Chapter 4

Left Ventricular Ischemia Due to Coronary Stenosis as an Unexpected Treatable Cause of Paroxysmal Atrial Fibrillation

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ABSTRACT

We present a patient with exercise-induced paroxysmal atrial fibrillation, who was eventually scheduled for a Cox-maze operation due to persistence of his complaints of fatigue, impaired exercise tolerance and predominantly exercise related irregular palpitations despite treatment with several anti-arrhythmic drugs. A pre-operative exercise stress test without anti-arrhythmic or negative chronotropic drugs, however, showed clear evidence of myocardial ischemia. After coronary angioplasty of a significant stenosis in the left anterior descending artery, there was no recurrence of atrial fibrillation during a follow up of seven months.
INTRODUCTION

Paroxysmal atrial fibrillation (PAF) is known to have many cardiac and non cardiac causes but also occurs in the absence of an underlying disease. Determining and subsequently treating the cause may eliminate or reduce PAF. Thus, this is the prime goal in the treatment of PAF. Unfortunately, treatment of an underlying disease very frequently does not terminate PAF. In these patients and in patients in which PAF is idiopathic (“lone”), therapy should be aimed at maintaining sinus rhythm in order to keep a normal ventricular rate, preserve the “atrial kick”, prevent thrombo-embolic complications, inhibit subjective symptoms and prevent PAF to become chronic. Several antiarrhythmic drugs are used for this purpose. However, pharmacological treatment in patients with AF sometimes is insufficiently effective. Nonpharmacologic therapies comprise ablation of the AV node in combination with a pacemaker, antiarrhythmic surgery, and an implantable atrial defibrillator. These options, however, should be considered as a last resort. In this report we present a patient presumed to have lone PAF who appeared to have PAF due to a rather uncommon but easily treatable cause.

CASE REPORT

A 44-year-old man presented to our hospital with a history of PAF. The paroxysms started four years ago in 1994, with no clear precedent. They occurred invariably during exercise but also occasionally were present in rest and during sleep. During such episodes, which lasted from minutes to up to 5 hours, the patient noticed palpitations, dizziness, shortness of breath and chest discomfort. He never experienced syncope. He had no previous heart disease nor did he suffer from hypertension, diabetes or hyperthyroidism. There was no family history of cardiac disease. He smoked 15 cigarettes daily and was not an excessive drinker. Figure 1 demonstrates an ECG recording with short bursts of atrial fibrillation during an exercise stress test. The ECG pretest showed normal sinus rhythm without arrhythmia or ST segment abnormalities. Initially he had been treated with sotalol 40 mg b.i.d. and later 80 mg b.i.d. Although duration and frequency of the paroxysms were reduced, the patient suffered from severe fatigue as a side effect. Consequently, in 1995, sotalol was stopped and the patient was subsequently treated with propafenone and later with flecainide. Effects of both treatments, unfortunately, were unsatisfactory. Complaints of paroxysmal irregular rapid palpitations remained present. Treatment with a selective beta-receptor blocking drug, bisoprolol, was not well tolerated, although it did ameliorate the symptoms. Eventually, treatment with sotalol was resumed at 40-mg dose b.i.d. shortly before he visited our outpatient center in January 1997.
Figure 1. Six-lead ECG recording of short bursts of atrial fibrillation during an exercise stress test. Note the fast irregular ventricular response during the short episodes of atrial fibrillation. Paper speed = 25 mm/s. During this exercise stress test the patient was receiving sotalol 40 mg b.i.d.
At that time the paroxysms were less frequent but still occurred 3 to 4 times a month. Furthermore, the patient suffered from serious sotalol-related side effects such as extreme fatigue, drowsiness, orthostatic hypotension, and impotence. The combination of paroxysm-associated complaints and these side effects impeded him to continue his job as a truck driver. A physical examination revealed no abnormalities. An echocardiography demonstrated good left ventricular function with end-diastolic and end-systolic dimensions of 55 and 35 mm, respectively, and mild left atrial dilatation (44 mm long axis view). There were no signs of mitral regurgitation or other valve disease. The result of blood tests showed no abnormalities, particularly no signs of hyperthyroidism. A standard 12-lead ECG was normal. An ambulatory 24-hour ECG demonstrated, besides bradycardia due to the sotalol, no arrhythmias. A repeated exercise stress test during sotalol 40 mg b.i.d. did not reveal any arrhythmias or ST-segment abnormalities.

Because of the persistence of symptoms in combination with unsatisfactory results and severe side effects of the pharmacological treatment and the inability to practice his profession, the patient decided that he wanted to undergo the proposed Cox-maze operation. In this procedure, which is the most widely used surgical method to treat atrial fibrillation, incisions are applied on both atria. In this way, conduction blocks are created in the atrial wall that do not allow reentrant wavelets to induce atrial fibrillation.

