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PREDICTORS OF ADRENOCORTICAL INSUFFICIENCY AMONG SEVERELY INJURED PATIENTS ATTENDING AL TUZ GENERAL HOSPITAL

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ABSTRACT

Background: Severe trauma is a leading cause of mortality and morbidity globally, placing a huge load on emergency and critical care systems. In response to severe damage, the body initiates a complicated neuroendocrine stress reactions to preserve hemodynamic stability, metabolic balance, and immunological control. Adrenocortical insufficiency might occur due to disruption of these reactions. **Objectives:** Is to identify clinical and laboratory predictors of adrenocortical insufficiency in severely injured patients at Al-Tuz General Hospital. **Methods:** The study is an observational, descriptive, prospective cross-sectional study. It was conducted between the 1st of March 2024 to the end of September 2025. All severely injured patients admitted to the emergency department or ICU at Tuz General Hospital in Salahiddin Governorate/Iraq during the study period were assessed for eligibility. The study included patients aged more than or equal to 18 years with severe trauma defined by an Injury Severity Score (ISS) ≥ 16. As well as those who admitted within 24 hours of injury and been hemodynamically unstable or requiring intensive monitoring. On the other hand, the study excluded patients with known history of adrenal insufficiency, chronic steroid therapy within the previous 3 months or patients with known pituitary or adrenal disease. Additionally, the study excluded pregnant patients and patients who died before hormonal assessment. **Results:** The study includes 40 patients severely injured, of them 10 patients had adrenocortical insufficiency matched according to gender with 30 patients without adrenocortical insufficiency. The mean age ± standard deviation of the study participants is 59.74 ± 7.64 days. Male: Female ratio was 1.22:1. Statistically significant difference between them regarding their mean age (P value <0.001), body temperature (P value < 0.001), mean arterial pressure (P value <0.001), development of sepsis (P value <0.001) and GCS (P value <0.001). Moreover, statistically significant difference between them regarding their urinary volume (P value <0.001), and serum sodium (P value < 0.001). **Conclusion:** The study found seven factors may contribute to ACI following severe injury: patients' age, body temperature, MAP, development of sepsis, abnormal GCS, urine volume, and serum sodium level. If these factors have been detected, frequent testing of plasma ACTH and cortisol levels is recommended in clinical settings.

KEYWORDS: Adrenal, Crisis, Iraq, Salahiddin, Trauma.

1. INTRODUCTION

Severe trauma is a leading cause of mortality and morbidity globally, placing a huge load on emergency and critical care systems.[1] In response to severe damage, the body initiates a complicated neuroendocrine stress reactions to preserve hemodynamic stability, metabolic balance, and immunological control.[2] The hypothalamic-pituitary-adrenal axis is activated as part of this response, resulting in increased cortisol

production, which is vital for survival in severely seriously ill or injured patients.[3]

Adrenocortical insufficiency (ACI), also known as critical illness-related corticosteroid insufficiency (CIRCI), arises when cortisol production is insufficient for the level of stress.[4] ACI has been linked to hypotension, higher vasopressor doses, poor resuscitation response, longer intensive care unit (ICU) stay, and increased death in critically injured patients. Despite its clinical significance, ACI is still underdiagnosed because of vague clinical symptoms and diagnostic criteria variation.[5-6]

Several characteristics have been identified as predictors of ACI in trauma patients, including advanced age, injury severity, traumatic brain damage, substantial blood loss, infection, extended hypotension, and the administration of certain drugs.[7] However, identified predictors differ depending on demographic and healthcare situation. Identifying accurate predictors of ACI in seriously injured patients is critical for early detection and management.[8-9]

In Iraq, there is little information on the frequency and predictors of adrenocortical insufficiency in trauma patients. Understanding these predictors in the local setting may aid in clinical decision-making and patient outcomes. As a result, the purpose of this study was to identify clinical and laboratory predictors of adrenocortical insufficiency in severely injured patients at Al-Tuz General Hospital.

