
Heart rate (beats/min)	83.40 ± 22.80	85.92 ± 16.34	0.655
	51 – 120	50 – 117	
Systolic blood pressure (mmHg)	82.80 ± 8.08	86.08 ± 9.08	0.184
	71 – 96	70 – 100	
Diastolic blood pressure (mmHg)	54.24 ± 9.22	57.88 ± 10.73	0.204
	41 – 73	41 – 76	
EF (%)	31.68 ± 4.71	32.44 ± 5.12	0.587
	25 – 39	25 – 40	

BMI: body mass index, EF: ejection fraction.

Stroke until day 30, duration of mechanical ventilation, length of ICU stay, duration of inotropic support, mortality and pulmonary congestion by US were insignificantly different between both groups.**[Table 4]**

Table 4: Outcome between both groups

		Group I (N = 25)	Group II (N = 25)	P value
Stroke until day 30		0 (0.0%)	2 (8.0%)	0.490
Duration of mechanical ventilation (days)	**Mean ± SD**	4.88 ± 2.44	5.40 ± 2.22	0.434
	Range	2 – 10	2 – 10	
Length of ICU stay (days)	**Mean ± SD**	6.76 ± 3.13	7.08 ± 2.83	0.706
	Range	2 – 11	2 – 12	
Duration of inotropic support (days)	**Mean ± SD**	3.92 ± 2.55	4.72 ± 2.15	0.236
	Range	1 – 10	2 – 10	
Mortality	**Died**	13 (52.0%)	16 (64.0%)	0.390
	Survived	12 (48.0%)	9 (36.0%)	
Pulmonary congestion by US (3 B lines or more)		18 (72.0%)	16 (64.0%)	0.544

US: ultrasound

Arterial lactate at 4h, 6h, 8h, 10h, 12h, 14h, 16h, 18h, 20h was significantly higher in group I than Group II (P value <0.05). and was insignificantly different between both groups at 0h, 2h, 22h, 24h, 26h, 28h, 30h.**[Figure 1]**

