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Brief Report

Anaphylactic shock after amiodarone infusion resulting in haemodynamic collapse requiring a temporary ventricular assist device

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Abstract Acute heart failure related to anaphylactic shock is often reversible and necessitates aggressive support to ensure full recovery. We report the case of a 15-year-old boy who developed severe ventricular dysfunction and haemodynamic instability after administration of amiodarone and required temporary mechanical circulatory support with a left ventricular assist device. He had full recovery of cardiac function and returned to baseline neurologic status. This is the first report of successful left ventricular assist device use for recovery from cardiovascular collapse due to anaphylaxis.

Keywords: Amiodarone; anaphylaxis; Heart Assist Device; ventricular dysfunction; heart failure

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LEFT VENTRICULAR DYSFUNCTION FOLLOWING anaphylactic shock is a well-reported phenomenon.[1–3] Animal models have shown that cardiac damage is a primary event in anaphylactic shock with the development of low cardiac output, high left ventricular end-diastolic pressure, and electrocardiographic evidence of myocardial ischaemia.[4] The aetiology of this clinical entity remains ill-defined.

Circulating epinephrine has been implicated as the aetiologic factor, and in dogs it has been shown that boluses of epinephrine immediately after anaphylactic shock do not accelerate recovery but are actually responsible for left ventricular dysfunction.[2] Akashi et al suggested that epinephrine can lead to multi-vessel epicardial or micro-vascular vasospasm and myocardial ischaemia, which results in myocardial stunning and global left ventricular dysfunction.[2,5] This phenomenon can occur even in the absence of underlying coronary artery narrowing. Alternatively, circulating epinephrine can lead to myocardial stunning not related to myocardial ischaemia. Lyon et al showed that high levels of circulating epinephrine trigger a switch in intracellular signal trafficking in ventricular cardiomyocytes from Gs protein to Gi protein signalling via the β_2-adrenoreceptor, which is negatively inotropic.[2,6]

Kounis syndrome or "allergic angina" describes an acute coronary syndrome occurring during an anaphylactic reaction thought to be secondary to mast cell degranulation and circulating inflammatory mediators.[2,7] Mast cell degranulation can lead to left ventricular dysfunction via a number of pathways. Histamine can produce coronary arterial vasoconstriction leading to myocardial ischaemia, especially in patients with underlying atherosclerosis. Alternatively, TNF-α and IL-1β have a direct negative inotropic effect leading to depressed myocardial contractility.[2,3]

Anaphylaxis due to amiodarone is rare with only two previous reported cases.[2,8] To our knowledge, this is the first report of anaphylactic shock due to amiodarone that resulted in severe haemodynamic collapse requiring mechanical circulatory support.

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Objective

To understand that acute-onset heart failure thought to be related to anaphylactic shock is reversible; and aggressive management strategies, including consideration for mechanical circulatory support, should be employed early.

Case description

Our patient is a 15-year-old boy (weight 76.2 kg, height 168 cm) with a history of tuberous sclerosis, and a large non-obstructive cardiac rhabdomyoma in the left ventricle involving the anterolateral free wall and interventricular septum. He had a history of tumour-associated ventricular tachycardia for which he had undergone failed ablation and subsequent implantable cardioverter defibrillator placement because of inducible unstable ventricular tachycardia. He had prior amiodarone exposures for recurrent ventricular tachycardia and was maintained on atenolol followed by sotalol. On the day of presentation, he developed palpitations and presented to an outside medical centre where he was found to be in a wide complex rhythm, later determined to be ventricular tachycardia, with stable haemodynamics that was initially treated with multiple doses of adenosine, a dose of diltiazem, and finally a dose of amiodarone. After receiving the amiodarone, he had acute onset of facial/lip/tongue swelling, hypotension, poor perfusion, and altered mental status, which was concerning for anaphylactic shock. He quickly progressed to pulseless electrical activity arrest and was successfully resuscitated to a perfusing sinus rhythm with volume, epinephrine, and intubation. He was then transported to our facility.

On arrival, he had marked facial oedema, was cold and mottled, had non-palpable distal pulses, faint femoral pulses, and was hypotensive with a pulse pressure of 10 mmHg (blood pressure 80/70). He continued to have haemodynamic instability and received multiple doses of epinephrine, bicarbonate, calcium, and was started on epinephrine and vasopressin infusions. He went into ventricular tachycardia three times, for which he was successfully defibrillated. An emergent bedside transthoracic echocardiogram was performed, which demonstrated severely depressed biventricular function. He continued to be inotrope resistant and was emergently taken to the operating room for a temporary left ventricular assist device, the centrifugal Rotaflow (Marquet). A median sternotomy was performed and the patient was cannulated via 36 bullet tip cannula at the right superior pulmonary vein-left atrial junction and a 20-french EOPA cannula in the ascending aorta. The patient was stabilised and transported to the cardiac intensive care unit. An electrocardiogram the following day revealed ST elevation in the inferior and lateral leads. He was started on methylprednisone for a 5-day course for anaphylactic shock. Approximately 60 hours after cannulation, a repeat echocardiogram revealed near normal biventricular systolic function and he was weaned and decannulated from mechanical support. Electrocardiographic changes had resolved by the time he was taken off support. He recovered from the event and was discharged home with normal systolic function and baseline neurologic status.

In the setting of previously normal systolic function, our working diagnosis has been either Kounis syndrome (allergic angina) or epinephrine mediated dysfunction in a patient with multiple cardiac rhabdomyomas.

Discussion

The indications for use of ventricular assist devices in the paediatric population are rapidly expanding as the technology improves and clinicians become more experienced with these life-saving devices. Current indications for use of ventricular assist devices include bridge to transplantation, bridge to recovery, bridge to decision, and in rare circumstances as a destination therapy.[9] Extracorporeal membrane oxygenation has served as the primary form of urgent mechanical circulatory support in paediatrics. In the short term, extracorporeal membrane oxygenation remains an adequate method of support. The incidence of complications and mortality related to extracorporeal membrane oxygenation increases with increasing duration of support, which becomes particularly problematic in patients awaiting cardiac transplantation.[4] Limiting circuit size without an oxygenator – as in a left ventricular assist device – has the potential to minimise thromboembolic events and amount of anticoagulation. Many newer ventricular assist devices have improved outcomes and can be used to support patients for longer periods of time with fewer complications.

Anaphylactic shock resulting in acute ventricular dysfunction is rare but very important to recognise because of its potentially fatal but reversible nature. The exact aetiology of ventricular dysfunction following anaphylactic shock is unknown, but the numerous reports related to this entity demonstrate that recovery of normal systolic function is the norm if the patient can be supported long enough. It is important for critical care physicians to be aware of the transient nature of the ventricular dysfunction and treat it aggressively, including consideration for the use of temporary mechanical circulatory support as a bridge to recovery.

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Conflicts of Interest

None.

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