Norepinephrine/vasopressin

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Lack of drug effect: case report

A 73-year-old man exhibited a lack of drug effect during treatment with norepinephrine and vasopressin for management of septic shock [*routes and dosages not stated*].

The man presented to the emergency department (ED) with anorexia and progressive weakness for a week. His medical history was significant for hypertension, hyperlipidemia, gout and diabetes mellitus. On admission, he was mildly hypoxic with oxygen saturation 90%. Therefore, he required oxygen supplementation via nasal cannula. His laboratory test showed leukopenia (1400/µL). Subsequently, chest x-ray demonstrated subtle diffused bilateral interstitial opacities. Later, he experienced a severe hypoxic respiratory failure and required mechanical ventilation. Therefore, he was shifted to ICU, for ventilatory management and monitoring of hemodynamics. It was considered that the acute respiratory distress syndrome was due to the suspected COVID-19. Subsequently, he was commenced on unspecified broad broad-spectrum antibiotics covering community-acquired pneumonia along with norepinephrine and vasopressin for management of septic shock. However, his respiratory status continued to worsen. Thereafter, he required FiO_2 of 60%. On hospital day 2 (HD), Point of care ultrasound (POCUS) revealed preserved systolic function of the left ventricle (LV) and a normal-sized right ventricle (RV). On the following day, he was diagnosed with COVID-19. Subsequently, he started receiving treatment with azithromycin and hydroxychloroquine. On HD 5, he developed pancytopenia, due to bone marrow suppression. On HD 6, he required increased vasopressor support, as his condition continued to worsen. Also, his troponin-I was increased (3.16 ng/mL). Subsequently, POCUS revealed diffused b-lines of the overlaying lungs, a severely dilated RV, dilated and non-variable inferior vena cava, and a hyperdynamic and underfilled LV with no apparent regional wall motion abnormalities. Also, POCUS of the lower extremities showed fully compressible deep veins at the femoral and popliteal level bilaterally. Later, it was noted that his haemoglobin level was decreasing, and therefore, treatment with unspecified antiplatelet and unspecified anticoagulation was stopped. He was transfused with packed red blood cells. On HD 7, he developed hepatic dysfunction and progressive renal failure. Also, his transaminases were elevated. Telemetry revealed ST-segment elevation, which was also confirmed from EKG. The chest x-ray showed progression of bilateral pulmonary opacities, and POCUS was unchanged. He was considered a poor candidate for thrombolytic and invasive intervention therapy.

Eventually, the man became bradycardic and hypotensive. His telemetry tracing progressed to asystole, and on the night of HD 7, he died due to asystole.

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