Section: General Surgery

Case Series

REVIEW OF CASES OF CONGENITAL HYPERTROPHIC PYLORIC STENOSIS IN ARUNACHAL PRADESH, INDIA: A CASE SERIES

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ABSTRACT

Congenital Hypertrophic Pyloric Stenosis (CHPS) is a common cause of gastric outlet obstruction in infants, classically presenting with projectile, non-bilious vomiting. Timely diagnosis and surgical management are essential for favorable outcomes.

We present a case series of six male infants with CHPS managed at a tertiary care hospital in Arunachal Pradesh. Clinical presentation, laboratory findings, imaging results, surgical management, and outcomes were analyzed.

All infants presented with projectile, non-bilious vomiting. Three had severe dehydration and electrolyte imbalance, while others had mild or no biochemical disturbances. A palpable pyloric mass was identified in most cases, though absent in one, where an upper GI contrast study confirmed the diagnosis after inconclusive ultrasonography. USG findings in other cases demonstrated pyloric muscle thickness between 5.6–7 mm and length 1.8–2.5 cm. All infants underwent open Ramstedt's pyloromyotomy, with uneventful postoperative recovery and favorable follow-up outcomes.

CHPS should be suspected in infants with persistent non-bilious vomiting. Early diagnosis using USG or contrast studies, correction of fluid-electrolyte imbalance, and timely pyloromyotomy result in excellent outcomes. Our case series reinforces the clinical spectrum of CHPS and the importance of prompt surgical management.

Keywords: Congenital hypertrophic pyloric stenosis, projectile vomiting, infant surgery, Ramstedt's pyloromyotomy, case series.

INTRODUCTION

Congenital Hypertrophic Pyloric Stenosis (CHPS), also known as Infantile Hypertrophic Pyloric Stenosis (IHPS), is the most common cause of gastric outlet obstruction in infants, with an incidence ranging from 1 to 4 per 1000 live births worldwide.[1] It typically presents between 2 and 8 weeks of life with projectile, non-bilious vomiting in previously healthy newborns. Progressive pyloric muscle hypertrophy leads to gastric outlet obstruction, resulting in dehydration and metabolic disturbances such as hypochloremic, hypokalemic metabolic alkalosis with paradoxical aciduria.[2]

Prior to recognition of this disease as an entity by Hirschsprung in 1888,[3] and description of pyloromyotomy by Ramstetd in 1911,[4] mortality rates exceeded 50 %.[5]

The onset of symptoms is usually abrupt and dramatic, presenting with non-bilious emesis resulting from hypertrophy and hyperplasia of the pylorus, usually between the second and eighth week of life.[6] A great amount of research has been conducted regarding this disease, but the exact etiology remains unknown. In 1961, the hypothesis of the multifactorial threshold model of inheritance was suggested.[7] In recent years, environmental factors have been associated with IHPS. Children from a smoking mother have a higher risk of IHPS.[8] Pesticides have also been reported as a potential cause of IHPS.[9] A combination of genetic and

environmental factors may contribute to the occurrence of IHPS.

Ultrasonography is the gold standard for diagnosis due to its high sensitivity and specificity. Ramstedt pyloromyotomy, first described over a century ago, remains the definitive surgical treatment with excellent long-term outcomes.[10]

This case series presents the clinical features, diagnostic approaches, and outcomes of infants with CHPS treated in Arunachal Pradesh, India.

MATERIALS AND METHODS

This is a retrospective case series of infants diagnosed with CHPS and treated at TRIHMS, Naharlagun, between January 2016 and December 2018.

Inclusion Criteria

Infants with confirmed diagnosis of CHPS who underwent surgery at TRIHMS during the study period.

Exclusion Criteria

Cases operated outside TRIHMS.

CHPS cases with associated congenital anomalies.

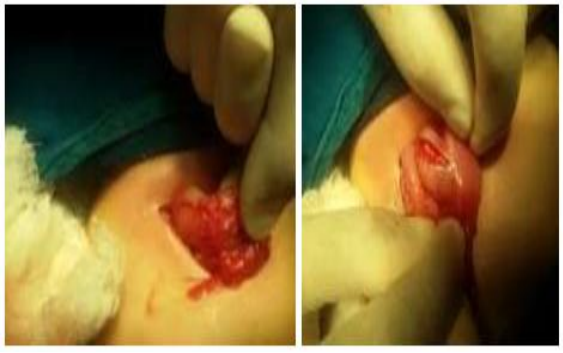
Data Collection

Patient demographics, clinical presentation, laboratory findings, imaging results, surgical procedure, and outcomes were extracted from medical records.

