

Superior Mesenteric Artery Syndrome Complicating Severe Acute Malnutrition Secondary to Pulmonary Tuberculosis in an Adolescent: A Case Report

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Case Report

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Abstract

Background:

Superior mesenteric artery (SMA) syndrome is a rare cause of upper gastrointestinal obstruction defined by extrinsic compression of the third portion of the duodenum between the SMA and the abdominal aorta, resulting from depletion of the retroperitoneal fat pad. It predominantly affects adolescents, is rarer in children, and carries a reported mortality of up to 33% when diagnosis is delayed.

Case Summary:

A 16-year-old male presented with a three-month history of progressive weight loss, anorexia, productive cough, night sweats, abdominal pain, and bilious vomiting. He had a household contact with confirmed pulmonary tuberculosis (TB). Admission anthropometry revealed a body mass index (BMI) of 12 kg/m² and mid-upper arm circumference (MUAC) of 13 cm, confirming severe acute malnutrition (SAM). Sputum GeneXpert was positive for *Mycobacterium tuberculosis*. CT scan confirmed SMA syndrome with an aortomesenteric angle of 22° and distance of 6 mm. The patient was managed conservatively with ReSoMal rehydration via nasogastric tube (NGT), electrolyte correction, enteral feeding initiated with F-75 formula and advanced to F-100 following stabilization, intravenous antibiotics, and four-drug anti-tuberculosis therapy (isoniazid, rifampicin, ethambutol, pyrazinamide) initiated after two weeks of nutritional stabilization. Refeeding syndrome was anticipated and monitored throughout nutritional rehabilitation. Planned surgery was deferred after clinical improvement. Bilious vomiting and abdominal pain resolved over four months; by seven months, the patient had achieved significant weight gain and was tolerating a full family diet.

Conclusion:

This case illustrates the bidirectional and compounding relationship between SAM driven by pulmonary TB and SMA syndrome. Conservative nutritional rehabilitation was curative, obviating surgery. Clinicians in TB-endemic, resource-limited settings should maintain a high index of suspicion for SMA syndrome when standard malnutrition management fails.

Trial Registration

Not applicable. This study was not a clinical trial.

Introduction

Superior mesenteric artery (SMA) syndrome is a rare but clinically significant cause of upper gastrointestinal obstruction, defined by extrinsic compression of the third portion of the duodenum

between the SMA anteriorly and the abdominal aorta and vertebral column posteriorly, resulting in partial or complete duodenal obstruction. (1, 2, 3, 4) The condition is also known by several eponyms and descriptive terms, including Wilkie's syndrome, cast syndrome, chronic duodenal ileus, and arteriomesenteric duodenal compression syndrome. (5, 6, 7)

First described by Carl Freiherr von Rokitsky in 1842 as an incidental autopsy finding, (7, 8, 9, 10) the syndrome was not fully characterized until 1921–1927, when David Wilkie defined its pathophysiological basis and published the first comprehensive case series of 75 patients. (4, 5, 7, 11) Radiologically, SMA syndrome is characterized by reduction of the aortomesenteric angle and distance, which together create a clamping effect on the transverse duodenum. (12, 13) The normal aortomesenteric angle ranges from 38–65° and distance of 10–28 mm; values below 25° and 8 mm are diagnostic.

The etiology is multifactorial. Congenital predisposing factors include a high or fixed insertion of the ligament of Treitz and a low origin of the SMA. Acquired causes include any condition leading to depletion of the retroperitoneal fat pad most importantly rapid or severe weight loss from wasting illnesses (malignancy, HIV/AIDS, malabsorption, tuberculosis), eating disorders, catabolic states (burns, trauma), prolonged bed rest, and complications of spinal or abdominal surgery. (14) The condition is rare in children and its exact pediatric incidence remains unknown. (7)

Clinically, SMA syndrome is challenging to diagnose because its symptoms epigastric pain, nausea, bilious vomiting, early satiety, abdominal distension, and postprandial discomfort are nonspecific and overlap with many common gastrointestinal conditions, frequently leading to diagnostic delay. (4, 15) Of note, in the pediatric population, rapid pubertal linear growth with disproportionate loss of adipose tissue can deplete the mesenteric fat pad and precipitate SMA syndrome even without overt weight loss a mechanism that is under-recognized and particularly relevant in adolescent males. Furthermore, while tuberculosis-associated wasting is a well-established driver of severe acute malnutrition (SAM), its role as an indirect precipitant of SMA syndrome via progressive fat pad depletion has rarely been reported. Management begins with conservative measures—fluid resuscitation, electrolyte correction, nasogastric decompression, and nutritional rehabilitation—reserving surgery for refractory cases. (12) Herein, we report a case of a 16-year-old male who presented with SAM secondary to pulmonary tuberculosis (TB), whose hospital course was complicated by SMA syndrome and was successfully managed with conservative nutritional rehabilitation, representing what we believe to be among the first reported cases of SMA syndrome precipitated by pulmonary TB-driven SAM in an adolescent from a resource-limited setting.

