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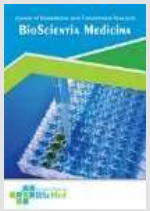

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Challenges in the Management of Hypertrophic Pyloric Stenosis in a Malnourished Infant with Pulmonary Tuberculosis: A Case Report

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A B S T R A C T

Background: Hypertrophic pyloric stenosis (HPS) is a common cause of gastric outlet obstruction in infants, but its management can be challenging in the presence of comorbidities like malnutrition and pulmonary tuberculosis (PTB). This case report highlights the complexities and considerations in managing an infant with HPS, PTB, and severe malnutrition. **Case presentation:** A 6-month-old male infant presented with lethargy, recurrent vomiting, and failure to thrive. He had a history of PTB on anti-tuberculosis treatment and was severely malnourished. Investigations revealed HPS, and he underwent a pyloromyotomy. Postoperatively, he required careful fluid management, nutritional support, and continued anti-tuberculosis therapy. **Conclusion:** This case highlights the challenges in managing HPS in an infant with PTB and severe malnutrition. It underscores the importance of a multidisciplinary approach involving pediatricians, surgeons, and nutritionists to ensure optimal outcomes in such complex cases.

1. Introduction

Hypertrophic pyloric stenosis (HPS) is a common cause of gastric outlet obstruction in infants, characterized by hypertrophy of the pyloric muscle, leading to the narrowing of the pyloric canal. This condition typically presents between the second and sixth week of life, with a higher incidence in males and first-born children. The incidence of HPS varies geographically, ranging from 2 to 4 per 1,000 live births annually. The etiology of HPS is multifactorial, with genetic and environmental factors playing a role. Genetic factors include a family history of HPS, while environmental factors include exposure to macrolide antibiotics, young maternal age, and infant feeding practices. The pathophysiology of HPS involves hypertrophy and hyperplasia of the circular and longitudinal muscles of the pylorus, leading to progressive narrowing of the pyloric canal.[1-4]

The clinical presentation of HPS typically includes non-bilious projectile vomiting, which occurs shortly after feeding. Other symptoms may include weight loss, dehydration, and constipation. The diagnosis of HPS is made based on clinical findings and imaging studies, such as ultrasound or upper gastrointestinal series. Ultrasonography is the gold standard for diagnosing HPS, with a sensitivity and specificity of

95% and 100%, respectively. The diagnostic criteria for HPS on ultrasound include a pyloric muscle thickness greater than or equal to 3 mm and a pyloric canal length greater than or equal to 15 mm.[5-7]

The management of HPS can be particularly challenging in the presence of comorbidities such as malnutrition and pulmonary tuberculosis (PTB). Malnutrition, a state of inadequate nutrition, can compromise the infant's growth and development, while PTB, an infectious disease caused by the bacterium Mycobacterium tuberculosis, can further exacerbate the infant's health status. This case report presents the complexities and considerations in managing an infant with HPS, PTB, and severe malnutrition.[8-10] The report aims to highlight the importance of a multidisciplinary approach involving pediatricians, surgeons, and nutritionists to ensure optimal outcomes in such complex cases.

2. Case Presentation

The patient, a 6-month-old male infant, presented with a history of feeding difficulties, frequent vomiting, and poor weight gain. These symptoms had been present for five months prior to admission, with the vomiting frequency increasing to 5-7 times per day and each vomit volume ranging from 3 to 5 cc. Despite these issues, the patient's weight had increased by 0.5 kg in one month. Four months prior to admission, the patient's parents noted he appeared skinnier and had a recurrent fever. He was diagnosed with pulmonary tuberculosis (PTB) at a district hospital due to contact with a neighbor with active TB. A Mantoux test was positive, and a chest x-ray showed perihilar thickening. He was started on anti-tuberculosis medication. Three months prior to admission, the patient's condition remained stable, with ongoing treatment for PTB. However, two weeks before admission, his vomiting became more frequent. One day before admission, he presented with lethargy, repeated vomiting, weakness, and cold hands and feet. He had not had any oral intake for one day and had not urinated in the past 5 hours. A referral letter from a pediatric clinic listed suspected diagnoses of GERD and pulmonary TB. The patient was taking anti-tuberculosis medication (continuation phase), domperidone, and vitamin B6. Upon examination, the patient was severely ill and somnolent, with a Glasgow Coma Scale (GCS) score of E3M6V5. His blood pressure was 60/30 mmHg, his pulse was 146 times/minute, and his respiratory rate was 35 times/minute. His temperature was 36.2°C, and his oxygen saturation (SpO_2) was 98%. His skin was cold and pale, with thin subcutaneous fat. Multiple lymph nodes were palpable. The patient also exhibited microcephaly and had a sunken fontanelle and eyes, with fewer tears. His mouth and lips were dry. Examination of his neck, chest, and heart revealed no significant abnormalities. His abdomen was not distended, and his liver was just palpable, with slow skin turgor. His extremities were cold, with a capillary refill time (CRT) of 4 seconds. Upon admission on May 8th, 2023, the patient remained severely ill and somnolent (GCS E3M6V5). A chest x-ray was suggestive of pulmonary TB, and an abdomen x-ray showed a 'single bubble' or 'caterpillar sign'. Blood tests revealed a hemoglobin level of 11.7 g/dL, leukocytes 8760/mm^3, hematocrit 32%, platelets 518,000/mm^3, and reticulocytes 3.69%. His serum potassium level was 2.4 mmol/L. The patient was diagnosed with shock hypovolemic ec repeated vomitus ec suspected hypertrophic pyloric stenosis. On May 9th-10th, 2023, the patient continued to experience nausea and vomiting with nasogastric tube (NGT) or oral intake. He remained moderately ill, with a blood pressure of 96/53 mmHg, a heart rate of 90 bpm, and a respiratory rate of 30 breaths/minute. His temperature was 36.9°C, and his SpO_2 was 99% on room air. His abdomen was not distended, and he had thin subcutaneous fat but good skin turgor. An abdominal ultrasound showed pyloric thickness with an antral nipple sign, a pylorus length of 24.3 mm, a single muscle thickness of 8 mm, and a pyloric thickness of 18.7 mm (Table 1).

