Case History No 1

DELTEX MEDICAL

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About a shock state resistant to catecholamines Oesophageal Doppler Monitoring

Mr X, a 40 year old male with no past medical history of note, was admitted to intensive care in a state of shock.

He had been found unconscious at home, lying flat on a hard surface, after having obviously taken a significant amount of sedative drugs (benzodiazepines, tricyclic antidepressants, barbiturates).

On admission he was found to be comatose with a Glasgow Coma Score of 3. His pupils were small and he was generally areflexic. Superficial examination showed signs of right-sided compression with phlyctenular lesions (Blisters). The patient had circulatory collapse with a tachycardia of 140 beats per minute. a systolic blood pressure of 60 mmHg and a mean pressure of only 40 mmHg. Treatment instituted by the SAMU consisted of initial intravascular volume expansion with 1.5 litres of crystalloid, endotracheal intubation and assisted ventilation. An electrocardiogram was unremarkable while echocardiography excluded a cardiogenic shock picture but did show signs of a non-dilated but hypokinetic ventricle. The patient underwent central venous cannulation, arterial catheterization and insertion of an oesophageal Doppler flow probe for cardiac output measurement. Of note, he was also profoundly hypoxaemic with a right lung ‘white-out’.

At this stage, several possible diagnoses should be considered:

- cardiac attack within the framework of drug intoxication. However, the absence of a conduction disorders on electro cardiography does not support this assumption.
- early septic shock on top of an aspiration pneumonia.
- an ischemia-reperfusion injury following a compartment syndrome.

The therapeutic decision lay with either further volume expansion or commencement of a catecholamine. In favour of continued hypovolaemia were the following; (1) a hypokinetic state noted on both echocardiography and oesophageal Doppler monitoring, (2) the presence of a respiratory swing with an obviously large pulse pressure variation (PPV) and a DPP of 35%, (3) the presence of a low cardiac output (down to 2.5 litres/minute). Moreover, the oesophageal flow waveform was relatively typical of an hypokinetic state. On the basis of these results, volume expansion was continued with a mixture of colloid and crystalloid, reaching a cumulative total of 7 litres.

The patient however remained severely hypotensive (mean arterial pressure 45 mmHg), and tachycardic (130 bpm) though the cardiac output jumped from 2.5 to 14 litres/min. He was anuric and hyperlactataemic with a lactate of 8 mmol/l.

We thus found ourselves now facing a hyperdynamic shock state. A new measurement of DPP (8%) confirmed adequate volume loading. Echocardiography confirms the new hyperkinetic state. We then decided to commence vasopressor therapy, initially with dopamine, then with noradrenaline, and finally with a combination of noradrenaline (10 mg/hr infusion rate) and adrenaline (10 mg/hr infusion rate). This treatment was completely ineffective as the mean arterial pressure remained at 40 mmHg, with an unchanged heart rate, an elevated cardiac output (12 l/min), and a severe metabolic acidosis (pH 7.02, lactate level 10 mmol/l). The patient then developed a severe bradycardia, at one point requiring external cardiac massage for three minutes.

In view of this refractory, catecholamine-resistant, shock state, we decided to administer the VASOPRESSIN analogue, GLYPRESSIN (terlipressin). After an intravenous injection of 1mg, the mean arterial pressure rose to 75 mmHg over the following 50 minutes, allowing progressive weaning of the catecholamine infusions. Diuresis resumed and the hyperlactactaemia improved. With the rise in blood pressure, cardiac output fell from 12 to 6 l/min. Throughout this period, blood flow was being continuously monitored by oesophageal Doppler. A later drop in blood pressure necessitated administration of a second bolus of GLYPRESSIN, enabling total cessation of all catecholamine drugs.

The haemodynamic picture at this time revealed a MAP of 85 mmHg (off catecholamines) and a cardiac output of 5 l/min. However, in parallel, ischemia of the extremities and abdominal distension was noted. Treatment with DOBUTAMINE 10 microg/kg/mn enabled a fall in mean arterial pressure to 65 mmHg and an increase in cardiac output. Subsequently, weaning was achieved in 72 hours, while the abdominal distension and digital ischaemia quickly disappeared.

Several comments may be made on this short clinical case. Firstly, GLYPRESSIN and VASOPRESSIN are potentially useful agents in our therapeutic battery of drugs. Secondly, the monitoring of cardiac output is absolutely vital during the administration of vasoconstrictor catecholamines. The oesophageal Doppler is a particularly useful tool in such cases. In our patient, it facilitated straightforward monitoring and efficient patient care. The insertion of an oximetry probe to monitor central venous O_2 saturation would have been interesting, however the concurrent severe coagulopathy in our patient contra-indicated its insertion. As our central venous access was femoral and no subclavian or jugular catheter was in place, we could not perform intermittent central venous blood sampling.

In Summary: potent vasopressor treatment enabled restoration of an optimal haemodynamic state, but it did induce secondary effects due to the reduction on flow with consequent ischaemic manifestations. The use of oesophageal Doppler monitoring permitted real time monitoring and titration of therapy.