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Case Report

Midgut Volvulus - A Rare Cause of Recurrent Acute Kidney Injury in Adolescence

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Keywords
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Pseudo-Bartter syndrome

Abbreviations
AKI - Acute kidney injury

Abstract

A15-year-old boy presented with recurrent episodes of non-bilious vomiting for the past 9 years. During these episodes he also had dehydration and oliguria. He required hospitalization during each episode that was marked by features of acute kidney injury. A plain radiograph showing ground glass opacity raised suspicion. Imaging studies confirmed intestinal malrotation with intermittent midgut volvulus. Following correction of malrotation by Ladd's procedure, he became asymptomatic. Delayed presentation of malrotation beyond infancy is rare. Recurrent acute kidney injury is an unusual complication of late manifesting malrotation in adolescence.

INTRODUCTION

Midgut volvulus due to intestinal malrotation usually presents in the early infancy. Delayed presentation leads to diagnostic dilemmas and hence increased morbidity and mortality. In this report, we describe a case of midgut malrotation with an extraordinary diagnostic delay leading to significant complications.

CASE REPORT

A 15-year-old boy, the second-born child of non-consanguineous parents, was admitted with recurrent bouts of vomiting for the past 9 years. Antenatal and perinatal periods were uneventful. He had appropriate development for the age. After his mother's death 10 years ago (cause unknown), he was living with his relatives and there was no reported child abuse during this period.

He presented with non-bilious, non-projectile, and non-hemorrhagic vomiting that occurred typically after food. Symptoms occurred semiannually, starting at the age of 6 years. The frequency of symptoms worsened progressively after the age of 12 years, with monthly episodes in the last 2 years and fortnightly episodes in the last few months. With the passage of time, the quantity of vomitus (currently 2-3 large cups) and its intensity (each episode lasting for 5-6 days at a stretch) have also

Table 1: Renal parameters at the previous and current inpatient admissions

Parameters	Mar 2023		April 2023		May 2023		Oct 2023	
	Adm	Dis	Adm	Dis	Adm	Dis	Adm	Dis
Blood Urea (mg/dl)	110	-	66	-	109	18	29	-
Serum Creatinine (mg/dl)	2.3	0.8	1.6	0.8	6.2	0.7	3.8	0.7
e-GFR (ml/min per 1.73 m^2)	22	63	32	63	8	72	13	72
Serum Sodium (mEq/l)	124	134	134	136	131	134	142	139
Serum potassium (mEq/l)	2.7	4.3	4.7	3.6	2.5	3.7	2.9	4.9
pH	7.59	7.35	7.39	7.31	7.56	7.39	7.47	-
Bicarbonate (mEq/L)	57	23	28	21	46	23	27	21
Ionized Calcium (mEq/l)	0.76	1.02	1.48	-	0.98	1.22	1.09	-

e-GFR- Estimated glomerular filtration rate, Adm - Day of admission, Dis- Day of Discharge

worsened. Few episodes of bilious vomiting, were observed recently, which were preceded by abdominal fullness and discomfort. He did not tolerate solid food, and preferred liquids instead.

For the last 4 months, fortnightly episodes of large quantity, bilious vomiting were associated with severe dehydration, reduction in the urine output and constipation. Three inpatient admissions each lasting for 8-10 days were required for correction of severe dehydration, metabolic alkalosis, hyponatremia, severe hypokalemia, and stage-3 acute kidney injury (AKI) (Table 1).

He self reported symptomatic relief during nil per oral therapy with nasogastric drainage. At a peripheral centre potassium chloride and indomethacin were given to correct recurrent hypokalemic metabolic alkalosis, although there was no documented polyuria. Diarrhea, jaundice, bulky or oily stools, headache, aura, seizures or altered sensorium were absent during the preceding 9 years. He denied using any traditional or over-the-counter medications. Poor growth was recorded during the recent past visits.

On presentation to our centre, he was severely dehydrated and hypotensive, but with a normal sensorium. Initial pulse rate was 110/min with low volume, respiratory rate was 22/min and blood pressure was 80/50 mm Hg. He was afebrile and was not dyspnoeic, maintaining SpO_2 99% on room air. Severe wasting and stunting were observed with a height of 139 cm (-3.1 standard deviation score - SDS), weight of 30.2 kg (-2.8 SDS), and body mass index of 15.6 kg/m^2 (-1.3 SDS).

The abdomen was soft with mild epigastric fullness, but without any visible peristalsis. Bowel sounds were diminished in the lower quadrants. The liver and spleen were not palpable. Hypovolemic shock corrected with bolus infusion of 20 ml/kg of isotonic fluids and it was followed by correction of dehydration.

An erect radiograph showed a gasless abdomen with a small fundal gas bubble. Barium swallow study demonstrated a grossly distended stomach with abrupt narrowing of the bowel at the proximal duodenum. (Fig. 1) A negligible amount of contrast was seen in the distal bowel.