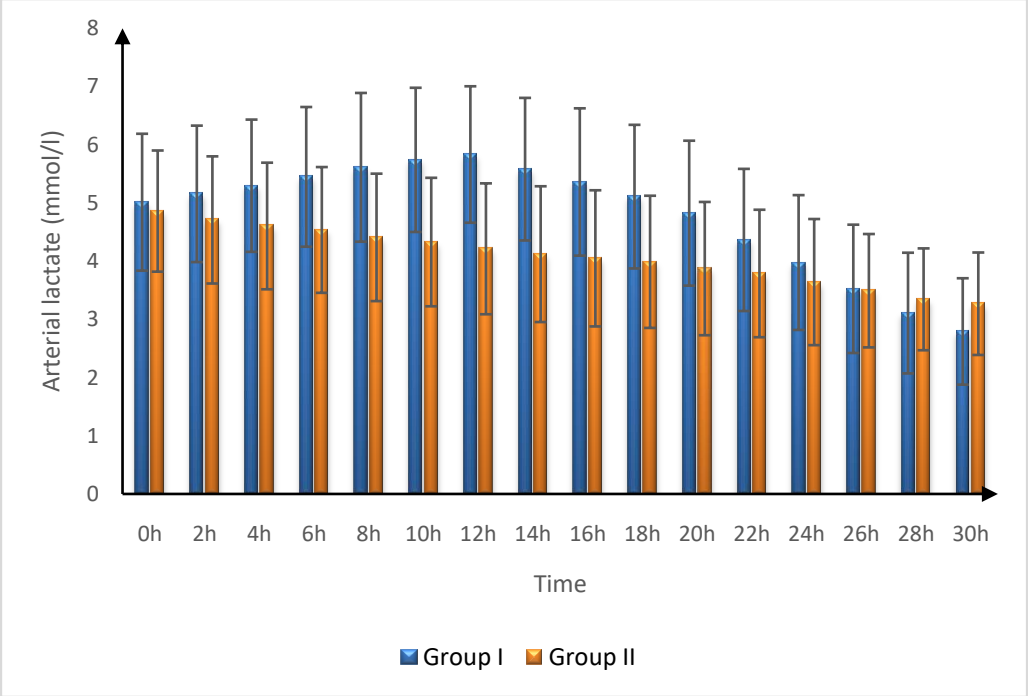


Figure 1: Arterial lactate in both groups

MAP at 2h, 4h, 6h, 8h, 10h, 12h, 14h, 16h, 18h, 20h was significantly higher in group I than Group II (P value <0.05). and was insignificantly different between both groups at 0h, 22h, 24h, 26h, 28h, 30h. **[Figure 2]**

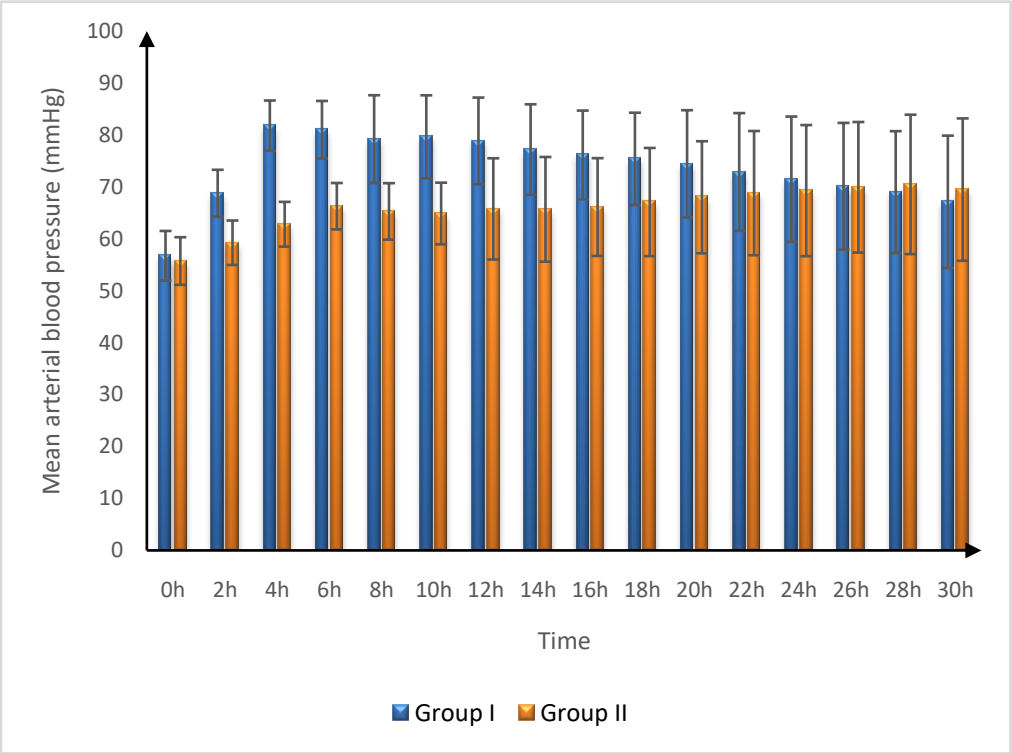
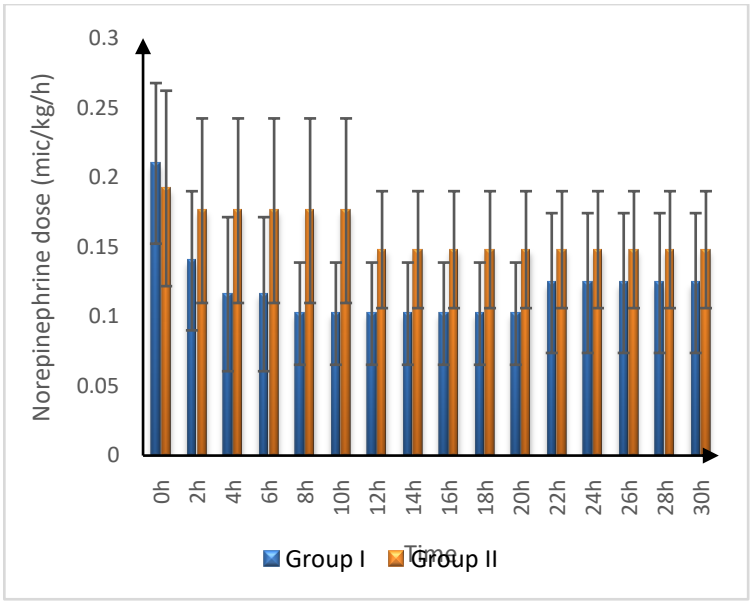
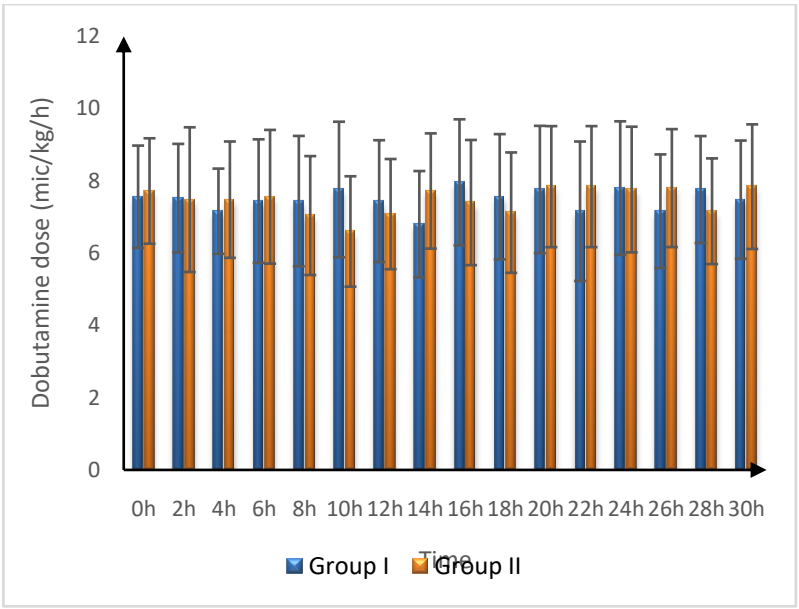


