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SUPRAVENTRICULAR TACHYCARDIA-INDUCED CARDIOGENIC SHOCK IN A NEONATE: A CASE REPORT

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INTRODUCTION

Shock is defined as the failure to deliver adequate oxygenated blood to tissues, re-sulting in a mismatch between oxygen supply and demand. Oxygen delivery is primarily determined by arterial oxygen content (dependent on hemoglobin con-centration and oxygen saturation) and cardiac output, which is the product of stroke volume and heart rate (Marcdante et Kliegman, 2019). Shock can be broadly classified into three progressive phases: com-pensated, uncompensated, and irreversible. In the compensated phase, cardiac ou-tput is maintained via sympathetic activation, increasing heart rate and systemic vascular resistance (Mardešić et al., 2016). Based on the underlying pathogenesis, shock can be classified: hypovolemic, cardiogenic, distributive (septic) and obstructive shock. Cardiogenic shock is pathophysiologically characterized by impai-red systolic function and reduced cardiac output, typically resulting from congenital heart defects, cardiomyopathies, or arrhythmias (Brissaud et al., 2016).

We report the case of a neonate admitted to our clinic in a state of cardiogenic shock caused by supraventricular tachycardia (SVT). On admission, the infant presented in sinus rhythm, which initially masked the underlying arrhythmia and complicated the diagnostic process. The diagnosis of SVT was confirmed only after haemodynamic stabilization, emphasizing the diagnostic challenges associated with undifferentiated shock in neonates.

CASE DESCRIPTION

A 27-day-old female neonate was brought to the emergency department due to respiratory distress, poor general condition, and grunting. The parents reported that the infant had awakened during the night with a high-pitched cry and assumed a fetal position, followed by labored breathing and peripheral cyanosis.

The infant was born at term (40 weeks' gestation) after a pregnancy complicated by gestational diabetes. Apgar scores were normal; birth weight was 3600 g, and length was 56 cm. She had shown normal neurological and clinical development and had been breastfed regularly.

On admission, the neonate was in shock. Clinical examination revealed mottled skin, cold and livid extremities, capillary refill time of 3 seconds, and bradycardia (~100 bpm). She was in respiratory distress, with narrow, isocoric pupils, and a soft anterior fontanelle level with the skull.. Pulmonary auscultation was unremarkable. Cardiac examination revealed a rhythmic, bradycardic heart rate with clearly audible tones and no murmurs. The abdomen was distended. The patient was hypotonic. She was immediately intubated and placed on mechanical ventilation. Initial venous blood gas analysis demonstrated severe mixed acidosis (pH 6.57, pCO_2 9.46 kPa, pO_2 4.9 kPa, SpO_2 15%, HCO_3^- 6.4 mmol/L, BE – 31.7, lactate >20 mmol/L). Boluses of crystalloids and sodium bicarbonate were administered, along with dopamine and dobutamine.

Septic shock was initially suspected. Lumbar puncture excluded meningitis, and laboratory markers were within normal ranges. Empiric triple antibiotic therapy (ampicillin, amikacin, and ceftazidime) was initiated. Chest X-ray was unremarkable.

Following hemodynamic stabilization, the patient showed improved vital signs (SpO_2 93%, HR 130 bpm, BP 84/36 [52] mmHg). Several hours later, she developed a narrow-complex tachycardia >250 bpm. ECG confirmed SVT at >300 bpm, which was successfully terminated with adenosine and amiodarone. Echocardiography revealed normal cardiac anatomy, while abdo-minal ultrasound showed a small amount of free fluid between bowel loops. On the first day of hospitalization, propranolol, gastric protection, neuroprotection (phe-nobarbital), and analgesia/sedation were initiated. Laboratory findings revealed markedly elevated proBNP (55,707 pg/mL), troponin (778 ng/L), and signifi-cantly elevated AST, ALT, and LDH le-vels.

On day two, the patient experienced a brief episode of bradycardia, which responded promptly to resuscitation. Serial echocardiography revealed a small pleural effusion, which later resolved. On day five, during extubation, the patient experienced a tonic-clonic seizure that ceased following administration of phenobarbital. EEG demonstrated mildly dysrhythmic patterns, and levetiracetam was added to the treatment. Cranial ultrasound was normal.

