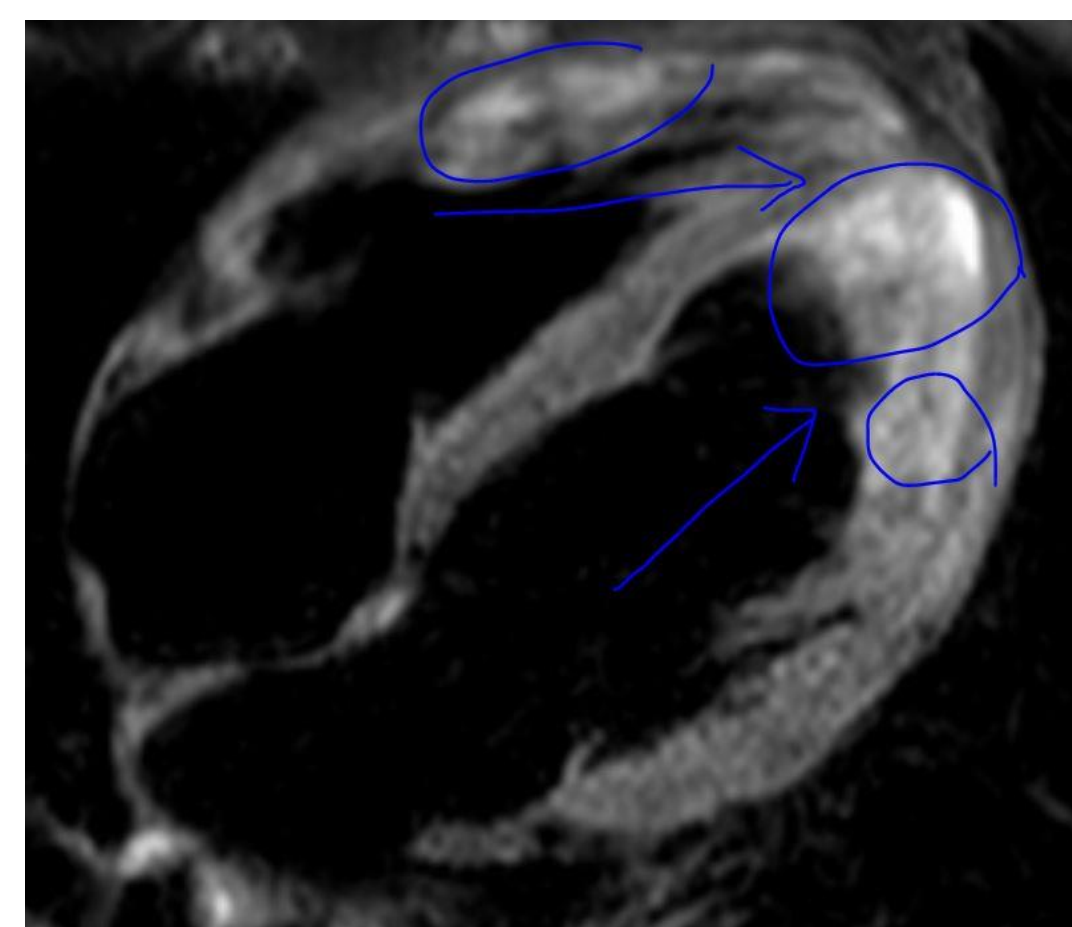
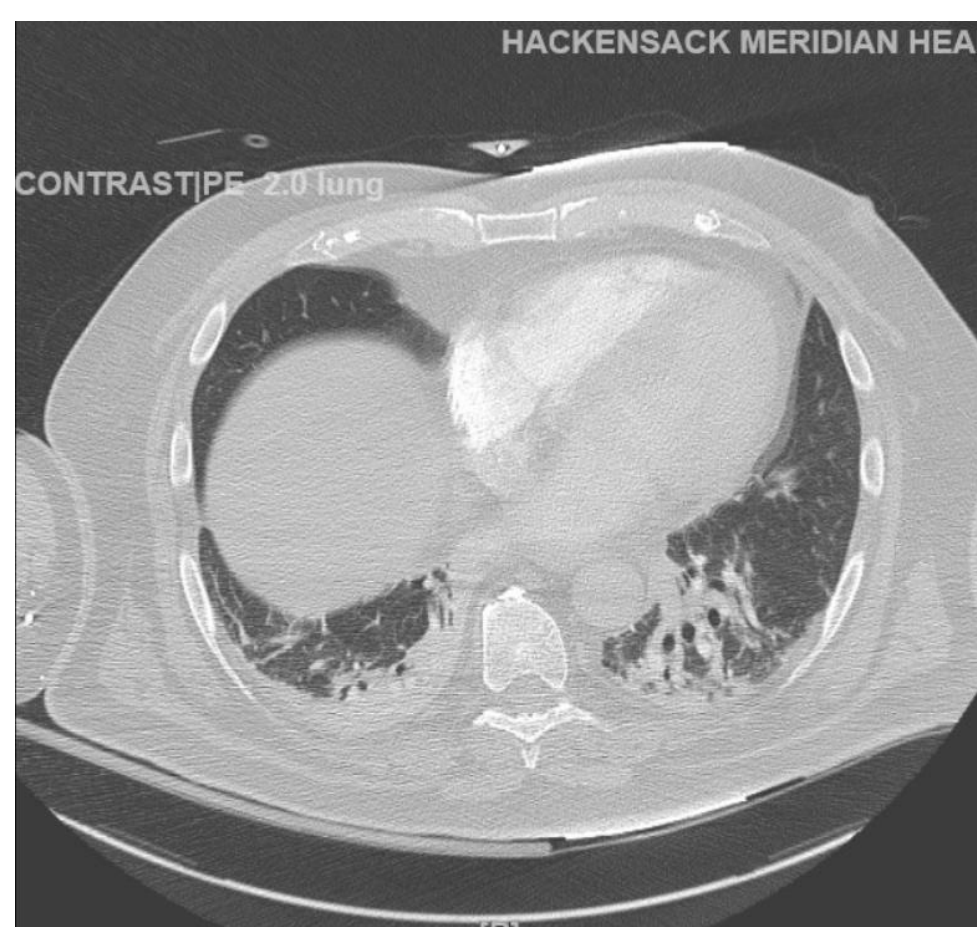