2. PATIENTS AND METHODS

The study is an observational, descriptive, prospective cross-sectional study. It was conducted between the 1st of March 2024 to the end of September 2025. All severely injured patients admitted to the emergency department or ICU at Tuz General Hospital in Salahiddin Governorate/Iraq during the study period were assessed for eligibility.

The study included patients aged more than or equal to 18 years with severe trauma defined by an Injury Severity Score (ISS) ≥ 16. As well as those who admitted within 24 hours of injury and been hemodynamically unstable or requiring intensive monitoring. Informed consent obtained from the patient or legal guardian. On the other hand, the study excluded patients with known history of adrenal insufficiency, chronic steroid therapy within the previous 3 months or patients with known pituitary or adrenal disease. Additionally, the study excluded pregnant patients and patients who died before hormonal assessment. The investigators conducted direct interviews with the patients or with their responsible relative to complete self-administered structured questionnaires. The questionnaire was composed from three parts. The first part for patients' demographic information; including patient age and gender. The second part for patients' clinical parameters such as blood pressure on admission, requirement for vasopressors, presence of shock, and development of sepsis. The third part for laboratory investigations including serum cortisol level measured within the first 24 hours of admission. Adrenocortical Insufficiency was defined as: a random serum cortisol level < 15 µg/dL, or an inadequate cortisol response to stress according to institutional or accepted critical care criteria. As well as, urine volume and serum sodium level.

Statistical analysis: the demographic, clinical findings, and surgical outcomes were recorded and analyzed in Microsoft Excel. Data were entered into SPSS version 30.0, a statistical analysis tool for social sciences. Scale variables were reported as mean and standard deviation, median and interquartile range, and compared using parametric testing. In all statistical tests and procedures, level of significance P value was set at ≤ 0.05 considered as significant difference or association.

3. RESULTS

The study includes 40 patients severely injured, of them 10 patients had adrenocortical insufficiency matched according to gender with 30 patients without adrenocortical insufficiency. The mean age ± standard deviation of the study participants is 59.74 ± 7.64 years. Male: Female ratio was 1.22:1.

Table 3.1 shows comparison between patients with adrenocortical insufficiency and those with adrenocortical insufficiency regarding their clinical characteristics. Statistically significant difference between them regarding their mean age (P value <0.001), body temperature (P value < 0.001), mean arterial pressure (P value <0.001), development of sepsis (P value <0.001) and GCS (P value <0.001).

Table 3.1: Comparison between patients with adrenocortical insufficiency and those with adrenocortical insufficiency (number=40).

Variable	ACI = 10	Non ACI = 30	P value
Patient age, mean ± standard deviation	67.38 ± 6.22	54.43 ± 8.87	**<0.001**
Body temperature, number (%): -Normal -Abnormal	 2 (20%) 8 (80%)	 15 (50%) 15 (50%)	**<0.001**
Mean arterial pressure number (%): -Normal -Abnormal	 1 (10%) 9 (90%)	 15 (50%) 15 (50%)	**<0.001**
Pulse, number (%): -Normal -Abnormal	 5 (50%) 5 (50%)	 17 (56.67%) 13 (43.33%)	0.122
Respiratory, number (%): -Normal -Abnormal	 9 (90%) 1 (10%)	 29 (96.67%) 1 (3.33%)	0.392
Development of sepsis, number (%): -Yes -No	 7 (70%) 3 (30)	 3 (10%) 27 (90%)	**<0.001**
Glascow coma score, median (interquartile range)	9 (6-11)	13 (12-15)	**<0.001**

Table 3.2 shows comparison between patient with adrenocortical insufficiency and those with adrenocortical insufficiency regarding their laboratory investigations. Statistically significant difference between them regarding their urinary volume (P value <0.001), and serum sodium (P value < 0.001).

Table 3.2: Comparison between patient with adrenocortical insufficiency and those with adrenocortical insufficiency regarding their laboratory investigations (number = 40).

