

Short Communication

Dealing with the Widow Maker Coronary Lesion in the 2020s

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INTRODUCTION

Blockage of the left anterior descending coronary artery is sometimes referred to as a “widow maker.” The artery supplies blood to up to half or more of the left ventricle. It can lead to sudden death or a massive myocardial infarction when occluded.

The best approach is prevention, aggressively looking at one’s coronary risk factors, doing a coronary calcium computed tomography scan, and alerting patients on prompt action if they develop any chest discomfort.

The following two recent cases we saw are illustrative of these points.

Case 1

A 68-year-old former college football player has been followed for a family history of cardiovascular disease and hypercholesterolemia.

A coronary calcium scan three years prior showed three areas of calcification in the left anterior descending artery, with a coronary calcium score of 47.

A stress test seven months before his current visit showed no ischemic ST-segment changes to 96% of age-predicted maximum heart rate. Premature atrial and ventricular beats were noted.

His blood lipids five months earlier, on 40 mg of atorvastatin, showed total cholesterol of 138 mg/dl, triglycerides 58 mg/dl, HDL of 55 mg/dl and an LDL cholesterol of 78 mg/dl.

One week before his present visit, he recalled a 30-second episode of chest discomfort while jogging. It did not reoccur with subsequent jogs. The evening before his recent visit, he experienced chest pain throughout the night.

His physical exam showed a blood pressure of 140/82 mmHg, and his pulse was regular at 63 beats per minute. The cardiopulmonary exam was normal.

An ECG showed an acute anterior wall ST-segment elevation (STEMI). He was transported to the cardiac catheterization laboratory, where he was noted to have total occlusion of the proximal to the mid-left anterior descending artery. An IVUS-

guided angioplasty and thrombectomy were performed with stent placement. He tolerated the procedure well and was discharged symptom-free the next day.

An echocardiogram revealed a normal ejection fraction of 61%, but hypokinesia of the mid-anterior, apical anterior, and apical inferior left ventricular wall.

Akinesia was noted in the mid-anteroseptal, mid-inferoseptal, and apical septal areas.

Case 2

A 48-year-old, high altitude mountain climber and adventure traveller noted the onset of exertional mid-anterior chest pain one week prior, relieved within several minutes of rest.

His father had experienced a heart attack at age 40 and had a history of hypercholesterolemia. Our patient had never bothered to have his blood lipids checked, assuming that he was well.

On physical exam, his blood pressure was 122/90 mmHg, and his pulse was 68 per minute and regular. His weight was 216 lbs. with a body mass index in the overweight category at 29.2 kg/m². His cardiopulmonary examination was normal.

His total cholesterol was elevated at 265 mg/dl, the LDL at 179 mg/dl, and the triglycerides at 157 mg/dl. His HDL cholesterol was normal at 54 mg/dl.

His resting ECG was normal. On the Bruce treadmill test, he had a duration of 5.31 minutes, and the test was stopped due to chest pain and ischemic ECG changes. The ST-segment was elevated in lead V1, suggesting a left anterior descending coronary lesion (Figure 1).

At cardiac catheterization, his mid-left anterior descending coronary artery was 95% stenosed. An intravascular ultrasound-guided stent was placed in the proximal and mid-left anterior descending artery.

He was discharged later the same day and has remained free of chest pain. His medications included dual antiplatelet therapy for at least six months, along with atorvastatin 40 mg to get his LDL cholesterol in the 40-70 mg/dl range.

