CASE REPORT

Cardiogenic shock in a neonate

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SUMMARY

We describe a case of a healthy male full-term neonate, 21 days old, admitted to the emergency room, presenting a severe cardiovascular collapse with an initial sinus rhythm. The first diagnostic hypothesis was of septic shock, having antibiotics, fluid resuscitation, inotropic drugs and ventilatory support started immediately. After achieving haemodynamic stability, a new cardiovascular collapse occurred with supraventricular tachycardia (SVT). The latter was successfully treated and the neonate did not suffer any organ damage. Cardiogenic shock should be considered despite being a much rarer cause of shock in neonates. SVT is promptly diagnosed when a cardiorespiratory monitor is available; however, the intermittent occurrence of the tachycardia episodes makes this diagnosis more difficult to recognise and manage.

BACKGROUND

Shock is a clinical disorder that challenges caregivers in the neonatal intensive care unit. Critically ill neonates may develop shock due to a variety of causes, but the predominant cause of shock in this population is sepsis. Treatment and supportive care of the neonate in shock is determined by the underlying cause with the ultimate goal of treatment being adequate perfusion of tissues so as to maintain adequate delivery of oxygen to cells and remove metabolic waste products.¹ Cardiogenic shock in neonates is a rare medical emergency. The most common aetiologies include congenital heart diseases with reduced cardiac output and systemic hypotension, cardiac muscle disorders, dysrhythmias and metabolic conditions.²

We report a case of a neonate who was admitted to the emergency department with cardiogenic shock due to a supraventricular tachycardia (SVT). On admission, he showed a sinus rhythm. The diagnosis of this arrhythmia was only established after haemodynamic stabilisation.

CASE PRESENTATION

A 21-day-old Caucasian full-term male neonate was admitted to the emergency room with shock. He had no relevant medical history and no family history of sudden death or arrhythmias. Parents reported irritability, grunting, poor feeding and respiratory distress in the last 12 h. There was no history of fever, weight loss or ingestion of recent medication. At observation, he presented poor general status with marked hypotonia, decreased level of consciousness, bradycardia (heart rate (HR) of 70 rpm), immeasurable blood pressure, tachypnoea and retractions. Percutaneous oxygen saturation was 75% at room air. He had a mottled blue

and white skin with prolonged capillary refill time (above 3 s) and weak pulses. Cardiac auscultation showed rhythmic heart sounds without murmurs. On pulmonary auscultation, he had symmetric entrance of air and discrete bilateral rales. He was immediately transferred to the paediatric and neonatal intensive care unit with an initial diagnostic hypothesis of septic shock. He started intravenous fluid resuscitation, inotropic drugs, broad-spectrum antibiotics and non-invasive ventilation support with subsequent re-establishment of normal blood pressure.

Laboratory tests revealed white cell count of 26 600/ μ L, neutrophils 42.4%, haemoglobin 9.7 g/dL, a normal platelet count and a C protein reactive level of 3.9 mg/L. There were no significant changes in sodium, potassium, calcium and magnesium levels. His initial arterial gasometry showed metabolic acidosis (pH 7.11, pCO₂ 15 mm Hg, bicarbonate 7.8 mEq/L and lactate 16 mmol/L) and mild hypoglycaemia (59 mg/dL). He also had an elevated blood urea nitrogen (71 mg/dL) and high limit creatinine (0.9 mg/dL), indicating evolution to a prerenal failure. After the first hour and a half the stabilisation, partial correction of his acidosis was achieved. Cardiac monitoring revealed a normal sinus rhythm. A transthoracic echocardiogram was performed that showed a normal structural heart with mild mitral regurgitation and no aortic coarctation. Chest radiograph showed a slightly enlarged heart (cardiothoracic ratio 0.56) with an apparently normal pulmonary parenchyma.

On the third hour of admission, he was tachycardic (HR 235 rpm) with signs of acute cardiocirculatory failure. He was intubated and ventilated with conventional mechanical ventilation. His ECG revealed SVT (regular rhythm, with no P waves and a narrow QRS complex tachycardia, no δ waves and normal T waves). Two intravenous bolus of adenosine were administered but with no response, followed by an intravenous perfusion of amiodarone with a reversion of SVT to a sinus rhythm. About 24 h after admission, while on amiodarone perfusion, he developed two further episodes of SVT (HR of roughly 300 rpm) which were successfully terminated with electric cardioversion (figure 1). On the same day, he presented other episodes of SVT that reverted spontaneously. The final diagnosis was of cardiogenic shock caused by a paroxysmal supraventricular tachycardia. He was progressively weaned off inotropic support and mechanical ventilation and, on the third day of admission, successfully extubated. He was evaluated by a paediatric cardiologist on the first day of admission and continued amiodarone perfusion. Transfontanelar ultrasonography revealed no ischaemic, haemorrhagic or ventricular alterations. Before

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Figure 1 Electrocardiogram strip of the moment of one of the electric cardioversions, showing the reversion of a supraventricular tachycardia to a normal sinus rhythm.

discharge, oral administration of amiodarone was started and the follow-up routine transthoracic echocardiogram was normal. He was discharged on day 10 of admission with oral amiodarone.

