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Multi-Organ Dysfunction with Supraventricular Tachycardia, Hypertrophic Cardiomyopathy, Multiple Genetic Variants and Necrotizing Enterocolitis in An Extremely Preterm Infant: A Case Report

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Abstract

Background: Extremely preterm infants born to mothers with severe preeclampsia and intrauterine fetal death face significant morbidity and mortality risks. Multi-organ dysfunction in these infants presents complex management challenges requiring coordinated intensive care.

Case Presentation: We describe a female neonate delivered at 31+5 weeks gestation (birth weight 1.98kg) via emergency cesarean delivery following maternal preeclampsia and co-twin intrauterine demise. The neonate experienced severe respiratory distress syndrome, persistent pulmonary hypertension, septic shock, necrotizing enterocolitis, acute kidney injury, neonatal seizures, supraventricular tachycardia, and patent foramen ovale with concentric left ventricular hypertrophy. Additional complications included subcortical leukomalacia with hypoxic-ischemic encephalopathy and genetic variants affecting multiple organ systems. After 14 weeks of intensive care management, discharge occurred in stable condition.

Conclusion: Extremely preterm neonates experiencing multiple organ dysfunction may achieve favorable outcomes through comprehensive intensive care and timely recognition of complications.

Keywords: Extremely preterm infant, Multi-organ dysfunction, Supraventricular tachycardia, Hypertrophic cardiomyopathy, Necrotizing enterocolitis, Multiple genetic variants.

Introduction

Delivery before 28 weeks gestation, classified as extremely preterm birth, occurs in approximately 0.4% of pregnancies yet contributes disproportionately to neonatal mortality and long-term morbidity.[1] Survival in this population varies between 60-90% based on gestational age at delivery and birth weight, with survivors frequently experiencing complex medical challenges[2]

Preeclampsia complicates 2-8% of pregnancies, characterized by new-onset hypertension and proteinuria beyond 20 weeks gestation[3] Multiple gestation pregnancies with severe preeclampsia pose amplified risks for both maternal and fetal well-being[4] The underlying pathophysiology involves defective placental implantation, resulting in maternal vascular dysfunction and systemic inflammation that compromises fetal growth and development[5] When intrauterine fetal death occurs alongside preeclampsia in twin pregnancies, the surviving fetus faces additional exposure to inflammatory cascades and thrombotic factors[6]

The syndrome of multiple organ failure in extremely preterm neonates encompasses a complex interplay of respiratory, cardiovascular, neurologic, gastrointestinal, and renal dysfunction[7] Immature organ systems demonstrate heightened susceptibility to hypoxic injury, inflammatory mediators, and iatrogenic complications from intensive interventions[8] Comprehending the relationships between maternal pathology, fetal conditions, and neonatal outcomes remains fundamental to advancing therapeutic approaches[9] Detailed case documentation of successful intensive care management provides valuable clinical insights and supports evidence-based practice development[10]

Case Presentation

A female infant was born at 31 weeks and 5 days gestation to a 28-year-old gravida 2, para 1 mother. The pregnancy was complicated by dichorionic diamniotic twins with intrauterine fetal death of twin 2 at 30 weeks gestation and severe maternal preeclampsia. Emergency cesarean section was performed due to maternal deterioration with hypertension (160/110 mmHg), proteinuria (4+ dipstick), and laboratory abnormalities.

The infant cried immediately at birth with APGAR scores of 6, 7, and 8 at 1, 5, and 10 minutes, respectively. 1.98 kg was the birth weight, which was suitable for the gestational age. Initial examination revealed no gross congenital anomalies. Within one hour, the infant developed respiratory distress with tachypnea, subcostal retractions, and decreased oxygen saturation (<90% on room air), necessitating NICU transfer. The parents provided written informed consent so that this case report may be published.

Clinical Course

Initial stabilization with oxygen via nasal prongs progressed to non-invasive ventilation, then endotracheal intubation due to increasing oxygen requirements (FiO_2 0.8) and respiratory fatigue. Chest radiography revealed bilateral ground-glass opacification consistent with respiratory distress syndrome. Surfactant (poractant alfa 200 mg/kg) was administered intratracheally.