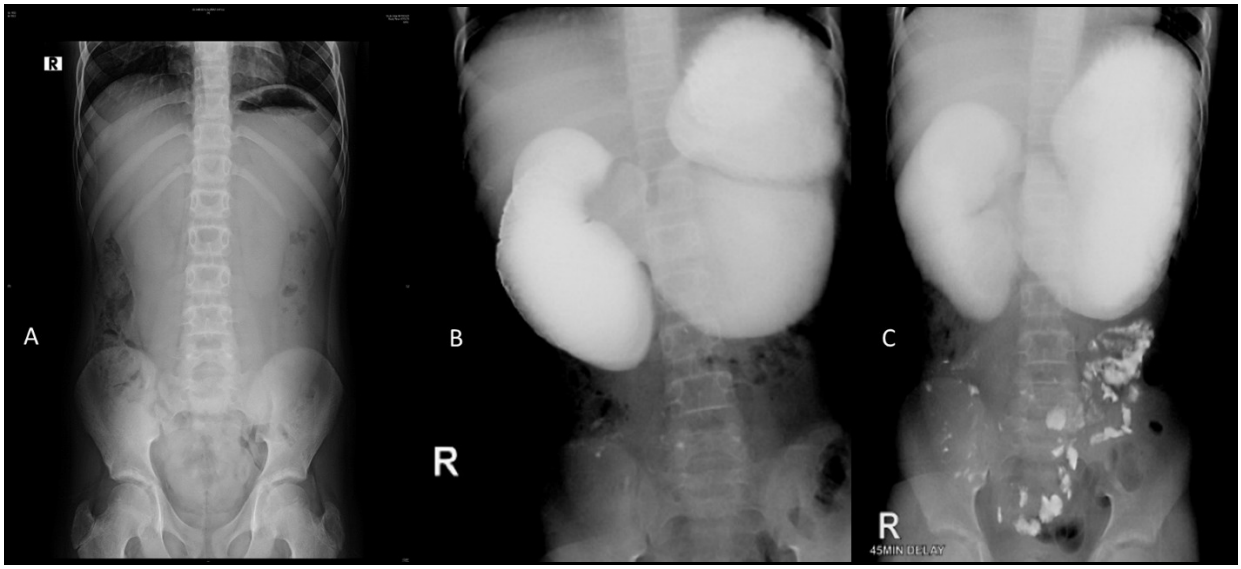

Fig 1: *Contrast radiography (A) Erect plain radiograph showing gasless abdomen; (B) Barium meal follow through showing massive dilatation of the stomach and the duodenum with abrupt cutoff at the third part of the duodenum. (C) Delayed picture showing minimal passage of contrast into the distal small bowel.*

Near-complete obstruction at the distal duodenum was recognized. MRI of the abdomen showed features of midgut malrotation with volvulus. (Fig. 2) Ultrasonography showed near-normal sized kidneys (right kidney length 74 mm; left kidney length 74 mm; -0.36 and -0.97 SDS respectively) with increased echogenicity, but without any hydroureteronephrosis.

He was finally diagnosed with recurrent metabolic alkalosis secondary to severe gastric losses, malrotation of the intestine with midgut volvulus, recurrent AKI in the context of acute-on-chronic kidney disease and chronic malnutrition.

After initial stabilization, a laparotomy was done. Small bowel volvulus with Ladd's band was found. After counterclockwise derotation, Ladd's band was divided, the mesenteric base was broadened and the small intestine was repositioned after straightening the duodenum. Postoperatively he recovered uneventfully and was discharged after a few days, with complete normalization of the metabolic parameters.

DISCUSSION

In this report, we present a patient who presented with multiple metabolic crises and recurrent reversible AKI due to midgut malrotation with volvulus. The absence of bilious vomiting and the episodic nature of the symptoms precluded an early diagnosis in this patient. It appears that the patient had had recurrent episodes of volvulus with transient derotation, leading to the episodic nature of symptoms.

Intestinal malrotation affects roughly 1 in 500 newborns, and volvulus occurs in approximately 1 in 2500 live births.[1] Normally, between 6 and 12 weeks of gestation, the intestine rotates 270^{o} with the superior mesenteric artery (SMA) as the axis. The duodeno-jejunal loop shifts to the left side of the SMA, while the ceco-colic loop moves to the right side, resulting in the final anatomic arrangement.[2] In cases of partial rotation (90^{o} - 180^{o}), the cecum remains in the mid-upper abdomen and becomes fixed to the right lateral wall by the Ladd's bands. These bands may compress the duodenum, leading to obstruction.

