To the Editor:

Although the incidence of ventricular fibrillation (VF) in non-ST segment elevation acute coronary syndrome (NSTE ACS) is low,\(^1\) VF after angioplasty in this context is extremely rare. We present the case of a 65 year old male, hypertensive and with dyslipidemia, admitted to the coronary service due to new onset angina, with a resting episode which ceased with sublingual nitroglycerine. The physical and laboratory tests displayed no significant findings, quantification of the MB fraction of the creatine kinase (CK-MB) was normal. The electrocardiogram highlighted minimal ST scooping in lead II and the echocardiogram disclosed a left ventricle with mild concentric hypertrophy and a preserved systolic function. He was treated with atenolol, aspirin, clopidogrel, atorvastatin, and heparin and nitroglycerine infusion. The patient had a repeat episode with ST scooping -0.5 mm in inferior leads. A coronary catheterisation was performed, which displayed sub-occlusive stenosis in the right mid coronary artery and severe distal vasospasm, dominant vessel with TIMI flow grade 2 (Figure 1A), with no significant lesions in the rest of the coronary tree. After administering intracoronary nitroglycerine, the vasospasm disappeared and the TIMI grade 3 flow was re-established, observing sub-occlusive residual stenosis in the mid-third with radiotranslucent appearance (Figure 1B). Next, treatment with tirofiban was commenced and a coronary angioplasty with a stent Flexmaster-F\(^1\) (Abbott\(^6\)) 4×26 mm implant performed, post-dilated with 4.5×20 mm balloon at 14 atm, with satisfactory angiographic result and no immediate complications (Figure 1C). The electrocardiogram after the angioplasty showed no significant alterations (Figure 2A). Two hours after the procedure with the patient completely asymptomatic, he presented an episode of VF (Figure 2B), immediately reverted with electrical cardioversion. Treatment with lidocaine was carried out for 24 h. The ECG after cardioversion displayed no ST segment elevation (Figure 2C). The laboratory test only displayed total CK elevation, with no significant CK-MB. Catheterisation was repeated 24 hours later and stent thrombosis excluded (Figure 1D) as well as alterations in other vessels. The patient evolved without complications, was discharged 5 days later and remained asymptomatic after a 10 month follow-up.
Among the possible trigger mechanisms, coronary vasospasm, with its subsequent reperfusion, could determine the appearance of malignant ventricular arrhythmias; also, the right coronary artery is associated with a greater risk of VF during primary angioplasty. In our case, it was a highly developed dominant right coronary sub-occlusive stenosis. It is possible that the sudden reperfusion of the vessel determined a great formation of free radicals and the ventricular arrhythmia was an expression of lesion due to reperfusion. Although the incidence of vasospasm after the stent implant is unclear, in a provocation test with acetylcholine severe vasospasm was induced in 19.6% and 8% of the patients with and without previous vasospasm, respectively. Likewise, Kim et al reported a case of VF caused by vasospasm a few hours after multiple drug eluting stent implants. In this case, the initial angiograph displayed spasm at a distance from the lesion; although the patient did not experience precordial chest pain before the occasion, neither did he display ST segment elevation after cardioversion; the possibility of silent vasospasm cannot be ruled out.

In conclusion, we reported an episode of VF without apparent trigger 2 hours after a successful angioplasty due to a NSTE ACS. This is probably an exceptional case, as predictors of this event have not been found in the literature.

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REFERENCES

To the Editor:

The case of an 80 year old male is presented. Type 2 diabetic, recently diagnosed with latent multiple myeloma and a history of severe right coronary lesion, revascularised through angioplasty and stent implant 2 years before. The echocardiography performed 2 years previously presented normal global and segmental contractility of the left ventricle (LV) and mild chronic pericardial effusion (maximum diameter, 8 mm). The patient attended the emergency unit with intense asthenia and maintained hypotension over the previous week.

In the physical exam, the patient presented a blood pressure of 90/40 mm Hg, heart rate 120 beats/ min, raised jugular venous pressure, lower limb oedema, and paradoxical pulse. The electrocardiogram displayed diffuse low voltage QRS and the chest x-ray, general cardiomegaly. With these findings, an emergency echocardiogram was performed (Figure), which confirmed the clinical suspicion of cardiac tamponade showing severe and diffuse pericardial effusion (maximum diameter, 53 mm), paradoxical movement in the interventricular septum, right atrial collapse and transmitral flow variation >25% with respiration. An emergency pericardiocentesis was performed, with immediate drainage of 1200 mL of serous-sanguineous fluid and rapid improvement of the clinical and haemodynamic parameters.

In the immediate echocardiographic monitoring, the normality of the diameters was checked and the LV systolic function (end diastolic diameter, 47 mm; LV ejection fraction of the LVEF >60%), with mild residual persistent pericardial effusion (maximum diameter, 11 mm). The cytologic, biochemical, microbiological, and immunological studies did not identify specific effusion aetiology.

Two days after the pericardiocentesis, the patient presented rapidly progressing dyspnea, with clinical symptomatology of pulmonary oedema and signs of low cardiac output. The emergency echocardiogram displayed severe global contractile dysfunction of the LV (LVEF, 13%). The ECG and myocardial enzymes obtained sequentially did not display data of acute ischemia.

The patient required treatment with intravenous furosemide and inotropic support with intravenous dobutamine and dopamine, which were then phased out. Contractility of the LV normalised on the sixth day (LVEF, 64%). A coronary catheterisation was performed which displayed a lesion in the posterolateral branch of the

Figure 2. A: electrocardiogram after angioplasty. B: ventricular fibrillation 2 hours later. C: electrocardiogram after cardioversion.