

Midgut volvulus on Ladd's bands in a young adult with a virgin abdomen, reported as normal rotation on computed tomography and twice misdiagnosed as gastroenteritis: a case report

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Case Report

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Abstract

Background:

Adult small bowel obstruction usually follows previous abdominal surgery. In a patient who has never been operated on, congenital anomaly enters the differential. Intestinal malrotation is normally corrected in infancy; when it survives undetected into adult life it may present for the first time as midgut volvulus, a strangulating emergency in which the interval to operation determines whether the small bowel is preserved.

Case presentation:

A previously well 21-year-old Asian man presented with five days of colicky abdominal pain, post-prandial vomiting and absolute constipation. He had no past medical history and no previous abdominal surgery. He attended an emergency department twice in the first three days, diagnosed as gastroenteritis on both occasions. At a third presentation his abdomen was soft, flat and non-distended, and an erect chest radiograph showed no free gas. He was admitted with acute kidney injury (creatinine 224 $\mu\text{mol/L}$). Computed tomography the following day showed marked gastric and proximal duodenal distension with an abrupt transition at the fourth part of the duodenum and a mesenteric whirlpool configuration, in keeping with midgut volvulus; the same report described the bowel as normally positioned without intestinal malrotation. At emergency laparotomy the small bowel was twisted around the root of the mesentery and obstructed at the third part of the duodenum by Ladd's bands arising on malrotated anatomy. The superior mesenteric vein was congested and dilated; the bowel was viable throughout. Detorsion, division of the bands, broadening of the mesenteric base and appendectomy were performed without resection. He was discharged on the fourth postoperative day, and at clinic review on day 18 was well, with normal renal function and a healed wound.

Conclusions:

Vomiting with absolute constipation in a virgin abdomen is mechanical obstruction until proven otherwise; gastroenteritis does not cause five days of obstipation. A soft, non-distended abdomen is expected in proximal small bowel obstruction and must not be used to withhold imaging. A computed tomography report stating that rotation is normal does not exclude malrotation, because acute torsion distorts the anatomical relationships on which that assessment depends; where a whirl sign is present, the rotational anatomy is determined at operation.

Background

Adhesions from previous abdominal or pelvic surgery account for the majority of adult small bowel obstruction, and the diagnostic reflex in an obstructed patient is to look for a scar. When there is no scar,

the differential shifts: incarcerated hernia, malignancy, inflammatory stricture, intussusception, gallstone ileus and congenital anomaly. Congenital peritoneal bands are an uncommon but recognized cause; in a recent institutional series with an accompanying literature review they accounted for seven of 269 small bowel obstruction admissions, and the diagnosis was almost never made before operation [1]. Current guidance for adhesive obstruction is built around patients with a surgical history and offers limited direction for the never-operated abdomen, in which a trial of conservative management risks temporizing a strangulating lesion [2].

Intestinal malrotation results from failure of the normal 270-degree counterclockwise rotation of the midgut about the superior mesenteric artery (SMA) during fetal development. Two consequences follow. The mesenteric base, normally a broad oblique attachment running from the duodenojejunal flexure to the ileocecal junction, is instead narrow and pedicle-like, permitting the midgut to twist about it. And peritoneal bands described by Ladd almost a century ago extend from the maldescended caecum and ascending colon across the anterior surface of the duodenum to the right posterolateral abdominal wall, producing extrinsic duodenal compression in their own right [3]. Most cases declare themselves in the neonatal period with bilious vomiting. A minority remain silent into adult life, and adult series are correspondingly small [4].

When malrotation does present in adulthood it is difficult to recognize. Chronic intermittent symptoms are easily attributed to functional disorders; acute presentation with volvulus is uncommon enough that it is rarely the first hypothesis; and the radiological signs, though well described, are imperfect [6]. Volvulus of a normally rotated midgut around an isolated congenital band is also reported, which further blurs the diagnostic boundary [7–9]. The practical consequence is delay, and delay in midgut volvulus is measured in bowel length.

We report a young man with a virgin abdomen who was twice diagnosed with gastroenteritis before computed tomography (CT) on the sixth day of illness demonstrated a mesenteric whirl and duodenal-level obstruction. That same CT report stated that there was no intestinal malrotation; at laparotomy the following day the volvulus was found to have occurred on Ladd's bands. The case illustrates both a familiar sequence of clinical misattribution and a less familiar radiological pitfall. It is reported in accordance with the CARE guidelines [11].

Case presentation

Patient information

A 21-year-old Asian man, a recent business graduate living alone, presented with a five-day history of abdominal pain and vomiting. He had no past medical history, took no regular medication and had no known allergies. He had never undergone abdominal surgery or sustained abdominal trauma. He did not drink alcohol and had stopped smoking three weeks earlier, having previously smoked approximately one cigarette per day. There was no family history of gastrointestinal disease. He was not taking a glucagon-

like peptide-1 receptor agonist. He described no antecedent history of chronic or intermittent abdominal pain, bloating or vomiting, and had regarded himself as entirely well until this illness.

