Stunned myocardium after hip arthroplasty

M. Mizogami1*, K. Shimo2, T. Taguchi3 and Y. Horita3

1Department of Anaesthesiology, Asahi University, 1851 Hozumi, Hozumi-cho, Motosu-gun, Gifu-ken, 5010296, Japan. 2Department of Anaesthesia and 3Department of Cardiology, Keiju Medical Center, 94 Tomioka-cho, Nanao-shi, Ishikawa-ken, 9268605, Japan

*Corresponding author

We report a case of ST-segment elevation occurring in all leads of the ECG during hip arthroplasty. It is thought that this resulted from a stunned myocardium because wall motion abnormalities were reversible, there was no evidence of fixed or vasospastic coronary occlusion and there was only a slight increase in serial cardiac enzymes. Treatment with nicorandil improved the patient’s cardiac function. A [123I]MIBG test revealed a high myocardial washout rate, suggesting that the stunned myocardium was caused by exposure to excessive norepinephrine induced by anaesthesia or surgery.

Br J Anaesth 2000; 84: 510–3

Keywords: heart, myocardial function; complications

Accepted for publication: November 22, 1999

Stunned myocardium is defined as prolonged, yet reversible, post-ischaemic ventricular dysfunction, usually without tissue necrosis.1 It has been described in animal models, and growing evidence suggests that it is a clinically relevant phenomenon in humans.2 The mechanism of stunned myocardium is unknown, although several factors, such as oxygen-derived free radicals,3 changes in calcium homeostasis,4 disruption of cardiac sympathetic neural responsiveness,5 neutrophil activation–migration6 or myocardial cell swelling and oedema8 may be responsible for the prolonged deficit in contractile function. We report an example of myocardial stunning during non-cardiac surgery in a patient on whom we performed a number of cardiac tests to confirm the diagnosis and to guide treatment. We report the findings of single-photon-emission computed tomographs (SPECT) and coronary arteriograms, and discuss the aetiology of stunned myocardium and the efficacy of verapamil or nicorandil in treating it.

Case report

A small, 75-yr-old woman (height 126 cm, weight 26 kg) was scheduled for urgent bipolar hip arthroplasty. Apart from diabetes mellitus, well controlled with insulin (10 units day−1), she had no history of cardiovascular disease. She was anaemic (Hb = 8.9 g dl−1). Physical examination and preoperative 12-lead electrocardiography (ECG) revealed no abnormal findings. Although the patient was diagnosed as having mild mitral regurgitation on transthoracic echocardiography, her left ventricular function was within normal limits. She was not premedicated before surgery. After establishing an intravenous infusion, the patient was connected to a 2-lead ECG (II, V5), pulse oximeter and non-invasive arterial pressure monitor. Following preoxygenation, thiopental 150 mg and vecuronium 6 mg were given intravenously, the trachea was intubated and general anaesthesia was maintained using 1.5–2.0% sevoflurane, nitrous oxide (4 litres min−1) and oxygen (2 litres min −1). After induction, a left radial catheter was inserted for arterial pressure monitoring. The patient’s arterial pressure (systolic pressure 130–160 mm Hg) and heart rate (70–80 beats min−1) remained stable during surgery. The operation took 1 h and was uneventful. With the initiation of transtracheal suction after spontaneous breathing had been established, a nodal bigeminy of sudden onset was noted on the ECG monitor. A few seconds later, even though the rhythm had returned to sinus, the monitor revealed ST-segment elevation in leads II and V5. We suspected the patient had suffered an acute myocardial infarction and immediately started i.v. nitroglycerine (1 µg kg−1 min−1). A 12-lead ECG revealed a Qs pattern in V1–2 and significant ST-segment elevation in I, II, aVL and V3–6. Approximately 1.5 h after its onset, the patient was transferred to the angiographic room. Left ventriculography (LVG) showed akinesis of the apex and hypokinesis of the anterior and inferior walls. The left ventricular ejection fraction (LVEF) was 27%. Coronary arteriograms revealed no evidence of fixed or vasospastic obstruction in the right or left coronary artery. However, a large area of no reflow phenomenon (slow radio-contrast run-off in the coronary arteriogram) was observed. We considered that thrombotic coronary occlusion had caused the myocardial infarction. After cardiac catheterization, the patient was taken to the intensive care unit (ICU) and given
Postoperative stunned myocardium

Fig 1 Twelve-lead electrocardiogram recorded at first onset of ST changes in the operating room (a) and during the second episode in the ICU (b).

Fig 2 Coronary arteriogram taken before (A) and after (B) the administration of intracoronary verapamil, showing improvement of slow radio-contrast run-off (no reflow phenomenon). There is no evidence of fixed or vasospastic obstruction in the left coronary artery. The description time of the whole left coronary artery is improved from 218 frames to 81 frames by the administration of intracoronary verapamil.

continuous i.v. heparin (30 000 units day⁻¹), isosorbide dinitrate (ISDN) (2 µg kg⁻¹ min⁻¹) and dopamine (3 µg kg⁻¹ min⁻¹). The ST-segment elevation in the precordial leads gradually decreased (Fig. 1).

Two days after the operation, while still in the ICU, the ECG monitor again showed ST-segment elevation. A 12-lead ECG revealed ST-segment elevation in all leads. Repeat emergency cardiac catheterization was performed. The left coronary arteriogram did not show left coronary obstruction, but the no reflow phenomenon was seen, as on the previous day. Verapamil was therefore injected directly into the coronary artery in an attempt to improve distal microvascular dysfunction and intramyocardial microvascular spasm. The no reflow phenomenon in the left coronary artery disappeared immediately. The patient was then treated with continuous i.v. nicorandil (4 mg h⁻¹) to avoid any bradycardia (a frequent side-effect of verapamil and beta-blockers). Serum creatine kinase (CK) increased to 306 units (it is normally <180 units), with an increase of muscle-brain isoenzyme (CK-MB) to 36 units (normally <20 units). Apart from aspiration of gastric contents (day 21 after the operation), the remainder of the patient’s ICU stay was uneventful and she was discharged from the ICU 33 days after the operation. LVG revealed improvement of the wall motion abnormality without hypokinesis of the apex and an LVEF of 58% 66 days after the operation (Fig. 2).

