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PREDICTORS OF RESPONSE TO FIXED-DOSE VASOPRESSIN IN ADULT PATIENTS WITH SEPTIC SHOCK
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Learning Objectives: Vasopressin (AVP) is often utilized for hemodynamic support in patients with septic shock. However, at this time, the most effective dose and the most appropriate patient to initiate therapy is in unknown. This study was conducted to determine the factors associated with hemodynamic response to fixed-dose AVP in patients with septic shock. Methods: This was a single center, retrospective cohort of patients who received fixed-dose fixed-dose AVP for septic shock for at least 6 hours after initiation of AVP. Results: There were 938 patients included: 426 responders (45%), 512 non-responders (55%). Baseline characteristics were well-balanced except for differences in baseline MAP (responders 69 ± 12 vs. non-responders 65 ± 12 mmHg; p < 0.001) and baseline lactate (responders 4.0 ± 3.6 mmol/L vs. non-responders 5.4 ± 4.4 mmol/L; p < 0.001). There was no difference in AVP dose between groups (overall mean 0.031 units/min). Responders had lower rates of in-hospital mortality (72% vs. 72%; p < 0.001) and ICU mortality (50% vs. 68%; p < 0.001). Responders also had increased ICU-free days at day 14 (2.3 ± 3.8 vs. 1.6 ± 3.3; p < 0.001) and hospital-free days at day 28 (4.2 ± 7.2 vs. 2.8 ± 6.0; p < 0.001). On univariable analysis, non-medical ICU location was associated with increased response odds (OR 1.70, 95% CI 1.18–2.46; p = 0.004) and lactate at AVP initiation was associated with decreased response odds (OR 0.93, 95% CI 0.89–0.97; p < 0.001). Factors associated with response included medical ICU location, APACHE III score, SOFA score, and baseline lactate. Conclusions: In this large cohort of patients with septic shock, 45% responded to the addition of fixed-dose AVP. Responders had improved outcomes compared to non-responders. The only factors found to be associated with AVP response were ICU location and lactate.

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CORRELATION OF CIRCULATING 8-OXODEOXYGUANOSINE WITH THE SEVERITY OF SEPTIC ORGAN DAMAGE
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Learning Objectives: Tissue 8-oxodeoxyguanosine (8-OH-dG) levels have been considered a parameter of oxidative DNA damage under an ischemic intracellular state and in the pathogenesis of persistent sepsis; therefore, multiple organ dysfunction occurs. However, correlation of circulating 8-OH-dG levels with septic organ damage has not been elucidated. Methods: To determine correlations between circulating and organ tissue 8-OH-dG levels, mean arterial pressure (MAP), AST, ALT, creatinine, and pH were measured. Fifty-two young male C57BL/6 mice were subjected to either 1 of 2 different cecal ligation and puncture procedures to produce mid- or high-grade sepsis. Heart, lung, liver, and aortic blood samples were obtained from mice 24 h after sepsis induction. Both tissue and circulating 8-OH-dG levels were estimated by high-performance liquid chromatography (HPLC). Results: In both the mid- and high-grade sepsis groups, circulating 8-OH-dG levels significantly increased up to 24 h based on sepsis severity; therefore, we used sample points from HPLC at 24 h after CLP. Correlation analysis for circulating 8-OH-dG revealed no changes in lung tissue for either group (lung: p = 0.17, r = 0.09); pH alone correlated with circulating 8-OH-dG levels (pH: p < 0.05, r = 0.80). Circulating 8-OH-dG levels increased in heart and liver tissue with sepsis severity (heart: p < 0.05, r = 0.79; liver: p < 0.05, r = 0.45); MAP, AST, ALT, and creatinine levels demonstrated similar trends (MAP: p < 0.05, r = 0.63; AST: p < 0.05, r = 0.80; ALT: p < 0.05, r = 0.57; creatinine: p < 0.05, r = 0.65). Thus, circulating 8-OH-dG levels are enhanced in damaged tissue with abundant mitochondria. Conclusions: Circulating 8-OH-dG may be an early direct marker of ischemic intracellular damage in septic organ damage.