On May 8th, 2023, in the Emergency Room (ER), the patient was administered oxygen at 1 liter per minute (LPM) and received a 5% Ringer's Lactate

Glucose (RLG) solution at 15 cc/kg body weight (57 cc) within 1 hour. This was done to evaluate hemodynamic stability after fluid therapy and hematological examinations were conducted, including a complete blood count, electrolyte panel, and arterial blood gas analysis. A nasogastric tube was inserted, and urinary catheterization and monitoring were initiated. The medical team prepared for potential Pediatric Intensive Care Unit (PICU) admission, but the parents refused due to the inability to accompany the patient. The parents were informed about the patient's condition. Later on May 8th, 2023, after the patient was transferred to the ward, Total Parenteral Nutrition (TPN) with D10% and electrolytes (maintenance) was initiated. The patient also received Aminoinfant at 0.5 g/kg body weight/day, folic acid 1x1 mg orally (PO), zinc 1x10 mg PO, Isoniazid (INH) 1x40 mg PO, Rifampicin 1x60 mg PO, and B6 1x1 tablet PO. A consultation with pediatric surgery was requested. From May 9th to 10th, 2023, TPN, medications, and supportive care were continued, and an abdominal ultrasound was performed. On May 11th and 12th, 2023, the same treatment and care were continued, and a pyloromyotomy was scheduled. Post-operatively, on May 13th, 2023, the patient received IVFD D10% with electrolytes and transitioned to NGT feeding after regaining consciousness. Medications included Ampicillin 4x200 mg intravenously (IV), Gentamicin 2x10 mg IV, Paracetamol 4x50 mg IV, and SF 10-30 cc/NGT (as tolerated). Medications and supportive care were continued, and a gradual increase in intake was planned. On May 14th and 15th, 2023, the patient received SF Gain 100 at 4x50 cc and 4x60 cc/NGT (as tolerated), and medications and supportive care continued. Discharge planning was initiated, and follow-up appointments were scheduled at various pediatric specialty clinics, including the Pediatric Gastrohepatology Clinic, Pediatric Surgery Clinic, Nutrition and Metabolic Disease Clinic, Pediatric Respiratory Clinic, and Pediatric Growth and Development Clinic (Table 2).

3. Discussion

Hypertrophic pyloric stenosis (HPS) is a fascinating yet perplexing condition that has intrigued medical professionals for centuries. Despite extensive research, the exact cause of this infantile disorder remains elusive, shrouded in a complex interplay of genetic predisposition, environmental influences, and intricate cellular mechanisms. Let's delve deeper into the current understanding of the etiology and pathophysiology of HPS, unraveling the intricate web of factors that contribute to its development. The role of genetics in HPS is undeniable. Studies have consistently shown a higher incidence of HPS in infants with a family history of the condition. This familial clustering suggests a strong genetic component, with multiple genes likely involved in its pathogenesis. While specific genes have been implicated, the exact mode of inheritance remains unclear. The risk of HPS is significantly higher if a sibling or parent has had the condition. The risk is even greater if the affected family member is male. This observation suggests a possible multifactorial inheritance pattern, where multiple genes and environmental factors interact to trigger the disease. Several genes have been investigated for their potential role in HPS. These include genes involved in muscle development, nerve signaling, and hormone regulation. However, no single gene has been definitively identified as the primary cause of HPS. Research suggests that variations in certain genes may increase susceptibility to HPS. These variations may affect the structure or function of proteins involved in muscle growth and development, potentially contributing to the abnormal thickening of the pyloric muscle. While genetics lays the foundation for susceptibility to HPS, environmental factors appear to play a crucial role in triggering its development. These factors may interact with genetic predispositions, influencing the timing and severity of the condition. Exposure to macrolide antibiotics, particularly erythromycin, in early infancy, has been associated with an increased risk of HPS.

Table 1. Timeline of the disease, including anamnesis, clinical findings, laboratory and imaging findings, and the diagnosis.

