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USE OF PERIPHERAL VASOPRESSORS IN EARLY SEPSIS-INDUCED HYPOTENSION ACROSS MICHIGAN HOSPITALS

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INTRODUCTION: Recent data suggest it may be safe to administer vasopressors via peripheral IV (PIV), challenging convention that vasopressors must be delivered centrally. Surviving Sepsis Campaign 2021 guidelines suggest using peripheral vasopressors as a bridge to central access. However, little is known about vasopressor initiation in practice.

METHODS: Cohort study of patients hospitalized with community-onset sepsis at 12 hospitals in the Hospital Medicine Safety Consortium (HMS) sepsis initiative. HMS is a Collaborative Quality Initiative sponsored by Blue Cross Blue Shield of Michigan. A random sample of adult sepsis hospitalizations between 11/2020-1/2022 were included. Data were abstracted by trained abstractors. We sought to determine how commonly vasopressors were initiated via PIV vs central access across hospitals. HMS-Sepsis is expanding to 69 hospitals. Here we present pilot data; full cohort analysis is in process.

RESULTS: Of 1,901 patients in the HMS-Sepsis registry at the time of pilot data analysis, 440 (23.1%) had hypotension (defined by mean arterial pressure< 65mmHg, systolic blood pressure< 90mmHg, and/or vasopressor initiation) within 3 hours of hospital arrival. Of these, 160 (36.4%) received vasopressors within 6 hours of hospital arrival. Route of initial vasopressor was PIV in 122 (76.3%), central access in 30 (18.8%), midline catheter in 1 (0.6%), oral (ie, midodrine) in 5 (3.1%), and unknown in 2 (1.3%). Across all hospitals, 50.0% to 91.7% of vasopressor initiation was via PIV (median 83.3%). Among 122 patients with vasopressor initiation via PIV, 66 (54.1%) received a 2nd vasopressor, after a median of 2.8 hrs [IQR 1, 8] from 1st vasopressor. Route of 2nd vasopressor was PIV in 27 (40.9%) and central access in 30 (45.4%). Time from hypotension to vasopressor initiation did not differ between patients receiving initial vasopressor via PIV vs central access (median 1.9 vs 2.1 hrs, p=0.79). Likewise, IV fluids within 6 hrs (median 2.0 vs 2.1L, p=0.78), hospitalization length (median 7 vs 6 days, p=0.31), and in-hospital mortality (33.6% vs 40.0%, p=0.51) were similar.

CONCLUSIONS: In this 12-hospital cohort, vasopressors were most frequently initiated peripherally. Outcomes were similar between patients in whom vasopressors were initiated via peripheral vs central access.

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PREVALENCE, RISK FACTORS, AND OUTCOMES ASSOCIATED WITH DELAYED SECOND DOSES OF ANTIBIOTICS IN SEPSIS

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INTRODUCTION: Best practice guidelines and quality measures emphasize timely administration of initial antibiotics in patients with sepsis but there are limited data on the prevalence, risk factors, and clinical impact of delays in subsequent doses.

METHODS: We retrospectively identified all adult patients admitted to a large academic hospital who triggered an electronic sepsis alert in the emergency department (ED), received ≥2 doses of vancomycin or an antipseudomonal beta-lactam, and were discharged with an ICD-10 sepsis code between January 2018-December 2019. We calculated the prevalence of delays in second doses of vancomycin or antipseudomonal beta-lactams, defined as ≥25% of the recommended dose interval (accounting for initial renal function), and conducted multivariable regression analyses to assess for risk factors for delays (including demographics, comorbidities, SOFA score, lactate, creatinine clearance, source of infection, time in the ED, ICU admission, ED vs non-ED location at time of 2nd dose, need for stress dose steroids) and the association between delays and short-term mortality (in-hospital death or discharge to hospice).

RESULTS: The cohort included 449 patients with sepsis, of whom 123 (27.4%) had delays in second doses of vancomycin or antipseudomonal beta-lactams. Short-term mortality occurred in 38 patients (30.9%) in the delayed group and 89 (27.3%) in the non-delayed group (p=0.45). On multivariable analysis, only location in a non-ED unit at the time second doses were due was significantly associated with delays (OR 2.75, 95% CI 1.20-6.32). In the mortality model, significant risk factors included malignancy (OR 2.11, 95% CI 1.26-3.53), respiratory infection (OR 1.91, 95% CI 1.15-3.17), and elevated SOFA score (OR 1.16, 95% CI 1.08-1.25), but not delayed second antibiotic doses (OR 1.19, 95% CI 0.69-2.05).

CONCLUSIONS: Over a quarter of patients treated for sepsis in the ED experienced delays in second doses of antibiotics; these delays were more likely when the patient had transferred out of the ED before the second dose was due, suggesting that transitions of care may have contributed. Delayed second doses were not significantly associated with mortality, although our study may be underpowered to detect small differences in this outcome.