The illness began on day 0 with generalized, colicky abdominal pain that he described as “a balloon being inflated”, together with vomiting after every meal. The pain was partially relieved by vomiting and radiated to the lumbar region. Vomiting occurred three to four times daily; the vomitus contained partially digested food, and there was no hematemesis. He had not opened his bowels since day 0 and was passing flatus only infrequently and with difficulty, the last occasion being early on the morning of his third presentation. He reported subjective fevers with rigors, frequent belching, an acidic taste in the mouth, progressive weakness and reduced oral intake. He described dark, reduced-volume urine with terminal stinging.

He attended his local emergency department twice within the first three days of the illness. On both occasions he was diagnosed with gastroenteritis and discharged with advice to increase oral fluid intake; he recalled being told that his kidney function was abnormal because of the vomiting. Because his symptoms did not settle, he travelled over 400 km to stay with relatives on day 4 and presented to a third emergency department that evening.

Clinical findings

On arrival he was alert and orientated but visibly weak and lethargic, shivering with pain. The chest was clear and heart sounds were normal. Mucous membranes were dry and capillary refill time was two seconds; peripheries were warm and there was no peripheral oedema. Calves were soft and non-tender.

The abdominal examination was, on every occasion it was recorded, largely unremarkable. At the third emergency department attendance the abdomen was documented as soft and not distended, with limited epigastric tenderness and quiet bowel sounds. On the following morning it was soft with tenderness confined to the left hypochondrium and audible bowel sounds. At surgical review after the CT it was recorded as “soft, flat, non-tender, no guarding, not distended”. At no point, including on the day of laparotomy, was distension, guarding or peritonism documented. The clinical timeline is summarized in Table 1.

Table 1
Clinical timeline from symptom onset to discharge

Day	Event
Day 0	Onset of generalized colicky abdominal pain and post-prandial vomiting. Last bowel motion. No previous abdominal surgery and no antecedent chronic abdominal symptoms.
Days 1–3	Two attendances at a local emergency department. Diagnosed as gastroenteritis on both occasions; discharged with advice to increase oral fluids. Informed that renal function was deranged.
Day 4	Travelled over 400 km to stay with relatives because symptoms persisted. Third emergency department attendance that evening. Abdomen soft, non-distended, quiet bowel sounds. Working impression: acute kidney injury secondary to vomiting and gastroenteritis, with bowel obstruction and urinary tract infection considered less likely. Erect chest radiograph: no free subdiaphragmatic gas. Venous blood gas: lactate not raised. Computed tomography discussed and felt not to be warranted at that stage. Intravenous crystalloid commenced. Referred to and accepted by internal medicine.
Day 5 (morning)	Medical clerking. White cell counts $13.6 \times 10^9/L$, urea 17.8 mmol/L, creatinine 224 $\mu\text{mol/L}$, bilirubin 70 $\mu\text{mol/L}$ with normal transaminases, lipase 98 U/L. Differential: intestinal obstruction, biliary pathology or acute pancreatitis, with acute kidney injury due to dehydration. Computed tomography requested. Kept nil by mouth.
Day 5 (afternoon)	Contrast-enhanced computed tomography: mechanical obstruction at the fourth part of the duodenum with a mesenteric whirlpool configuration, in keeping with midgut volvulus; bowel reported as normally positioned with no intestinal malrotation; no evidence of ischemia or perforation. Issued as a significant finding. Surgical review: abdomen soft, flat, non-tender. Nasogastric tube on free drainage, urinary catheter, staged fluid resuscitation, analgesia, proton pump inhibitor, intravenous antibiotics, thromboprophylaxis. Listed for theatre.
Day 6	Emergency midline laparotomy: detorsion of midgut volvulus, division of Ladd's bands overlying the third part of the duodenum, broadening of the mesenteric base and appendicectomy. Bowel viable throughout; no resection. Estimated blood loss < 10 mL.
Day 7 (POD 1)	No vomiting, afebrile. Nasogastric tube spigoted, free fluids commenced, mobilized to chair. Creatinine peaked at 244 $\mu\text{mol/L}$ pre-operatively on day 6.
Day 8 (POD 2)	Tolerating fluids, not yet passing flatus. Nasogastric tube and urinary catheter removed; soft diet commenced. Self-limiting pyrexia that evening.
Day 9 (POD 3)	Flatus passed. Tolerating soft diet, passing urine normally. Renal function improving.
Day 10 (POD 4)	Abdomen soft and non-tender; abdominal radiograph reviewed by the consultant surgeon with no concerns. Eating, drinking and mobilizing. Discharged on oral co-amoxiclav.
Day 18	Surgical clinic review. Well, eating normally and back to usual activities. Renal function returned to normal; midline wound clean and well healed; skin clips removed. Appendix histology reported as unremarkable.
POD postoperative day	

Diagnostic assessment

Initial investigations showed a neutrophil-predominant leukocytosis, a marked pre-renal picture and an isolated hyperbilirubinemia with normal transaminases and alkaline phosphatase (Table 2). Lipase was 98 U/L and not diagnostic of pancreatitis. Venous blood gas analysis showed no lactatemia. An erect chest radiograph showed no consolidation and no free subdiaphragmatic gas.