Hemodynamic instability with poor perfusion and hypotension developed, requiring inotropic support with dopamine (5-10 µg/kg/min) and epinephrine (0.1 µg/kg/min). Laboratory studies revealed leukocytosis with left shift and elevated inflammatory markers. Initial broad-spectrum antibiotics (ampicillin and gentamicin) were started, but clinical deterioration with septic shock necessitated escalation to meropenem and vancomycin.

Persistent pulmonary hypertension confirmed by echocardiography was treated with intravenous sildenafil (0.5 mg/kg every 6 hours). Neonatal seizures were confirmed by continuous EEG monitoring and treated with phenobarbital loading dose (20 mg/kg) followed by maintenance therapy (5 mg/kg/day).

On day 5, the infant developed supraventricular tachycardia with heart rate persistently >220/min (range 220-260 bpm) accompanied by narrow QRS complexes (<0.08 seconds) on 12-lead electrocardiography. Clinical manifestations included poor feeding, irritability, increased work of breathing, and progressive hemodynamic compromise with decreased peripheral perfusion, prolonged capillary refill time (>3 seconds), and weak pulse volume. The arrhythmia was associated with metabolic acidosis (pH 7.25, base deficit -8) and elevated lactate (4.2 mmol/L), indicating tissue hypoperfusion.

The SVT episode was preceded by mild hypokalemia (3.2 mEq/L) and hypomagnesemia (1.4 mg/dL). Initial management with rapid-acting adenosine (0.1 mg/kg IV push via central venous catheter) failed to terminate the arrhythmia, with no response after the first dose. A second adenosine dose (0.2 mg/kg IV push) administered 2 minutes later also proved unsuccessful, possibly due to shortened half-life in the preterm circulation or concurrent theophylline therapy for apnea of prematurity. Vagal maneuvers including ice application to the face were attempted but ineffective due to patient age and clinical instability.

Given the persistent tachyarrhythmia lasting >2 hours and progressive clinical deterioration with signs of congestive heart failure, pharmacological intervention was prioritized over electrical cardioversion. Synchronized cardioversion was not performed due to several considerations specific to extremely preterm neonates: the patient's critically low birth weight (1.98 kg) posed technical challenges for appropriate paddle size and energy delivery, the hemodynamic status remained stable enough to attempt pharmacological conversion, and the risks of general anesthesia or deep sedation in an already critically ill preterm infant were deemed excessive. Additionally, the narrow QRS morphology and clinical presentation suggested typical SVT amenable to pharmacological termination rather than atrial flutter or other arrhythmias requiring electrical intervention.

Amiodarone loading dose (5 mg/kg IV over 1 hour) followed by continuous infusion (5 mg/kg/day) was initiated. The arrhythmia successfully converted to normal sinus rhythm within 6 hours of amiodarone initiation. Electrolyte correction with potassium and magnesium supplementation was performed concurrently. The infant required continuous cardiac monitoring with telemetry, serial 12-lead ECGs every 6 hours, and echocardiographic assessment to monitor for amiodarone-related cardiac effects. The amiodarone was gradually weaned over 10 days with careful rhythm monitoring, and no recurrence of supraventricular tachycardia was observed during the remainder of hospitalization or at follow-up visits.

Necrotizing enterocolitis was diagnosed based on clinical signs and pneumatosis intestinalis, managed conservatively with bowel rest and antibiotics. Acute kidney injury with decreased urine output and rising creatinine was treated with furosemide (1 mg/kg every 12 hours). Two-dimensional echocardiography revealed patent foramen ovale measuring 2.1 mm, left ventricular ejection fraction of 75% with concentric left ventricular hypertrophy and mild mitral and tricuspid regurgitation, managed with propranolol (1 mg/kg/day). Left inguinal

hernia was successfully repaired via left herniotomy with no complications.

Blood culture grew Pseudomonas aeruginosa, treated with targeted antibiotic therapy. The infant developed bronchopulmonary dysplasia requiring dexamethasone and bronchodilator therapy. Ophthalmologic examination revealed retinopathy of prematurity (Zone 2, incomplete vascularization without plus disease).

Diagnostic Assessment

Serial investigations revealed initial arterial blood gas pH 7.28, pCO_2 52 mmHg, base deficit -6.

Procalcitonin 3.2 ng/mL and CRP 24 mg/L were the highest levels of inflammatory indicators. Minimum hemoglobin was 7.8 g/dL, requiring transfusion. Peak creatinine reached 1.4 mg/dL.

