

Successful resuscitation of class 4 anaphylaxis guided by transthoracic echocardiography

Suxamethonium administration is known to be associated with severe anaphylaxis during anaesthesia. With her consent, we describe a patient who experienced a class 4 anaphylactic reaction who responded poorly to adrenaline administration, but was subsequently stabilised with fluid and vasopressor therapy guided by point-of-care echocardiography.

A 68-year-old female was scheduled for urgent removal of an oesophageal foreign body. She disclosed no drug allergies or regular medication use, and her only past medical history was hypertension. A general anaesthetic administered six years previously for hysteroscopy, dilatation and curettage was reported as uneventful. Suxamethonium was chosen to facilitate intubation in order to prevent aspiration of retained oesophageal secretions. Immediately following administration of the drug, a long sinus pause followed by multiple atrial ectopics and sinus tachycardia of 160/minute was observed. Her femoral pulse could not be palpated.

Successful tracheal intubation was rapidly expedited. Rapid crystalloid fluid resuscitation was initiated via the in situ 18-gauge intravenous (IV) cannula concurrently with adrenaline administration. After four IV boluses given in doses of 1 mg followed by one of 2 mg, it was possible to palpate a femoral pulse. An adrenaline infusion of 300 $\mu\text{g}/\text{minute}$ was then instituted through the peripheral IV. Once arterial access was obtained, it was clear that the therapy thus far was inadequate to sustain a mean arterial pressure above 40 mmHg. In addition, the patient's sinus tachycardia persisted at a rate above 150/minute with increasingly frequent ventricular and atrial ectopics. Volume resuscitation had been limited at this point to one litre of crystalloid due to the size of the in situ cannula and difficulty in obtaining larger bore IV access. A parasternal left ventricular short axis view of the heart was obtained with a portable ultrasound machine (LOGIQ e Ultrasound, GE Healthcare, NY, USA) and a 2 to 5 MHz phased array ultrasound probe (3S, GE Healthcare, NY, USA), showing an under-volumed left ventricle (end-diastolic diameter of 2.4 cm) with low end-systolic volume (end-systolic diameter of 1.3 cm) and good systolic function (fractional shortening of 46%), which was suggestive of normal contractility and a vasodilated circulation (Figure 1). With this haemodynamic information, the adrenaline infusion was slowly weaned and a noradrenaline infusion started at 20 $\mu\text{g}/\text{minute}$. A further 2 l of crystalloid and 300 ml of 20% human albumin were also rapidly infused. The mean arterial pressure was quickly stabilised to

70 mmHg and central venous access was obtained. Inotrope requirements were reduced to adrenaline at 3 $\mu\text{g}/\text{minute}$ with noradrenaline at 12 $\mu\text{g}/\text{minute}$.

At this point the joint decision was made with the surgical team to proceed with the removal of the foreign body, as it had been retained for more than 24 hours and posed a danger of oesophageal perforation and mediastinitis. Inspection by rigid oesophagoscopy revealed minor oesophageal mucosal trauma and no sign of a foreign body, and it was assumed to have been passed. The patient was then referred to the intensive care unit for ongoing care; the offer of pulmonary artery catheter insertion was declined by the intensive care treating team as the patient's mean arterial pressure and inotrope requirements had been stable. By 48 hours her circulation required no inotropic or vasopressor support, although she remained intubated because of significant supraglottic swelling noted on nasendoscopy after a pre-extubation leak test had failed.

A class 4 anaphylactic reaction refers to circulatory or respiratory arrest in the context of suspected massive mast cell degranulation¹. The resulting

