The echocardiogram from my prior palpitations).

The cardiac catheterization report suggested no blockages and no dissections. The cardiac MRI suggested an area of myocardial damage where the myocardium is fed by the left circumflex artery. Of interest was that the damage was from the outside in, not a typical pattern with ischemia. A repeat MRI 6 weeks later again found this defect more prominently, suggesting permanent myocardial damage, not just stunning.

The discharge diagnosis from the treating physician was non-ST elevation myocardial infarction secondary to coronary vasospasm. I consulted a cardiologist for a second opinion, who diagnosed small vessel disease in the area of the left circumflex artery and likely ischemic disease given a poor lipid profile.

My treatment regimen was 325 mg aspirin once a day for the first 6 weeks, then 81 mg aspirin with 240 mg sustained-release diltiazem and 40 mg simvastatin once a day. I received a prescription for sublingual nitroglycerin as needed and typical post-MI discharge instructions, including exercise and weight loss.

Discussion

Why was MI excluded from the initial differential by me, my colleague and the ED team? Second, what is the correct diagnosis? Third, what is the appropriate management for this case?

Heart disease is the leading cause of death in women today.1 Studies show that women — especially young women — are often less likely to perceive their cardiac symptoms as seriously as men do.2 Women often present with atypical symptoms, and diagnostic studies are less likely to provide a correct diagnosis.3 As a result, the diagnosis of MI is often missed, and women are less likely to receive evidence-based care. In my case, the pain started in my back and over my top teeth. The chest pain became more classic in nature and even radiated up the neck, but remained over my top teeth. I was not placed on a critical pathway for the first 3 hours in the ER.

My final diagnosis remains acute myocardial infarction. Further understanding of the pathology that led to this event is unlikely. Ischemia, coronary vasospasm or some type of rare focal myocarditis remain the top differentials. My abnormal lipid panel and possible small vessel disease make ischemia likely. However, given my postpartum hormonal state, coronary vasospasm is highly suspect. Additionally, my only other cardiac symptoms were during my first pregnancy. Furthermore, studies have linked migraine sufferers with coronary vasospasm.4 Finally, while the MRI images were strongly suggestive of a focal myocarditis, my history does not support this diagnosis with an acute onset and offset of pain. Unless I experience a second event, it is unlikely that this puzzle will be solved.

Without a thorough understanding of the underlying pathology in my case, secondary prevention is challenging. Whether to treat with a beta blocker or calcium channel blocker or to add clopidogrel depends on whether you believe the disease is ischemic or vasospastic. Deciding on a lipid goal depends on whether you believe this could have been a form of myocarditis. Even determining whether this event represents a risk for my children is difficult.

It has been said that the health care provider who treats himself or herself is a fool, and that it is bad business to treat friends and family. When you are too close to the situation, you cannot be objective. In this case, it may have cost cardiac muscle.

References