Table 2
Serial laboratory results from admission to the third postoperative day

Parameter	Day 5 (adm.)	Day 5 (pre-op)	Day 6 (pre-op)	Day 7 POD 1	Day 8 POD 2	Day 9 POD 3
Hemoglobin (g/L)	ND	174	ND	131	125	127
White cell count ($\times 10^9/L$)	13.6	14.3	ND	9.6	9.0	7.5
Neutrophils ($\times 10^9/L$)	10.6	11.0	ND	6.9	6.3	5.2
Platelets ($\times 10^9/L$)	ND	277	ND	183	174	203
Sodium (mmol/L)	ND	ND	137	139	139	137
Potassium (mmol/L)	ND	ND	3.6	3.5	3.9	3.8
Urea (mmol/L)	17.8	ND	15.5	12.0	9.0	7.2
Creatinine ($\mu\text{mol/L}$)	224	ND	244	216	187	178
eGFR (mL/min)	ND	ND	31	36	43	46
Bilirubin ($\mu\text{mol/L}$)	70	ND	ND	ND	ND	ND
ALT / ALP (U/L)	34 / 102	ND	ND	ND	ND	ND
Lipase (U/L)	98	ND	ND	ND	ND	ND
CRP (mg/L)	ND	ND	2	48	53	55
adm. admission, ALP alkaline phosphatase, ALT alanine aminotransferase, CRP C-reactive protein, eGFR estimated glomerular filtration rate, ND not done on that sample, POD postoperative day, pre-op pre-operative						

The combination of a soft abdomen, a normal lactate and a chest radiograph without free gas was interpreted at the receiving hospital as evidence against obstruction, and CT was deferred after discussion with the on-call registrar in favor of repeat blood tests and an erect chest radiograph. CT was requested the following morning by the admitting medical team on the grounds that the differential remained unresolved.

Contrast-enhanced CT of the abdomen and pelvis performed on day 5 demonstrated marked distension of the stomach and proximal duodenum with an abrupt transition at the level of the fourth part of the duodenum, consistent with mechanical obstruction (Fig. 1). There was twisting of the mesenteric root and the associated mesenteric vessels with a characteristic whirlpool configuration, together with twisting of the proximal small bowel loops, in keeping with midgut volvulus (Fig. 2). The remainder of the small and large bowel was described as normally positioned, without evidence of intestinal malrotation. There was associated mesenteric oedema, small-volume free fluid and mesenteric lymphadenopathy. Bowel wall enhancement was preserved, and there was no pneumatosis, portal venous gas or free intraperitoneal air; there was no definite CT evidence of bowel ischemia. The liver, gallbladder, biliary tree, pancreas, spleen, adrenal glands, kidneys and major vessels were normal. The report was issued as a significant finding with a recommendation for urgent surgical review.

Therapeutic intervention

Therapeutic intervention

Following the CT report the patient was reviewed by the surgical team, kept nil by mouth with sips of water, and resuscitated with staged intravenous crystalloid. A nasogastric tube was placed on free drainage with four-hourly aspiration, a urinary catheter was inserted with hourly urine output monitoring, and analgesia, a proton pump inhibitor, intravenous antibiotics and venous thromboembolism prophylaxis were commenced.

Emergency laparotomy was performed on day 6 under general anesthesia through a midline incision. The small bowel was found twisted around the root of the mesentery, with the transition point at the third part of the duodenum. Ladd's bands were found running from a maldescended caecum across the second and third parts of the duodenum, arising in the context of intestinal malrotation. The superior mesenteric vein (SMV) was congested and dilated. Small and large bowel were pink, viable and healthy; small bowel distal to the transition point was collapsed. No internal hernial defect was present.

Detorsion was performed, followed by division of the bands using blunt dissection and a harmonic scalpel, with broadening of the mesenteric base. After detorsion the small bowel progressively returned to normal caliber and no bowel resection was required. Appendicectomy was performed, recorded in the operative note as being undertaken to avoid future diagnostic confusion given the presence of intestinal malrotation. Haemostasis was achieved and the abdomen closed by mass closure with two No. 1 polydioxanone sutures; 20 mL of 0.5% levobupivacaine was infiltrated and the skin was closed with staples. Estimated blood loss was less than 10 mL.