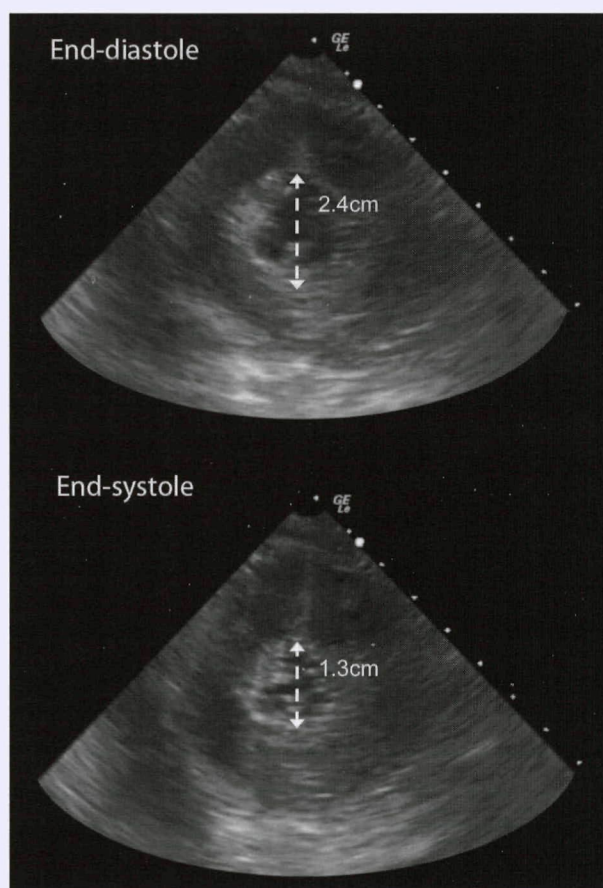


Figure 1: Parasternal left ventricular mid-papillary short axis views at end-diastole and end-systole.

'redistributive' intravascular hypovolaemia contributes to the development of shock. Most cases respond to adrenaline in doses recommended by current Australian Resuscitation Council guidelines for treatment of cardiorespiratory arrest. Adrenaline has been used successfully for many years in the resuscitation of patients suffering anaphylaxis² and is the mainstay of therapy for its effect on mast cell stabilisation. In addition, its simultaneous counteraction of the negative cardiac and circulatory effects of the anaphylactic response² makes adrenaline a logical choice. In this case and others that have been reported³ however, early introduction of vasoconstrictor therapy has succeeded where the exclusive use of volume resuscitation and adrenaline has failed, suggesting that in a distinct subset of patients the primary problem is vasodilation. The recent Australian and New Zealand Anaesthetic Allergy Group guidelines published in 2013 include the *Refractory Management* guidelines, where administration of vasoconstrictors such as noradrenaline and vasopressin are recommended in the face of persistent hypotension, in addition to ongoing adrenaline and volume administration. Echocardiography is included in the recommended approach to refractory anaphylaxis as monitoring to be considered to assess preload.

In our case, the rapid and unequivocal information obtained from a single echocardiographic view identified the primary need for aggressive volume expansion rather than focusing on adrenaline administration alone. Vasoconstrictor administration was of benefit in our case as low systemic vascular resistance was also identified and permitted early normalisation of perfusion pressure, where poor IV access had impaired the delivery of volume resuscitation. Other means of cardiac output monitoring—for example, the Vigileo™ monitor and FloTrac™ sensor (Edwards Lifesciences, CA, USA)—are simple to mobilise but still take time to set up and calibrate, and surrogate measures of preload such as stroke volume variation are subject to confounders. These markers of preload may be more difficult to interpret in the context of a rapidly changing haemodynamic state. These same limitations apply to central venous pressure as an indicator of volume state. When the equipment and expertise is available, echocardiography provides near-instantaneous information about preload, systolic function and systemic vascular resistance. While systolic function was unaffected in our case, intra-aortic balloon pump support has been required to augment systolic function in other reported cases of class 4 anaphylaxis⁴. Most often, however, circulatory failure results from profound vasodilation and massive intravascular volume depletion.

We consider that, with timely attention to the relative contribution of each of these factors, the potential adverse effects of unnecessarily large doses of adrenaline—namely, arrhythmogenesis⁵, coronary ischaemia⁶ and hyperlactataemia—can be avoided. The Australian and New Zealand Anaesthetic Allergy Group currently recommends echocardiography solely to assess volume state; we recommend that the indication for echocardiography be prioritised and its application broadened to include assessment of the relative requirements of volume state, inotropy and vasoconstriction.

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Tracheostomy tube cuff herniation: a cause of airway obstruction in home care patients with long-term tracheostomy

Tracheostomy is a frequently performed procedure in intensive care units for long-term airway maintenance, prolonged ventilator support and prevention of complications related to long-term translaryngeal intubation¹. Keeping the tracheostomy cuff inflated not only reduces leaks around the tracheostomy tube but reduces the likelihood of aspiration in patients who are either obtunded or else have an absent gag reflex. Maintaining an appropriate cuff seal is pivotal in patients who are being ventilated long-term and are difficult to wean. These goals, however, could be jeopardised because of cuff malfunction occurring for numerous reasons, including asymmetrical cuff inflation²⁻⁴.

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