Figure 2: Mean arterial blood pressure in both groups

NE at 2h, 4h, 6h, 8h, 10h, 12h, 14h, 16h, 18h, 20h was significantly decreased in group I than Group II (P value <0.05). and was insignificantly different between both groups at 0h, 22h, 24h, 26h, 28h, 30h. Dobutamine dose at 10h was significantly increased in group I than Group II, was significantly decreased in group I than Group II at 14h (P value <0.05) and was insignificantly different between both groups at 0h, 2h, 4h, 6h, 8h, 12h, 16h, 18h, 20h, 22h, 24h, 26h, 28h, 30h. **[Figure 3]**



(A)



(B)

Figure 3: Doses of (A) NE (mic/kg/h) and (B) Dobutamine (mic/kg/h) between both groups

ALT and AST at 0h, 24h, 48h were insignificantly different between both groups. Serum creatinine at 24h, 48h was significantly increased in group I than Group II (p value <0.05) and was insignificantly different between both groups at 0h. **[Table 5]**

Table 5: ALT, AST and serum creatinine between both groups

		Group I (N = 25)		Group II (N = 25)		P value
		Mean	**± SD**	**Mean**	**± SD**	
ALT	0h	292.20	66.35	309.24	66.42	0.369
	24h	283.72	57.37	281.32	61.47	0.887
	48h	298.80	60.48	291.88	57.12	0.679
AST	0h	290.04	54.18	298.32	54.90	0.594
	24h	316.60	59.12	306.20	50.45	0.507
	48h	313.68	67.81	298.40	64.68	0.419
Serum creatinine	0h	2.28	0.39	2.17	0.39	0.314
	24h	2.37	0.43	1.94	0.37	**<0.001***
	48h	2.25	0.46	1.85	0.39	**0.002***

ALT: alanine aminotransferase, AST: aspartate aminotransferase, *Statistically significant as p value<0.05.

Discussion

Cardiogenic shock continues to be the leading cause of death in hospitalised patients with acute myocardial infarction (MI). It affects 7 to 9 percent of all patients with acute MI. In spite of optimal medical treatment, prompt revascularization, and advancements in

mechanical support, post-MI cardiogenic shock mortality continues to surpass 40 percent. Unfortunately, despite the high death rate, there have been few recent advancements in the management of cardiogenic shock, and research into alternative therapeutic approaches is required[4].

In the present study, prior stroke, prior myocardial infarction, and prior PCI were insignificantly different between both groups, except DM that was significantly increased in group I than Group II.Conforming to our results, **Fuernauet al.**[5] found that prior stroke, prior myocardial infarction, and prior PCI were insignificantly different MTH treated and control group.

Our study found that type of MI, extent, left main stenosis, DES used, TIMI flow before PCI, TIMI flow after PCI, ECMO, intra-aortic, sepsis and pneumonia were insignificantly different between both groups. In spite of optimal medical treatment, prompt revascularization, and advancements in mechanical support, post-MI cardiogenic shock mortality continues to surpass 40 percent. Unfortunately, despite the high death rate, there have been few recent advancements in the management of cardiogenic shock, and research into alternative therapeutic approaches is required. The results of our analysis are in line with **Fuernauet al.**[5]who declared that left main stenosis, TIMI flow before PCI, TIMI flow after PCI, intra-aortic, and pneumonia showed no significant difference between MTH and the control group.

Heart rate, systolic blood pressure, diastolic blood pressure, and EF were not statistically different between the two groups in the present study.However, **Zobel et al.**[6] studied The hemodynamic effects of mild therapeutic hypothermia were explored in 20 consecutive individuals with cardiogenic shock after successful resuscitation from out-of-hospital cardiac arrest. Using a propensity score, a historic normothermic control group was matched (one-to-one). Using an endovascular cooling device, patients were cooled to 33°C for 24 hours, while hemodynamic characteristics were continually measured using pulse contour analysis. Using echocardiography to evaluate cardiac performance, it was determined that mild therapeutic hypothermia caused a substantial drop in heart rate from 74 to 64 beats per minute (p .05). In addition, the study revealed an increase in ejection percent from 43 4% to 55 4%. The small sample size that was included may account for this contradiction.Similar to our findings, **Fuernauet al.**[5]notedno significant changes between MTH and the control group in terms of systolic and diastolic blood pressure.

In our study, stroke until day 30, duration of mechanical ventilation, length of ICU stay, duration of catecholamine support and mortality were insignificantly different between both groups.Conforming to our results, **Annbornet al.**[7]revealed that (ICU) and 30-day mortality rates did not change significantly between 33°C and 36°C temperature management. In contrast, **Zobel et al.**[6] documented that catecholamine use was significantly lower in cardiogenic shock individuals treated to hypothermia; the small sample size that was included may account for this contradiction.

In the present study, arterial lactate at 4h, 6h, 8h, 10h, 12h, 14h, 16h, 18h, 20h was significantly increased in group I than Group II (P value<0.05). and was insignificantly different between both groups at 0h, 2h, 22h, 24h, 26h, 28h, 30h.In contrast, **Fuernauet al.**[5] results declared arterial lactate dropped with time in both groups, with the MTH group experiencing a slower and flatter decline. The vast sample size may shed light on this disparity.