Follow-up and outcomes

The postoperative course was uncomplicated. On the first postoperative day (POD) the patient was afebrile and free of vomiting; the nasogastric tube was spigoted and free fluids introduced. The

nasogastric tube and urinary catheter were removed on POD 2 and a soft diet was tolerated. A single self-limiting pyrexia occurred on the evening of POD 2, with a C-reactive protein (CRP) trajectory (2 to 48 to 53 to 55 mg/L) consistent with the expected postoperative acute-phase response rather than sepsis. Flatus was passed on POD 3.

Renal function improved steadily with resuscitation and relief of the obstruction: creatinine fell from a pre-operative peak of 244 $\mu\text{mol/L}$ to 178 $\mu\text{mol/L}$ by POD 3, with a corresponding rise in estimated glomerular filtration rate (eGFR) from 31 to 46 mL/min. Hemoglobin fell from 174 g/L pre-operatively to 125–127 g/L, a change attributable to correction of hemoconcentration rather than to blood loss, which was negligible.

On POD 4 the abdomen was soft and non-tender, an abdominal radiograph was reviewed by the consultant surgeon without concern, and the patient was eating, drinking and mobilizing. He was discharged on oral co-amoxiclav with a surgical clinic appointment eight days later for repeat renal function and removal of skin clips.

He was reviewed in the surgical clinic on day 18. He was well, eating normally and had resumed his usual activities. Renal function had returned to normal, completing recovery from an acute kidney injury that had peaked at 244 $\mu\text{mol/L}$ before operation, and the midline wound was clean and well healed; the skin clips were removed. Histological examination of the appendicectomy specimen showed no abnormality.

Discussion

This case is instructive less for the rarity of the underlying anomaly than for the sequence of individually reasonable decisions that delayed operation for a strangulating volvulus by six days in a 21-year-old. Three of those decisions were clinical and are examined below. A fourth was radiological, and is in some ways the more interesting: the CT that made the diagnosis of volvulus simultaneously reported that the patient did not have the condition that caused it.

Gastroenteritis does not cause five days of obstipation

The patient was diagnosed with gastroenteritis twice. Gastroenteritis is a common and usually correct explanation for vomiting in a young adult, but it is a diagnosis of exclusion in a patient who has stopped opening their bowels. Diarrhea is its defining feature; five days of absolute constipation with only infrequent, effortful flatus is the opposite finding and should have redirected the differential towards mechanical obstruction. Two further features in the history pointed the same way: vomiting immediately after every meal, and colicky pain relieved by vomiting. Both describe a fixed obstacle to gastric emptying rather than mucosal inflammation. The label, once applied, proved durable it survived a second attendance, a journey of over 400 km and a third presentation. This is diagnostic momentum, and it is

best interrupted by restating the differential from first principles at each new contact rather than inheriting it from the previous discharge.

A soft abdomen does not exclude proximal small bowel obstruction

The most striking feature of this record is that the abdomen was documented as soft and non-distended on every examination, including after the CT diagnosis had been made and on the day of laparotomy. This is not an anomaly; it is the expected finding. Distension in small bowel obstruction is produced by the column of gas and fluid proximal to the obstruction. When the obstruction lies at the third or fourth part of the duodenum that column is short and is emptied repeatedly by vomiting, so the abdomen remains flat. The triad taught for obstruction pain, distension, vomiting describes distal obstruction; proximal obstruction substitutes early, profuse vomiting and metabolic derangement for distension, and its physical signs are correspondingly poor. Quiet bowel sounds, recorded at the third presentation, are similarly unhelpful in either direction.

The practical implication is that in a patient with persistent vomiting and obstipation, the decision to image should be driven by the history and the biochemistry rather than by the palpating hand. Here the biochemistry was itself a red flag: a creatinine of 224 $\mu\text{mol/L}$ and a urea of 17.8 mmol/L in a previously healthy 21-year-old represent a fluid deficit that a self-limiting viral illness would be unlikely to produce over five days in a patient with free access to oral fluids.

The erect chest radiograph answers a different question

An erect chest radiograph was obtained and reported as showing no free subdiaphragmatic gas, and this appears in the record alongside a normal lactate as part of the reassurance that supported deferring CT. Both tests exclude late complications rather than the underlying diagnosis. A chest radiograph excludes pneumoperitoneum, and pneumoperitoneum is absent in the overwhelming majority of closed-loop obstructions until the bowel has already perforated. Serum lactate is insensitive for early mesenteric venous congestion and is typically normal until ischemia is established and transmural; a normal lactate in a strangulating volvulus is common and should never justify observation. The same caution applies to the CT itself: it showed preserved bowel wall enhancement and no pneumatosis or portal venous gas, and the report explicitly stated that there was no definite evidence of ischemia yet the SMV was found congested and dilated at operation. CT signs of established ischemia are specific but late; their absence does not confer time.

When computed tomography reports no malrotation in a patient who has it

The CT report in this case was, in the respect that mattered most, entirely correct: it identified the mechanical transition point at the fourth part of the duodenum, described the whirlpool configuration of the mesenteric vessels, concluded that this represented midgut volvulus, and recommended urgent

surgical review as a significant finding. That report is what secured the operation. In one respect, however, it was misleading: it stated that the bowel was normally positioned without evidence of intestinal malrotation, and the operative findings were of Ladd's bands arising on malrotated anatomy.