Two-dimensional echocardiography revealed patent foramen ovale measuring 2.1 mm, left ventricular ejection fraction of 75% with concentric left ventricular hypertrophy, and mild mitral and tricuspid regurgitation. Electroencephalography demonstrated normal background activity with no epileptiform discharges.

Cranial ultrasonography showed no intraventricular hemorrhage. Neurosonography revealed multiple anechoic cystic lesions involving cortex along bilateral fronto-parietal regions (right > left), with the largest measuring 10×13 mm, suggestive of subcortical leukomalacia with hypoxic-ischemic encephalopathy and choroid plexus cyst.

Whole genome exome sequencing revealed variants suggestive of pituitary hormone deficiency combined type 4, thyroid dyshormonogenesis type 3, autoimmune disease multisystem infantile-onset type 1, and hyper-IgE syndrome type 1 (autosomal dominant) with predisposition to recurrent infections.

Management

Respiratory support included mechanical ventilation with lung-protective strategies, surfactant replacement, and bronchodilator therapy. Cardiovascular management required inotropic support, antiarrhythmic therapy with amiodarone, beta-blocker therapy with propranolol, and pulmonary vasodilator therapy with sildenafil. Antimicrobial management consisted of empirical broad-spectrum antibiotics followed by targeted therapy. Neurological management included anticonvulsant therapy with phenobarbital and continuous EEG monitoring. Nutritional support involved parenteral nutrition during acute phases, followed by gradual advancement to enteral feeds.

Outcome and Follow-up

The infant was discharged home at 44 weeks corrected gestational age weighing 3.2 kg in stable condition. Ongoing medications included propranolol for cardiac management and iron supplementation. All acute complications resolved, with plans for long-term neurodevelopmental, endocrine, and immunologic follow-up.

At 6 months corrected age follow-up, the infant demonstrated age-appropriate developmental milestones including social smiling, head control, and visual tracking. Two-dimensional echocardiography showed complete resolution of patent foramen ovale with normal left ventricular function (LVEF 65%) and no residual hypertrophy. Electrocardiography revealed normal sinus rhythm with age-appropriate intervals and no conduction abnormalities. Growth parameters showed steady catch-up growth with weight at 25th percentile for corrected age. Neurological examination was normal with appropriate muscle tone and reflexes. Ophthalmologic examination showed complete regression of retinopathy of prematurity with normal retinal vascularization.

Growth hormone levels and thyroid function were found to be normal by endocrine assessment. Immunologic assessment showed normal immunoglobulin levels with no evidence of recurrent infections.

Discussion

The complicated treatment issues that arise when treating extremely preterm newborns with various organ failure brought on by maternal preeclampsia are demonstrated by this instance. The constellation of medical complications demonstrates the heightened vulnerability of extremely preterm neonates to systemic involvement across multiple organ systems [5]

Pulmonary Management: Respiratory distress syndrome occurs in virtually all neonates delivered before 32 weeks gestation[6] Persistent pulmonary hypertension development necessitated sildenafil intervention[7], while subsequent bronchopulmonary dysplasia required judicious corticosteroid therapy[8,9] Contemporary BPD management approaches have undergone significant evolution, with dexamethasone timing and duration requiring careful risk-benefit evaluation[10]

Cardiac Manifestations: Hemodynamic compromise in extremely preterm neonates represents complex cardiovascular transitional difficulties[11] The documented patent foramen ovale with concentric left ventricular hypertrophy may reflect adaptive mechanisms to chronic illness, persistent pulmonary hypertension, or underlying genetic predisposition [12]

Supraventricular tachycardia in preterm neonates is a rare but potentially life-threatening arrhythmia, occurring in approximately 0.1-0.4% of NICU admissions[13] The presentation in this case was particularly challenging, with failure of initial adenosine therapy necessitating alternative management strategies. The persistence of tachyarrhythmia despite adequate adenosine dosing (0.1-0.2 mg/kg) may be attributed to several factors including altered pharmacokinetics in preterm infants, concurrent use of methylxanthines (theophylline), reduced adenosine receptor sensitivity, or underlying myocardial dysfunction [14]