The infant was discharged in stable condi-tion on day 13 with continued beta-blocker and antiepileptic therapy.

DISCUSSION

Cardiogenic shock in neonates, although rare, is a critical emergency with high morbidity and mortality. It most commonly arises from congenital heart defects, myocarditis, or arrhythmias such as su-praventricular

tachycardia (SVT). The immature neonatal myocardium is particu-larly vulnerable to volume and pressure overload, and even mild myocardial dysfunction can result in profound circulatory failure. Unlike in older children and adults, neonates often present with non-specific signs - such as poor feeding, irritability, vomiting, or cyanosis - which can delay diagnosis. (Brissaud et al., 2016). Early recognition and differentiation from other types of shock are crucial for survival and favorable long-term outcomes.

A study conducted at the British Columbia Children's Hospital revealed that, in neonates presenting with undifferentiated decompensated shock, cardiogenic causes - particularly arrhythmias like SVT are mo-re frequently identified than infectious etiologies (Chan et al., 2019). This finding underscores the importance of maintaining a high index of suspicion for arrhythmic causes of shock even in the absence of classical signs of infection.

SVT is among the most common ar-rhythmias requiring emergency cardiovas-cular intervention in neonates. Its inciden-ce is estimated at 0.06 per 1,000 live births within the first month of life and 0.25 per 1,000 by the end of the first year (Kamel et al., 2022). Clinical manifestations of SVT are often nonspecific, which can delay diagnosis and appropriate manage-ment. If left untreated, sustained SVT can lead to myocardial dysfunction, hemodynamic compromise, cardiogenic shock, and ultimately heart failure.

Several case reports have highlighted SVT as a primary cause of cardiogenic shock in neonates. A report by Viveiros et al. described a neonate who, similarly to our patient, initially presented in sinus rhythm and developed SVT only after hemodynamic stabilization. Conversely, a Croatian case report presented a neonate diagnosed with SVT and cardiogenic shock upon ad-mission, illustrating the variability in clinical presentation of this condition (Viveiros et al., 2013; Doder et al., 2022).

Further supporting these observations, a retrospective study by Gilljam et al. (2008) found that nearly half (48%) of neonates under 30 days old with SVT exhibited signs of heart failure. Importantly, the same study demonstrated that by one year of age, 52% of these infants remained free of arrhythmia without requiring further antiarrhythmic therapy. These findings highlight the potential for spontaneous resolution of SVT during infancy, particularly in cases without underlying structural heart disease.

Additionally, neonates diagnosed with SVT before 60 days of age and without a Wolff-Parkinson-White (WPW) pattern on electrocardiogram are considered to have a low risk of recurrence. In 90% of such cases, SVT does not recur by one year of age. In those treated with antiarrhythmic therapy, recurrence rates fall to only 5% after cessation of treatment, emphasizing the importance of individualized management strategies based on risk stratification (Mah et al., (2017).

In our case, the initially misleading presentation in sinus rhythm delayed recognition of the underlying arrhythmic cause. The patient's rapid deterioration and development of SVT after stabilization emphasize the diagnostic challenges associated with undifferentiated shock in neonates. Early ECG monitoring and consideration of arrhythmia, even in the absence of typical features, were pivotal in achieving a favorable outcome.

CONCLUSION

This case, therefore, highlights the need for comprehensive assessment and timely intervention in neonates with cardiogenic shock, especially when the etiology is not immediately apparent. Although SVT-induced cardiogenic shock is rare, it is a critical diagnosis that can be made with prompt electrocardiographic evaluation and appropriate pharmacologic intervention. Early diagnosis and treatment are key to preventing long-term sequelae, including developmental delays and organ dysfunction.

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Contribution of individual authors:

Iva Mandić: conception, writing the first draft, manuscript preparation, execution.
Svjetlana Mikulić: conception, organization, manuscript preparation, analysis, design.
Helena Karlović: organization, review and critique.
Marija Novaković Bošnjak: organization, review and critique
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