This divergence is worth examining rather than glossing, because it is predictable. The radiological assessment of rotation rests on the position of the duodenojejunal flexure, the position of the caecum, and the relationship of the SMV to the SMA the vein normally lying to the right of the artery, with inversion described as a sign of malrotation [6]. Each of these is degraded in the presence of an acute volvulus. The mesenteric twist physically rotates the vessels about one another, so the venous–arterial relationship reflects the torsion rather than the underlying rotation. The duodenum is grossly distended and its distal course obscured at precisely the level where the flexure would be assessed. Cecal position in an adult is variable even without rotational anomaly, and a mobile or high caecum is not in itself diagnostic. The signs are in any case imperfect: inversion of the mesenteric vessels is neither perfectly sensitive nor perfectly specific, and a normal vascular relationship does not exclude malrotation [6].

The practical rule that follows is narrow but useful. A CT statement that there is no malrotation, made in the presence of a mesenteric whirl, should be regarded as provisional. It does not alter management the whirl sign mandates operation either way, and has been shown to be associated with a substantially increased likelihood of requiring operative intervention in small bowel obstruction [5] but it should not be allowed to shape the surgeon's expectation of what will be found, nor to be carried forward into the discharge summary as though the rotational anatomy had been settled. In a patient presenting acutely with volvulus, that question is answered in theatre. Where it matters for subsequent counselling and the acute anatomy is unclear, an elective upper gastrointestinal contrast study once the patient has recovered will demonstrate the position of the duodenojejunal flexure in a decompressed abdomen.

Adult intestinal malrotation and Ladd's bands

Ladd described the bands that bear his name almost a century ago, together with the operation that addresses them [3]. They extend from the maldescended caecum and proximal ascending colon across the anterior surface of the duodenum to the right posterolateral abdominal wall, and they contribute to obstruction by direct extrinsic compression. They are not, however, the principal danger. That lies in the narrowed mesenteric base that accompanies failed rotation, which converts the broad oblique root of the normal mesentery into a pedicle about which the entire midgut can twist, taking the superior mesenteric vessels with it. This patient exhibited both components: obstruction at the third part of the duodenum where the bands crossed it, and torsion of the small bowel about the mesenteric root, with a congested and dilated SMV as evidence of the venous outflow obstruction that precedes infarction.

Presentation at 21 years with no antecedent symptoms is uncommon but not unknown; adult malrotation is increasingly recognized, and contemporary series describe both chronic symptomatic presentations and acute volvulus, with laparoscopic correction now well established in the elective setting [4, 10]. What distinguishes the adult presentation is that malrotation is not on the mental list. The patient has lived for two decades without trouble, which is itself read as reassurance, and the abdomen

is virgin, which removes the commonest cause of obstruction without suggesting the correct one. Volvulus of a normally rotated midgut on an isolated congenital band is also described and would have produced an identical clinical and radiological picture [7–9]; the distinction is made at operation, not before it.

That this patient came to theatre with viable bowel after six days of symptoms and a congested mesenteric vein was fortunate rather than inevitable. Published reports of adult midgut volvulus include patients who required extensive small bowel resection and were left intestinally insufficient, and the determinant in most of them was the interval to operation [7, 8].

How complete should the operation be?

The classical Ladd's procedure comprises counterclockwise detorsion, division of Ladd's bands, broadening of the mesenteric base by division of the peritoneum between the duodenum and the colonic mesentery, appendicectomy, and final placement of the small bowel in the right abdomen with the colon on the left [3, 4, 10]. The first four were performed here. The operative note does not record the final positioning of the bowel, and in an emergency laparotomy for an obstructed, edematous midgut with an acute kidney injury there are sound reasons for limiting the dissection once the volvulus has been relieved and viability secured.

The appendicectomy, in this context, is straightforward to justify. It is a standard component of the Ladd's procedure precisely because the caecum does not return to the right iliac fossa, so that a future appendicitis would present in an unexpected location and be missed or diagnosed late. Histological examination of the specimen showed no abnormality.

The question that remains open is what the patient should now be told. A patient in whom malrotation has been identified and the volvulus relieved retains an abnormal mesenteric base unless it has been adequately broadened, and recurrent volvulus after a Ladd's procedure, while uncommon, is described. He is 21 years old and may present anywhere in the world over the next sixty years. He should therefore leave the service with three things in writing and in terms he can repeat: that he has intestinal malrotation; that his appendix has been removed; and that any future episode of severe abdominal pain with vomiting requires urgent assessment and early cross-sectional imaging, with volvulus explicitly considered. A copy of the operative note is the single most useful document he can carry.

Associated metabolic findings

Two laboratory findings contributed to the diagnostic uncertainty and are worth brief comment. The acute kidney injury (AKI) documented as stage 2, with a peak creatinine of 244 $\mu\text{mol/L}$ against a presumed normal baseline for a 21-year-old man was pre-renal, driven by protracted vomiting, third-space sequestration into obstructed bowel and reduced intake. It was the finding that secured his admission, and it was correctly attributed to volume depletion; the error lay in accepting gastroenteritis as the reason for that depletion. Notably, creatinine continued to rise after admission, peaking on the

morning of surgery and falling only once the obstruction was relieved a reminder that ongoing losses into obstructed bowel are not fully corrected by intravenous fluids alone.

The isolated hyperbilirubinemia of 70 $\mu\text{mol/L}$ with entirely normal transaminases and alkaline phosphatase raised the possibility of biliary pathology and appears in the admitting differential. An unconjugated hyperbilirubinemia of this pattern in a young, fasting, dehydrated patient is most consistent with an underlying Gilbert's syndrome unmasked by starvation, and CT confirmed a normal gallbladder and a non-dilated biliary tree. Recognizing the pattern would have removed one competing diagnosis at an early stage. Fractionation of bilirubin, with repeat testing once fed and rehydrated, would be reasonable at follow-up.

Limitations

This is a single retrospective case report assembled from contemporaneous clinical records and inherits their limitations. The two initial emergency department attendances occurred at a different institution and those records were not available to us, so the assessments performed there are described only as reported by the patient. The determination of intestinal malrotation rests on the operating surgeon's intraoperative assessment, which he has since reconfirmed on review of the case; the CT report described normally positioned bowel, and no upper gastrointestinal contrast study was performed, so there is no independent confirmation. The operative note does not record the position of the caecum, the position of the duodenojejunal flexure or the width of the mesenteric base, and we would emphasize the value of documenting these explicitly whenever a rotational anomaly is identified, since the label attached in the operative note is inherited by every clinician who reads it subsequently.

Key learning points

- Gastroenteritis does not cause five days of absolute constipation. Vomiting with obstipation is mechanical obstruction until proven otherwise, particularly when vomiting relieves the pain.
- A soft, non-distended abdomen is the expected finding in proximal small bowel obstruction, because the obstructed segment is decompressed by vomiting. Examination cannot be used to withhold cross-sectional imaging.
- An erect chest radiograph without free gas and a normal serum lactate exclude perforation and established ischemia respectively. Neither excludes obstruction or strangulation, and neither justifies observation.
- The CT whirl sign is a surgical emergency. Absence of CT features of ischemia does not confer time those features are late and insensitive.
- A CT report of "no malrotation" in the presence of a mesenteric whirl is provisional. Acute torsion distorts the superior mesenteric vessel relationship and obscures the duodenojejunal flexure, and the underlying rotation is determined at operation, not on the workstation.
- Intestinal malrotation can remain silent for two decades and present as acute midgut volvulus in a virgin abdomen. A long asymptomatic history is not reassurance.

- When a rotational anomaly is found, record cecal position, duodenojejunal flexure position, mesenteric base width and the origin and insertion of every band divided. The label used in the operative note is inherited by every clinician who reads it afterwards.
- A young patient with malrotation should leave the service knowing the diagnosis, that the appendix has been removed, and that future severe abdominal pain with vomiting needs urgent imaging with volvulus explicitly considered.

Conclusions

Intestinal malrotation presenting for the first time in adulthood as midgut volvulus is uncommon, easily missed, and unforgiving of delay. The diagnostic difficulty in this case arose from three familiar traps a plausible early label that was never revisited, a soft abdomen in a proximal obstruction, and negative tests for late complications read as evidence against the underlying diagnosis and from one less familiar: a CT report that correctly diagnosed the volvulus while stating that the patient did not have the anomaly that produced it. In a patient with vomiting and absolute constipation and no previous abdominal surgery, mechanical obstruction should be assumed and CT obtained early. Where CT demonstrates a mesenteric whirl, operation should not await radiological evidence of ischemia, and a statement that rotation is normal should be treated as provisional until the abdomen is open.

Patient's perspective

I had never had anything serious wrong with me before this, so when the pain and the vomiting started, I assumed it was something I had eaten. I went to hospital twice near where I live and was told both times that it was a stomach bug and that I should drink more water. I tried, but I could not keep anything down, and I was not opening my bowels at all. By the time I travelled down to stay with my family I felt very weak.

What surprised me most was being told afterwards that this was something I had been born with. I had always thought of myself as a healthy person, and it was strange to hear that the problem had been there my whole life without ever causing me any trouble until now. Nobody had any reason to look for it, and I had never had a single symptom that would have made me go and ask.

I was frightened when I was told I needed an emergency operation, but the surgeons explained what they were going to do and why it could not wait, and that helped. The recovery has gone well. My kidney tests are back to normal, the wound has healed and I am eating normally again. I am relieved that it was found when it was, and glad that it is dealt with.