The pathophysiology of SVT in extremely preterm infants differs from term neonates, with immature conduction systems, altered autonomic innervation, and increased susceptibility to electrolyte disturbances [15] Precipitating factors in this case included hypokalemia and hypomagnesemia, which can alter cardiac action potentials and facilitate re-entrant circuits. The associated metabolic acidosis and elevated lactate levels indicated significant hemodynamic compromise requiring urgent intervention, as prolonged SVT can lead to cardiac dysfunction and cardiogenic shock in neonates [16]

Amiodarone, while not first-line therapy in neonates due to potential thyroid, pulmonary, and hepatic toxicity, proved effective in rhythm conversion when adenosine failed[17] The choice of amiodarone over other antiarrhythmics like propranolol or digoxin was justified by the hemodynamic instability and need for rapid rhythm control. Electrical cardioversion was not attempted in this case due to multiple factors specific to extremely preterm neonates: technical challenges associated with very low birth weight (appropriate paddle size selection and energy delivery calculations), relative hemodynamic stability allowing time for pharmacological intervention, and significant anesthetic risks in a critically ill preterm infant with multi-organ dysfunction[17] The narrow QRS morphology and typical SVT presentation also suggested high likelihood of pharmacological termination success.

The loading dose strategy (5 mg/kg over 1 hour) followed by maintenance infusion represents current evidence-based practice for neonatal SVT management

when adenosine fails[18] Cardioversion in neonates requires specialized pediatric equipment, experienced personnel, and careful energy dosing (0.5-1 J/kg initially), making pharmacological conversion the preferred first-line approach when hemodynamically tolerated[19] The successful management without recurrence suggests that the arrhythmia may have been triggered by transient factors including electrolyte imbalances, hypoxia, inflammatory mediators, or catecholamine administration rather than structural cardiac abnormalities or accessory pathways[20]

Infectious Disease: Both early and late-onset sepsis complicated the clinical course[14] The emergence of Pseudomonas aeruginosa highlights antimicrobial resistance challenges in NICU settings[15] Progression to septic shock required aggressive hemodynamic support and targeted antibiotic therapy[16]

Neurological Considerations: Neonatal seizures carry significant prognostic implications and require continuous EEG monitoring for accurate diagnosis[17] Phenobarbital remains first-line therapy despite concerns regarding neurodevelopmental effects[18] Normal EEG findings were reassuring, though the presence of subcortical leukomalacia with hypoxic-ischemic encephalopathy on neurosonography indicates significant white matter injury requiring long-term surveillance[19] The genetic variants identified, including those associated with pituitary hormone deficiency and autoimmune conditions, may contribute to the complex clinical phenotype and require ongoing endocrine and immunologic monitoring[20]

Gastrointestinal and Surgical Issues: Conservative management of necrotizing enterocolitis avoided surgical intervention[21] Successful left herniotomy for inguinal hernia repair demonstrates the feasibility of surgical interventions when indicated in extremely preterm infants[22] Acute kidney injury, common in critically ill preterm infants, required careful fluid and diuretic management[23] Nutritional support balance between adequate growth and gastrointestinal tolerance remains challenging[24]

Long-term Implications and Follow-up: Despite favorable short-term outcomes, 40-50% of extremely preterm survivors experience significant neurodevelopmental impairments[25] The encouraging 6-month follow-up findings, including normal cardiac function, appropriate developmental milestones, and absence of recurrent infections, suggest a favorable trajectory[26] However, the presence of subcortical leukomalacia and genetic variants affecting multiple systems necessitates continued comprehensive follow-up including neurodevelopmental, endocrine, and immunologic surveillance[27] Complete regression of retinopathy of prematurity was reassuring, though continued ophthalmologic monitoring remains important[28] Systematic follow-up programs are crucial for early identification of potential developmental delays, particularly given the initial white matter changes[29]

Clinical Significance: This case supports continued aggressive intervention in extremely preterm infants while acknowledging substantial resource requirements. The evolution of neonatal intensive care, including newer therapies and improved ventilation strategies, has contributed to improved survival rates. However, the complexity of care underscores the importance of regionalized perinatal care systems and multidisciplinary expertise including genetics and specialized surgical services when indicated.

Conclusion

This case illustrates that extremely preterm neonates with severe multiple organ dysfunction can achieve favorable short-term outcomes through comprehensive, evidence-

based intensive care management. Successful treatment required prompt complication recognition, immediate therapeutic interventions, and coordinated multidisciplinary care involving neonatology, cardiology, neurology, genetics, and pediatric surgery subspecialties.