Timeline	Anamnesis	Clinical finding	Laboratory and Imaging Finding	Diagnosis
5 months before admission	Difficulties feeding, frequent vomiting (5-7 times/day), vomit volume 3-5 cc, decreased intake, weight gain of 0.5 kg in one month	-	-	-
4 months before admission	Skinnier appearance, recurrent fever, not as tall as other babies the same age, diagnosed with lung TB in district hospital due to contact with a neighbor with active TB	-	Positive Mantoux test, thickened perihilar in chest x-ray	Pulmonary TB
3 months before admission	Treated with anti-TB medication [intensified phase]	-	-	Pulmonary TB (on treatment)
2 weeks before admission	Vomiting becomes more frequent	-	-	-
1 day before admission	Lethargic, repeated vomiting, weakness, feeling cold, no oral intake for 1 day, cold hands and feet, last urination 5 hours prior, suspected GERD and pulmonary TB (referral letter), taking ATD (continuation phase), Domperidone, and Vitamin B6	Severely ill, somnolent (GCS E3M6V5), BP 60/30 mmHg, pulse 146 times/minute, weak and feeble pulse, respiratory rate 35 times/minute, temperature 36.2°C, SpO_2 98%, cold and pale skin, thin subcutaneous fat, multiple palpable lymph nodes, microcephaly, sunken fontanelle, sunken eyes, fewer tears, dry mucosa of mouth and lips, no neck stiffness, JVP 5-2 cmH_2O, normal chest shape, prominent ribs, symmetrical chest movement, vesicular breathing sounds, no rhonchi or wheezing, ictus cordis visible and palpable, heart sounds regular with no murmur, no abdominal distension, slow skin turgor, liver just palpable, spleen not palpable, no rebound tenderness, extremities cold with CRT 4 seconds, no rash or edema	-	Shock hypovolemic ec repeated vomitus ec suspected Hypertrophic Pyloric Stenosis
At admission (May 8th, 2023)	-	Severely ill, somnolent (GCS E3M6V5)	Chest x-ray suggestive of pulmonary TB, abdomen x-ray shows 'single bubble' or 'caterpillar sign'; Hb 11.7 g/dL, leukocytes 8760/mm^3, Hct 32%, platelets 518,000/mm^3, reticulocytes 3.69%, DC 0/1/0/40/55/4, PT 11.8 seconds, APTT 24.1 seconds, albumin 3.4 g/dL, total bilirubin 0.4 mg/dL, direct bilirubin 0.2 mg/dL, indirect bilirubin 0.2 mg/dL, AST 32 U/L, ALT 35 U/L, urea 11 mg/dL, creatinine 0.3 mg/dL, blood glucose 93 mg/dL, sodium 134 mmol/L, potassium 2.4 mmol/L, chloride 92 mmol/L	Shock Hypovolemic ec repeated vomitus ec suspected Hypertrophic Pyloric Stenosis, Suspected Hypertrophic Pyloric Stenosis (HPS), Malnourished marasmic type I with shock, Pulmonary TB maintenance phase, Hypokalemia, Incomplete immunization, Failure to Thrive
May 9th-10th, 2023	Nausea and vomiting with NGT or oral intake, no cough or fever, still fasting	Moderately ill, BP 96/53 mmHg, HR 90 bpm, RR 30 breaths/minute, temperature 36.9°C, SpO_2 99% on room air, no abdominal distension, thin subcutaneous fat, good skin turgor, no rebound tenderness, warm extremities with good perfusion, no edema	Sodium 134 mmol/L, potassium 4.0 mmol/L, chloride 98 mmol/L; Abdominal ultrasound shows pyloric thickness with antral nipple sign, pylorus length 24.3 mm, single muscle thickness 8 mm, pyloric thickness 18.7 mm	Shock hypovolemic ec repeated vomitus (history) ec Hypertrophic Pyloric Stenosis (HPS), Malnourished marasmic type III, Pulmonary TB maintenance phase, Hypokalemia (history), Incomplete immunization, Failure to Thrive

