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Case report

Lactate and base deficit in trauma: Prognostic value☆

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ABSTRACT

Objectives: Clinical case discussion and non-systematic literature review on lactate and base deficit in trauma, its pathophysiology and prognostic value.
Materials and method: The case of a polytraumatized patient that underwent major vascular and orthopedic surgery, ICU management and outcomes is discussed with the approval of the Ethics Committee of our Institution. The literature search included Pub Med, Scielo and Bireme.
Results: Lactate and base deficit are early follow-up clinical tools in trauma for identifying anaerobic metabolism, in addition to evaluating and changing the resuscitation strategy. This model is applicable to cardiovascular surgery.
Conclusions: Both in trauma and cardiovascular surgery, lactate and base deficit are biomarkers that need to be quantified very early and in a serial manner. They are independent predictive factors for mortality in trauma patients in the first 48 h.

Similarly, the base deficit allows for an early staging of patients in shock and for establishing with a high probability the need for blood by-products or mass transfusion.

Further studies are required for normotensive patients.

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Lactato y deficit de bases en trauma: valor pronóstico

Palabras clave:
Ácido Láctico
Heridas y traumatísmos
Choque
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RESUMEN

Objetivos: Presentación de un caso clínico y revisión no sistemática de la literatura sobrelactato y déficit de bases en trauma, su fisiopatología y su valor pronóstico.
Material y método: Con autorización del comité de ética de nuestra institución, se presenta el caso de un paciente politraumatizado sometido a cirugía vascular mayor y ortopédica, su manejo en la UCI y su desenlace. La búsqueda bibliográfica se realizó en Pub Med, Scielo y Bireme.

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Resultados: El lactato y el déficit de bases son herramientas clínicas de seguimiento muy temprano en trauma para detectar metabolismo anaeróbico. Igualmente evaluar y modificarla estrategia de reanimación. Este modelo es aplicable a cirugía cardiovascular.

Conclusiones: En trauma y cirugía cardiovascular, el lactato y el déficit de bases constituyen biomarcadores que se deben cuantificar de manera muy temprana y seriada, constituyendo un factor predictivo independiente de mortalidad dentro de las primeras 48 h en los pacientes con trauma. Igualmente, el déficit de base permite una estratificación temprana de los pacientes que se presentan en estado de choque y determinar con alta probabilidad su necesidad de hemoderivados o transfusión masiva. Se requieren más estudios relacionados con los pacientes normotensos.

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Introduction

Inadequate or insufficient oxygen delivery results in anaerobic metabolism. The level of anearobiosis is proportional to the depth and severity of the hemorrhagic shock reflected by the base deficit and the level of lactate. In the presence of oxygen in the mitochondria, per every molecule of glucose, 36 ATP are produced during oxidative phosphorylation, in addition to water and carbon dioxide. In anaerobic conditions, pyruvate accumulates due to the failure of the pyruvate dehydrogenase enzyme to turn it into acetyl CoA. The excessive amount of pyruvate is converted into lactate through the action of lactic dehydrogenase. This system generates only 2 ATP molecules. Lactate is then used as metabolic fuel through Cori cycle or lactic acid. Lactate is an indicator sensitive to the presence and the severity of anaerobic metabolism. Its normal serum concentration is <2 mmol/L. Actually, two categories of lactic acidosis have been described: Type A where lactic acidosis occurs with tissue hypoxia and Type B, where lactic acidosis occurs without tissue hypoxia (Tables 1 and 2 and Fig. 1).[1–3]

Table 1 – Causes of lactic acidosis type A (clinical evidence of tissue hypoxia).

Shock (hypovolemic, cardiogenic, septic)
Tissue hypoperfusion
Severe hypoxemia
Severe anemia
Carbon monoxide poisoning
Severe asthma

Source: Authors'.

Table 2 – Causes of lactic acidosis type B (no clinical evidence of tissue hypoxia).

Medical causes	Diabetes mellitus, pheochromocytoma, thiamine deficiency
Toxic agents	Ethanol, methanol, salicylates, sorbitol
Inborn metabolic errors	Pyruvate dehydrogenase deficiency, oxidative phosphorylation defects, glucose 6-phosphate deficit
Miscellaneous	Hypoglycemia

Source: Authors'.

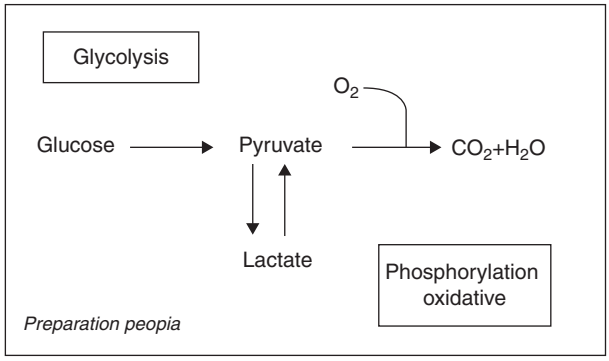

Fig. 1 – Glucose metabolism.
Source: Authors'.

Case discussion

The Ethics Committee of our organization authorized this case discussion of a 30-year old male patient who was the driver in a car crash but did not loose consciousness. The physical examination revealed chest trauma with dissection of the descending aorta, left femur fracture, fracture of the left distal radius and of the fifth left hand digit, in addition to pneumothorax and pulmonary contusion of the left lung. The patient was managed with closed thoracotomy (see Fig. 2).

The transesophageal ultrasound evidenced a normal biventricular systolic function.

Surgical management: descending aortic graft, bleeding packing.

The patient was admitted to the ICU with a left-sided double lumen endotracheal tube and continuous cardiac output monitoring. The surgery lasted for 10 h; clamp time of 320 min and extracorporeal circulation of 349 min with a beating heart. Complications: bleeding and coagulopathy. 15 units of platelets, 8 units of plasma, and 6 units of red blood cells were transfused. The orthopedist placed an external tutor in the left femur.

The resuscitation in the ICU was accomplished using isotonic crystals, hypertonic agents (3% hypertonic saline solution) and colloids (20% albumin). Sedation with fentanyl for RASS-2. Table 3 depicts the lactate behavior, base deficit, vasopressor support, inotropes, invasive ventilation and gasimetry during surgery and in the ICU.

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