Key factors contributing to the positive outcome included immediate advanced neonatal life support preventing severe hypoxic injury, aggressive hemodynamic management maintaining organ perfusion, prompt antimicrobial therapy preventing irreversible septic shock, and comprehensive monitoring minimizing secondary organ damage. The case highlights the importance of newer therapeutic modalities including sildenafil for persistent pulmonary hypertension and structured bronchopulmonary dysplasia management approaches.

However, the favorable short-term outcome must be interpreted cautiously. Long-term neurodevelopmental outcomes in extremely preterm infants remain concerning, with 40-50% experiencing major developmental impairments. The encouraging 6-month follow-up with normal developmental milestones and cardiac function resolution provides hope, but continued vigilance is essential. The presence of multiple risk factors including initial seizures, prolonged mechanical ventilation, sepsis, white matter injury, and genetic variants necessitates particularly close developmental surveillance through systematic follow-up programs.

This case emphasizes the critical importance of family-centered care and communication throughout intensive care periods. The substantial resources required for such complex care highlight the importance of evidence-based resource allocation and regionalized care systems. While prognosis for extremely preterm infants with multi-organ dysfunction remains guarded, this case provides evidence that aggressive intervention can result in survival with resolution of acute complications when supported by advanced technology, evidence-based medicine, skilled healthcare teams, and comprehensive family support systems.

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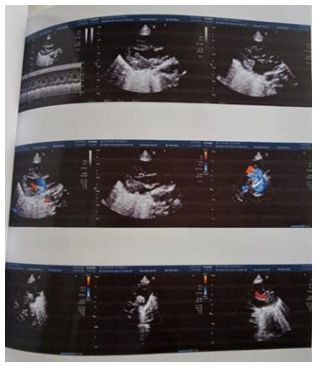
Legend Figures

Figure 1:

Figure 2:



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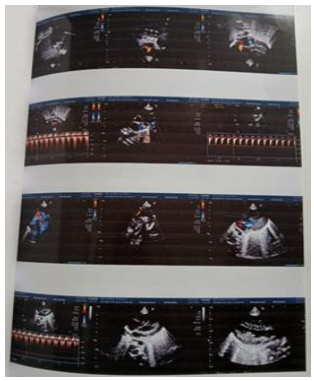
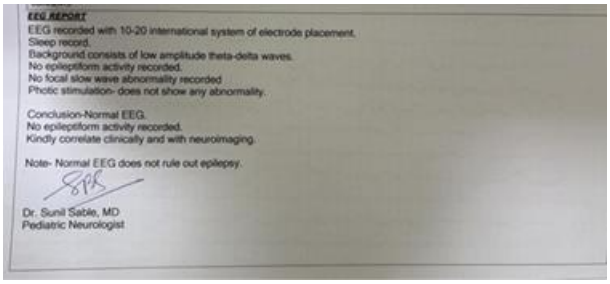


Figure 4:


EEG REPORT
EEG recorded with 10-20 international system of electrode placement.
Sleep record.
Background consists of low amplitude theta-delta waves.
No epileptiform activity recorded.
No focal slow wave abnormality recorded
Photic stimulation- does not show any abnormality.

Conclusion-Normal EEG.
No epileptiform activity recorded.
Kindly correlate clinically and with neuroimaging.

Note- Normal EEG does not rule out epilepsy.

Dr. Sunil Sable, MD
Pediatric Neurologist

Figure 5:

Figure 6:

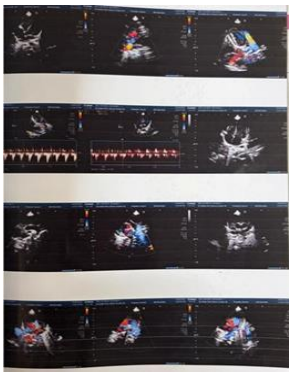


Figure 7:

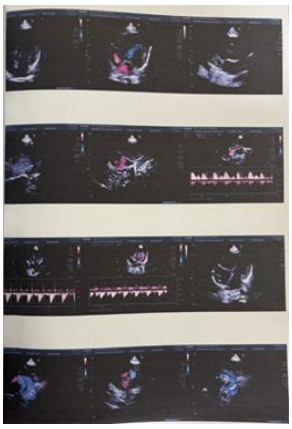


Figure 8: