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Severe myocardial depression following intravenous nimodipine for aneurysmal subarachnoid haemorrhage

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Sir: We report a case of aneurysmal subarachnoid haemorrhage (SAH) treated with intravenous nimodipine. The patient had myocardial dysfunction which did not respond to high doses of inotropes and vasopressor. Discontinuation of nimodipine improved myocardial function, suggesting that nimodipine worsens the myocardial dysfunction accompanying SAH. Nimodipine was reintroduced after improvement in myocardial function, and was tolerated.

A 52-year-old, previously fit and healthy lady was admitted to our intensive care unit following World Federation of Neurosurgeons grade 5 SAH. An ECG showed antero-lateral wall ischaemia. She was ventilated and needed fluid resuscitation and noradrenaline 0.5 µg/kg per min, to achieve a mean arterial pressure (MAP) of more than 75 mmHg. Nimodipine was commenced at a dose of 0.5 mg/h with a target dose of 2.0 mg/h. Dopamine was commenced (7.5 µg/kg per min) and later replaced with dobutamine (19 µg/kg per min) based on clinical worsening and pulmonary artery (PA) catheter findings (cardiac index 2.6 l/m² per min, pulmonary artery occlusion pressure of 20 mmHg and mixed venous saturation (SVO₂) 61%). The dose of norepinephrine was 0.88 µg/kg per min. Though there was no frank pulmonary oedema, the lung compliance was poor with a FiO₂ of 0.9.

Dobutamine did not improve the cardiac index satisfactorily. At this stage, nimodipine (administered at the rate of 1.2 mg/h) was discontinued, while dobutamine and norepinephrine were maintained at the same dosage. The cardiac index improved from 3 to 3.5 l/m² per min and SVO₂ to 65% over the next hour. Urine output remained over 1 ml/kg per h with resolution of metabolic acidosis.

After a period of stability (3 h), nimodipine was reintroduced at a dose of 0.5 mg/h. This resulted in a fall in the cardiac index to 2.5 l/m² per min and SVO₂

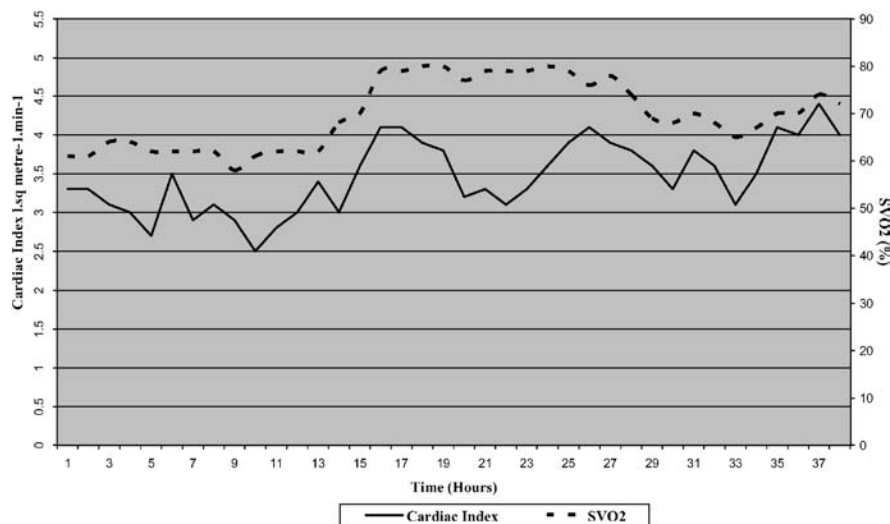


Fig. 1 Cardiac index and SVO₂ plotted against time. Nimodipine was discontinued between 5 and 8 h and reintroduced at 8 h for 30 min. Reduction in dobutamine dosage was attempted between 31 and 33 h

to 58% within 30 min and, therefore, was discontinued. The cardiac index and SVO₂ improved over the next 4 h. Over the next 24 h, the dobutamine and norepinephrine requirement remained unchanged at 19 and 0.88 µg/kg per min, respectively. The cardiac index and SVO₂ remained mostly above 3.5 l/m² per min and more than 70%, respectively (Fig. 1). The urine output was good, with correction of metabolic acidosis and improvement in oxygenation. She had a tracheostomy on day 2 and sedation was subsequently reduced to assess her neurological status. Intravenous nimodipine was reintroduced 48 h after it had been discontinued, and was tolerated well up to the recommended dose of 2 mg/h. The PA catheter was removed on day 4, dobutamine and noradrenaline stopped by day 5 and enteral nimodipine commenced on day 5. On day 8, an anterior communicating artery aneurysm was coiled. Her neurological recovery was poor and she was discharged to the referring hospital for rehabilitation.

Three recent publications have demonstrated an improvement in myocardial dysfunction with treatment based on PA catheter findings in cases with SAH [1, 2, 3]. Nimodipine following SAH caused hypotension and arrhythmias necessitating dose-reduction or discontinuation [4, 5]. Following ischaemic cerebral infarctions, nimodipine was associated with hypotension (especially in hypertensive subjects on 2 mg/h), poor neurological outcome and increased mortality [6].

Diltiazem [7] and nifedipine [8] used to reduce post myocardial infarction cardiac events have been shown to increase mortality in the subgroup of patients with congestive cardiac failure.

In view of the myocardial dysfunction associated with aneurysmal SAH and evidence against calcium-channel blockers following acute myocardial infarction, we have a strong case for more invasive monitoring with probable discontinuation or dose-reduction of nimodipine till the myocardium recovers from the acute insult.

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