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Hypovolemic Shock: How Does Lactic Acid Affect the Heart?

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ABSTRACT

Hypovolemic shock is due to a critical loss in the effective circulating blood volume with systemic hypoperfusion. If left untreated, hypovolemic shock can lead to ischemic injury of vital organs, leading to multi-system organ failure and death. Hypovolemic shock is a potentially life-threatening condition (1,2). There are five causes of hypovolemic shock: hemorrhage, trauma, surgical intervention, burns, and fluid loss caused by vomiting or diarrhea. This woman was involved in a motor vehicle accident, which resulted in a traumatic crash (2,3). This was caused by blood loss to the abdomen, as the physical examination suggested (3). Early recognition and appropriate management are essential. Hypovolemic shock results from depletion of intravascular volume, whether by extracellular fluid loss or blood loss (1,2). The pre-shock stage is characterized by compensatory mechanisms with increased sympathetic tone resulting in increased heart rate, increased cardiac contractility, and peripheral vasoconstriction (2,3).

KEYWORDS: Hemorrhagic shock; Massive transfusion; Trauma, Coagulopathy; Damage control resuscitation.

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INTRODUCTION

Hypovolemic shock is a potentially life-threatening condition. Early recognition and appropriate management are essential (1,3). Hypovolemic shock is circulatory failure due to effective intravascular volume loss (fluids or blood) (1,3,4). This effective circulatory volume loss leads to tissue hypoperfusion and tissue hypoxia (3,4). If left untreated, hypovolemic shock can lead to ischemic injury of vital organs, leading to multiorgan failure (MOF) (2,3).

CASE PRESENTATION

A 26-year-old woman is taken to the emergency room by ambulance after a serious automobile accident. She's unconscious. Her blood pressure is 64/40 mmHg, her heart rate is 150 beats per minute. The woman is intubated and given manual ventilation. No evidence of head injury Her pupils are 2mm and responsive. She withdraws when faced with painful stimulation. Cardiac examination reveals no murmurs, gallops, or rubs. Her lungs are clear to auscultation. Her abdomen is tense with reduced intestinal sounds. Extremities are cold and clammy with thread-like pulses. She has surveillance and medical management in an ambulance. Despite aggressive resuscitation with blood and fluids in the trauma unit, the patient entered her 86-minute resuscitation refractory to her pulse recovery with 4 cardiopulmonary arrests, leaving the patient dead. Resuscitation was performed with a trauma surgeon, emergency room doctor, and family doctor as a team. The Resuscitation Control Algorithm was met using the range of available blood products. Prompt identification of the mechanism of injury, clinical and tactical

decision making, and immediate advanced medical care through various pre-hospital and intra-hospital medical evacuation platforms culminated in negative outcomes following the previous 4-vehicle crash.

DISCUSSION

There are five causes of hypovolemic shock: hemorrhage, trauma, surgical intervention, burns, and fluid loss caused by vomiting or diarrhea. This woman was involved in a motor vehicle accident, which resulted in a traumatic crash (3,4). This was caused by blood loss to the abdomen, as the physical examination suggested (3).

The four pathophysiologic types of shock are hypovolemic, distributive, cardiogenic, and obstructive (2-5). Depending on the age of the patient, the history of severe trauma, and the signs, the most probable type in this case is hypovolemic shock. In hypovolemic shock, reduced blood volume leads to inadequate risk of tissues (3,4). This causes increased anaerobic glycolysis and lactic acid production (1-4). The fact of having anaerobic glycolysis and production of lactic acid decreases the vascular response capacity in stress ranges for the cells (5,6).

Lactic acidosis depresses the myocardium, reduces the responsiveness of the peripheral vasculature to catecholamines, and can cause coma (7,8). The decrease in mean arterial pressure reduces activation of the arterial baroreceptor, which results in an increase in vasomotor discharge (9,10). This produces generalized vasoconstriction. The vasoconstriction is seen in the skin through coldness and pallor (11,12).

CONCLUSIONS

Hyperlactatemia can be viewed as part of the stress response including increased metabolic rate, sympathetic nervous system activation, accelerated glycolysis and a modified bioenergetic supply (10,11).

In animals with cardiogenic shock and in patients with cardiogenic shock, a marked increment in glycolysis and gluconeogenesis associated with hyperlactatemia was described (11,12).

In healthy subjects and in cardiogenic shock, it was observed, using an infusion of labelled lactate, that 50% of this lactate was oxidized and 20% used for glucose synthesis, without differences between the two subgroups (13,14). All these data strongly suggest that lactate is a source of energy in stress conditions (12-14).

Ethical Statements: According to Colombiana law, case reports do not need to be approved by the Ethics Committee; however, the work complies with the ethical guidelines of the Helsinki declaration and the Oviedo convention, as well as the ethical standards of the University (Universidad Santiago de Cali. Departamento de Urgencias, Hospital San Juan de DIOS).

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Conflict of interest: None declared.

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