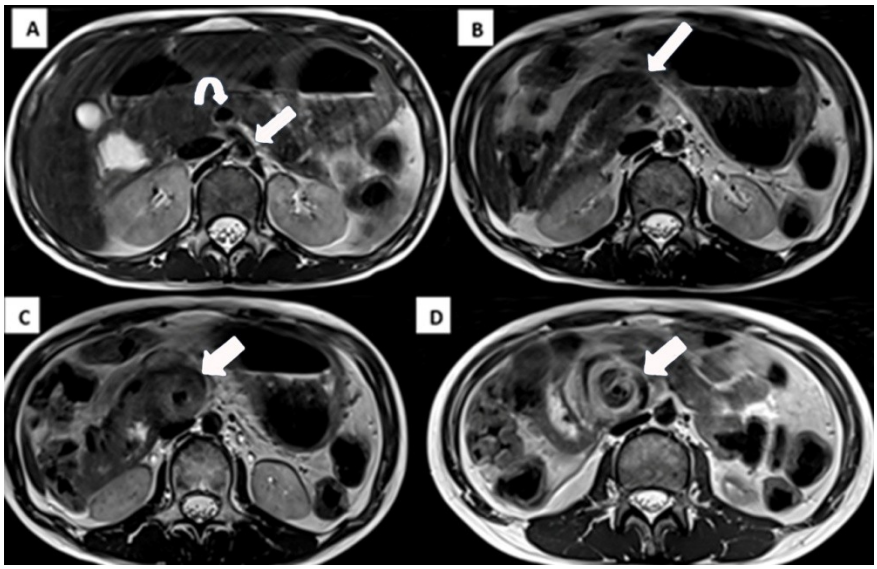

Fig 2: *Magnetic rersonance imaging (T2 high-resolution axial sections through the upper abdomen) showing the superior mesenteric artery (white arrow) coursing to the right of the superior mesenteric vein (bent arrow) (Panel A). The third part of the duodenum (arrow) does not cross the midline (Panel B & C) and is seen spiralling with the mesentery forming a "cork-screw" appearance (arrows in the panels C & D). These features are typical of midgut volvulus due to intestinal malrotation.*

The classical clinical presentation of midgut malrotation is that of bilious vomiting in the neo-natal period or infancy. An untreated malrotation may be complicated by midgut volvulus which in turn leads to hypovolemia, shock, perforation, peritonitis, bowel necrosis and systemic complications. Rarely, spontaneous derotation may cause intermittent relief of symptoms. The diagnosis is eventually reached by acute crisis, and rarely mortality occurs.

Some patients experience poor weight gain and have difficulty in tolerating solid food. This subtle clue is very important for early diagnosis and intervention.[4] Recurrent metabolic alkalosis with AKI is a rare presentation of midgut malrotation that can mimic some of the salt-wasting tubulopathies such as Bartter syndrome and Gitelman syndrome. Ultrasound is the first-line diagnostic imaging of choice in midgut volvulus. Its sensitivity is 94% and specificity is 100%. In cases that go undetected upper GI contrast studies, CT or MRI may be useful. [5]

The management of midgut volvulus involves several key steps. Preoperatively, brief, effective fluid resuscitation is needed to address dehydration or shock. Undue delay in surgical correction of the volvulus can lead to bowel gangrene. Nasogastric tube is necessary for decompression of the gastrointestinal tract. Broad-spectrum antibiotics should be administered to cover the bowel flora. The primary treatment choice involves an emergency laparotomy and Ladd's procedure, during which the volvulus is derotated and the mesentery is widened to prevent recurrence.[6] It is imperative to exclude other associated gastro-intestinal anomalies (e.g. duodenal web, intestinal pseudo-obstruction, ciliary dyskinesias). Extra-intestinal anomalies such as genitourinary, cardiovascular and craniofacial abnormalities may rarely coexist with midgut malrotation.[2]

REFERENCES

[1] Svetanoff WJ, Srivatsa S, Diefenbach K, Nwomeh BC. Diagnosis and management of intestinal rotational abnormalities with or without volvulus in the pediatric population. Semin Pediatr Surg. 2022 Feb;31(1):151141.

[2] Martin V, Shaw-Smith C. Review of genetic factors in intestinal malrotation. Pediatr Surg Int. 2010 Aug; 26(8): 769-81.

[3] Shah MR, Levin TL, Blumer SL, Berdon WE, Jan DM, Yousefzadeh DK. Volvulus of the entire small bowel with normal bowel fixation simulating malrotation and midgut volvulus. Pediatr Radiol. 2015 Dec; 45(13): 1953-1956.

[4] Karlslätt KS, Husberg B, Ullberg U, Nordenskjöld A, Wester T. Intestinal malrotation in children: Clinical presentation and outcomes. Eur J Pediatr Surg. 2024 Jun; 34 (3): 228-235.
[5] Nguyen HN, Kulkarni M, Jose J, Sisson A, Brandt ML, Sammer MBK, Pammi M. Ultrasound for the diagnosis of malrotation and volvulus in children and adolescents: a systematic review and meta-analysis. Arch Dis Child. 2021 Dec; 106(12): 1171-1178.
[6] Langer JC. Intestinal Rotation Abnormalities and Midgut Volvulus. Surg Clin North Am. 2017 Feb; 97(1): 147-159.

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