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Pediatric Sepsis in Congenital Hypertrophic Pylorus Stenosis Patient: A Case Report

Alegra Rifani Masharto[1*], Arie Utariani[2], Kohar Hari Santoso[2]

[1]Fellowship Program of Pediatric Anesthesia Department, Airlangga University, Surabaya, Indonesia

[2]Anesthesiology and Reanimation Department, Airlangga University, Surabaya, Indonesia

*Corresponding author details: Alegra Rifani Masharto; alegrarifani@gmail.com

ABSTRACT

Sepsis remains a leading cause of morbidity and mortality in children worldwide. Hypertrophic pyloric stenosis (HPS) is a common cause of gastric outlet obstruction in infants that can cause sepsis. Characterized by progressive thickening of the pyloric muscle, it typically presents in the first few weeks of life with non-bilious projectile vomiting, dehydration, and weight loss. Sepsis may develop secondary to severe dehydration, metabolic alkalosis, delayed diagnosis, or underlying infection. This case report article reviews treatment strategies for sepsis in pyloric stenosis pediatric-specific considerations.

Keywords: pediatric sepsis; hypertrophic pylorus stenosis; pediatric anesthesia.

INTRODUCTION

Sepsis is responsible for nearly 3 million deaths annually, with the highest burden in low to middle-income countries. Neonates and infants under one year are at the highest risk. Risk factors include prematurity, immunodeficiency, or congenital diseases.

Hypertrophic pyloric stenosis (HPS) is a condition involving hypertrophy of the pyloric muscle leading to gastric outlet obstruction. It is most commonly diagnosed in infants between 2 and 8 weeks of age and represents one of the most frequent indications for surgery in early infancy. This illness affects approximately 2 to 5 per 1,000 live births with a male-to-female ratio of approximately 4:1. First-born children are at higher risk, and there may be a familial predisposition.

Characterized by progressive thickening of the pyloric muscle, it typically presents with non-bilious projectile vomiting, dehydration, and weight loss. It is typically a benign and treatable cause of vomiting in infants; complications such as sepsis are rare but potentially life-threatening. Although HPS is not an infectious process, several mechanisms can contribute to the development of sepsis, such as severe dehydration and hypovolemia reduce perfusion to vital organs, prolonged vomiting, and hospitalization without definitive management may increase the risk of nosocomial infections, and inadequate nutritional support weakens the immune response. Delayed diagnosis allows for progressive physiological deterioration, sometimes masking early signs of infection.

CASE REPORT

A 3-month-old baby boy, weighing 3 kilograms, was diagnosed with pylorus stenosis. The patient was found with non-bilious projectile vomiting more than 3 times a day, severe weight loss, and severe dehydration with symptoms of fever in the last 14 days reaching 39 °C.

The patient's hemodynamics were unstable in the emergency department. Airway was clear with respiratory rate 26 times/minute, oxygen saturation 99%, and heart rate 163 beats/minute. The patient also did not have urine output for the last 24 hours. The patient was given Ringer's lactate 20 ml/bw as resuscitation. The laboratory findings were unfortunately abnormal, with anemia, leukocytosis, thrombocytopenia, and hypoalbuminemia. Liver and renal function tests also worsen with electrolyte imbalance, with severe low potassium and hyponatremia. The patient's phoenix score was 1.

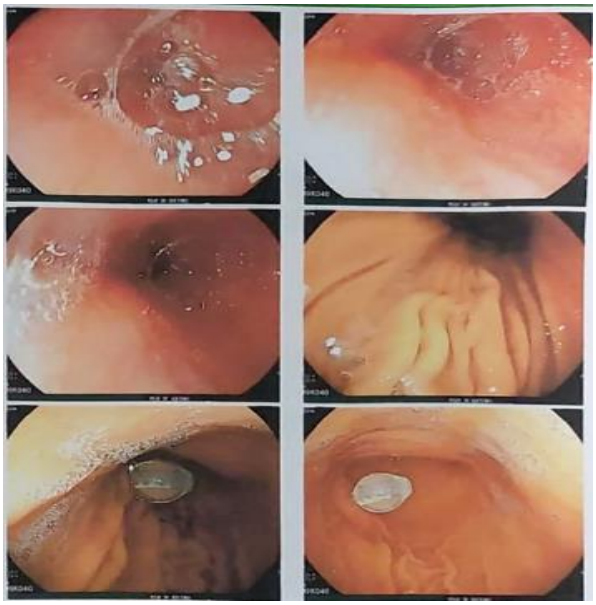

FIGURE 1: Endoscopic diagnostic findings.

The patient was then scheduled to have pyloromyotomy after being stabilized first, then going to the pediatric intensive care unit (PICU) after surgery for intensive care treatment. Patient was premedicated with midazolam 0.05 mg/bw. Propofol 1 mg/bw was used as an induction agent with fentanyl 2 mcg/bw as an analgetic. The muscle relaxant was atracurium 0.5 mg/bw with sevoflurane as an inhalation maintenance agent.

Surgery was done in 1 hour with a stable hemodynamic state. Bleeding was 10 ml, and urine output was within normal limits. Patient was given Ringer's lactate as maintenance fluid 4 ml/bw/hour. With postoperative analgetic, metamizole was administered 3 times/day based on body weight. Antibiotics were given ceftriaxone 2 times/day based on body weight and bacterial culture. But, due to the late treatment of pylorus stenosis, the patient underwent tracheostomy because of prolonged intubation.

DISCUSSION

Sepsis as a complication of hypertrophic pyloric stenosis is infrequent but clinically significant. While HPS is generally recognized and treated effectively with surgical intervention, delays in diagnosis or inadequate preoperative management may lead to systemic complications, including sepsis. The pathophysiological link lies in the profound dehydration and metabolic derangements seen in prolonged cases. Severe hypovolemia can result in impaired perfusion of the gastrointestinal tract, potentially facilitating bacterial translocation and systemic infection.

In this context, early clinical signs of sepsis, such as fever, tachycardia disproportionate to dehydration, and altered mental status, may be subtle or masked by the symptoms of HPS itself. This presents a diagnostic challenge, as HPS typically does not involve fever, and the presence of such symptoms should prompt clinicians to consider coexisting or secondary infection.

Management strategies must focus on stabilization before surgical correction. Fluid resuscitation and correction of metabolic alkalosis are standard in HPS, but the addition of empiric broad-spectrum antibiotics is warranted when sepsis is suspected. Hemodynamic monitoring and careful timing of surgery are critical; operating on a child with uncompensated sepsis increases the risk of perioperative complications, including shock and poor wound healing.

The presence of sepsis in HPS highlights the importance of maintaining a high index of suspicion, especially in infants presenting with prolonged symptoms or signs of systemic compromise. Multidisciplinary care involving pediatricians, surgeons, and intensivists ensures optimal outcomes. With early recognition and proper management, the prognosis remains favorable. However, delays in diagnosis or treatment can lead to prolonged hospitalization, multiorgan dysfunction, or even death.

CONCLUSIONS

Although pyloric stenosis is a common and treatable cause of vomiting in early infancy, sepsis is an uncommon complication of pyloric stenosis. That is why clinicians must be vigilant for signs of systemic infection, especially in cases with delayed diagnosis or poor hydration status. Prompt resuscitation, targeted antibiotics, and careful surgical timing are essential to improve outcomes. Awareness of early signs, adherence to resuscitation protocols, and ongoing evaluation of response to treatment are essential for reducing mortality and morbidity in pediatric-specific strategies must be emphasized in training and clinical practice. In this case report, we can see that sepsis can be treated in pylorus stenosis with critical care treatment and early recognition of pediatric sepsis.

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