Induced hypothermia in cardiogenic shock: a case report

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Abstract

Induced hypothermia is a technique that has gained popularity as an adjuvant therapy for patients with traumatic brain injuries, refractory arrhythmias, cardiac arrest and myocardial infarction. Induced hypothermia in patients with cardiogenic shock could improve the recovery of a stunned myocardium by decreasing its metabolic demands. We report a patient who presented with cardiogenic shock after surgical replacement of both aortic and mitral valves. The patient was placed in induced hypothermia for 34 hours. The clinical course was followed using measurements of arterial blood gas concentrations, serum lactate levels and transthoracic echocardiograms. During mechanical ventilation, the patient was monitored using a bedside bispectral index state of consciousness and temperature was measured using a Swan-Ganz catheter. The requirements for vasoactive drugs declined considerably during the hypothermic and rewarming periods. Echocardiography showed improvements in the left ventricle ejection fraction from 30% to 60% in the rewarming period with improvement in the mobility of both the anterior and septal walls. Induced hypothermia could be considered as part of the management strategy for patients with cardiac surgery, extracorporeal circulation and cardiogenic shock.

Key words:Induced hypothermia, mitral valve replacement, aortic valve replacement, extracorporeal circulation, cardiogenic shock, vasoactive drugs.

Introduction

Induced hypothermia (IH) has been used for over 60 years (1) as an adjuvant therapy in a group of critically ill patients. Described by Fay in the 1940s, (2) IH has been used in operating rooms since the early 1950s for patients undergoing cardiac surgery and neurosurgery. The use of IH after cardiac arrest was first reported in 1957 by Benson et al. (3) The beneficial effects of hypothermia in animal tests during periods of ischemia result in a wide range of biological effects and it is not a simple mechanism of action. When IH is used in any clinical event, it is important to understand the mechanisms involved in its impact. There are four different stages of hypothermia: mild (34-35.9 ºC), moderate (32-33.9 ºC), moderately deep (30-31.9 ºC) and profound (below 30 ºC). (4) The duration of cooling varies considerably, from 2-3 days up to 10 days in special situations. The rewarming mechanism is often a major challenge for the management of the patients in inadequate conditions, given the added discomfort produced by shivering. When conventional techniques are used, the control of rewarming and subsequent conditions of normothermia will take the patient to formal recovery from IH despite the underlying disease.

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Cardiovascular and hemodynamic effects during moderate hypothermia decrease the heart rate with a concomitant increase in cardiac output and myocardial contractility in euvolemic patients. In general, the systolic function is improved but diastolic dysfunction has been reported. (5-9) It has been reported that by increasing the heart rate in patients under normothermic conditions, both myocardial contractility and cardiac output increase. Under IH conditions, such an elevation of the heart rate does not occur; neither does contractility improve. (10)

Case report

We report a 63-year-old man from New York and presently a resident of Ixtapa (Mexico). He had a medical history of aortic valve replacement (St Jude prosthesis) for aortic stenosis in September 2005, longstanding hypertension and recently diagnosed chronic lymphocytic leukemia. The patient had been treated in a clinic in Michoacan (Mexico) for an acute event of fever and was found to have leukocytosis of 42,000/mL. An echocardiogram was performed with a maximum gradient of 53 mmHg and an average gradient of 26 mmHg. An image suggestive of vegetation in the aortic prosthesis was noted and the patient was admitted to our hospital. On admission, an echocardiogram showed a hyperechoic image of material measuring 14x14 mm stuck to the left back disc, decreasing the mobility of the prosthesis. It had an average pressure gradient of 62 mmHg and a flow acceleration of 6.2 m/s with significant mitral regurgitation and a pulmonary aortic systolic pressure (PASP) of 58 mmHg. It was given Parsonnet (11) score of 5 points and a Euroscore (12) of 4 points.

