from AstraZeneca, Bristol Myers Squibb, Merck Sharp & Dohme, and Sanofi Synthelabo.

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Ischemia: Substrate or Trigger?  

Isquemia, ¿sustrato o desencadenante?

To the Editor,

Sustained monomorphic ventricular tachycardia (SMVT) in the setting of an anterior acute myocardial infarction (AMI) is rare. We present a case that illustrates the diagnostic, prognostic and therapeutic implications of this entity.

The patient was a 47-year-old male smoker, with type 2 diabetes without prior episodes of chest pain, who had experienced several syncope episodes at home. He had been treated by the emergency services, with documented SMVT at 140 bpm, with left bundle-branch block morphology and superior axis (Fig. 1A). Sinus rhythm was restored by electrical cardioversion. There was

Figure 1. A: 12-lead electrocardiogram during sustained monomorphic ventricular tachycardia at 140 bpm, with left bundle-branch block morphology, superior axis and fusion complexes. B: 1-lead telemetry tracing of sustained monomorphic ventricular tachycardia at 160 bpm. C: 12-lead electrocardiogram in sinus rhythm with a mild residual decrease in ST-segment elevation in inferior wall leads.

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The presence of SMVT in the acute phase of AMI is rare and is usually associated with very extensive necrosis in an anterior location and congestive heart failure. In the study by Mont et al., only 1.9% of patients with AMI showed SMVT in the first 48 h and the percentage is even lower (1.1%) if only an inferior location is taken into account. Interestingly, the factors that these authors identified as predisposing to SMVT (extensive necrosis, worse Killip class and bifascicular block) were not present in our patient. Given such an atypical clinical profile, it does not appear reasonable to expect similar results to those described regarding mortality (43%) and the recurrence of SMVT (17%), which may be closely related to ventricular dysfunction, nor does this profile appear to fit within the framework described in clinical practice guidelines. However, this atypical presentation should not modify the usual therapeutic strategy for urgent reperfusion and conventional medical treatment of AMI, nor does it entail indications for an implantable cardioverter defibrillator since arrhythmic events occur in the acute phase and LVEF is not very depressed.

The interest of this case lies in the patient’s clinical characteristics and the diagnostic approach for subsequent management, since this was guided by the pathophysiological mechanism underlying the episodes of SMVT.

If it is thought that there is a previous anatomical substrate—whether idiopathic, or caused by a silent infarction (which is possible in a diabetic patient)—on which the ischemia has acted as a trigger, correcting the ischemia will not eliminate the risk of recurrent SMVT. The absence of inducibility does not rule out the presence of a substrate; however, because this may progress to transmural infarction, it does not appear reasonable to consider ablation (of the substrate), at least in the acute phase of AMI.

However, if it is thought that the ventricular tachycardia episodes are secondary to a correctable cause, ischemia, which would have created a functional substrate different from the electrical instability that often causes far more severe polymorphic ventricular tachycardia or ventricular fibrillation (ischemia is not often a determining factor in the development of SMVT), then the principal treatment would be revascularization.

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