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THE EFFECT OF OBESITY ON MORTALITY IN TRAUMA PATIENTS WITH SEPSIS
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Learning Objectives: There is an obesity epidemic in the United States with over one-third of the adult population having a body mass index (BMI) of 30 kg/m2 or greater. To date there has been no study that assesses the impact of obesity in trauma patients that develop sepsis. The purpose of this study is to evaluate the effect of obesity on in-hospital mortality in this specific population. Methods: This was a retrospective cohort study including all adult trauma patients that developed sepsis with ≥72 hours of antimicrobial therapy from January 2013 to December 2014. Obese patients (BMI ≥ 30 kg/m2) were compared to non-obese patients and data collection included: baseline demographics, comorbidities, Injury Severity Score (ISS), source of infection, antimicrobial data, and clinical outcomes. The primary outcome was in-hospital mortality between obese vs. non-obese patients. Results: A total of 1,971 patients were screened with 251 patients meeting inclusion criteria (182 (73%) non-obese vs. 69 (27%) obese patients). Baseline characteristics were similar between the two groups regarding age, ISS, and mechanism of trauma. The obese group had more male patients (91% vs. 81%; p = 0.04) and a history significant for hypertension (37.7% vs. 19.8%; p = 0.003), hyperlipidemia (18.8% vs. 6.6%; p = 0.004), diabetes (26.1% vs. 7.7%; p = 0.001), and coronary artery disease (14.5% vs. 3.8%; p = 0.003). No difference in hospital mortality was found between groups (non-obese: 19.8% vs. obese: 15.9%; p = 0.588). Obese patients had a higher incidence of septic shock (49.3% vs. 35.2%; p = 0.044), a longer hospital length of stay (LOS) (26.5 vs. 22.9 days; p = 0.023), and a longer time to clinical resolution of infection (13.5 vs. 9 days; p = 0.02) compared to non-obese patients. Conclusions: Sepsis in obese trauma patients is not associated with an increased risk of in-hospital mortality, but may be associated with higher incidence of septic shock, prolonged hospital LOS, and longer time to infection resolution. Larger, prospective studies are needed to validate these findings.

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PREDICTIVE MODELS OF FEVER AND MORTALITY IN HOSPITALIZED PATIENTS WITH NEUTROPENIA
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Learning Objectives: Febrile neutropenia is a medical emergency associated with high morbidity and mortality. Since early intervention can decrease mortality in these critically ill patients, a validated early warning score (EWS) could lead to improved outcomes. Consequently, we aimed to create optimized predictive models of fever and mortality in hospitalized patients with neutropenia. Methods: Using the University of Virginia electronic health record (EHR) data, we identified neutropenic patients 218 years who developed fever during their hospitalization. Neutropenia was defined as an absolute neutrophil count <500 cells/mm³. Onset of fever was defined as the first recorded instance of temperature >38.0°C. We then built multivariate logistic regression models using clinical laboratory and vital sign data to predict 1) neutrophic fever up to 24 hours prior to fever onset and 2) mortality. The models were trained and evaluated using 10-fold cross-validation. Results: We identified 503 patients with neutropenia admitted to UVA from 2010 to 2015. Fever occurred in 341 (68%) patients and 80 (16%) patients died. The final neutropenic fever model had a c-statistic of 0.77 and included the following variables: temperature, systolic and diastolic blood pressures, heart and respiratory rates, white blood cell concentration, and blood urea nitrogen. The model identified patients a mean of 9.5 hours before onset of fever. The final neutropenic mortality model had a c-statistic of 0.80 and included the following variables: pulse rate, sodium, glucose, magnesium, total bilirubin, and total protein. Conclusions: We created predictive models for neutropenic fever and mortality which could be implemented into the EHR as an EWS to facilitate early interventions and improved outcomes in this critically ill patient population.