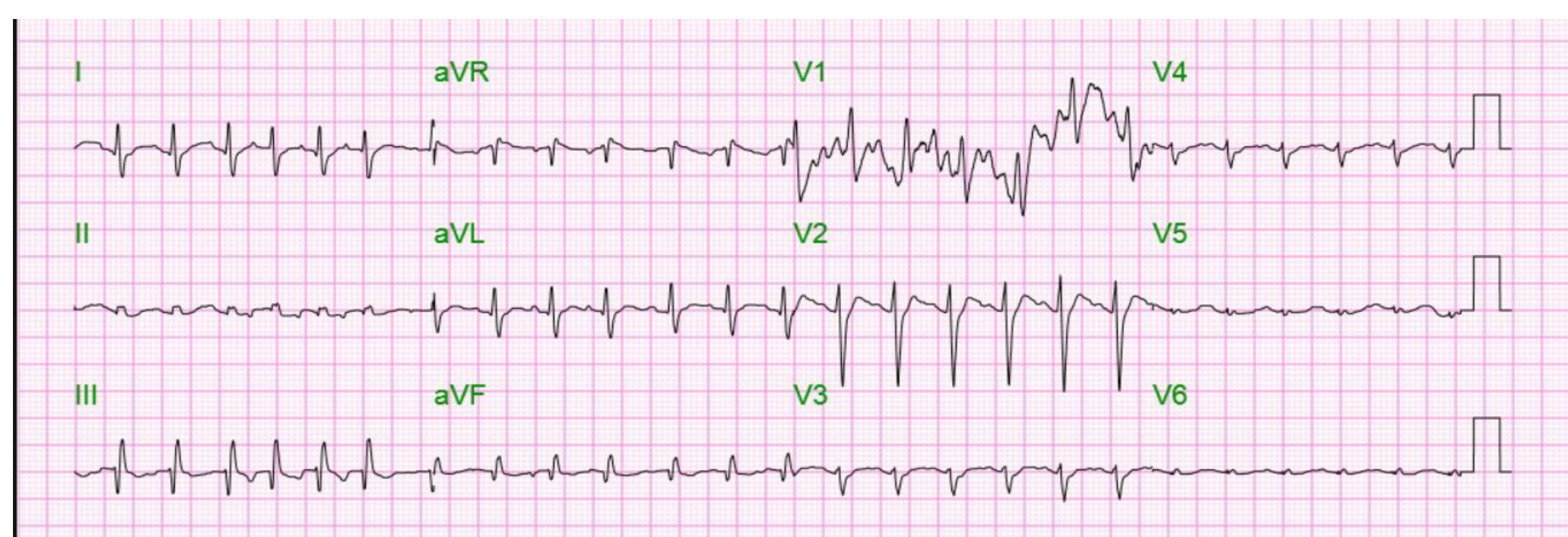