The patient was admitted for the maze procedure in May 1997. Sotalol was discontinued before admission. As part of the preoperative protocol, an exercise stress test and coronary angiography were performed. At that time, sotalol had been discontinued for 5 days. The standard 12-lead ECG showed no abnormalities prior to the exercise stress test (Figure 2). Remarkably, whereas no arrhythmias occurred, the 12-lead ECG during the exercise stress test demonstrated ST-segment depression in leads V3 through V5, indicating the presence of myocardial ischemia (Figure 3). The patient experienced neither chest pain nor palpitations at that moment. The coronary angiogram demonstrated a 70-90% stenosis in the proximal part of the left anterior descending artery (LAD). Angiography of the right coronary artery (RCA) and the circumflex artery (CX) revealed no abnormalities. A decision was made to postpone the maze procedure in order to first treat the patient’s coronary artery disease. Percutaneous transluminal coronary angioplasty (PTCA) with placement of two stents in the proximal LAD was performed. The patient was discharged 4 days later without any antiarrhythmic medication. Thereafter, the patient never again experienced a paroxysm of atrial fibrillation, whereas until shortly before the angioplasty he experienced episodes of palpitations 3 to 4 times a month. An exercise stress test four months after the PTCA procedure showed neither arrhythmias nor signs of myocardial ischemia. A repeated echocardiographic examination 7 months after the procedure demonstrated a reduction in left atrial size from 44 mm to 36 mm (long-axis view). Currently he has been asymptomatic and off antiarrhythmic drugs for 7 months.
Figure 2. Standard 12-lead ECG recording during exercise stress test. ECG at baseline. No abnormalities are present, particularly no STT segment abnormalities.
Figure 3. Standard 12-lead ECG recording during exercise stress test. ECG at a workload of 170 Watts. Sinus tachycardia with ST-segment depressions in leads V3 through V5, indicating the presence of myocardial ischemia. Paper speed = 25 mm/s. During this exercise stress test the patient was receiving no antiarrhythmic drugs.
DISCUSSION

The above mentioned report indicates an unusual cause of PAF. PAF was ischemia related in this patient. After PTCA of a significant LAD stenosis, the patient no longer suffered from PAF during 7 months of follow-up, while previously he had suffered from paroxysms at least once a week. An LAD occlusion will not cause atrial ischemia, since the atria are vascularized by branches descending from the RCA and CX. Additionally, the left atrial myocardium is directly oxygenated from oxygen-rich blood inside the left atrium. The probable cause of exercise-induced PAF in this patient was left ventricular ischemia. Ischemia, caused by an increase of oxygen demand during exercise, may have resulted in an acute impairment of left ventricular function. This in turn may have caused a rise in left ventricular end-diastolic pressure and, as a consequence, an increase in left atrial pressure. Exercise may thus have created stretching of the left atrial wall. The finding that this patient had an increased left atrial diameter at echocardiography supports this hypothesis, although the repeated induction of atrial fibrillation may have caused a tachycardia-induced atrial cardiomyopathy which may have contributed to the dilatation of the atria. The reduction of left atrial size after 7 months of follow-up supports both the elimination of atrial fibrillation and the restoration of normal left ventricular function. The presence of stretching of the left atrial wall caused by an LAD stenosis is supported by a study of Sigwart et al., in which left atrial function in 32 patients during acute ischemia caused by PTCA of a proximal LAD stenosis was studied. They demonstrated that mean left atrial pressure increased from 11 ± 2 to 29 ± 2 mmHg after only 30 seconds of balloon occlusion. It is well known that myocardial mechanical changes (e.g., stretch) modulate electrophysiologic properties. This process is called mechanoelectric feedback or contraction-excitation coupling. Numerous animal studies have shown that acute atrial stretching alters the atrial effective refractory period (AERP). Although, in two recent studies, there is disagreement about whether the AERP prolongs or shortens, both studies observed an increased inducibility of atrial fibrillation after acute atrial stretching. Additionally, as PAF predominantly occurred during exercise in the present patient, tachycardia induced shortening of AERP may have further facilitated the genesis of PAF. Moreover, the irregular and fast ventricular rhythm itself may have further impaired cardiac output.

Another indication that ventricular ischemia was the probable cause of PAF is the fact that bisoprolol and sotalol, although having serious side effects, were most efficacious in reducing the frequency and duration of the paroxysms compared to flecainide and propafenone in this patient. During exercise, these drugs, both with beta-receptor blocking properties, inhibit the heart rate and thus the oxygen demand of the ventricular myocardium, preventing ischemia and resulting in preservation of left ventricular function. On the other hand, beta-receptor blocking drugs may have masked the cause
of PAF in the present case. When the patient was treated with sotalol, the exercise stress test demonstrated no signs of ischemia. However, only 3 months later clear signs of ischemia were present during an exercise stress test without drugs.

There are numerous patients with an isolated LAD stenosis who do not experience atrial fibrillation. Although one could expect that during exercise a similar rise in left atrial pressure occurs, PAF is relatively unusual in these patients. We hypothesize that, in contrast to most patients with coronary artery disease, the present patient, in addition to a coronary stenosis, had an "arrhythmogenic area" in the left atrium that may have been triggered by stretch in combination with sympathetic stimulation. Recently, the existence of "focal atrial fibrillation" has gained interest. In this arrhythmia, a surface ECG pattern of atrial fibrillation or irregular atrial tachycardia is present due to a focal rapidly firing source of electrical activation. The focus often is situated in the pulmonary vein region. Evidence has now emerged that this region also is an important source of ectopic beats which can initiate atrial fibrillation. It is very well possible that our patient suffered from focal atrial fibrillation since he demonstrated the short bursts of rapid atrial activity that are common in this type of atrial fibrillation (Figure 1). However, we neither performed an electrophysiologic study to confirm this diagnosis nor considered radio frequency ablation of the arrhythmogenic focus because at that time little was known about this variant of atrial fibrillation.

In conclusion, we describe an unusual cause of PAF. Not atrial ischemia, but ventricular ischemia inducing an impaired left ventricular function with an increased left ventricular end-diastolic pressure and increased left atrial pressure may have triggered paroxysms of atrial fibrillation in the present patient. Exercise-induced atrial fibrillation occurs only rarely. It may occur in the setting of adrenergic atrial fibrillation in the presence of an underlying heart disease. In patients with adrenergic atrial fibrillation, sympathetic stimulation per se may provoke a paroxysm. However, our patient experienced no recurrence of PAF after PTCA. This indicates that stimulation of the sympathetic system, although it may have contributed, was not solely responsible for the occurrence of PAF in the present case.

References