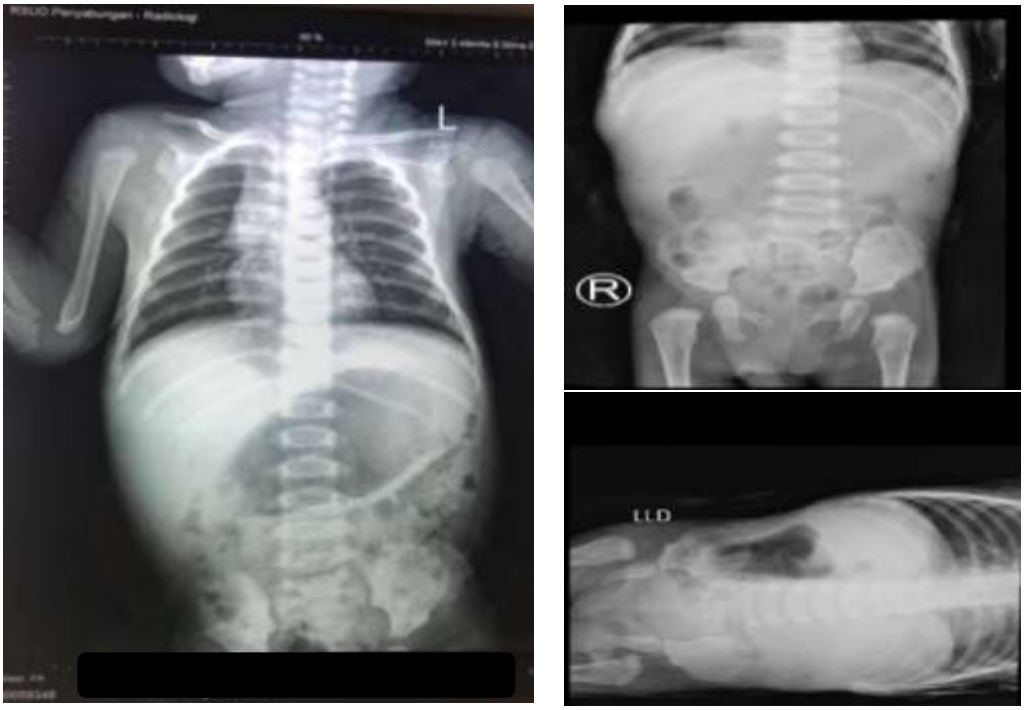

Figure 1. Chest X-ray and abdomen X-ray (May 8th,2023). Chest X-ray: Suggestive to pulmonal tuberculosis. Abdominal X-ray: On plain abdominal image appears gastric dilatation accompanied by widening of the notch provides a 'single bubble' or 'caterpillar sign.

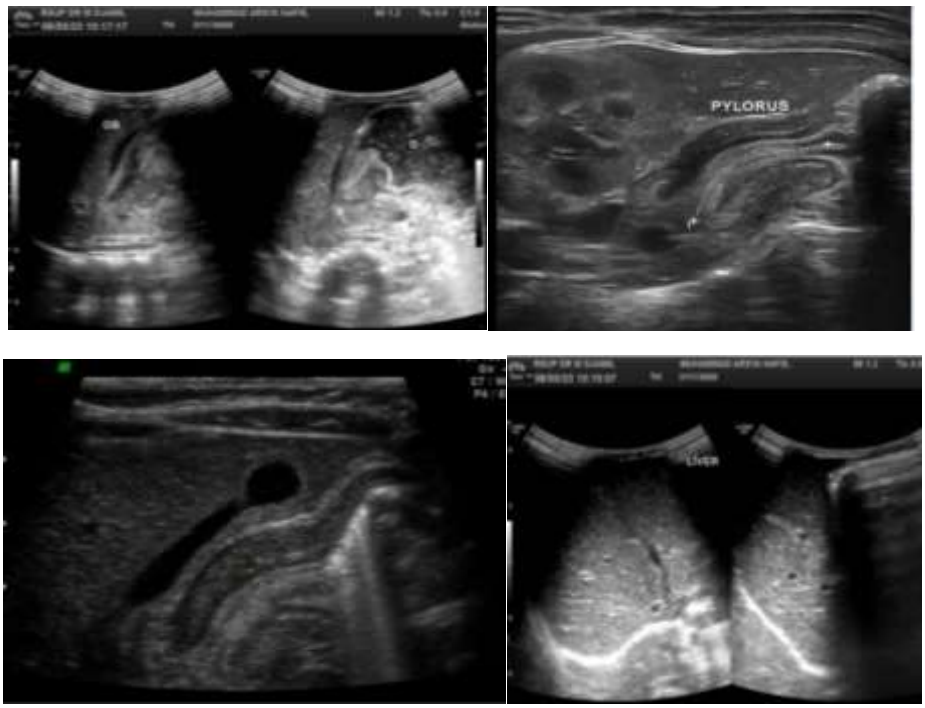

Figure 2. Abdominal Ultrasound (USG) May 10th 2023. Pyloric thickness with antral nipple sign, pylorus length 24.3 mm, with single muscle thickness 8 mm and pyloric thickness 18.7 mm. Hepar, gald bladder, lien: shape and size normal. Pancreas and Aorta: Covered by a dilated gastric shadow. USG: Hypertrophic Pyloric Stenosis.

Table 2. Treatment and follow-up.

Timeline	Treatment	Follow-up
May 8th, 2023 (ER)	Oxygen 1 LPM; RLG 5% 15 cc/kg body weight (57 cc) within 1 hour	Evaluation of hemodynamic stability after fluid therapy; Hematological examination, including complete blood count and electrolyte panel, as well as arterial blood gas analysis; Insertion of nasogastric tube, urinary catheterization, and monitoring; Preparation for potential PICU admission; Informing parents about the patient's condition
May 8th, 2023 (Ward)	TPN D 10% + electrolytes (maintenance); Aminoinfant 0.5 g/kg body weight/day; Folic acid 1x1 mg PO; Zinc 1x10 mg PO; INH 1x40 mg PO; Rifampicin 1x60 mg PO; B6 1x1 tab PO	Consultation with pediatric surgery
May 9th-10th, 2023	Continued TPN, medications, and supportive care	Abdominal ultrasound
May 11th-12th, 2023	Continued TPN, medications, and supportive care	Scheduled pyloromyotomy
May 13th, 2023	Post-operative care: IVFD D10% + electrolytes (transition to NGT feeding after consciousness); Ampicillin 4x200 mg IV; Gentamicin 2x10 mg IV; Paracetamol 4x50 mg IV; SF 10-30 cc/NGT (as tolerated); Continued medications and supportive care	Gradual increase of intake
May 14th-15th, 2023	SF Gain 100 4x50 cc and 4x60 cc/NGT (as tolerated); Continued medications and supportive care	Discharge planning; Follow-up appointments scheduled at the Pediatric Gastrohepatology Clinic, Pediatric Surgery Clinic, Nutrition and Metabolic Disease Clinic, Pediatric Respiratory Clinic, and Pediatric Growth and Development Clinic

* ER: Emergency Room.

The mechanism behind this association is unclear, but it is hypothesized that these antibiotics may disrupt the normal development of the pyloric muscle. Some studies suggest that infant feeding practices, such as early introduction of formula feeding or bottle feeding, may increase the risk of HPS. However, the evidence is not conclusive, and further research is needed to clarify the role of feeding practices. Maternal factors, such as young maternal age, smoking during pregnancy, and certain maternal infections, have also been linked to an increased risk of HPS. These factors may influence the fetal environment and potentially disrupt the normal development of the pyloric muscle. At the cellular level, HPS is characterized by abnormal growth of the smooth muscle cells within the pylorus. This growth involves both hypertrophy (increase in cell size) and hyperplasia (increase in cell number), leading to a progressive thickening of the pyloric muscle and narrowing of the pyloric canal. The smooth muscle cells within the pylorus undergo significant changes in HPS. They become enlarged and increase in number, resulting in a thickened and elongated pyloric muscle. This excessive growth leads to a functional obstruction, preventing the passage of

food from the stomach into the small intestine. Research suggests a possible role of neurotransmitters, such as nitric oxide and vasoactive intestinal peptide, in the development of HPS. These neurotransmitters are involved in regulating muscle contraction and relaxation. Imbalances in their levels may contribute to the abnormal muscle growth seen in HPS. Hormones, such as gastrin and cholecystokinin, are also thought to play a role in the pathophysiology of HPS. These hormones stimulate gastric motility and secretion. Alterations in their levels or their receptors may contribute to the dysmotility and obstruction observed in HPS. Growth factors, such as epidermal growth factor and transforming growth factor-beta, are involved in regulating cell growth and differentiation. Dysregulation of these growth factors may contribute to the abnormal muscle growth seen in HPS. Hypertrophic pyloric stenosis (HPS) is a condition that typically manifests in the first few weeks of life, with the majority of cases presenting between 3 and 5 weeks of age. Recognizing the clinical signs of HPS is crucial for early diagnosis and prompt intervention, preventing potential complications and ensuring the infant's well-being. While the hallmark symptom of HPS is projectile vomiting, a constellation of other clinical manifestations may accompany this telltale sign. Projectile vomiting is the most striking and characteristic symptom of HPS. It is often described as forceful vomiting, with the vomitus projecting several feet away from the infant. This forceful expulsion is due to the increased pressure within the stomach as it contracts against the obstructed pyloric canal. The timing of vomiting is also noteworthy. Typically, projectile vomiting occurs shortly after feeding, as the stomach attempts to empty its contents but is met with resistance from the narrowed pylorus. The vomitus is characteristically non-bilious, meaning it does not contain bile. This is because the obstruction is located proximal to the ampulla of Vater, the point where bile enters the digestive tract. While projectile vomiting is a hallmark of HPS, it is important to note that not all infants with HPS will exhibit this classic presentation. Some infants may have less forceful vomiting or may only regurgitate small amounts of milk. Therefore, a careful evaluation of other clinical signs is essential to avoid misdiagnosis. Frequent vomiting can lead to significant fluid loss, resulting in dehydration. Dehydration is a serious condition that can have detrimental effects on an infant's health. Recognizing the signs of dehydration is crucial for prompt intervention and prevention of complications. Infants with dehydration produce less urine than usual. The urine may also be darker in color and have a stronger odor. The fontanelle, the soft spot on the top of an infant's head, may appear sunken or depressed in cases of dehydration. The mouth and tongue may appear dry, and the lips may be cracked. Dehydration can cause lethargy, decreased activity, and irritability. In severe cases of dehydration, the heart rate may increase, and the blood pressure may decrease. The inability to retain adequate nutrition due to persistent vomiting can hinder growth and development, leading to failure to thrive. Failure to thrive is a term used to describe inadequate weight gain or weight loss in infants and children. Infants with HPS may not gain weight appropriately or may even lose weight. The rate of growth may slow down, and the infant may fall behind on growth charts. In severe cases, failure to thrive can lead to developmental delays. Reduced passage of stomach contents into the intestines can result in decreased stool frequency and constipation. Constipation is a common symptom in infants with HPS, often accompanying vomiting and dehydration. The infant may have fewer bowel movements than usual. The stools may be small, hard, and difficult to pass. In some cases, visible peristaltic waves (wave-like muscle contractions) may be observed across the infant's abdomen as the stomach attempts to force contents through the narrowed pylorus. These waves are often described as moving from left to right across the upper abdomen. Visible peristaltic waves are not always present in infants with HPS, but when observed, they can be a strong indicator of gastric outlet obstruction. On physical examination, a firm, olive-shaped mass