A second aortic valve replacement was performed using a 23 mm St Jude prosthetic valve and a mitral valve replacement with a 27 mm St Jude prosthetic valve. Surgery required an extracorporeal circulation time of 127 minutes and an aortic clamping time of 101 minutes. The patient developed hemodynamic instability during the withdrawal of extracorporeal circulation and required intra-aortic balloon counterpulsation. The hemodynamic values became unstable; inotropic and vasopressor support was required using vasopressin, norepinephrine, adrenaline, levosimendan infusion and neo-synephrine boluses (**Figure 1**). A maximum blood pressure of 90/60 mmHg was reached. Arterial blood gases showed metabolic acidosis (**Table 1**). Perioperative myocardial infarction was documented (creatine phosphokinase MB [CPK-MB] 382.8 nanograms/mL; myoglobin150.9 nanograms/mL and troponin I 37.46 nanograms/mL). By 5 hours postoperatively, the patient reached a temperature of 38.8 °C, followed by complex partial and secondarily generalized seizures that were resolved with intravenous midazolam.

We decided to apply therapeutic IH using ice packs placed randomly around the head, torso and extremities as well as enteric fluid infusion (-5 °C), achieving a temperature of 33.2 °C, maintained between 32 °C and 33.5 °C. The first echocardiogram (**Figure 2**) was performed in the early postoperative period showing severe mobility disorders, apex akinesia, anterior wall akinesia, hypokinesia of the left ventricle and a left ventricle ejection fraction (LVEF) of 30%.

During IH, the requirement of vasoactive drugs was reduced, the cardiac output changed to 3.9 L and a considerable improvement in the blood gas parameters was observed (**Table 1**; e.g., a serum lactate value of 1.9 mmol/L). A new echocardiogram was performed and showed improvement of the LVEF to 42%, PASP to 40 mmHg and better motility of both the anterior and septal walls.

The patient remained under IH for 34 hours and the rewarming phase was then started immediately following internationally established algorithms to attain a body temperature of 36 °C in about 8 hours without complications. The patient's blood gas parameters are described in **Table 1**. Vasoactive drugs were discontinued progressively. A follow-up echocardiogram showed significant improvement in wall motility and an LVEF of 62% (**Figure 3**). The patient was weaned from mechanical ventilation, continuing in a noninvasive modality using bilevel positive pressure ventilation (BiPAp Vision; Philips Respironics). Finally, his cardiogenic shock was resolved.

Discussion

Two studies have described the use of IH in comatose patients after massive cardiac arrest and cardiogenic shock. (13–14) Oddo et al. reported that the outcome measures of a patient in cardiac arrest and cardiogenic shock did not correlate with those of 74 patients treated with IH, regardless of the initial rhythm or hemodynamic status. (15)

Theoretically, IH causes hypotension and myocardial dysfunction. Maintain a patient at a temperature below 30 °C decreases myocardial contractility, and the hypovolemia caused by hypothermia induces 'cold diuresis' with a combination of increased venous return and activation of atrial natriuretic peptide. It also decreases antidiuretic hormone levels and causes renal tubular dysfunction. (16)
A study with eight patients who underwent open-heart surgery who were placed under IH showed that five of these patients were discharged from the hospital after satisfactory outcomes and returned to their normal lives. (17)

In the present case, IH was used to decrease the patient's metabolic rate and allow time for the stunned myocardium to recover. This permitted improvements in the hemodynamic parameters and even less use of vasoactive amines during hypothermia and rewarming. Echocardiographic recovery was evident, the potential length of hospital stay was reduced and the patient's clinical course was satisfactory.
This case suggests that IH could be considered as part of the management strategy for patients undergoing cardiac surgery with extracorporeal circulation and intra-aortic balloon counterpulsation.

