DEVICE THERAPY

CLINICAL DECISION-MAKING

Severe Coronary Artery Spasm and Sudden Cardiac Death: Is there a Consensus for Management?

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ABSTRACT. Severe coronary artery spasm causing ventricular fibrillation (VF), with either normal or diseased coronary vasculature, has been described. A 54-year-old male with known coronary artery disease developed an episode of retrosternal chest pain at rest and subsequently lost consciousness during emergency medical technician (EMT) evaluation and was found to be in VF. He was defibrillated four times during the advanced cardiovascular life support (ACLS) protocol before sustained restoration of normal sinus rhythm. Coronary angiography revealed severe diffuse coronary artery spasm and was reversed with intracoronary nitroglycerine. The patient was started on long-acting nitrates and calcium channel blockers and had an implantable cardioverter-defibrillator (ICD) placed because of the unknown risk of recurrence. He was discharged the following day and has been asymptomatic on follow up visits.

KEYWORDS. ventricular fibrillation, ventricular tachycardia, coronary artery spasm, automatic implantable cardioverter defibrillator (AICD).

Introduction

Coronary artery spasm has been known to induce myocardial ischemia and potentially fatal ventricular arrhythmias, which can lead to sudden cardiac death.1–3 The recommended treatment for patients with evidence of coronary artery spasm is the combination of long-acting nitrates and calcium channel blockers; however, there are no data guiding the management of patients with coronary artery disease leading to sudden cardiac death. This is likely due to the relatively small number of patients with isolated coronary artery spasm.4 We describe a patient with severe diffuse coronary artery spasm leading to sudden cardiac death.

Case presentation

A 54-year-old male with known coronary artery disease had undergone percutaneous coronary intervention and drug eluting stent deployment to his left anterior descending (LAD) and right coronary arteries 4 years prior to presentation. A comprehensive cardiac workup had been performed 2 months earlier at another institution for non-exertional chest discomfort and occasional palpitations, including an electrophysiology study, stress myocardial perfusion imaging, and a coronary angiogram, all of which were unremarkable. The patient activated EMS after an episode of retrosternal chest pain at rest. During EMT evaluation, the patient lost consciousness and was found to be in ventricular fibrillation (VF). The ACLS protocol was initiated and he was defibrillated four times before successful restoration of normal sinus rhythm. No ischemic electrocardiogram (ECG) changes were evident, and there was no laboratory evidence of myocardial infarction. Urine toxicology screening was negative for cocaine. The VF was presumed to be

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secondary to myocardial ischemia associated with the patient’s coronary disease. Angiography demonstrated patent stents, but severe diffuse coronary artery spasm (Figure 1), which was relieved with intracoronary delivery of nitroglycerine (Figure 2). Left ventricular systolic function was normal. The patient was started on long-acting nitrates and calcium channel blockers and had an implantable cardioverter-defibrillator (ICD) placed. He was discharged the following day and has been asymptomatic on follow up visits.

Discussion

Although the pathogenesis of coronary artery spasm has not been fully elucidated, endothelial dysfunction and enhanced vascular smooth muscle contractility are considered to be the major underlying mechanism.5,6 The etiology for development of VF with coronary artery spasm has been related to profound myocardial ischemia7 or can be as a result of a reperfusion arrhythmia upon spontaneous relief of the coronary spasm.8

Figure 1: Severe diffuse coronary artery spasm of mid-distal left anterior descending coronary artery distal to patent prior stent site.
There are no data guiding management of patients with coronary artery spasm leading to sudden cardiac death; however, medical therapy with calcium channel blockers, long-acting nitrates, 3-hydroxy-3-methylglutaryl-coenzyme A (HMG CoA) reductase inhibitors (statins), smoking cessation, and avoidance of β-blocker drug therapy have been recommended. There are few controlled trials assessing the efficacy of these therapies, in isolation or in combination, and data are scarce on the optimal therapy as well as the duration of therapy for patients with coronary artery disease leading to sudden cardiac death.

A study reported by Lee et al. showed that despite combination therapy at maximal dose, provocative testing remained positive in 12.8% of patients, representing the subtherapeutic effect of medical management. Al-Sayegh et al. reported that despite medical treatment, 5–30% of patients continue to have angina attacks and even myocardial infarction and rarely sudden cardiac death. Therapy with multiple vasodilators, including two or more calcium channel blockers, along with ICD implantation has been proposed for such refractory coronary artery disease leading to lethal arrhythmias.

In a retrospective observational trial, Matsue et al. examined apparently consecutive patients from three Japanese hospitals who had received a secondary prevention ICD after resuscitation from an episode of

Figure 2: Coronary artery spasm relieved following intracoronary delivery of nitroglycerine.
life-threatening VT/VF in the absence of structural heart disease or coronary artery disease who had spasm of a major epicardial coronary artery induced with acetylcholine challenge. They determined that ICD therapy is effective for the termination of VF and for secondary prevention of sudden cardiac death in patients with coronary artery spasm who have survived an episode of life-threatening VT/VF, however, it is not yet clear that the risk of recurrent VT/VF is enduring in this patient population.

Conclusion

There are currently no data guiding management of patients with coronary artery spasm leading to sudden cardiac death. ICD placement is recommended for resuscitated VT arrest in this patient group as the risk of recurrent ventricular arrhythmias on medical therapy is not predictable; however, this remains controversial. The need remains for further research on the pathophysiology, management, and prevention of intractable coronary artery disease spasm.

References