may be palpable in the upper abdomen, representing the hypertrophied pylorus. This mass is typically located in the right upper quadrant of the abdomen, just below the liver edge. The olive-shaped mass is not always easily palpable, especially in infants who are crying or tense. However, when present, it is a highly suggestive sign of HPS. While the classic presentation of HPS is relatively straightforward, it is important to recognize that the clinical picture can vary among infants. Some infants may present with subtle or atypical symptoms, making diagnosis more challenging. Therefore, a high index of suspicion is crucial in any infant presenting with persistent vomiting, especially if accompanied by signs of dehydration, failure to thrive, or constipation. A thorough history and physical examination, coupled with appropriate imaging studies, are essential for accurate diagnosis and timely intervention. Hypertrophic pyloric stenosis (HPS) is a condition that often presents with characteristic clinical features, raising suspicion for the diagnosis. However, confirming the suspicion requires a systematic diagnostic approach that integrates information from the infant's history, physical examination, and imaging studies. A comprehensive evaluation is essential not only to establish the diagnosis but also to assess the severity of the condition and guide appropriate management. Obtaining a detailed history from the infant's caregivers is the cornerstone of the diagnostic process. The history should focus on eliciting information about the onset and characteristics of the infant's symptoms, feeding patterns, and growth history. The timing of vomiting in relation to feeding, the forcefulness of the vomiting episodes, and the appearance of the vomitus (bilious or non-bilious) are crucial details that can help differentiate HPS from other causes of vomiting. Inquiries about the infant's feeding habits, including the type of milk (breast milk or formula), frequency and volume of feeds, and any difficulties with feeding, can provide valuable insights. Assessing the infant's growth history, including birth weight, current weight, and any recent weight loss or failure to thrive, is essential. A family history of HPS or other gastrointestinal disorders can increase the index of suspicion for HPS. A thorough physical examination is an integral part of the diagnostic evaluation for HPS. The examination should focus on assessing the infant's hydration status, nutritional status, and abdominal findings. Signs of dehydration, such as decreased urine output, sunken fontanelle, dry mucous membranes, and lethargy, should be carefully assessed. The infant's weight, length, and head circumference should be measured and plotted on growth charts to assess for failure to thrive. The abdomen should be gently palpated to assess for distention, tenderness, and the presence of a palpable olive-shaped mass in the right upper quadrant. Visible peristaltic waves may also be observed in some cases. While the history and physical examination can provide strong clues for the diagnosis of HPS, imaging studies are essential for confirming the diagnosis and assessing the severity of the condition. Ultrasonography is the gold standard for diagnosing HPS. It is a non-invasive, readily available, and highly accurate imaging modality that allows for direct visualization of the pylorus. The thickness of the pyloric muscle is measured on ultrasound. A PMT of 3 mm or greater is considered diagnostic of HPS. The length of the pyloric canal is also measured on ultrasound. A PCL of 15 mm or greater is considered diagnostic of HPS. The PMR is calculated by dividing the PMT by the PCL. A PMR of 0.4 or greater is suggestive of HPS. In addition to confirming the diagnosis, ultrasonography can also provide information about the severity of the stenosis and guide treatment decisions. In some cases, an upper gastrointestinal series (UGI) may be performed as an alternative to ultrasonography. This involves giving the infant a contrast agent to drink and then taking X-rays to visualize the passage of the contrast through the digestive tract. The pyloric canal appears narrowed or elongated on the X-ray images. A thin stream of contrast may be seen passing through the narrowed pyloric canal, creating a characteristic "string sign." The stomach may retain the contrast agent for a

prolonged period, indicating delayed gastric emptying. While UGI can provide valuable information, it is generally considered less accurate than ultrasonography and is associated with radiation exposure. Therefore, ultrasonography is typically the preferred imaging modality for diagnosing HPS. The diagnosis of HPS is typically made based on a combination of clinical findings and imaging studies. A classic presentation of projectile vomiting, dehydration, and a palpable olive-shaped mass, coupled with characteristic ultrasound findings of a thickened and elongated pylorus, strongly supports the diagnosis of HPS. In cases where the clinical presentation is less clear-cut or ultrasound findings are equivocal, an UGI may be performed to further evaluate the pylorus and confirm the diagnosis. Hypertrophic pyloric stenosis (HPS) is a condition that, once definitively diagnosed, requires prompt and effective treatment to relieve the gastric outlet obstruction and restore normal digestive function. The cornerstone of HPS treatment is surgical intervention, specifically a procedure known as pyloromyotomy. However, the journey to successful treatment extends beyond the surgical procedure itself, encompassing crucial pre-operative and post-operative care to ensure the infant's well-being and optimize outcomes. Pyloromyotomy is a surgical procedure designed to alleviate the obstruction caused by the thickened pyloric muscle. The procedure involves making a small incision in the abdomen, either through an open or laparoscopic approach, and carefully cutting through the thickened muscle fibers of the pylorus. This effectively widens the pyloric canal, restoring the normal flow of stomach contents into the small intestine. Traditionally, pyloromyotomy was performed through an open incision, typically in the right upper quadrant of the abdomen. While effective, this approach is associated with a larger incision and potentially more discomfort for the infant. Laparoscopic pyloromyotomy has become increasingly popular in recent years. This minimally invasive approach utilizes small incisions and specialized instruments, resulting in less pain, faster recovery, and minimal scarring. The choice between open and laparoscopic pyloromyotomy depends on various factors, including the surgeon's experience, the infant's overall health, and the availability of resources. Before proceeding with pyloromyotomy, it is essential to stabilize the infant's condition and address any underlying imbalances. This pre-operative management is crucial to minimize surgical risks and ensure the infant's optimal health before surgery. Infants with HPS often present with dehydration and electrolyte imbalances due to persistent vomiting. Intravenous fluids are administered to correct these imbalances and restore hydration. Electrolyte levels, particularly potassium and chloride, are closely monitored and corrected as needed. Vomiting can also lead to metabolic alkalosis, a condition characterized by an increase in blood pH. Addressing this acid-base imbalance is important before surgery. In cases of severe malnutrition or failure to thrive, nutritional support may be initiated before surgery. This may involve providing intravenous nutrition or introducing small, frequent feeds through a nasogastric tube. Following pyloromyotomy, most infants recover quickly and can resume normal feeding within a few days. However, careful post-operative care is essential to ensure a smooth recovery and prevent complications. Adequate pain relief is crucial for the infant's comfort and to facilitate early feeding. Pain medications may be administered intravenously or orally, depending on the infant's needs. Continued monitoring of fluid and electrolyte balance is necessary to ensure the infant remains hydrated and electrolyte levels remain within normal limits. Feeding is typically started gradually, with small, frequent feeds, to allow the stomach to adjust. The volume and frequency of feeds are gradually increased as tolerated. Proper wound care is essential to prevent infection. The surgical incision is kept clean and dry, and any dressings are changed as needed. The infant is closely monitored for any signs of complications, such as wound infection, bleeding, or persistent vomiting. The prognosis for infants with HPS is excellent following pyloromyotomy. The

procedure is highly effective in relieving the obstruction and allowing for normal growth and development. Most infants experience complete resolution of their symptoms and go on to thrive. The presence of comorbidities, such as malnutrition and pulmonary tuberculosis (PTB), as seen in this case, can significantly complicate the management of HPS. These comorbidities can increase the risk of surgical complications, impair wound healing, and affect the infant's overall ability to tolerate the stress of surgery and anesthesia. Therefore, careful pre-operative assessment and optimization are crucial in these cases to minimize risks and ensure the best possible outcome.[11-16]

Pulmonary tuberculosis (PTB), a contagious disease caused by the bacterium Mycobacterium tuberculosis, remains a significant global health concern. Understanding the intricacies of its transmission and pathogenesis is crucial for effective prevention and control efforts. This detailed exploration delves into the mechanisms by which PTB spreads and develops, shedding light on the factors that influence susceptibility and disease progression. PTB is primarily transmitted through the air, making it a highly contagious disease. Individuals with active pulmonary TB, particularly those with untreated or poorly controlled disease, release microscopic droplets containing Mycobacterium tuberculosis bacteria when they cough, sneeze, speak, sing, or even laugh. These droplets, laden with infectious particles, can remain suspended in the air for extended periods, increasing the likelihood of inhalation by susceptible individuals. The higher the concentration of bacteria in the air, the greater the risk of transmission. Close proximity to an individual with active TB, especially in poorly ventilated or crowded spaces, increases the likelihood of inhaling infectious droplets. Prolonged exposure to an individual with active TB increases the risk of transmission. Individuals living in the same household or spending significant time in close contact with an infected person are at higher risk. The immune status of the individual plays a crucial role in determining susceptibility to PTB. Infants, young children, and individuals with weakened immune systems are more vulnerable to infection. Once inhaled, the microscopic droplets containing Mycobacterium tuberculosis bacteria travel through the respiratory tract, reaching the alveoli, the tiny air sacs in the lungs responsible for gas exchange. Here, the bacteria encounter the first line of defense of the immune system. In healthy individuals with robust immune systems, the initial immune response is typically effective in containing the infection. Macrophages, a type of white blood cell, engulf and attempt to destroy the bacteria. This process often leads to the formation of granulomas, small, organized collections of immune cells that wall off the bacteria, preventing further spread. In many cases, the immune system successfully contains the infection, but the bacteria are not completely eradicated. This results in latent tuberculosis infection (LTBI), a state where the bacteria remain dormant within the body, causing no symptoms and not being contagious. However, individuals with LTBI can reactivate the infection later in life, particularly if their immune system becomes weakened. In some individuals, particularly those with immature or compromised immune systems, the initial immune response is not sufficient to contain the infection. The bacteria can multiply and spread within the lungs, causing active tuberculosis disease. This can lead to a range of symptoms, including cough, fever, weight loss, and fatigue. In severe cases, the bacteria can disseminate through the bloodstream to other organs, causing extrapulmonary TB. This can affect various organs, including the lymph nodes, bones, kidneys, and brain. The strength of the immune system is a crucial determinant of disease progression. Infants, young children, and individuals with weakened immune systems, such as those with HIV infection or malnutrition, are more susceptible to developing active TB. The virulence of the Mycobacterium tuberculosis strain can also influence disease progression. Some strains are more aggressive and more likely to cause active disease. Genetic factors may also play a role in susceptibility to TB. Certain genetic variations may influence the immune