Case Presentation

A 16-year-old male with no known prior medical history presented to the emergency department with a three-month history of progressive weight loss, anorexia, easy fatigability, and diffuse abdominal pain, followed by on-and-off bilious vomiting. Associated symptoms included a productive cough with whitish sputum and drenching night sweats. Bowel habit was preserved. Of epidemiological significance, his

uncle had been diagnosed with pulmonary tuberculosis (TB) three months prior and had frequented the household before diagnosis, representing a clear source of household exposure.

At a local health facility, the patient had been empirically treated with azithromycin and loratadine without improvement, and was subsequently initiated on ready-to-use therapeutic food (RUTF; Plumpy'Nut) for malnutrition. He was unable to meet his daily requirements and vomiting became progressively more frequent. Neither the underlying tuberculosis nor the evolving severe acute malnutrition (SAM) was appropriately investigated or managed at the primary level.

On admission, the patient was emaciated and acutely ill-looking, with tachycardia and markedly sunken eyes. Anthropometric assessment revealed a body mass index (BMI) of 12 kg/m² and a mid-upper arm circumference (MUAC) of 13 cm, confirming SAM. Chest auscultation revealed bilateral coarse crepitations over the posterior lower lung fields. He was admitted with the working diagnoses of: (1) severe acute malnutrition with dehydration; (2) intestinal obstruction, to be excluded; and (3) disseminated tuberculosis involving the lungs and gastrointestinal tract, to be excluded.

Laboratory investigations demonstrated normocytic-to-microcytic anemia (hemoglobin 7.5 g/dL, MCV 75 fL), leukocyte count of 6,000/μL (neutrophils 45%), and thrombocytopenia (platelets 115,000/μL). Serum electrolytes showed mild hyponatremia (Na 132 mEq/L) and borderline hypokalemia (K 3.0 mEq/L). Renal and liver function tests, random blood sugar, urinalysis, stool microscopy, stool GeneXpert, and stool H. pylori antigen were all unremarkable. HIV serology was negative. Sputum GeneXpert was positive for Mycobacterium tuberculosis. Chest X-ray demonstrated bilateral pulmonary infiltrations. Abdominal ultrasound showed gastric distension with gas but no mass or fluid collection. Upper GI endoscopy revealed dilatation of the stomach and the first and second parts of the duodenum without an intraluminal obstructing lesion.

Subsequent CT scan of the abdomen confirmed extrinsic vascular compression of the third part of the duodenum consistent with SMA syndrome, with an aortomesenteric angle of 22° (measured on the mid-sagittal reconstruction between the abdominal aorta and the SMA) and an aortomesenteric distance of 6 mm (measured on the axial view at the level of the third duodenal segment), both below the established diagnostic thresholds of 25° and 8 mm respectively (**Figures 2: a and b**).

The patient was rehydrated with ReSoMal via nasogastric tube (NGT) per SAM protocol, and serum electrolytes were actively corrected; the mild hyponatremia and borderline hypokalemia identified on admission normalized within the first 48 hours of management. Enteral feeding was commenced with F-75 formula, subsequently advanced to F-100 following clinical stabilization, in keeping with the standard two-phase SAM nutritional rehabilitation protocol. The step-up approach from F-75 to F-100 was deliberately phased to minimize the risk of refeeding syndrome, with close electrolyte monitoring throughout. Intravenous ampicillin and gentamicin were initiated for infection management. Ondansetron was administered as needed for vomiting and intravenous omeprazole for gastric acid suppression. After two weeks of clinical stabilization, four-drug anti-tuberculosis therapy—isoniazid (INH), rifampicin, ethambutol, and pyrazinamide was initiated via NGT and later transitioned to oral

intake; the two-week delay before commencing ATT was intentional, allowing nutritional stabilization and reducing the risk of drug-related adverse effects in a severely malnourished patient. Given early clinical improvement with nutritional rehabilitation, the initially planned surgical intervention was deferred. NGT formula feeding was continued for six weeks, after which RUTF and a supplementary diet were introduced gradually. Given early clinical improvement with nutritional rehabilitation, the initially planned surgical intervention was deferred. NGT formula feeding was continued for six weeks, after which RUTF and a supplementary diet were introduced gradually.