Abbreviations

AKI
Acute kidney injury

ALP
Alkaline phosphatase
ALT
Alanine aminotransferase
CRP
C-reactive protein
CT
Computed tomography
eGFR
Estimated glomerular filtration rate
POD
Postoperative day
SMA
Superior mesenteric artery
SMV
Superior mesenteric vein

Declarations

Ethics approval and consent to participate

Not applicable. Ethical approval is not required for the publication of a single anonymised case report at our institution.

Consent for publication

Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Prior publication.

This manuscript has not been published elsewhere and is not under consideration by another journal.

Competing interests

The authors declare that they have no competing interests.

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Author Contribution

H.A. conceived the report, collected the clinical, laboratory and radiological data, prepared the figures and drafted the manuscript. A.K. and M.A. were involved in the clinical care of the patient and revised the manuscript critically for important intellectual content. P.C.V. and A.I. contributed to data collection and to revision of the manuscript. P.H. performed the operation, provided and verified the operative findings, supervised the work and revised the manuscript critically. All authors reviewed and approved the final manuscript.

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Availability of data and materials

All data generated or analyzed during this case are included in this published article.

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Figures

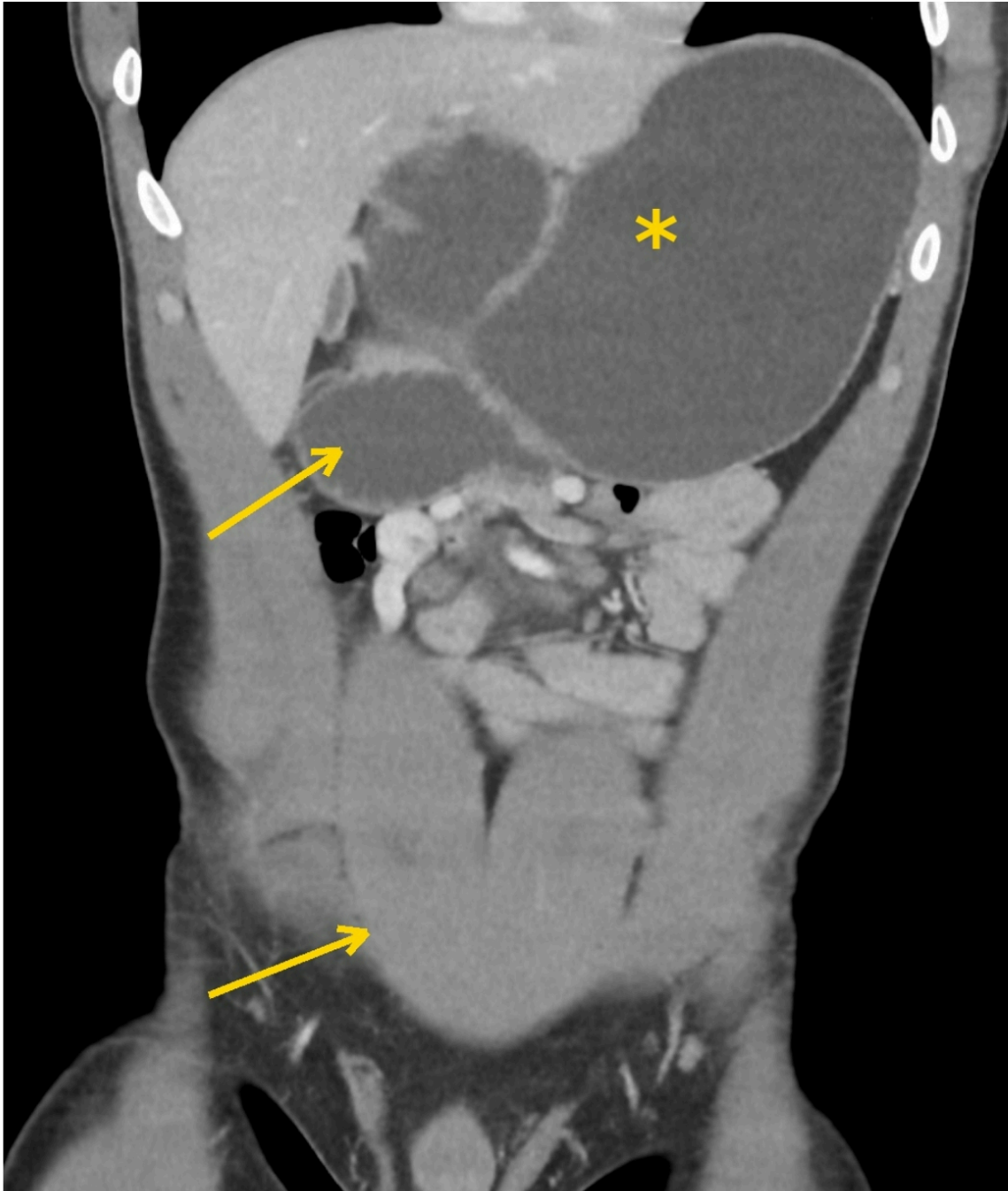


Figure 1

Coronal reformatted contrast-enhanced computed tomography of the abdomen, demonstrating marked distension of the fluid-filled stomach (asterisk) and of the proximal duodenum (upper arrow), with an abrupt change in calibre below this and collapsed small bowel loops throughout the lower abdomen (lower arrow). The appearances are those of a high mechanical small bowel obstruction.

