A 34- year old man presented to an outside hospital with 16 hours of worsening right flank pain, nausea, and vomiting. He had a past medical history of human immunodeficiency virus (HIV) and hospitalization 12 weeks prior for a painful vesicular rash consistent with herpes zoster infection. On the current presentation he reported 3 months of right sided rib and flank pain that had been getting acutely worse over the past 16 hours. He also had new onset non-bloody non-bilious emesis which prompted him to seek urgent evaluation. At the outside hospital a CT of the chest, abdomen, and pelvis showed innumerable rounded consolidative opacities throughout the lungs, some of which were cavitary, and multiple superficial lesions overlying the lower pelvis and groin, raising concern for tuberculosis or metastatic malignancy. His ECG showed sinus rhythm with 1-2mm ST segment elevations in leads V2-V5 with T wave inversions, as well as T wave inversions in leads II, III, and aVF (figure 1). The patient received 325mg aspirin and was urgently transferred to our institution.

On arrival to our hospital, the patient was emergently evaluated by the cardiology service. He reported ongoing right flank pain without further nausea or vomiting. He did not have any previous or current chest pain or shortness of breath, abdominal pain or dysuria. He noted unintentional 35 pound weight loss and night sweats over the past 3 months as well as non-productive cough without hemoptysis. His only daily medication was gabapentin for zoster pain, and he was still at the contemplative phase for initiating antiretroviral therapy for HIV. He was a former smoker with a 5 pack year history, and denied IV drug use. His family history was notable for a mother with a myocardial infarction at age 50.

On exam he was afebrile and hypertensive with blood pressure 170/90. He appeared uncomfortable and cachectic with dry mucous membranes and oral thrush. His cardiovascular exam was notable for an absence of jugular venous distension, absent cardiac murmurs, rubs, or gallops, with strong distal pulses and no lower extremity edema. He had pulmonary crackles at the left lung base and marked clubbing of bilateral digits. He had prominent bilateral inguinal lymphadenopathy. A repeat ECG showed sinus rhythm with 2mm ST elevations in leads V3-V5 with deep T wave inversions, as well as T wave inversions in the inferior leads, II, III, and aVF (figure 2). His laboratory results were notable for a hemoglobin of 12.4 g/dL, sodium 130 mmol/L, aspartate aminotransferase 88 U/L, lactate dehydrogenase (LDH) 3943 uL (normal 171-308), an HIV viral load of 49,000 copies/mL, and an absolute CD4 count of 73. A troponin I was positive at 0.695 ng/mL (reference range <0.033) with an elevated B-type natriuretic peptide 506 pg/mL.

The patient's ECG raised concern for an acute antero-lateral myocardial infarction. However, his age, lack of chest pain or dyspnea, and only mild troponin elevation despite more than 16 hours of ongoing discomfort argued against an acute plaque rupture and against acute ST elevation myocardial infarction. Given his HIV status, several months of constitutional symptoms, CT scan findings, and markedly elevated LDH we had higher suspicion for an infectious process or malignancy. We therefore decided not to pursue urgent cardiac catheterization and instead obtained a transthoracic echocardiogram (TTE) to evaluate for an alternative explanation. This study demonstrated moderately increased left ventricular (LV) wall thickness, low normal LV systolic function with ejection fraction 50-55% with low reflectance material layering on the interventricular septum within the right ventricular (RV) cavity toward the RV apex, at the base of the RV near the tricuspid annulus as well as within the right ventricular outflow tract and left atrium (figures 3 and 4). Based on the patient's history, presentation, and these echocardiographic findings, our differential diagnosis included secondary cardiac malignancy, primary cardiac malignancy, cardiac thrombus with potential emboli to the coronary arteries, systemic infection with myocardial involvement, and less likely acute coronary syndrome or infiltrative disease such as sarcoidosis. In order to better characterize the "low reflectance material" seen on echocardiogram, a cardiac MRI was ordered.

The patient concurrently underwent a bronchoscopy which demonstrated pneumocystis pneumonia, CMV, candida, and mycobacterium avian complex. Bronchoscopy and induced sputa returned negative for tuberculosis. Given his B symptoms, markedly elevated LDH, HIV with now AIDS defining illness pneumocystis, a biopsy of the right thigh mass was performed to evaluate for AIDS associated malignancy. After this procedure, the patient refused further testing including cardiac MRI, citing his frustration with being in the hospital and left the hospital against medical advice.

