Takotsubo Cardiomyopathy: Severe Hyponatremia from SIADH, a Real Heart-Breaker

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Intro: Takotsubo cardiomyopathy (TTC), is defined as a sudden, temporary, stress-induced systolic abnormality of the left ventricular apex. TTC occurs predominantly in Asian or Caucasian postmenopausal women, following exposure to unexpected physical or emotional stress. In addition to triggers for Takotsubo cardiomyopathy can include medical, surgical, and pharmacological etiologies. We present a case who was admitted for severe hyponatremia secondary to SIADH concomitantly diagnosed with Takotsubo cardiomyopathy.

Case: A 73-year-old female with history of major depressive disorder & hypertension, presented with encephalopathy. On the morning of admission, she was found displaying left-sided eye twitching, staring spells, and incoherent speech. Home medicines included hydrochlorothiazide, sertraline, and amitriptyline. Vital signs were stable. Initial EKG was unremarkable. Patient was emotionally labile, oriented to name only, and had no focal neurologic deficits. The patient's pertinent admission labs included WBC 29,300 uL, Na 105 mmol/L, serum osm 237 mOsm/kg, urine osm 425 mOsm/kg, and urine Na 42 mOsm/dL. CT head was unremarkable. Patient was diagnosed with SIADH and started on hypertonic saline in the ICU. Sodium levels recovered appropriately, however shortly after admission, she developed new-onset shortness of breath, hypoxia, and diffuse bibasilar crackles. Troponins peaked at 2,441 pg/mL (reference <12 pg/mL) within 24 hours. BNP was 1,909 pg/mL (reference <100 pg/mL). Chest CT confirmed bilateral ground-glass opacities and small bilateral effusions. A transthoracic echocardiogram was obtained and revealed an ejection fraction of <20%, abnormal LV strain without thrombus, and hypokinetic contraction of the basal heart wall segments with complete akinesis of apical wall segments, consistent with Takotsubo cardiomyopathy. Three weeks prior, she had chest pain that prompted a coronary angiogram which revealed no obstruction and an ejection fraction of > 55%. Patient was monitored further on the floor and recovered gradually with supportive care, including fluid restriction and diuresis.

Discussion: There are multiple hypotheses for the pathophysiology of TTC; catecholamine cardiac toxicity and microvascular dysfunction are the most widely accepted proposed mechanisms. Excess catecholamine levels following a stressful event are suggested to lead to myocardial stunning via calcium overload, production of reactive oxidative species, and mitochondrial dysfunction following activation of beta adrenergic receptors. Fortunately. Takotsubo cardiomyopathy typically has a favorable prognosis with supportive treatment. Therefore, less common etiologies such as hyponatremia should be considered and addressed to attain that strong prognosis.