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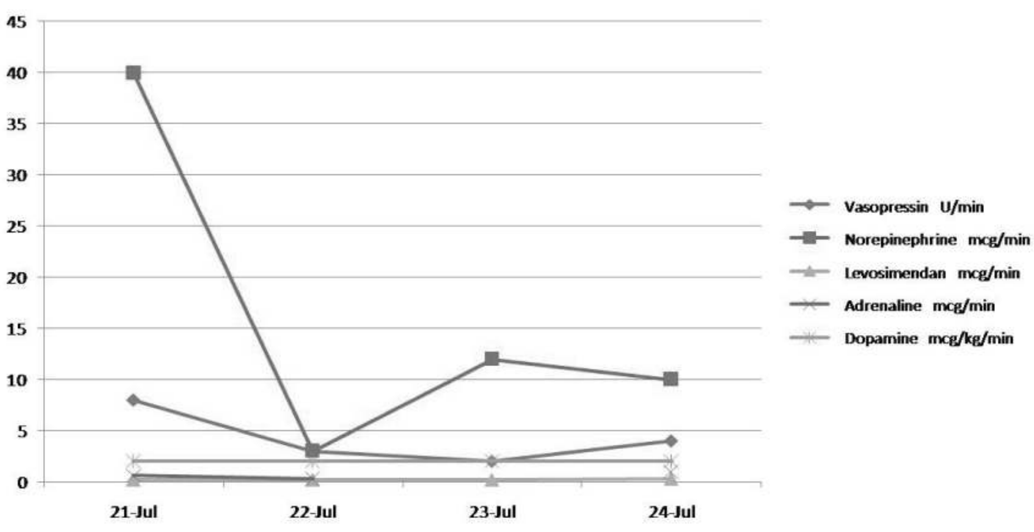
Table 1. Changes in body temperature, arterial gas and serum biochemistry measurements

Date	Temperature (°C)	pH	PaO_2 mmHg	PCO_2 mmHg	HCO_3 mmol/L	Lactate mmol/L
20-August	37.6	7.33	228	32	16.8	7.9
21-August	38.3	7.28	199	33.6	15.5	8.8
	39.3	7.36	219	32.5	18.3	12.3
	37.2	7.34	183	32.2	19.5	10.7
	35.8	7.51	234	26.6	21.3	8.7
	34.7	7.52	257	31.5	26.1	6
	34.2	7.57	159	26.8	25.1	3.1
	34	7.44	120	38.9	26.1	1.9
	33.6	7.44	170	38.3	25.8	1.5
22-August	33.2	7.48	157	32.7	24.1	1.5
	33.2	7.47	170	52.8	22.7	1.3
	33.4	7.44	143	32.6	22.2	1.6
	34.4	7.42	154	35.3	22.8	2.6
	35.3	7.45	137	31	21.5	3
	36.3	7.4	122	32.9	20.3	4
	36.5	7.4	120	31.4	19.4	5
	36.7	7.4	139	2.8	19.2	5.4
23-August	36.1	7.42	139	36.8	23.7	2.5
	36	7.48	129	34.1	24.8	1.4
	36.9	7.44	134	36.3	24.5	1.5
	36.8	7.5	133	32.6	25.2	2
24-August	37.2	7.5	134	28.7	25.8	2.1
	37.7	7.49	95.5	35.5	26.9	2

Legend: Postoperative values are shown in light shades. The values measured during hypothermia are shown in medium shades and those during the rewarming and normothermic periods are in dark shades. PaO2=partial pressure of oxygen; PaCO2=partial pressure of carbon dioxide; HCO3=bicarbonate

Figure 1. Dosages of vasoactive drugs administered before, during and after induced hypothermia (IH)



Legend: U/h=Units/hour; mcg/kg/min=micrograms per kilogram per minute

Figure 2. The first echocardiogram performed immediately after the surgical procedure and before IH

