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Short Communication

The Role of Calcium in the Polytraumatized Patient

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Polytrauma is a complex and severe condition that can lead to death, characterized by mechanisms such as blunt or penetrating trauma causing multiple injuries to the human body. Polytrauma is the leading cause of death in the first four decades of life in Brazil and is a global epidemic.

Management of the polytraumatized patient must be swift and systematic to ensure that no injury goes unnoticed. The care of polytrauma patients has been continuously studied and has undergone various modifications to improve patient survival. The lethal triad of trauma is well established worldwide and emphasizes that not only visible injuries should be treated but also that correcting the patient's metabolic disturbances is crucial. Hypothermia, acidosis, and coagulopathy have been recognized as evident causes of death in trauma; however, over recent years, hypocalcemia has been identified as closely associated with trauma mortality. Calcium replacement in polytraumatized patients can be a critical factor in increasing survival chances and a factor in reducing the length of hospital stay.

Ionic calcium is a cofactor for the activation of factors II, VII, IX, and X in the coagulation cascade, as well as for the activation of proteins C and S. Additionally, it is required for platelet adhesion at the site of injury.

Beyond coagulopathy, calcium is also correlated with the other two elements of the lethal triad of trauma. Calcium is essential for cardiac contractility and vascular resistance, meaning it is directly related to afterload. A reduction in calcium levels leads to decreased cardiac contractility and afterload, which results in further tissue hypoxia and exacerbates lactic acidosis. Hypothermia decreases hepatic metabolism, and in patients who have received blood products, citrate accumulation occurs, as its metabolism is hepatic. Citrate chelates calcium, further worsening hypocalcemia. It is also noteworthy that hypothermia leads to vasoconstriction and decreased cardiac contractility, which will further reduce afterload, already compromised by hypocalcemia.

Metabolic acidosis, a critical component of the lethal triad in trauma, arises from excessive lactic acid production, typically seen in hypovolemic or hemodiluted patients due to impaired tissue perfusion. Inadequate oxygen supply triggers anaerobic metabolism, leading to lactic acid accumulation and a subsequent drop in blood pH. A decline in pH from 7.4 to 7.0 can reduce the activity of activated factor VII by up to 90%, significantly impairing the coagulation cascade. Furthermore, aggressive fluid resuscitation, especially with large volumes of normal saline, can induce hyperchloremic acidosis and exacerbate hemodilution.

Temperature control is vital for metabolic processes and coagulation, as hypothermia (core temperature <35°C) markedly decreases the activity of clotting factors. Post-trauma hypothermia can be driven by multiple factors, including exposure to cold environments, wet clothing, open body cavities, hypovolemic shock, and the infusion of cold or room-temperature fluids.

In conclusion, calcium plays a pivotal role in the management of polytraumatized patients, significantly influencing their survival and recovery. Beyond its essential function in coagulation, where it activates critical factors in the clotting cascade and facilitates platelet adhesion, calcium is crucial for maintaining cardiac contractility and vascular resistance.

Hypocalcemia, often exacerbated by trauma-related factors such as hypothermia, hemodilution, and citrate accumulation from blood product transfusions, can severely impair these processes. Its deficiency not only worsens coagulopathy but also contributes to tissue hypoxia and metabolic acidosis, compounding the challenges posed by the lethal triad of trauma. Timely calcium replacement is therefore critical, as it can help stabilize hemodynamics, enhance coagulation efficiency, and improve overall outcomes in polytrauma patients [1-17].

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