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LONGITUDINAL CLINICAL AND LABORATORY PATTERNS OF CORTICO-METABOLIC DYSREGULATION IN CRITICALLY ILL PATIENTS WITH SEPSIS AND SEPTIC SHOCK

Data Kekutia[1], Levan Ratiani[1,2], Tamar Didbaridze[1,2],

[1]Tbilisi State Medical University

[2]First University Clinic, Tbilisi, Georgia.

Background: Sepsis and septic shock are accompanied by profound inflammatory, neuroendocrine, hemodynamic, and metabolic alterations. Changes in hypothalamic–pituitary–adrenal signaling, corticosteroid availability, catecholamine activity, glucose metabolism, cellular energetics, and electrolyte homeostasis may contribute to heterogeneous patterns of systemic stress adaptation. Direct endocrine phenotyping is rarely available in routine intensive care; therefore, longitudinal clinical and laboratory trajectories may provide complementary information on cortico-metabolic dysregulation.

Objective: To characterize longitudinal clinical and laboratory patterns associated with cortico-metabolic dysregulation in critically ill patients with sepsis and septic shock.

Methods: A retrospective, de-identified longitudinal case-series analysis was performed using adult intensive care records from a tertiary hospital. Eighteen unique records were screened; 16 patients with explicitly documented sepsis (A41.9) and/or septic shock (R57.2) were included in the primary analysis. Serial blood-gas parameters, glucose, lactate, sodium, potassium, ionized calcium, chloride, renal and inflammatory markers, hemodynamic status, respiratory failure, and organ-support requirements were reviewed. Routine laboratory variables were interpreted as components of an integrated systemic stress phenotype rather than specific diagnostic markers of adrenal dysfunction.

Results: Median age was 65 years (range 23–76). Shock was documented in 13/16 patients (81.3%), including explicitly coded septic shock in 6/16 (37.5%). Acute respiratory failure occurred in all 16 patients, and cardiac arrest was documented in 11/16 (68.8%). Among available serial measurements, pH ranged from 6.925 to 7.773, glucose from 52 to 661 mg/dL, lactate from 0.77

to 15.27 mmol/L, potassium from 2.38 to 6.58 mmol/L, and ionized calcium from 0.45 to 1.11 mmol/L. Marked inter- and intra-patient heterogeneity was evident. Some patients developed profound hyperglycemic, hyperlactatemic acidosis with major electrolyte abnormalities, whereas others experienced severe clinical deterioration despite only modest lactate elevation. Recurrent mixed acid–base disturbances, dysglycemia, hypokalemia, hyperkalemia, and hypocalcemia demonstrated rapid temporal variability during critical illness.

Conclusion: Sepsis and septic shock were associated with highly heterogeneous longitudinal metabolic and homeostatic trajectories. Integrated interpretation of glucose, lactate, acid–base status, electrolytes, renal function, inflammation, and hemodynamic changes may better characterize systemic stress dysregulation than isolated laboratory measurements. These patterns should not be considered diagnostic of adrenal dysfunction, but they provide a rationale for prospective studies combining routine ICU trajectories with direct cortisol, ACTH, corticosteroid-binding globulin, and free-cortisol assessment.

Keywords: Sepsis; septic shock; stress hyperglycemia; lactate; electrolyte disturbances.