**1)A CASE OF STEMI SECONDARY TO CAPECITABINE. HAVE YOU CHECKED THE MEDICATION HISTORY?**

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**Background:** Capecitabine is one of the common chemotherapeutics used in the treatment of solid tumors. It is believed to have a safer profile when compared to 5 fluoro-uracil. However, cardiotoxicity has been reported.

**Case Presentation:** A 68-year-old female who presented to the emergency department with sudden onset retrosternal chest pain for three hours while sleeping. She was started recently on Capecitabine for Stage IV rectal adenocarcinoma with lung metastases. Her past medical history was remarkable for colon cancer status post colectomy seven years ago, hypertension, polysubstance abuse & hepatitis C. Vital signs were within normal limits. Electrocardiogram (EKG) showed diffuse ST- elevation (STE), that resolved after 30 mins. She had recurrent symptoms after four hours, repeated EKG showed STE of the anterolateral precordial leads which resolved after 15 mins.

**Decision making:**

Patient was started on sublingual nitroglycerin, followed by nitroglycerin drip. Three sets of troponin I came back normal. Left heart catheterization showed single vessel moderate nonobstructive coronary artery disease (50% -distal left anterior descending artery). An Echocardiogram showed normal left ventricular size and function without any regional wall motion abnormalities. The patient’s presentation is explained by the recent use of Capecitabine. The transient STE during the chest pain episodes in the absence of significant epicardial coronary artery disease on LHC are supportive. Proposed mechanisms include coronary artery vasospasm, vascular endothelial dysfunction, direct toxicity on the myocardium & thrombogenicity. Risk factors include frequency of administration, duration of treatment, decreased renal clearance & concomitant cardiotoxic chemotherapeutics. Management includes discontinuation of the chemotherapeutic agents & coronary vasodilator agents. The patient was discharged on amlodipine & Imdur. Further doses of Capecitabine were discontinued.

**Conclusion:** We report a patient with recurrent ST elevation due to Capecitabine cardiotoxicity. Clinicians should maintain high level of suspicion for coronary vasospasm in patients who were recently started on Capecitabine.

**2) ATYPICAL ADULT-ONSET STILL’S DISEASE PRESENTING WITH MYOPERICARDITIS**

**Authors: Merna Shata**

**Background:** Adult-onset Still’s Disease (AOSD) is a rare acute systemic inflammatory disorder characterized by high spiking fever, neutrophilic leukocytosis, maculopapular rash, arthritis & rarely serositis. Skin rash & arthritis are often typical features. This is a case with atypical presentation where skin rash & arthritis were not manifested.

**Case:** A 38-year-old male presented with fever (103 F), sore throat & pleuritic chest pain. On exam, he was hypotensive, tachycardic & tachypneic. Initial lab work showed high troponin (2.34 ng/ml), echo showed a newly decreased biventricular dysfunction with EF of 40-45% & mild pericardial effusion. Patient was admitted to the ICU for myopericarditis & acute heart failure requiring vasopressors. He was started on dexamethasone & colchicine then successfully weaned off pressors and transferred to the floor. Patient kept having high spiking fever with tachycardia, sore throat & pleuritic chest pain. He also had respiratory distress with a single attack of hemoptysis. Chest CT showed bilateral ground glass opacity with bilateral pleural effusion. Extensive infectious workup was negative for bacterial, viral or fungal agents. Advanced rheumatological, hematological & oncological workup revealed leukocytosis > 30000 with neutrophilia, ferritin > 40000, high IL2, negative (ANA, rheumatoid factor (RF), dsDNA and ANCAs). Bone marrow biopsy was negative for HLH, pleural fluid was exudative & BAL was negative for alveolar hemorrhage.

**Decision-Making**: Patient was started on IV steroids for a possible autoimmune phenomenon & naproxen for pericarditis. Repeated echo showed improved EF, but fever & chest pain persisted. While the patient didn’t have any skin rash or arthritis, he had several features suggestive of AOSD including high grade fever, elevated ferritin, serositis, negative ANA, negative RF & sore throat. The decision was made to start him on methotrexate along with steroid taper after which he started to improve.

**Conclusion:** AOSD is an uncommon disease, where the diagnosis can be difficult to establish given the complexity of symptoms. It is also an uncommon cause for myopericarditis and should be considered after excluding more common etiologies.

**3) AMIODARONE INDUCED MYXEDEMA COMA PRESENTING WITH CARDIOGENIC SHOCK AND JUNCTIONAL RHYTHM**

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**Background:** Amiodarone is a commonly used antiarrhythmic agent. It has variable effects on thyroid function, including hypothyroidism, hyperthyroidism, and, in few reported cases myxedema coma which is a life-threatening emergency.

**Case:** A 76-year-old male with a past medical history of hypertension and atrial fibrillation, who was recently started on amiodarone about 2 months before presenting with generalized weakness. He was found to be hypotensive and bradycardic with a heart rate in the 20s-30s beats per minute. His temperature was 35.9 Celsius. On physical examination: he was somnolent, his extremities were cold, no thyroid enlargement was felt, and no signs of volume overload were observed. His initial electrocardiogram revealed junctional rhythm with interventricular conduction delay. He received atropine with no improvement. He was started on dopamine and norepinephrine. Laboratory investigations were significant for elevated TSH (thyroid-stimulating hormone): 180 uIU/mL, decreased free T4 (thyroxine): <0.40 ng/dL and decreased free T3 (Triiodothyronine): 2.3 pg/mL. He also had hyponatremia, elevated creatinine, and hyperkalemia. Troponins were within normal limits and blood cultures were negative. Echocardiography revealed an ejection fraction of 35-45% with no evidence of pericardial effusion.

**Decision‐making:** Based on his presentation and laboratory findings he was diagnosed with myxedema coma and was started on intravenous (IV) T4 (Levothyroxine), and IV T3 (Liothyronine) in addition to IV Hydrocortisone until the coexistence of adrenal insufficiency was ruled out. His heart rate improved, and his electrocardiogram changes resolved. He was weaned off vasopressors and his electrolytes and kidney function went back to his baseline. Five days after starting IV thyroid hormone replacement his TSH and T4 improved to 34.99 uIU/mL and 0.75 ng/dL, respectively. He was discharged home on oral Levothyroxine 88 mcg daily.

**Conclusion:** Myxedema coma is a life-threatening emergency that has been reported in a few cases of patients taking amiodarone. It is important to check thyroid function before starting amiodarone and in cases of cardiogenic shock or rhythm disturbances.