response to the bacteria, affecting the likelihood of developing active disease. Environmental factors, such as malnutrition, overcrowding, and poor sanitation, can increase the risk of TB transmission and disease progression. The pathogenesis of PTB is a complex interplay between the host's immune system and the Mycobacterium tuberculosis bacterium. The outcome of this interaction depends on a delicate balance between the virulence of the bacteria and the strength of the host's immune response. Pulmonary tuberculosis (PTB) in infants presents a unique diagnostic challenge due to the often subtle and nonspecific nature of its clinical manifestations. Unlike older children and adults, who may exhibit more classic symptoms like persistent cough, weight loss, and night sweats, infants with PTB often present with a constellation of vague symptoms that can mimic other common childhood illnesses. Recognizing these subtle clues is crucial for early diagnosis and prompt initiation of treatment, preventing potential complications and ensuring the best possible outcome for the infant. Fever is a frequent finding in infants with PTB, often presenting as a persistent or recurrent elevation in body temperature. However, fever is a nonspecific symptom that can be caused by a myriad of other conditions, including viral infections, urinary tract infections, and even teething. Therefore, the presence of fever alone is not sufficient to diagnose PTB. A persistent cough, which may be dry or productive, is another common symptom of PTB in infants. The cough may be accompanied by other respiratory symptoms, such as rapid breathing, wheezing, or grunting. However, these respiratory symptoms can also be seen in other respiratory infections, such as bronchiolitis or pneumonia. PTB can affect the infant's appetite and nutrient absorption, leading to poor weight gain or even weight loss. This can be a subtle but important clue, especially in infants who are otherwise healthy and have no other obvious cause for poor growth. In some cases, infants with PTB may exhibit signs of respiratory distress, such as rapid breathing, grunting, nasal flaring, or retractions (pulling in of the chest muscles during breathing). These signs indicate that the infant is having difficulty breathing and may require urgent medical attention. Infants with PTB may appear lethargic, irritable, or have decreased activity levels. These nonspecific symptoms can be easily overlooked, but they can be important indicators of underlying illness. Diagnosing PTB in infants can be particularly challenging due to the nonspecific nature of the symptoms and the difficulty in obtaining sputum samples for laboratory testing. Infants are often unable to produce sputum, and even if they can, the samples may be contaminated with saliva, making it difficult to isolate the TB bacteria. A thorough clinical evaluation, including a detailed history and physical examination, is the foundation of the diagnostic process. The history should focus on eliciting information about the infant's symptoms, exposure to individuals with active TB, and any other relevant medical history. A history of contact with individuals with active TB is a significant risk factor for PTB in infants. This includes household contacts, close relatives, or caregivers with active TB. Travel to areas with high TB prevalence can also increase the risk of infection. Infants who have not received the BCG vaccine, which provides some protection against TB, are at higher risk of developing the disease. A chest X-ray can reveal abnormalities suggestive of PTB, such as infiltrates (patches of increased density), consolidation (areas of lung tissue filled with fluid), or enlarged lymph nodes. However, chest X-ray findings in infants with PTB can be variable and may not always be definitive. The TST, also known as the Mantoux test, involves injecting a small amount of tuberculin purified protein derivative (PPD) under the skin. A positive reaction, indicated by induration (hardening) at the injection site after 48 to 72 hours, suggests exposure to TB bacteria. However, the TST may not be reliable in infants with weakened immune systems or those who have received the BCG vaccine. IGRAs are blood tests that measure the immune response to TB bacteria. They are more specific than the TST and can be helpful in diagnosing TB in infants, especially those with weakened immune

systems or those who have received the BCG vaccine. If possible, obtaining samples for microbiological confirmation is crucial for definitive diagnosis. This may involve gastric aspirates (samples of stomach contents), induced sputum (obtained by stimulating the infant to cough), or bronchoalveolar lavage (a procedure that involves washing the lungs with fluid and collecting the fluid for analysis). These samples are examined for the presence of TB bacteria using microscopy, culture, or molecular tests.[17-20]

4. Conclusion

This case report presents the challenges in managing a 6-month-old infant with hypertrophic pyloric stenosis (HPS), pulmonary tuberculosis (PTB), and severe malnutrition. The convergence of these conditions necessitates a multidisciplinary approach involving pediatricians, surgeons, and nutritionists. The infant's successful management involved initial stabilization, followed by a pyloromyotomy to address the HPS. Postoperatively, careful fluid management, nutritional support, and continued anti-tuberculosis therapy were crucial. This case underscores the importance of early diagnosis, prompt intervention, and comprehensive care in managing infants with complex medical conditions.

5. References

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