In our study, MAP at 2h, 4h, 6h, 8h, 10h, 12h, 14h, 16h, 18h, 20h was significantly increased in group I than Group II (P value <0.05) and was insignificantly different between both groups at 0h, 22h, 24h, 26h, 28h, 30h.As opposed to our results, **Young et al.**[8]between May 2007 and March 2012, 188 consecutive patients with effective ROSC after cardiac arrest were treated with TH at Vanderbilt University Medical Center. All patients were chilled externally using an active surface-cooling device for a total of 24 hours post ROSC to maintain a core body temperature of 32–34 degrees Celsius, after which they were actively rewarmed at a rate of 0.25 degrees Celsius per hour. Our institution's standard TH protocol suggests a MAP target of 80–90 mmHg and norepinephrine as the initial vasopressor of choice for treating hypotension. Prior to and during TH, patients obtained comparable MAP levels (80,3 versus 83.7 mmHg; p=0.11). This difference may be linked to the larger used sample size and the study design.In contrast to our results, **Fuernauet al.**[5] observed no differences in MAP between MTH treated group and the control group. This gap could be justified by the huge sample size that was recruited.

In our study, NE at 2h, 4h, 6h, 8h, 10h, 12h, 14h, 16h, 18h, 20h was significantly decreased in group I than Group II (P value <0.05) and was insignificantly different between both groups at 0h, 22h, 24h, 26h, 28h, 30h.Also, **Zobel et al.**[6]observednearly all patients (80%) required increasing doses of norepinephrine throughout normothermic treatment, but

for the control group, the rate of norepinephrine infusion could be dramatically reduced upon induction of hypothermia.

In our present study, Dobutamine dose at 10h was significantly increased in group I than Group II, was significantly decreased in group I than Group II at 14h (P value <0.05) and was insignificantly different between both groups at 0h, 2h, 4h, 6h, 8h, 12h, 16h, 18h, 20h, 22h, 24h, 26h, 28h, 30h.Comparable to our results, **Fuernauet al.**[5] highlighted that dobutamine had a faster decline in the MTH group than the control group.Our results were consistent with **Jacobshagenet al.**[9]study**.** In the study they investigated the influence of therapeutic hypothermia on hemodynamic parameters in resuscitated patients and on contractility in failing human myocardium. They studied the hemodynamic data of 200 survivors of cardiac arrest during hypothermia. Before and after hypothermia, dobutamine administration rates were not statistically different, according to the study (P = 0.23).

Serum creatinine at 24h, 48h was significantly increased in group I than Group II (p value <0.05) and was insignificantly different between both groups at 0h. In contrast to our results, **Fuernauet al.**[5] results showed serum creatine that did not differ between treatment groups (MTH versus control). Large, recruited sample size could justify this variance. However, **Stegman et al.**[10]following assessing the serial hemodynamics of 14 consecutive patients with cardiogenic shock after cardiac arrest who were treated with TH, the serum creatinine did not change substantially from baseline to 24 hours (1.52 1.0 vs. 1.45 0.6 mg/dl, p=0.63). The short duration of serum creatinine level monitoring may justify this criticism.

In our study, ALT at 0h, 24h, 48h was insignificantly different between both groups.AST at 0h, 24h, 48h was insignificantly different between both groups.In line with our results, **Mulalicet al.**101 individuals diagnosed with acute stroke were recruited and treated. The first group (n=40) received conventional treatment as well as therapeutic hypothermia, whereas the second group (n=61) received only conventional care. The body was cooled to the goal body temperature of 34°C to 35°C for up to 24 hours. The outcome (survival or death) of treatment was followed, and the National Institutes of Health Stroke Scale (NIHSS) and Glasgow Coma Scale were used to measure the level of awareness (GCS). The levels of aspartate aminotransferase (AST) and alanine aminotransferase (ALT) were measured upon admission, 24 hours later, and upon discharge. Although patients treated with therapeutic hypothermia had lower AST and ALT readings after 24 hours (from 32.50 to

31.00 IU/mL and from 27.50 to 26.50 IU/mL, respectively), the difference was not statistically significant.

Conclusions:

Therapeutic hypothermia (TH) did not enhance short-term outcomes for post-AMI cardiogenic shock patients.

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Conflict of Interest: Nil

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