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CASE REPORT

Resuscitation from adrenaline resistant electro-mechanical dissociation facilitated by levosimendan in a young man with idiopathic dilated cardiomyopathy☆

Eleftheria P. Tsagalou, John N. Nanas*

University of Athens School of Medicine, Department of Clinical Therapeutics, Alexandra Hospital, Athens, Greece

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Summary A 32-year-old man with severe congestive heart failure due to idiopathic cardiomyopathy developed ventricular tachycardia followed by electro-mechanical dissociation. High doses of conventional inotropic medications failed to restore haemodynamics. The additional infusion of levosimendan in conjunction with external chest compressions for 2.5 h restored haemodynamics, followed by complete recovery, including normal neurological function. The anti-stunning properties of levosimendan probably attenuated post-ischaemic myocardial dysfunction and helped to restore normal cardiac output.

Introduction

The goal of cardiopulmonary resuscitation (CPR) is to maintain perfusion to vital organs, pending restoration of a spontaneous circulation. Vasopressor agents are administered to enhance coronary perfusion pressure[1] while efforts are being made to correct reversible clinical factors and create optimal conditions for the restoration of efficient cardiac function. This is particularly significant in the presence of severe congestive heart failure (CHF), where any additional cause of cardiac dysfunction, for example stunning of globally ischaemic myocardium, may be detrimental.[2] Levosimendan is a new inotropic agent shown to possess anti-stunning properties.[3]

We report the case of a young man in severe CHF due to idiopathic dilated cardiomyopathy (IDC), who was successfully resuscitated from cardiac arrest with pulseless electrical activity (PEA) after 2.5 h of CPR with the addition of the calcium sensitizer levosimendan.

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* Corresponding author at: Makedonias 24, 104 33 Athens, Greece. Tel.: +30 210 8236877; fax: +30 210 8226857/6044611.
E-mail address: jnanas@ath.forthnet.gr (J.N. Nanas).

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Case report

A 32-year-old man suffering from CHF due to IDC, and with a left ventricular ejection fraction of 32%, was referred to our hospital for management of frequent temporary episodes of ventricular tachycardia (VT), manifested by paroxysmal lightheadedness and fatigue. Upon admission to the coronary care unit the patient was stable and asymptomatic. Arterial blood pressure was 90/65 mmHg and the electrocardiogram revealed sinus rhythm with frequent premature ventricular complexes, couplets and frequent runs of non-sustained VT. Electrolytes and arterial blood gases were within normal limits. Amiodarone was infused at a rate of 150 mg/h and vital signs were monitored. Sustained VT developed 2 h later, a 100 mg bolus of lidocaine was infused without effect, and synchronized cardioversion was planned. However, the tachycardia ended spontaneously as the patient's level of consciousness fell. Despite the return of apparent sinus rhythm the patient remained pulseless and mentally confused, then lost consciousness a few minutes later, despite the immediate intravenous administration of noradrenaline 0.1 μg/kg/min, dobutamine 10 μg/kg/min, and five consecutive 1 mg boluses of adrenaline. After prompt tracheal intubation and initiation of manual chest compressions, levosimendan was infused in a dose of 0.3 μg/kg/min. Pulseless electrical activity was interrupted by four episodes of ventricular fibrillation (VF), each of which was defibrillated promptly with 200, 360, 200 and 200 J shocks, respectively. A bolus of 300 mg of amiodarone was administered and, after approximately 30 min, the episodes of ventricular arrhythmias abated, and normal sinus rhythm without a perceptible pulse returned. Sodium bicarbonate was infused to maintain the arterial pH above 7.3. The right femoral artery was cannulated to monitor the arterial blood pressure generated by CPR, and the administration of maximum doses of adrenaline (epinephrine), noradrenaline (nor epinephrine), dobutamine, and levosimendan in a dose of 0.3 μg/kg/min was continued throughout the resuscitation efforts. The return of spontaneous circulation was checked every 10–15 min.