Abdominal pain and bilious vomiting resolved over approximately four months. At six months of follow-up, the patient was tolerating a full family diet without recurrence of symptoms, and anti-TB therapy was completed. By seven months from initial presentation, repeat investigations were within normal limits and significant weight gain had been achieved. The underlying etiology of SAM was attributed to secondary malnutrition driven by pulmonary tuberculosis, which—compounded by delayed diagnosis and inadequate primary-level management precipitated the development of SMA syndrome.

Discussion

SMA syndrome is a rare cause of upper gastrointestinal obstruction resulting from compression of the third portion of the duodenum between the SMA anteriorly and the aorta posteriorly, due to loss of the intervening retroperitoneal fat pad. (1, 2, 5) The normal aortomesenteric angle ranges from 38–65° and distance 10–28 mm; values below 25° and 8 mm, respectively, are diagnostic. (7, 11, 17) In our patient, the angle was reduced to 22° and the distance to 6 mm, consistent with published thresholds. The estimated incidence is 0.013–0.3%, rising to 2.4% in high-risk groups; the condition predominantly affects adolescents and young adults aged 10–39 years, with a female predominance (male-to-female ratio 3:2) and a median diagnosis age of 17 years, though it is rarer in children. (2, 4, 7, 11, 12, 18, 19, 20, 21)

Etiology is multifactorial. The most common precipitant is significant weight loss from any cause—malignancy, HIV/AIDS, malabsorption, tuberculosis, eating disorders, burns, or catabolic states—which depletes the mesenteric fat pad and narrows the aortomesenteric angle. (2, 5, 8, 10, 14, 19, 22, 23, 24) In the pediatric population, pubertal growth spurts with disproportionate lean mass gain and adipose loss represent an important, under-recognized predisposing mechanism. (1, 19, 25) Congenital variants (low SMA origin, high ligament of Treitz insertion) and postoperative anatomy (scoliosis correction, bariatric surgery) complete the etiological spectrum. (7, 8, 11) Notably, there is a bidirectional relationship between SMA syndrome and SAM: weight loss from SAM precipitates SMA syndrome, while food aversion from obstructive symptoms perpetuates malnutrition. In our patient, SAM driven by pulmonary tuberculosis clearly preceded SMA syndrome. At 16 years of age, our patient was additionally at the peak age for pubertal lean mass accretion with concomitant adipose depletion a physiological state that very likely compounded the TB-driven fat pad loss and lowered the threshold for duodenal compression. To our knowledge, SMA syndrome precipitated by pulmonary TB-driven SAM in an adolescent from a resource-limited sub-Saharan African setting has not been previously described; this case therefore adds

a novel etiological dimension to the published literature. Up to 40% of cases have no identifiable cause. (8)

Clinical presentation is nonspecific, with classical features of postprandial abdominal pain (58%), nausea (50%), vomiting (83%), and weight loss in virtually all reported cases. (19) Symptoms worsen in the supine position and are relieved by the prone, knee-chest, or left lateral decubitus position. (7) Our patient's weight loss, bilious vomiting, and postprandial pain overlapped extensively with SAM and pulmonary TB, contributing to diagnostic delay. A high index of clinical suspicion is essential, particularly when standard malnutrition management fails to produce improvement. (2, 19, 7)

Diagnosis requires clinical suspicion confirmed radiologically. CT scan with intravenous contrast is the current standard, directly measuring the aortomesenteric angle and distance and excluding alternative pathology; an angle $< 25^\circ$ and distance < 8 mm are highly suggestive. (4, 12, 19) Upper GI barium studies demonstrate duodenal dilatation, antiperistaltic contrast flow, and delayed transit (4–6 hours) with positional relief. (8, 22, 24) Upper GI endoscopy excludes intraluminal lesions; our patient demonstrated gastric and duodenal dilatation. BMI alone does not reliably reflect visceral fat; CT or MRI is preferred for body fat assessment in suspected cases. (8, 15) The differential diagnosis is broad and includes anorexia nervosa, peptic ulcer disease, functional dyspepsia, cyclic vomiting syndrome, pancreatitis, gastric outlet obstruction, pseudoobstruction, and duodenal pathology from tuberculosis, neoplasia, or extrinsic compression. (7, 10, 19)