Variable	ACI = 10 (%)	Non ACI = 30 (%)	P value
Urinary volume: -Normal -Abnormal	 2 (20%) 8 (80%)	 23 (76.67%) 7 (23.33%)	**<0.001**
Serum sodium level: -Normal -Abnormal	 1 (10%) 9 (90%)	 24 (80%) 6 (20%)	**<0.001**

4. DISCUSSION

Redistributing energy and making energy more accessible are examples of how appropriate reactions to acute stress would promote survival.[10] Damage to the HPA axis or secondary ACI after multiple traumas could inhibit the body's defensive stress response, impact hemodynamics, and worsen cerebral inflammation and neurodegeneration.[11] Predicting and managing high-risk individuals with ACI is critical for body function recovery.

This study concentrates on the clinical and biochemical predictor of ACI among patients with severe injury. Among nine factors included, the study found the age of patients with ACI was significantly higher than patients with no ACI. A study on patients with traumatic brain injury (TBI) reported that the incidence of ACI in older patients (≥60 years) was significantly higher than in younger patients (<60 years).[12] This increased susceptibility in older patients may be due to a higher prevalence of existing comorbidities and a potentially reduced functional capacity of the hypothalamic-pituitary-adrenal (HPA) axis with age.

Regarding vital signs, the study found patients with ACI exhibited more abnormal body temperature specifically hypothermia in comparison to patients with no ACI, this suggests that patients with ACI experience a dysregulation of their normal thermoregulatory responses, leading to a higher incidence of significant, and potentially life-threatening, deviations from normal body temperature (both low and high). Comparable results obtained from other studies.[12-13] The same with mean arterial pressure, the study found patients with ACI significant had higher percentage of abnormal MAP than patients with no ACI particularly a higher likelihood of hypotension. This hypotension is often resistant to standard fluid resuscitation and vasopressor support. Parikshak et al[14] and Shenker et al[15] showed similar findings.

The present study showed patient with ACI significantly having more sepsis than patient with no ACI, this might

due to decreased HPA axis function, abnormal cortisol metabolism, and tissue corticosteroid resistance. These lead to an excessive protracted proinflammatory response, particularly in ICU patients. Which runs with Fredrick et al[16] study results. Additionally, the present study found patients with ACI had significantly lower GCS than patients with no ACI. Lower GCS scores are typically associated with more severe injuries, worse neurological outcomes, and higher fatality rates. The presence of ACI in critically ill or seriously injured individuals suggests a failure of the body's normal stress response and is linked to poorer clinical outcomes, which correspond to a lower GCS score. An adrenal crisis, a severe symptom of ACI, might potentially result in coma if left untreated. Which in agreement with other studies' results.[17-18]

On the other hand; regarding the laboratory findings, the study found patients with ACI had significantly higher percentage of abnormal urine volume (either polyuria, excessive urination, or oliguria, decreased urination) and abnormal serum sodium level than patients with no ACI. This might due to disruption of the central regulating mechanism of fluid balance via various neuro-humoral factors such as antidiuretic hormone (ADH) and aldosterone. Furthermore, mineralocorticoids deficiency leads to increased urinary sodium loss and hypovolemia, which result in hypotension and reduced renal perfusion, triggering oliguria. Feng et al[12] showed similar findings.

When evaluating the results, it is important to take into account the limitations of the current study. First, the results could be more difficult to generalize to other groups due to the limited sample size. Second, the study was carried out at a single hospital, which probably reduced the findings' external validity.

5. CONCLUSIONS AND RECOMMENDATIONS

The study found seven factors may contribute to ACI following severe injury: patients' age, body temperature, MAP, development of sepsis, abnormal GCS, urine volume, and serum sodium level. If these factors have been detected, frequent testing of plasma ACTH and cortisol levels is recommended in clinical settings.

CONFLICT OF INTERTEST

The authors of this study report no conflicts of interest.

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