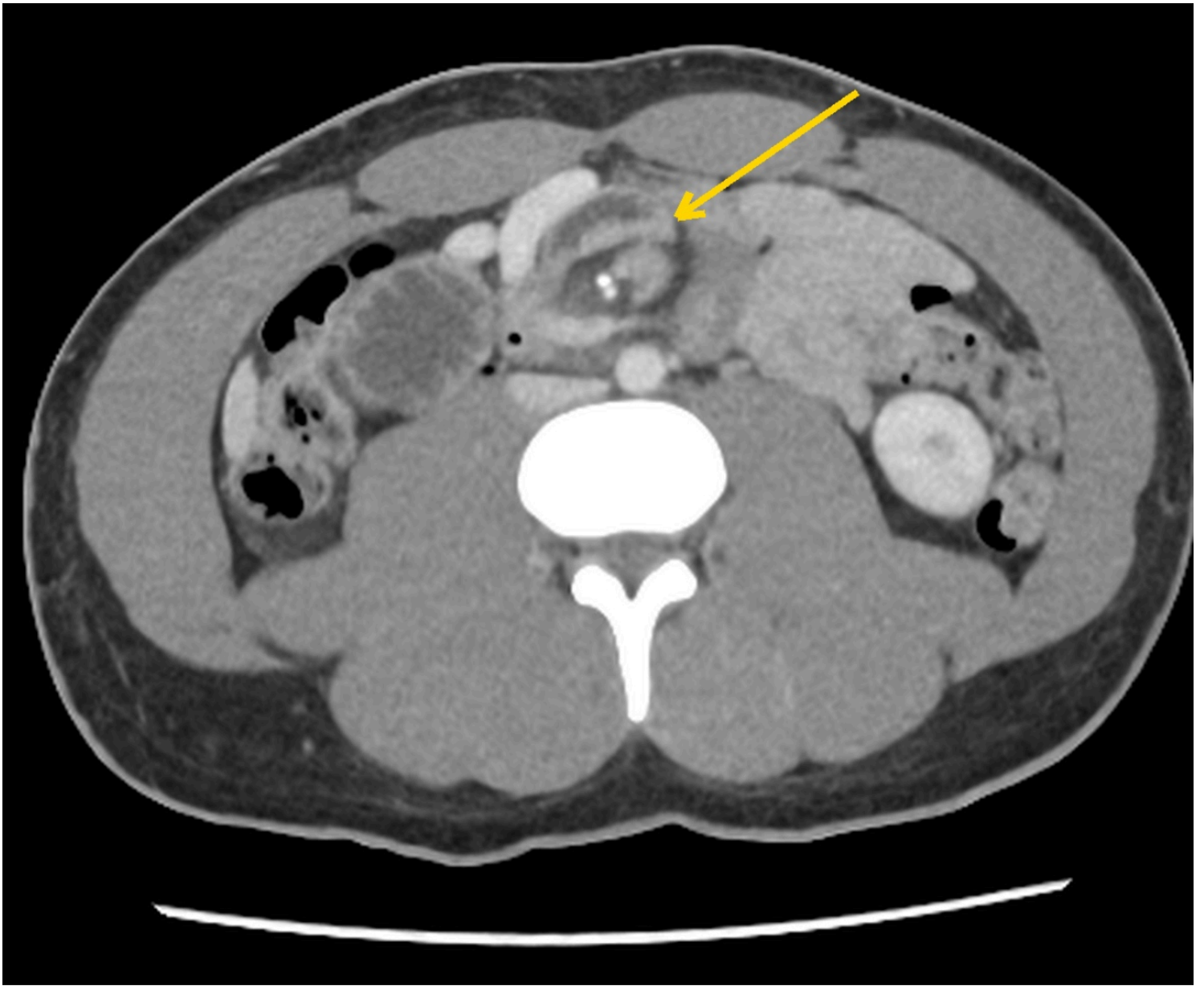


Figure 2

Axial contrast-enhanced computed tomography at the level of the mesenteric root, demonstrating the whirlpool sign (arrow): mesenteric fat and vessels spiralling about the mesenteric axis, with the superior mesenteric vessels seen end-on at the centre of the whorl. This is the direct radiological sign of midgut volvulus. Small bowel distal to the obstruction is collapsed, bowel wall enhancement is preserved, and there is no pneumatosis or portal venous gas.

Supplementary Files

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- [CAREchecklist.docx](#)