Conservative management is first-line and achieves remission in approximately 83% of cases. (1, 8, 12, 15, 16, 19, 21, 22, 23, 25, 26, 27) It comprises nasogastric decompression, fluid resuscitation, electrolyte correction, and nutritional rehabilitation via nasojejun tube (used in $\sim 50\%$ of cases) or total parenteral nutrition (TPN) for complete obstruction ($\sim 70\%$ as adjunct), with gradual caloric advancement to avoid refeeding syndrome a potentially fatal complication of rapid nutritional repletion in severely malnourished patients, characterized by dangerous electrolyte shifts (particularly hypophosphatemia, hypokalemia, and hypomagnesemia) triggered by insulin-mediated cellular uptake. (7, 19) Postural maneuvers provide symptomatic relief; prokinetics are contraindicated when obstruction is present, as increasing gastrointestinal motility against a fixed mechanical obstruction aggravates intraluminal pressure, worsens pain, and risks perforation. (7, 8, 22) No definite time limit has been described for conservative management. (3, 5, 8, 14) A 6–8 weeks trial is generally recommended before surgical referral. (1, 7) In our patient, the two-phase enteral feeding protocol (F-75 advancing to F-100) was employed specifically to mitigate refeeding risk, followed by RUTF supplementation. Full resolution and significant weight gain were achieved by seven months without requiring surgery. Surgical options—laparoscopic duodenojejunostomy (preferred in pediatric patients), gastrojejunostomy, or Strong's operation—are reserved for refractory cases and carry success rates exceeding 92% with 0% recurrence at 12 months. (1, 4, 5, 7, 12, 16, 17, 19, 22, 28, 24)

Delayed diagnosis carries serious consequences including progressive malnutrition, electrolyte abnormalities, gastric perforation, gastrointestinal hemorrhage (5, 17), and has a reported mortality of up

to 33% (17). This case underscores that in any adolescent presenting with refractory vomiting, weight loss, and malnutrition particularly in a tuberculosis-endemic, resource-limited setting SMA syndrome should be considered early in the diagnostic workup. Recognition of the compounding triad of SAM, pulmonary tuberculosis, and SMA syndrome, and timely CT evaluation, can be life-saving.

Conclusion

We report a rare case of SMA syndrome complicating severe acute malnutrition secondary to pulmonary tuberculosis in a 16-year-old male, successfully managed with conservative nutritional rehabilitation. This case highlights three important clinical lessons: (1) SMA syndrome and SAM share a bidirectional, compounding relationship that can obscure and delay both diagnoses; (2) pulmonary tuberculosis should be recognized as an underlying driver of malnutrition severe enough to precipitate SMA syndrome; and (3) conservative nutritional management, when applied systematically and with patience including a deliberate step-up feeding protocol to mitigate refeeding syndrome can achieve full resolution and avoid surgery even in resource-limited settings. Clinicians in endemic regions should maintain a high index of suspicion for SMA syndrome in any adolescent in whom standard malnutrition management fails to produce improvement. We recommend that SAM management guidelines in TB-endemic, resource-limited settings explicitly acknowledge SMA syndrome as a potential mechanical complication requiring early CT evaluation when clinical response to nutritional rehabilitation is unexpectedly poor.

Abbreviations

ATT – Anti Tuberculosis Therapy

BMI – Body mass index

CT – Computed tomography

GI – Gastrointestinal

HIV – Human immunodeficiency virus

INH – Isoniazid

MRA – Magnetic resonance angiography

MUAC – Mid-upper arm circumference

NGT – Nasogastric tube

PICT – Provider-initiated counselling and testing (HIV)

RUTF – Ready-to-use therapeutic food

SAM — Severe acute malnutrition

SMA — Superior mesenteric artery

SMAS — Superior mesenteric artery syndrome

TB — Tuberculosis

TPN — Total parenteral nutrition

Declarations

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Author Contributions

All authors were involved in the concept design, preparation, critical analysis, and revision of the manuscript, and were involved in the patient's management.

Declaration of Conflicting Interests

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Ethical Approval and Consent to Participate

The institution does not require ethical approval for the publication of a single case report. Written informed consent was obtained from the patient's legal guardian for publication of this case report and any accompanying images.

Trial Registration

Not applicable. This study was not a clinical trial.

Data Availability Statement

All data sets supporting the conclusions of this case report are available from the corresponding author upon reasonable request.