Case Presentations

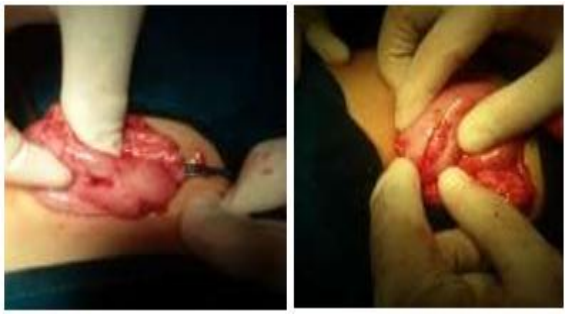
Case 1

A 1-month-old male infant presented with projectile, non-bilious vomiting for 3 weeks. Clinical examination revealed dehydration and a palpable pyloric mass. Biochemical tests showed electrolyte imbalance. Ultrasonography (USG) demonstrated a pyloric length of 2.5 cm and wall thickness of 7 mm. An open pyloromyotomy was performed with good postoperative recovery. The infant resumed feeds soon after surgery and was discharged early.



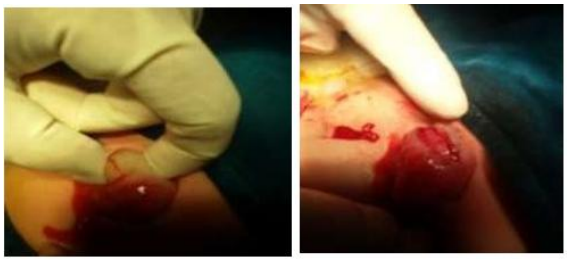
Case 2

A 3.5-month-old male infant presented with non-bilious vomiting since 2 weeks after birth. There was no dehydration or anemia, and biochemical parameters were within normal limits. USG revealed a pyloric length of 2 cm with wall thickening. At surgery, a thickened pyloric mass was visualized and palpated. Pyloromyotomy was performed successfully, with an uneventful recovery. The patient remained well during follow-up.



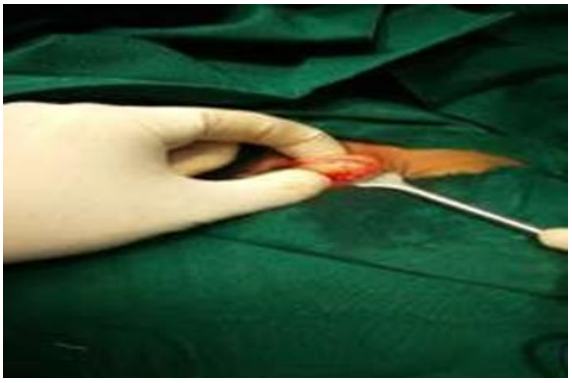
Case 3

A 1-month-old male infant presented with projectile, non-bilious vomiting associated with severe dehydration, malnutrition, and pallor. On abdominal examination, a visible gastric peristalsis and a palpable pyloric mass were noted. Laboratory investigations revealed hypokalemia and hypochloremia. USG showed a pyloric length of 2 cm and thickness of 6 mm. The patient underwent laparotomy and pyloromyotomy, with successful recovery.



Case 4

A 28-day-old male infant presented with projectile, non-bilious vomiting. The infant was otherwise healthy, with mild dehydration but no malnutrition. On examination, a palpable pyloric mass was noted, although it was not visibly apparent. USG confirmed a pyloric length of 1.8 cm and thickness of 5.6 mm. An open pyloromyotomy was performed, resulting in excellent recovery without complications.



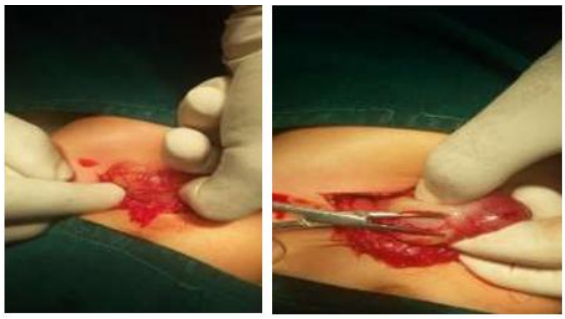
Case 5

A 28-day-old male infant presented with projectile, non-bilious vomiting. The infant was severely dehydrated and malnourished, with abdominal

distension. A visible and palpable pyloric mass was observed. Biochemical analysis showed dyselectrolytemia. USG revealed a pyloric length of 2.5 cm and thickness of 6.8 mm. The infant underwent pyloromyotomy and had an uneventful recovery with good follow-up.

Case 6

A 3-week-old male infant presented with projectile, non-bilious vomiting for 2 weeks. On examination, mild dehydration was present, but no visible or palpable abdominal mass was noted. USG was inconclusive; however, an upper GI contrast study confirmed CHPS. Ramstedt's operation was performed, resulting in good postoperative recovery.



DISCUSSION

Congenital Hypertrophic Pyloric Stenosis (CHPS) is a relatively common cause of gastric outlet obstruction in infants, typically presenting between the second and eighth week of life. In our series of six cases, all patients were male infants, consistent with the well-established male predominance reported in literature, with a male-to-female ratio of approximately 4:1.[11]

Clinical Presentation

The classical symptom in CHPS is progressive, projectile, non-bilious vomiting, which was universally present in our patients. Dehydration and electrolyte disturbances (hypokalemic, hypochloremic metabolic alkalosis) were observed in severely affected infants (Cases 1, 3, and 5). Such metabolic abnormalities are characteristic of prolonged vomiting and delayed diagnosis.[12] Visible gastric peristalsis and a palpable pyloric mass ("olive") were noted in most cases, in line with published data where the mass is clinically appreciable in up to 80% of cases.[13] However, in Case 6, no mass was palpable, highlighting the occasional diagnostic challenge when classical signs are absent.

Diagnostic Imaging

Ultrasonography (USG) is considered the gold standard for diagnosis, with cut-off values of pyloric muscle thickness ≥3 mm and length ≥15–18 mm [14]. In our series, all infants except one had diagnostic USG findings, with pyloric lengths ranging 1.8–2.5 cm and thicknesses 5.6–7 mm, consistent with accepted thresholds. In one case (Case 6), USG was inconclusive, and diagnosis was confirmed with an upper GI contrast study, which remains a reliable alternative when sonography is equivocal.[15]

Surgical Management

All infants underwent surgical correction via Ramstedt's pyloromyotomy, which remains the treatment of choice for CHPS. In our series, the procedure was performed successfully by the open approach, with excellent outcomes. Literature increasingly supports the laparoscopic approach as a safe alternative, offering advantages such as reduced postoperative pain and quicker recovery, though both methods have comparable success and complication rates.[16,17]

Outcomes

Postoperative recovery was uneventful in all cases, with early initiation of feeds and good follow-up outcomes. This aligns with reports that pyloromyotomy has a high success rate with minimal morbidity and mortality when performed promptly after correction of dehydration and electrolyte imbalance.[18]

Strengths and Limitations

Our series highlights the spectrum of presentations, from well-nourished infants to severely dehydrated and malnourished cases, demonstrating the importance of early recognition. Limitations include the small sample size and single-institution experience, which restrict generalizability. However, such reports add valuable insight into regional clinical patterns and management outcomes.

CONCLUSION

Congenital Hypertrophic Pyloric Stenosis (CHPS) remains an important cause of gastric outlet obstruction in infants, typically presenting with projectile, non-bilious vomiting. Our series highlights the variability in presentation, ranging from well-nourished infants with minimal dehydration to severely malnourished cases with profound electrolyte imbalance. Ultrasonography is the diagnostic modality of choice, but an upper GI contrast study may be valuable when USG findings are equivocal. Surgical correction with Ramstedt's pyloromyotomy remains the gold standard, with excellent outcomes when preceded by appropriate preoperative stabilization. Early recognition and

timely surgical intervention are key to reducing morbidity

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