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CASE REPORT

Anaphylactic shock: The advantages of intra aortic balloon counter pulsation for the treatment of heart failure[☆]

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Summary The authors report a patient with a history of angina pectoris who developed anaphylactic shock that was complicated by a heart failure due to focal heart ischaemia. Early coronary angiography confirmed the diagnosis of localised coronary hypoperfusion. Intra aortic balloon counter pulsation succeeded in the restoration of coronary blood flow and haemodynamic stability. The authors discuss the opportunity of such treatment when a focal coronary hypoperfusion is diagnosed.

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Implication statement

Myocardial ischaemia and heart failure may complicate anaphylactic shock. This case report confirms that, in cases of focal ischaemia, coronary angiography should be performed early and intra aortic balloon counter pulsation may be helpful for the treatment of the heart failure.

Introduction

Anaphylactic shock under anaesthesia has an incidence of 1/13,000 and may be life threatening,¹ and associated heart failure has been reported in patients with healthy or diseased coronary arteries.² The management of heart failure in anaphylaxis and notably the advantages of intra aortic balloon counter pulsation are not well documented.^{2,3} We report on a patient, who developed anaphylactic shock complicated by cardiogenic shock, during general anaesthesia, and, who was treated effectively by intra aortic balloon counter pulsation.

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Case report

Mr B., 69 years old, was due for repair of a large hernia on the linea alba. During the pre operative anaesthetic consultation, a history of stable angina, treated with amlodipine and aspirin was mentioned. Three years earlier, the patient had presented with unstable angina, which was treated with the insertion of two stents, one in the interventricular artery and the other in the right coronary artery. No other problem was apparent from questioning, notably no allergy problem, or from clinical examination.

After premedication with hydroxyzine (100 mg) 1 h before entering the operating room, and after pre-oxygenation, anaesthesia was induced by intravenous injection of sufentanil (0.6 µg/kg), propofol (2.5 mg/kg) and rocuronium (0.4 mg/kg). Three minutes after induction of anaesthesia, major bronchospasm occurred and manual ventilation of the patient became difficult in spite of salbutamol via the tracheal tube and inhalation of sevoflurane. While the bronchospasm improved, the patient developed bradycardia (40 b/min), hypotension (systolic pressure 40 mmHg) and reduced expired carbon dioxide (10 mmHg). Intravenous administration of adrenaline (epinephrine) followed by nor adrenaline (norepinephrine) resulted in restoration of blood pressure (systolic arterial pressure between 110 and 130 mmHg) and heart rate (heart rate around 90 b/min) but the patient remained haemodynamically unstable.

The ECG monitor showed ST segment changes and an electrocardiogram (ECG) showed a Pardee's wave in limb leads II, II and AVF (ST elevation of more than 4 mm in each derivation), raising fears of thrombosis of the right coronary artery. Ninety minutes after the onset of the presumed anaphylactic shock, coronary angiography was carried out. This showed a stenosis (50%) proximal to the right coronary stent, and severe hypoperfusion of this area. Angioplasty was not considered to be indicated and intra aortic balloon counter pulsation was initiated. This allowed a stable haemodynamic condition to be restored rapidly with improvement of coronary perfusion, and the vasopressors were gradually discontinued over a period of 6 h. The patient was extubated 2 h after coronary angiography and intra aortic balloon counter pulsation was removed after 24 h. The subsequent progress was uneventful. The plasma MB creatine phosphokinase fraction showed a slight rise (63 ng/ml normal values is <4 ng/ml) at 6 h. An ECG carried out later revealed the appearance of a Q wave in the inferior leads and cardiac ultrasound examination showed hypokinesia in the same area. The ejec-

tion fraction was comparable to the preoperative value.

The initial diagnosis of anaphylactic shock was confirmed by the plasma tryptase (1419 µg/l) and histamine (>2000 nmol/l) levels, tested during the acute phase, and allergy tests carried out later confirmed an allergy to rocuronium.

A few months later the patient was operated on under general anesthesia for repair of the hernia on the linea alba and no complications were reported.

Discussion

The classic electrical signs of myocardial ischaemia may be present in cases of anaphylactic shock and rhythm disorders. It may be that amlodipine treatment in the patient reduced the risk of rhythm disorders.⁴ However the action of the calcium antagonist combined with the halogenated inhalational anesthetics could have accentuated the initial shock by increasing the vaso-paralysis induced by anaphylactic shock. The patient was in a very precarious haemodynamic condition, presenting not only with low arterial pressure, but also very low EtCO₂ indicating a major drop in cardiac output. Diffuse signs of ischaemia are more frequent in this context.

This report refers to a case of localized ischaemia in a patient in whom the ECG signs could have suggested previous thrombosis of the right coronary artery. The low arterial pressure and consequent myocardial ischaemia combined with the vasoconstrictive action of the epinephrine could explain the appearance of the abnormal T waves and ST segment.⁵ Even in subjects without coronary disease the administration of adrenaline results in abnormal signs in the ST segment, suggestive of heart failure.⁶

In anaphylactic shock the systematic vasoplegia may induce severe arterial hypotension. In addition the mediators produced during the anaphylactic reaction also have coronary artery constrictive effects. The combination of these two phenomena induces a major decrease in coronary perfusion and thus myocardial ischaemia.² This ischaemia may be further accentuated by the associated hypoxia due to the pulmonary effects of anaphylaxis. The increase in airway pressure due to the bronchospasm may also amplify the drop in coronary perfusion pressure. It is then possible to understand why specific ECG signs appear in relation to coronary pathology in a specific area. In this context intra aortic balloon counter pulsation is appropriate for restoring coronary perfusion

by increasing the arterial pressure in the region affected.

Conclusion

This clinical case highlights the advantages of carrying out early coronary angiography when acute heart failure arises during anaphylactic shock, together with intra aortic balloon counter pulsation for the management of heart failure.

Conflict of interest

None.

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