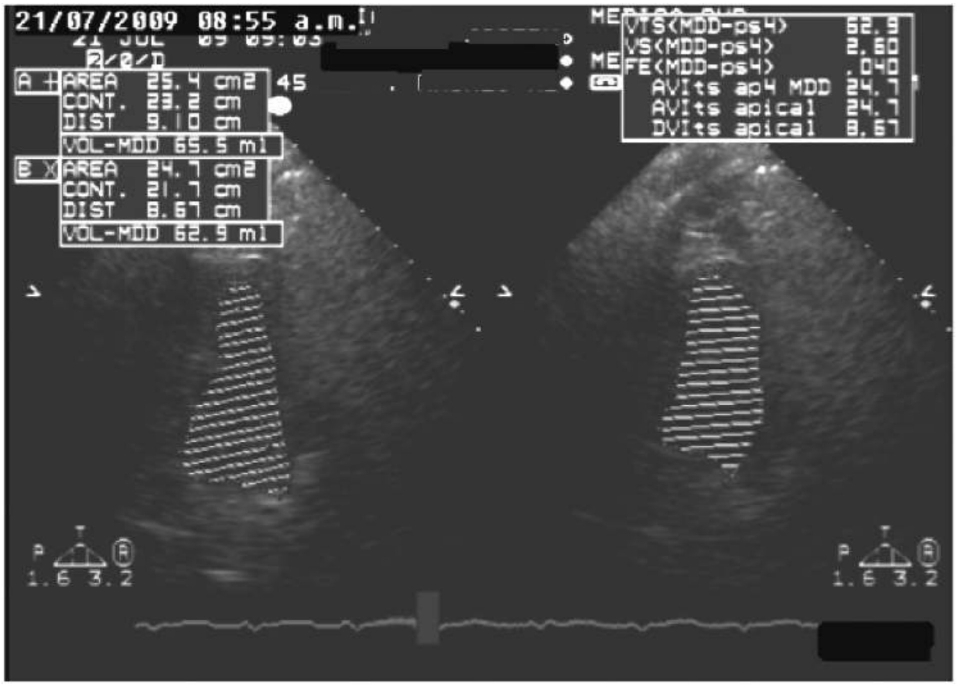
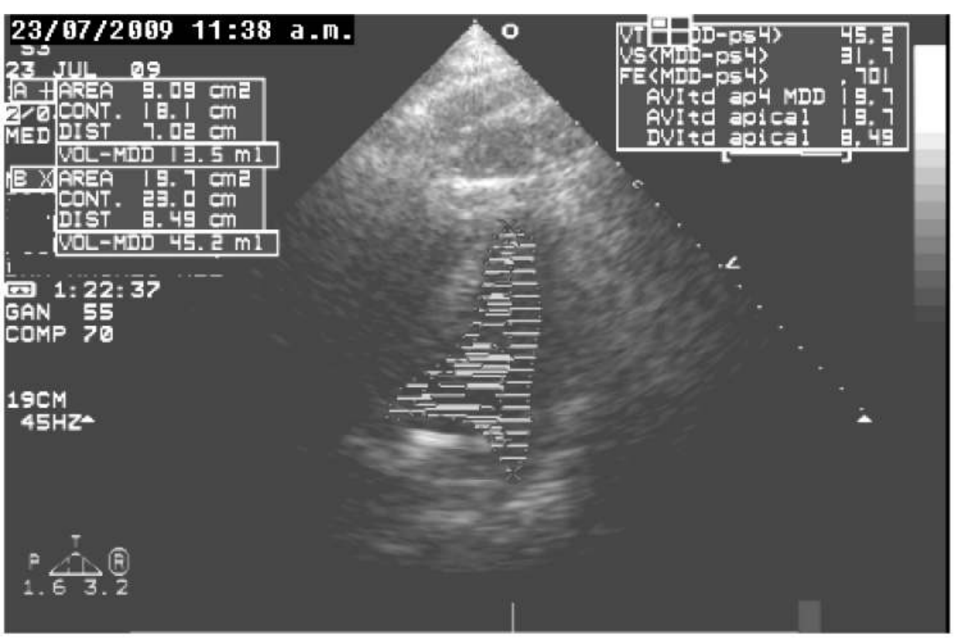


Figure 3. Echocardiogram performed before the rewarming showing improvements compared with **Figure 2**



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