Several days post discharge, the patient's right thigh biopsy returned demonstrating atypical lymphocytes, immunohistochemistry CD 3+, CD8+, CD43+, EBER + by ISH with ki 67 90% proliferation index, confirming diagnosis of extranodal NK/T- cell lymphoma, nasal type. Bronchiolar lavage cytology returned with the same results, which confirmed the diagnosis of stage IV high grade NK/T- cell lymphoma, a rapidly progressive malignancy with poor prognosis. He was urgently readmitted to the hospital for initiation of mSMILE (dexamethasone, methotrexate, ifosfamide, peg L-asparaginase, etopiside). After two cycles of chemotherapy, a PET showed marked improvement in lung opacities with remaining nodular opacities in the lower lobes with hypermetabolic activity. There was no increased metabolic activity in the myocardium. A repeat echocardiogram showed normalization of LV wall thickness, preserved LV function, regression of previous extensive low reflectance material in the myocardium and heart chambers (figures 3 and 4). After 3 cycles of chemotherapy his ECG showed sinus rhythm with resolution of ST segment elevations with remaining T wave inversions in leads V4 and V5 only (figure 5). The remarkable near normalization of the patient's ECG and echocardiogram with treatment of his NK/T- cell lymphoma are highly suggestive of lymphomatous involvement of the myocardium that regressed with systemic chemotherapy. The patient has completed 4 cycles of chemotherapy and now awaits re-staging PET CT and stem cell transplant.

In this case, the ECG is a distractor in a complex case of a severely immunocompromised patient who was ultimately diagnosed with a rare and life -threatening malignancy. On review of the literature, there have only been 19 cases of malignancy with secondary cardiac involvement with STEMI-like ECG changes and evidence of myocardial involvement on imaging[1]. While it is essential to consider a life threatening acute coronary event in a patient with ST elevations on ECG, it is equally as important to evaluate the patient, consider the clinical context and other potential etiologies before directly proceeding to the catheterization lab. Additional urgent imaging, in our case a bedside TTE, lead us to favor an infiltrative disease and promptly move to the next steps to determine the underlying etiology. Our patient was ultimately diagnosed with NK/T- cell lymphoma, which is a Non-Hodgkin lymphoma that is relatively rare, associated with Epstein-Barr Virus, and is seen most frequently in Asia, South and Central America, and occasionally Western countries. While it was initially thought to be rare in the HIV population and less common then the B cell lymphomas, there is mounting evidence that NK/T- cell lymphoma is being found more frequently in the HIV population, particularly in males with CD4 count less than 200[2]. NK/T- cell lymphomas present most commonly in the nasal cavity, but can also occur in other sites including the gastrointestinal tract, orbit, and testes. While lymphomatous involvement of the heart has been detected in 14-24% of lymphoma patients on autopsy, NK/T- cell lymphoma is not commonly known to have myocardial involvement[3,4]. There are case reports of myocardial involvement of NK-T cell lymphoma with diagnosis made after development of arrhythmia (ventricular tachycardia, supraventricular tachycardia) during administration of chemotherapy, however there have not been previously described cases of NK/T- cell lymphoma with initial ST elevations on presenting ECG[5,6,7] .

This patient's low health literacy added an additional layer of complexity to an already rare and challenging presentation. It was his lack of understanding of the severity of his illnesses that led him to delay initiation of antiretroviral therapy for HIV, and delayed diagnosis and expedited treatment of his advanced stage malignancy. It is notable that the patient did not have advanced imaging such as a cardiac MRI or an initial staging PET CT prior to initiation of chemotherapy. While it would have been helpful to have the information from these tests, it ultimately did not change the patient's diagnosis or further delay his initiation of live saving chemotherapy. Since his initial presentation, his multidisciplinary team of providers has worked together to eliminate unnecessary testing, consolidate doctors' appointments and trips to the hospital in an effort to provide patient centered care, proposing a care plan that the patient is able to understand and that allows him to stay engaged in his care. As noted in a recent AHA scientific statement, it is crucial that we work to address barriers imposed by limited health literacy to decrease health disparities and improve patient outcomes[8]. Our case not only highlights the challenges to approaching a rare cause of a common ECG finding, but also the complexity of caring for a patient with low health literacy.