After 2.5 h of CPR a blood pressure of 65/45 mmHg had returned during continued administration of vasopressor and inotropic agents in high doses, and chest compressions were discontinued. Over the following 2 h, the arterial blood pressure increased gradually, vasopressor administration was reduced, and the patient regained consciousness and was extubated. Detailed neurological examination revealed no deficit. Adrenaline and noradrenaline were discontinued approximately 4 h later, while dobutamine and levosimendan were continued for 24 h.

Mild post-resuscitation renal and liver dysfunction manifested by rises in serum creatinine, and in alanine and aspartate transaminase resolved in the following days. The patient was discharged after 20 days of hospitalization in an excellent neurological state on a regimen of amiodarone, enalapril, spironolactone, furosemide and digoxin. At 17 months of follow-up, he was alive, in a normal neurological state, and in a stable clinical condition.

Discussion

This is, to our knowledge, the first report of successful resuscitation from prolonged electromechanical dissociation (EMD) with the inotropic support of a calcium sensitizer. While the main cause of cardiac arrest is a malignant ventricular arrhythmia,[4] spontaneous or active restoration of apparent sinus rhythm is not invariably associated with recovery of effective circulatory function. This post-arrhythmia PEA may be a manifestation of a separate life-threatening event, such as prolonged myocardial ischaemia that caused VT or VF. On the other hand, when VT/VF is the precipitating event, PEA may result from severe cardiac dysfunction developing during the tachyarrhythmia. Post-arrhythmia myocardial dysfunction caused by a decreased responsiveness of the myofilaments to Ca^{2+}, secondary to VF or VT, or to global cardiac ischaemia,[5] is an alternative explanation. The prompt administration of agents with anti-stunning properties during the resuscitation may be life-saving in this setting. However, these drugs preferably should not increase myocardial oxygen consumption, since this is detrimental in the setting of low coronary perfusion during resuscitation. This is particularly applicable to adrenaline which, when administered in high doses during cardiac arrest, did not increase the rate of survival to hospital discharge[6] despite higher rates of return of spontaneous circulation. The adverse effect of adrenaline on myocardial viability due to an increase in myocardial oxygen consumption may explain this lack of effectiveness. In animal models of cardiac arrest and resuscitation, dobutamine, a selective beta-1 adrenergic receptor agonist, improved indices of systolic and diastolic function at the cost of increased cardiac work and oxygen demand in a dose of 10 μg/kg/min though imposed a lower energy cost when administered in a dose of 5 μg/kg/min.[7] Also in an animal model

of cardiac arrest, milrinone abolished refractory post-DC shock PEA and attenuated left ventricular dysfunction, though its effects on myocardial oxygen consumption were not evaluated in that study.[8] On the other hand, calcium sensitizers were more effective than phosphodiesterase inhibitors in improving the mechanical function of stunned myocardium when tested in a pig model.[9]

Levosimendan was administered without a loading dose because of severe hypotension. However, in an animal model of intracoronary administration its anti-stunning effect was dose-dependent.[4] Since the blood concentrations of levosimendan increase during a continuous intravenous infusion, to reach steady-state within 4 h,[10] insufficient blood concentrations probably accounted for the delay in the restoration of cardiac systolic function. Furthermore, the positive inotropic effects of levosimendan, together with the high coronary perfusion pressure achieved by the high doses of vasopressors may have prevented the development of ''stoned heart'', allowing the maintenance of an adequate cardiac output during resuscitation, and the prevention of serious organ damage despite its prolonged duration.

Conclusions

This case demonstrates that levosimendan, as part of a complex resuscitation procedure and associated with prolonged CPR, can facilitate resuscitation from cardiac arrest due to PEA. The inotropic and positive inotropic effects without an increase in myocardial oxygen requirements make levosimendan a drug of choice for the management of post-ischaemic cardiac dysfunction. However, controlled studies will be needed to confirm our observations and identify the optimal mode of levosimendan administration.

Conflict of interest statement

None of the authors has any financial or personal relationship with individuals or organizations that could bias their work inappropriately.

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