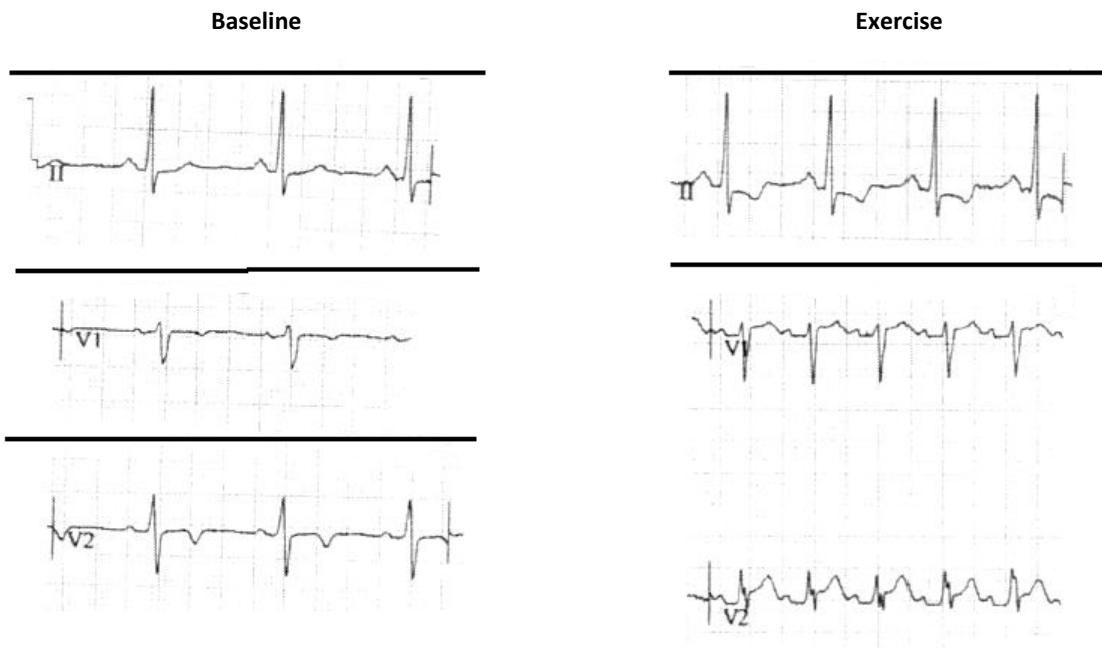


Figure 1. Exercise ECG in case #2 showing ischemic changes in the inferior leads and transient ST elevation in the anterior chest leads.

DISCUSSION

Blockage of the proximal or mid-left anterior descending coronary artery is a high risk for catastrophic results, given the large area of blood supply involved to the left ventricle. The first sign/symptom can be sudden cardiac death.

These two patients each made a major mistake. The first spent a whole night with severe chest pain without notifying anyone. Typically, we advise such patients to call 911 and be transported to the emergency room for pain lasting over 10-15 minutes, especially if not relieved by nitroglycerin-SL.

Case #2 is an example of the Jim Fixx [1] or ostrich approach, wherein one ignores checking blood lipids or doing any exercise stress testing, despite a strong family history of early cardiovascular disease. His coronary lesion was likely due to years of a very high LDL cholesterol level, along with possible genetic contributions.

An early prospective randomized trial compared stenting to internal mammary artery grafting (the SIMA trial) with fairly similar results [2]. The improved restenosis rate with more updated stenting has made this the way most patients prefer to go. We agree with Dr. Holmes that a shared discussion with the patient should be implemented [3]. Cardiac surgeon, Robert Guyton, makes a case for a possible longer survival benefit using the left anterior mammary artery (LIMA) graft to the LAD (over 90% 20-year patency) [4]. Case #2 chose to have the stent and aggressive lipid management and was discharged pain-free within a day.

CONCLUSION

Two athletic men were seen in the same week for so-called “widow maker” left anterior descending coronary artery lesions. Fortunately, both survived, despite each making a serious error in their management (one allowing hours to pass before seeking medical attention for severe chest pain, the other assuming he was well in the absence of symptoms, despite a family history of early coronary artery disease).

It behoves us, physicians, to make sure every patient is educated on how to proceed with new-onset chest pain, look for coronary risk factors, and consider coronary artery calcium screening in patients certainly by age 40.

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