OUTCOME AND FOLLOW-UP

During follow-up by a paediatric cardiologist, oral amiodarone was maintained until 6 months after discharge and replaced by a β blocker (sotalol). Thyroid function tests were normal. Currently, with 3 years of age, he continues the same antiarrhythmic medication (sotalol, 160 mg/day) and his parents describe sporadic short-lived (<10 min) episodes of tachycardia that are well tolerated. The subsequent ECG records have been normal.

DISCUSSION

Cardiogenic shock is uncommon among children when compared with adults and a high index of suspicion is mandatory. Cardiogenic shock results from pump failure manifested physiologically by decreased systolic function and depressed cardiac output. The three general categories are cardiomyopathies, arrhythmias and obstructive disorders.³

In children, SVT is the most common rhythm disturbance. The prevalence of SVT is not well defined, but is estimated to be between 1 in 250 and 25 000 children⁴ and often caused by an accessory atrioventricular pathway. Children may exhibit a range of clinical findings from no symptoms to cardiogenic shock, depending on the duration of SVT. A key component on the assessment of a new-born with shock and tachycardia is the categorisation of the QRS complex as narrow or wide from the cardiorespiratory (CR) monitor or 12-lead ECG. In the presence of a narrow complex tachycardia, it is fundamental to determine whether there is a p wave, so as to distinguish from sinus tachycardia. Approximately 90% of paediatric patients who have non-sinus narrow QRS complex tachycardia will have an atrioventricular re-entrant (typical) SVT.⁵ Episodes of SVT are usually paroxysmal and are characterised by abrupt onset and termination. Heart failure has been reported in about half of the neonatal cases of SVT as illustrated in a retrospective case series of infants below 30 days of age with SVT in which 52 of 109 patients had some clinical evidence of heart failure. In the same study, a lack of arrhythmia, antiarrhythmic medication or late recurrence of arrhythmia is described in 52% at 1 year of age.⁶ The acute treatment of a single episode of SVT has generally an excellent prognosis. An antiarrhythmic prophylaxis of SVT recurrences is usually recommended during the first year of life. Although many efficient drugs are available for the SVT treatment, a careful risk-benefit analysis of each case should suggest the correct drug choice.⁷

The diagnosis of SVT is promptly established if a cardiorespiratory monitor or 12-lead ECG examination is available. However, in our case, this diagnosis was not possible to make on admission because the neonate demonstrated an initial sinus rhythm. After haemodynamic stabilisation, he developed SVT and the cardiovascular collapse retrospectively was presumed to have been caused by a prolonged episode of SVT.

A review of the literature revealed two reports of five infants who presented with cardiogenic shock and a normal sinus rhythm and in whom SVT was only subsequently discovered.^{8 9} One possible explanation for this is that the metabolic derangements caused by severe haemodynamic compromise could affect cardiac conduction through the aberrant pathway, resulting in resolution of the tachycardia.⁸

In conclusion, when managing a neonate in shock, although the definitive goal is to treat the underlying cause,¹ prompt supportive treatment and measures to restore blood flow and oxygen delivery to tissues is essential and, in this patient, it contributed for the good prognosis achieved. SVT in early infancy is dangerous and potentially fatal if not treated early and appropriately.

Learning points

- ▶ Cardiogenic shock is a rare disturbance in neonates, but should be kept in mind.
- ▶ The major causes of cardiogenic shock in children are cardiomyopathies, arrhythmias and obstructive disorders.
- ▶ Supraventricular tachycardia (SVT) is the most common rhythm disturbance in children.
- ▶ The challenge of the diagnosis of a cardiogenic shock due to an SVT is the immediate identification of an intermittent tachycardia episode (that alternates with a sinus rhythm).
- ▶ The priority in a neonatal shock presentation is of supportive treatment to restore blood flow and oxygen delivery to tissues with the definitive goal being the treatment of the underlying cause.

Competing interests None.

Patient consent Obtained.

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