Rate Dependent ST Elevations in a Chronic Left Ventricular Aneurysm

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Introduction

Myocardial infarction (MI) is one the leading causes of death in developed countries.[1] The initial ECG is imperative as it can quickly diagnose a ST segment elevation myocardial infarction (STEMI), an MI that requires emergent revascularization in the catheterization laboratory. One of the important mechanical complications of a STEMI is a left ventricular aneurysm. This usually occurs in the anterior wall of the left ventricle after a left anterior descending artery occlusion. Occasionally, patients with a left ventricular aneurysm will have persistently elevated ST segments. However, the incidence of this complication has significantly decreased due to the use of percutaneous coronary intervention.[2] Despite this, it is still important complication to recognize.

Patient Presentation

63-year-old female with history of traumatic left anterior descending artery occlusion resulting in a left ventricular aneurysm (Figure 3 and 4), atrial flutter status post ablation and hypothyroidism presented for palpitations and a feeling that her heart was "not in sync." She was leisurely walking when her symptoms started. Her symptoms had remained constant for roughly one hour. She took a dose of her atenolol with no improvement in her symptoms. She denied any chest pain, shortness of breath, or any exertional symptoms. She did admit to drinking 3.5 cups of coffee that day, which was more than she usually drank.

Her initially ECG demonstrated the following changes (Figure 1).

She was found to be in a course atrial fibrillation with ST elevations in leads V3-V6. She then converted back to sinus rhythm on her own with improvement of the ST elevations (Figure 2). She was subsequently discharged with follow up with cardiology.

Figures

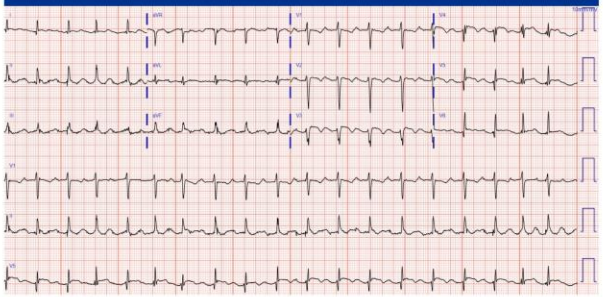

Figure 1: Initial presenting ECG

Figures

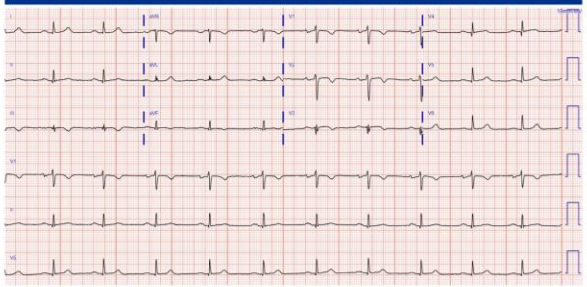

Figure 2: Follow up ECG after conversion to normal sinus rhythm.

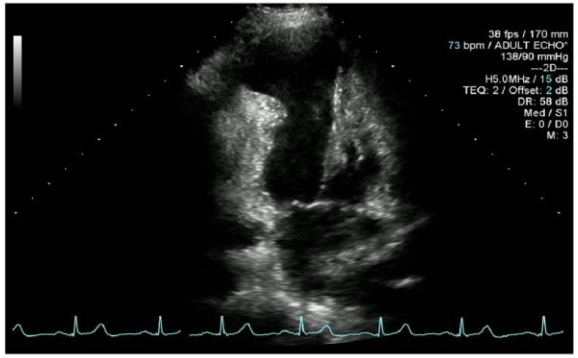

Figure 3: 2-chamber view

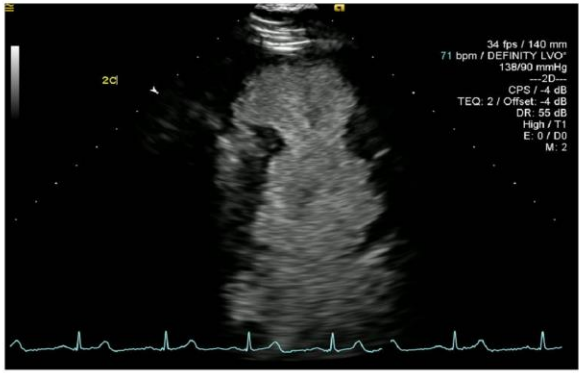

Figure 4: 2-chamber view with contrast

Discussion

Twenty years ago, the patient developed a traumatic left anterior descending artery occlusion. She was involved in a skiing accident where she was thrown off the side of a cliff. During that presentation, she developed worsening chest pressure, but it was initially thought to be musculoskeletal in nature from the fall. She continued to have chest pressure for several more days until she went into the emergency room where she was found to have an occlusion of her left anterior descending artery with apical aneurysm. Her subsequent ECGs over the years demonstrated intermittent ST elevations, but none as severe as shown in her current presentation (Figure 1).

Background

A left ventricular aneurysm is usually caused by total occlusion of a coronary artery with no collateralization, leading to a transmural myocardial infarction. Once this area has healed, the results is a thin, well delineated, fibrous scar that is usually devoid of muscle. The diagnosis of a LV aneurysm is primarily based off two dimensional echocardiography. Typically, aneurysm dilation with thinning of the affected wall is seen. The aneurysm has a broad neck and can either be akinetic or can be dyskinetic, ballooning out during systole.[2] (Figures 3 and 4)

ECG manifestations

Left ventricular aneurysms can demonstrate intermittent or persistent ST elevations, most commonly in the precordial leads. This is usually accompanied by well-developed Q waves as well as no reciprocal ST depressions. The T waves are typically small compared to the amplitude of the QRS complex.[2]

Mechanism for ST elevations

The exact pathophysiological mechanism for ST elevations is not known. ST elevations tend to correlate with dyskinesia of the scarred myocardium ballooning out during systole. Because of this established relationship, it is thought that the stretch of the myocardium causes the ST elevations.[3] This hypothesis could be applicable to the presented patient in that a more hyperdynamic ventricle secondary to tachycardia would result in greater stretch and dyskinesia of the apical aneurysm.

Conclusion

The most feared presentation of ST elevations occurs with a ST elevation myocardial infarction, but it can be present in other conditions, such as left ventricular aneurysm. The ST elevations in left ventricular aneurysms can be intermittent, persistent, or in the unique case of our patient, rate dependent.

References

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