With the agreement of the patient’s family, SPECT was performed at regular intervals to confirm the diagnosis and determine treatment. Three types of imaging, with thallium-201 chloride (²⁰¹TCl), iodine-123 betamethyl-iodophenylpentadecanoic acid ([¹²³I]BMIPP) and iodine-123 metaiodobenzylguanidine ([¹²³I]MIBG), gave information about myocardial perfusion, fat metabolism and sympathetic nerve activity respectively. Comparison of the ²⁰¹TCl and [¹²³I]BMIPP tomographs taken in the immediate (days 5–15 after operation) and late phases (days 46–53 after operation) of recovery showed complete resolution of the perfusion and fat metabolism defects at the apex. The [¹²³I]MIBG tomographs revealed a high (61%) washout rate (normal = 18–30%/3 h) in the immediate phase over all the left ventricle; damage was limited to the apex in
Fig 3  Bull’s eye expression of myocardial imaging in the immediate (A) and late (B) phases. Marked defects in the apical area revealed by 201TlCl and [123I]BMIPP were completely restored in the late phase. An increase in sympathetic tone over the left ventricle was observed as a high washout rate (61%) of [123I]MIBG in the immediate phase. Ant., anterior wall; Sept., septum; Lat., lateral wall; Inf., inferior wall.

201TlCl and [123I]BMIPP images. The former finding also improved in the late phase of recovery (Fig. 3).

Discussion
This patient is thought to have suffered a stunned myocardium; the evidence for this is that: wall motion abnormalities were reversible; there was no indication of fixed or vasospastic coronary occlusion; and there was only a slight increase in serial cardiac enzymes despite the severe ST-segment elevation, especially in the second attack.

[123I]MIBG is transported into sympathetic nerve terminals and stored in neuronal vesicles in the same way as norepinephrine, making it possible to investigate sympathetic nerve distribution in the heart in vivo.9 10 The washout rate was calculated as the percent change in cardiac [123I]MIBG activity over time. A higher washout rate indicates increased activity of the myocardial sympathetic nervous system.11 The changes in 201TlCl and [123I]BMIPP imaging were confined to the apex in the immediate phase, suggesting damaged myocardium in this area in accordance with the findings of the LVG and ECG. In contrast, a high washout rate was observed over the whole of the left ventricle in the immediate phase on [123I]MIBG imaging. If the findings of [123I]MIBG imaging resulted from myocardial damage, 201TlCl and [123I]BMIPP confirmed that it must be limited to the apex of the left ventricle. This increased sympathetic activity could have caused the stunned myocardium rather than resulting from the damage caused by it. We suggest that increased sympathetic tone induced by anaesthesia or by surgical stimulation led to an increase in the release of norepinephrine in the heart. This is also thought to cause neurogenic stunned myocardium in subarachnoid haemorrhage.12

The coronary arteriogram of this patient also demonstrated the no reflow phenomenon as the slow run-off of radio-contrast suggested that the blood flow to the stunned area had been markedly reduced, although there was no angiographic evidence of fixed or vasospastic obstruction in the coronary artery. This phenomenon is thought to result from increased microvascular impedance to flow. It has been observed in clinical practice after recanalization of an infarct-related artery by either thrombolysis or balloon angioplasty.13 14 The term ‘no reflow phenomenon’ is usually associated with myocardial infarction and occurs secondary to vascular disruption in the local ischaemic area or tissue oedema. Thus it may not be appropriate to apply this term to the wide area of changes in the left ventricle in this case. However, the slow radio-contrast run-off observed would be classified as grade 2 flow according to Thrombolysis in Myocardial Infarction (TIMI) criteria. Grade 2 flow is considered to be compatible with the no reflow phenomenon.15 Recent studies have suggested that TIMI grade 2 reflow results from microvascular dysfunction in the ischaemic region and may be regarded as reperfusion failure. The underlying mechanisms remain unknown. Piana and colleagues demonstrated that flow-restricting spasm of the distal microvascular bed is responsible.16 In those studies, the hypothesis of distal spasm as an underlying aetiology is supported by the poor response to nitroglycerine. The distal vessels may lack a sufficient local sulhydryl pool to convert nitroglycerine to its active nitrosothiol form. Calcium channel antagonists may not be effective because they only act directly on vascular smooth muscle. In our patient, the stunned myocardium was refractory to nitroglycerine and ISDN, although it responded promptly to the administration of intracoronary verapamil. The beneficial effect of beta-blockers, whose cardioprotective properties can be explained by their effects on lipid metabolism and generation of oxygen free radicals, have also been reported in the treatment of this phenomenon.17 18 However, this patient’s severe cardiac dysfunction did not permit us to use beta-blockers or calcium channel antagonists because of the risk of bradycardia developing. Thus we gave a continuous...
infusion of nicorandil, a nicotinamide nitrate with potassium channel-opening activity, in the ICU. This drug seemed to prevent recurrence of ST-segment elevation and enhanced recovery from the stunned myocardium. Nicorandil is thought to restore myocardial perfusion to the non reflow area by reducing the resistance of the distal microvascular bed by an ATP-sensitive potassium channel-opening effect, the beneficial actions of which on the stunned myocardium are not shared by the nitrovasodilator. These findings suggest that distal microvascular spasm may be the aetiology in this case.

References