Soroush Seifirad (presenting author), Zaza Cohen, David Blady, Bijal Mehta and Ali Elsayed  
Department of Internal Medicine, Hackensack Meridian Health Mountainside Medical Center

## INTRODUCTION

Non ischemic recurrent ventricular tachycardia (VT) can be a rare presentation of Myasthenia Gravis.



ECG (baseline: Only subtle abnl id inf leads  
CT: Atelectasis vs pneumonia  
Cardiac MRI : Delayed gadolinium enhancement, non ischemic pattern apical and inferolateral wall.

## CASE PRESENTATION

A 72-year-old male with past medical history of diabetes mellitus Type 2 and granulomatosis with polyangiitis presented after a **VT arrest**; patient was shocked and intubated in the field. ECG revealed **normal sinus rhythm** with no evidence of heart block or ischemia. Echo revealed an ejection fraction of 55-60% with **no significant regional wall motion abnormalities**. The patient had another episode of VT in the Intensive Care Unit (ICU) which was medically managed with amiodarone. Subsequent coronary catheterization showed **normal coronary arteries** without occlusion. Computed tomography (CT) Angiogram of the Chest was negative for proximal pulmonary emboli, revealing only atelectasis in both lower lobes and right upper lobe. The patient had an Implantable Cardioverter-Defibrillator (ICD) placed as definitive treatment for refractory VT. Further history from the patient's wife revealed weeks of fatigue and generalized weakness, 2 weeks of poor oral intake, difficulty swallowing fluids and solids with spells of coughing/choking, 10 lb weight loss and one episode of fall due to weakness. She did not report any shortness of breath, recent fever, loss of consciousness, diarrhea, droopy eyelids, diplopia or seizures. Given the findings of nonspecific weakness, dysphagia, atelectasis on CT scan, and normal coronary angiography and normal electrolytes, myasthenia gravis was considered as a unifying diagnosis. The patient was found to have **very high Acetylcholine receptor (ACHR) binding protein and high Acetylcholine blocker and Acetylcholine modulator antibodies**. The patient was referred to a neurologist, treatment was started for myasthenia gravis, and amiodarone was discontinued. Further follow up with the family revealed **that the treatment stopped VT attacks, and the patient had a significant functional improvement**. Further work ups revealed **Anti Kv 1.4 antibody** a very specific antibody for cardiac involvement in myasthenia gravis.

## CONCLUSION

This case demonstrates the importance of a thorough and complete history, as that is what ultimately elucidated the cause of refractory VT, a very rare cardiac manifestation of myasthenia gravis.

### References:

1. Mavrogeni S, et al. Silent myocarditis in myasthenia gravis: Role of cardiovascular magnetic resonance imaging. *Int J Cardiol* 2016; 202: 629–630.
2. Sato K . Et al. Myocarditis in Myasthenia Gravis - Multiparametric Evaluation Using Cardiac Magnetic Resonance Imaging. *Circ. J.* 2022 Oct 25;86(11):1788. doi: 10.1253/circj.CJ-22-0346
3. Calin C et al. Cardiac involvement in myasthenia gravis—is there a specific pattern? *Rom. J. Int. Med.* 2009;47(2):179-89.
4. Sakamoto et al. A case of myasthenia gravis with cardiac fibrosis and easily provoked sustained ventricular tachycardia
5. Mygland, A. et al. Heart muscle antibodies in myasthenia gravis. *Autoimmunity*, 10 (1991), pp. 263-267
6. Mygland, A. et al. Anti-cardiac ryanodine receptor antibodies in thymoma-associated myasthenia gravis. *Autoimmunity*, 17 (1994), pp. 327-331.