APMB - Atti della Accademia Peloritana dei Pericolanti
Classe di Scienze Medico Biologiche
Vol. 111(1) 2023

ISSN 1828-6550

DOI: 10.13129/1828-6550/APMB.111.1.2023.CCS3

Clinical Case Seminar **CCS3 (1-4)**

A case of a shocking rhythm

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Abstract

We report a case of a 40-day-old patient admitted to the neonatal and paediatric intensive care unit for severe cardiovascular failure with an initial sinus rhythm. The first diagnostic hypothesis was septic shock, thus antibiotics, fluid resuscitation, inotropic drugs and ventilatory supportwere immediately started. After achieving haemodynamic stability, a new cardiovascular failure occurred with supraventricular tachycardia (SVT), making diagnosis of cardiogenic shock. Cardiogenic shock should be considered, although it is a rare cause of shock in children. SVT may be a cause of cardiogenic shock, therefore it should be diagnosed whit the aid of a cardiorespiratory monitor, which represents a useful device in the differential diagnosis of the various types of shock.

Key words: newborn;supraventricular tachycardia; cardiogenic shock

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Introduction

Shock is an acute circulatory failure syndrome in which tissue hypoperfusion leads to insufficient supply of oxygen and nutrients to the cells, resulting in activation of anaerobic metabolism and accumulation of lactic acid [1]. The most common causes of shock in neonates and paediatrics are septic shock and hypovolemic shock [2]. Less common but associated with higher mortality is cardiogenic shock [1]. We report a case of aninfant who was admitted to the neonatal and pediatric intensive care unit 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 an initial haemodynamic stabilisation.

Case presentation

A previously healthy 40-day-old caucasian full-term female was admitted to the emergency department with a state of shock. She had no relevant past medical history and no family history of sudden death or arrhythmias. Parents then reported irritability, poor feeding and respiratory distress in the last 12 h. There was no history of fever or ingestion of recent medication Parents also reported episode of sweating and pallor during feeding in the last month. At clinical observation, she presented compromised general status with marked hypotonia, decreased

level of consciousness, tachycardia (heart rate 180 rpm), immensurable blood pressure, tachypnoea and retractions. Percutaneous oxygen saturation (SaO2) was 60% at room air. She had a cyanotic skin with prolonged capillary refill time (>5 second) and weak pulses. Cardiac auscultation showed rhythmic heart sounds without murmurs. On pulmonary auscultation, she had symmetric entrance of air with bilateral rales. Abdominal physical examination showed an epatomegaly. She was immediately transferred to the paediatric and neonatal intensive care unit with an initial diagnostic hypothesis of septic shock. She started intravenous fluid resuscitation, inotropic drugs, broad-spectrum antibiotics and invasive ventilation support with subsequent re-establishment of normal blood pressure. Her initial arterial blood gas showed severe acidosis (pH 6.57, pCO_2 60.7 mmHg, pO2 51 mmHg, bicarbonate 3.8 mEq/L, excess bases -32.5 and lactate 232 mmol/L). She was intubated and ventilated with conventional mechanical ventilation. Correction of acidosis with bicarbonate infusion was necessary, trough a central venous catheter. Laboratory tests revealed white cell count of 31500/μL, neutrophils 35%, haemoglobin 12.1 g/ dL, normal platelet count and negative C-protein reactive level. There were not significant changes in electrolytes levels. Transthoracic echocardiogram showed a normal structural heart with mild mitral regurgitation and a pump function at the lower limits of the normal (Ejection fraction 59%). Chest X-ray showed multiple parenchymal thickening and widening of the right mediastinal profile. After one hour, she was tachycardic (HR 280 rpm). Her ECG-monitor revealed SVT (regular rhythm, with no P waves and a narrow QRS complex tachycardia, no δ waves and normal T waves). First bolus of intravenous adenosine (0.1 mg/kg), followed by other two boluses with higher dose (0.2 mg/kg) were necessary to re-establish sinusalrythm. Diagnosis of cardiogenic shock caused by a paroxysmal supraventricular tachycardiawas made. She was evaluated by a pediatriccadiologist, who indicated beta-blocker therapy with propanolol. The patient was progressively weaned-off inotropic support and mechanical ventilation and, on the fourth day of admission, successfully extubated. Other diagnostic investigations performed during hospitalization did not reveal any organ impairments, therefore she was discharged after 7 days with beta blocker therapy.

Discussion

Cardiogenic shock is an acute state of organ hypoperfusion,following cardiac failure, which can result in decreased systolic function and depressed cardiac output [3]. The main causes in paediatrics are congenital heart disease, arrhythmias and cardiomyopathy [4].

Cardiogenic shock is uncommon among neonates and children., representing 5 – 13% of cases of shock in paediatric emergencies, with a high mortality rates, about 35 – 50% [5].

SVT is the most common arrhytmias in children, with an estimated incidence of 1 in every 250 to 1000 pediatric patients[6].In most cases the cause is an accessory atrioventricular pathway [7]. In children, clinical feature may vary from no symptoms to cardiogenic shock, depending on the duration of SVT and the age. The diagnosis is often incidental, due to the finding of tachycardia during a routine visit. In some cases, parents report aspecific symptoms, such as poor feeding, pallor, sweating, irritability. Episodes of SVT are usually paroxysmal and are characterised by abrupt onset and ending. 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 about 47.7% of patients had some clinical evidence of heart failure [8]. The diagnosis of SVT is promptly established if a

cardiorespiratory monitor or 12-lead ECG examination is available [9]. However, in our case, this diagnosis was not made on admission due to the presence of an initial sinus rhythm. After haemodynamic stabilisation, she developed SVT, thus, the cardiogenic shock retrospectively was presumed to have been caused by a prolonged episode of SVT. Regarding the treatment for acute SVT, the most important decision is led by the presence of hemodynamic compromise. Hemodynamically unstable patients with a tachyarrhythmia and signs of shock should receive immediate synchronized cardioversion. In stable patients with SVT, vagal manoeuvres, such as Valsalva or diving reflex, have been shown to successfully terminate SVT in 20%–63% of cases[10]. Adenosine is an atrioventricular (AV) nodal blocking agent and is the initial drug of choice for conversion of SVT. The starting dose is 0.1 mg/kg (to a maximum of 6 mg), followed by 0.2 mg/kg (to a maximum of 12 mg). If medical therapy with adenosine is unsuccessful, or stable SVT deteriorates to unstable SVT, synchronized cardioversion is needed [10]. In our case, despite the partially compromised haemodynamic conditions, the patient responded to a third bolus of adenosine with adequate dosage. Beta-blockers, particularly propranolol, have become established as the most common first-line agent for maintenance therapy in infants, continuing treatment for 6 months to a year followed by a period of weaning [11].

In conclusion, identifying the underlying cause of a state of shock can be difficult and often results in a exclusion diagnosis. Regardless of the underlying cause, initial management of shock focuses on respiratory support and cardiovascular stabilization, which are related to a good prognosis. Cardiogenic shock should always be considered in the differential diagnosis of shock states in children, even if it is rarer. Monitoring devices, such as the ECG monitor, provide an important aid in the recognition and management of shock states, as well as in differential diagnostics. SVT, often characterized by aspecific symptoms, can be the cause of cardiogenic shock and should be promptly recognized and treated.

Conflicts of Interest: There is no potential conflict of interest, and the authors have nothing to disclose. This work was not supported by any grant. Informed consent was obtained

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Communicated Dec 15, 20; received February 21, 2023; revised and accepted February 28 2023; published on line April 7, 2023