Letters to the Editor

Dobutamine-induced and spontaneous sustained ventricular tachycardia in recent myocardial infarction

Dobutamine stress echocardiography is increasingly used for detection of myocardial viability and ischaemia in patients with coronary artery disease\(^1\,^2\), the test may be frequently complicated by ventricular arrhythmias, but sustained ventricular tachycardia is rarely reported\(^3\).

We describe a patient with recent myocardial infarction who had dobutamine-induced and then spontaneous ventricular tachycardia, suggesting that the development of sustained ventricular tachycardia during the test may identify patients at high risk of subsequent major arrhythmic events.

A 39-year-old man was admitted with an acute anterior infarction and treated with streptokinase. The clinical course was complicated by mild left ventricular failure and by acute pericarditis. A 2-D echocardiogram showed a large akinetic area involving the interventricular septum, the anterior wall and the apex, with depressed left ventricular function and an ejection fraction of 33%.

On the 10th day after admission a dobutamine-atropine stress echocardiogram was carried out to evaluate myocardial viability and ischaemia in the infarct zone. No significant improvement of contractility in the akinetic area occurred after low-dose dobutamine; increase in ST-segment elevation in the anterior leads and apical dyskinesia with no anginal pain progressively developed. At the maximal dose of 40 \( \mu \text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1} \) followed by atropine 0.5 mg, sustained ventricular tachycardia at a rate of 200 beats \( \text{min}^{-1} \) with a left bundle branch block and inferior QRS axis deviation morphology developed (Fig. 1, top); the arrhythmia was well tolerated by the patient and was converted to sinus rhythm with intravenous ajmaline (20 mg) followed by chest thump. On the 15th day, repeated episodes of sustained ventricular tachycardia at a rate of 140 beats \( \text{min}^{-1} \) and with a different morphology (right bundle branch block with inferior QRS axis deviation) occurred (Fig. 1, bottom) and required treatment with intravenous ajmaline and oral amiodarone (800 mg \( \cdot \text{day}^{-1} \) followed by 400 mg \( \cdot \text{day}^{-1} \)). On the 30th day after the infarction during a bicycle ergometer exercise test, sustained ventricular tachycardia at a higher rate (240 beats \( \text{min}^{-1} \)) developed and was converted to sinus rhythm with chest thump. Beta-blocking treatment with metoprolol (100 mg \( \cdot \text{day}^{-1} \)) was added and no ventricular arrhythmias were documented on repeat exercise and during 6-month follow-up.

Dobutamine stress echocardiography is frequently associated with non-sustained ventricular arrhythmias\(^4\,^5\), but sustained ventricular tachycardias are reported in <0.5% of patients\(^6\,^9\). Our patient had a recent large anterior infarction and depressed left ventricular function; these findings are associated with a higher risk of developing ventricular tachycardia after myocardial infarction and are likely to increase the risk of dobutamine-induced arrhythmias. Dobutamine may elicit sustained ventricular arrhythmias either by impairing the balance between myocardial oxygen supply and demand in the infarct border zone or by eliciting catecholamine-dependent ventricular extrasystoles that may act as a trigger on the arrhythmogenic substrate of infarction; the latter mechanism seems to be the most important in this case, since no signs of ischaemia developed during the test. The fact that sustained ventricular tachycardia recurred during exercise and was prevented by beta-blocking treatment suggests that increased sympathetic tone played an important role in triggering ventricular arrhythmias. It is noteworthy that early after the dobutamine-induced arrhythmia, sustained ventricular tachycardia recurred both spontaneously and during exercise; thus, it may be hypothesized that in patients with myocardial infarction the occurrence of ventricular tachycardia during dobutamine stress test may be predictive of major arrhythmic events during the follow-up.

M. PREVITALI
L. LANZARINI
R. FETIVEAU
A. POLI
P. DIOTALLEVI
Division of Cardiology,
IRCCS-Policlinico S. Matteo,
Piazza Golgi, 1,
27100 Pavia, Italy
Internal mammary artery spasm immediately after grafting to the left anterior descending artery: diagnosis and treatment

Internal mammary artery (IMA) spasm may result in transmural anterior myocardial infarction[1]. We report a case of sudden inferior ischaemia 5 h after aorto-coronary bypass. Following emergency angiographic verification of a mammary artery spasm, an additional venous bypass of the distal left anterior descending (LAD) coronary artery was performed which relieved the ischaemia and rescued the myocardium at risk.

The left IMA has become the conduit of choice for bypass grafting of the LAD coronary artery. As perioperative IMA spasm may result in myocardial infarction[1], a number of techniques[2,3] are employed intraoperatively to increase the flow in the recently implanted IMA.

We report the case of a patient with inferior ischaemia due to early post-operative IMA spasm. A 63-year-old man had triple vessel disease; the ejection fraction of the left ventricle was 63% and a stress exercise test revealed significant ST segment depression in leads V4 to V6. At surgery, two saphenous vein grafts were used to bypass the right posterio-lateral and posterior descending and the left posterio-lateral and intermediate coronary arteries. Following mild hydrostatic dilatation with papaverine in accordance with the technique of Mills and Breau[4], the left IMA was implanted into the LAD. The post-bypass electrocardiogram (ECG) was normal and the cardiac index was 3.71 m².min⁻¹. Nifedipine (10 µg.min⁻¹) was infused throughout the procedure and continued in the intensive care unit. The mean arterial blood pressure was maintained between 70 and 90 mmHg. The patient did well until 5 h after surgery when he became haemodynamically unstable and low cardiac output was diagnosed. A 12-lead ECG revealed new ST segment elevations in leads II, III and aVF. An emergency cardiac catheterization was performed. Coronary arteriography showed patent grafts, although the IMA was in severe spasm (Fig. 1). While the nifedipine infusion was continued, nitroglycerin was administered directly into the proximal IMA, resulting in an immediate increase in the size of the IMA. This normalized the ECG in leads II, III and aVF (Fig. 1). Several minutes later, the IMA went into spasm again, leading to inferior ischaemia on the ECG. Despite two further intra-mammary nitroglycerin challenges, the IMA remained in spasm. Whilst haemodynamically stable, the patient underwent an immediate cardiopulmonary bypass and the distal third of the LAD was incised without aortic cross-clamping. The anastomosis between the IMA and the LAD was found to be patent. There was no antegrade flow from the IMA. A saphenous vein graft was implanted into the distal LAD and anastomosed to the ascending aorta. Weaning from cardiopulmonary bypass was uneventful. Postoperative cardiac enzymes and ECG were normal. Tranoeosphageal echocardiography revealed normal left ventricular function with normal segmental wall motions, indicating that permanent myocardial damage had not occurred. The patient was discharged on the 6th postoperative day.

Despite a number of preventive measures, including intraluminal administration of papaverine, continuous infusion of a calcium antagonist and maintenance of aortic pressure, spontaneous IMA spasm may occasionally occur in the early and late postoperative phases[1,2]. This may result in haemodynamic instability and possibly trans-myocardial infarction[1]. Interestingly, in the current case, the IMA spasm led to inferior ischaemia which was clearly demonstrated by the immediate postoperative bypass angiogram performed under continuous 12-lead electrocardiography. Postoperative ECG, cardiac enzymes and tranoeosphageal echocardiography indicated that a distal vein bypass to the LAD, when performed immediately, may result in rescue of the ischaemic myocardium.

P. R. VOGT
O. HESS
M. I. TURINA
Clinic for Cardiovascular Surgery,
University Hospital,
Rämistrasse 100,
CH-8091 Zurich, Switzerland

References