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Figures

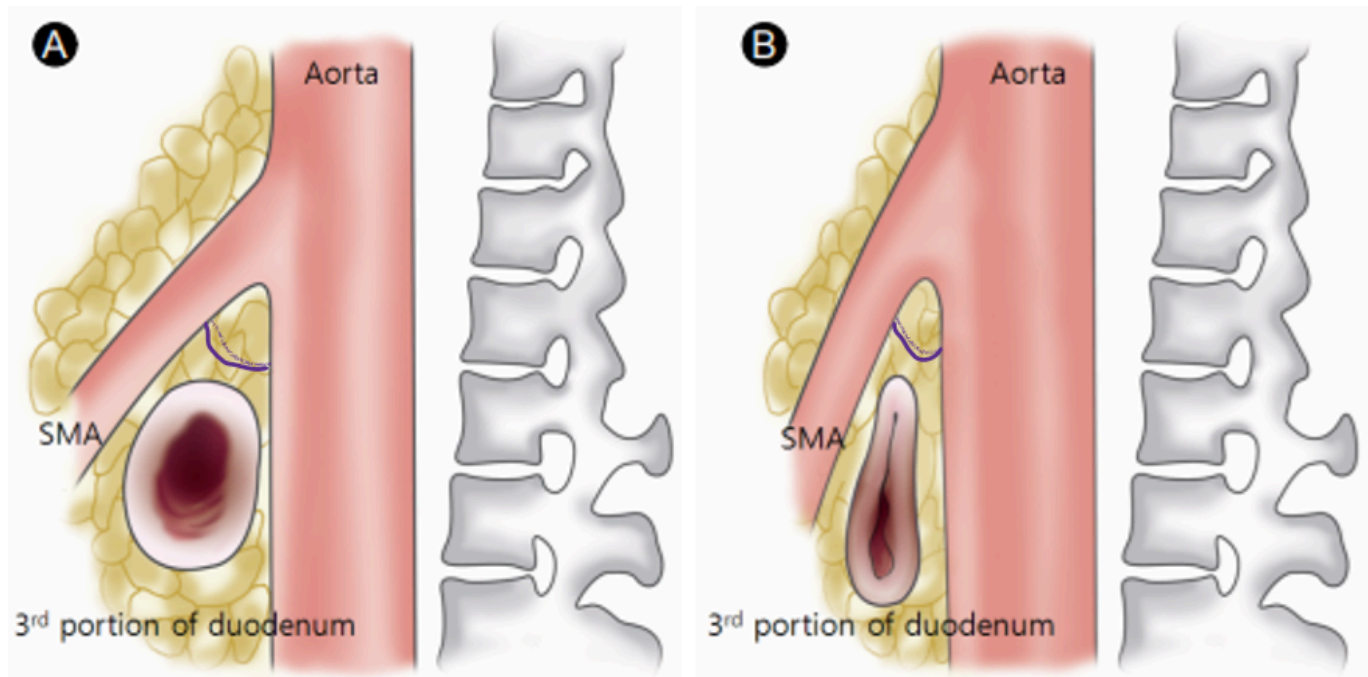
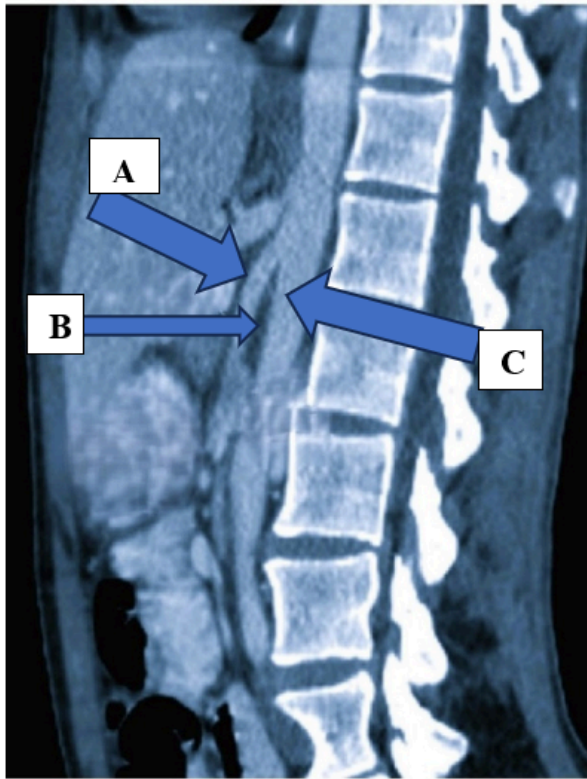
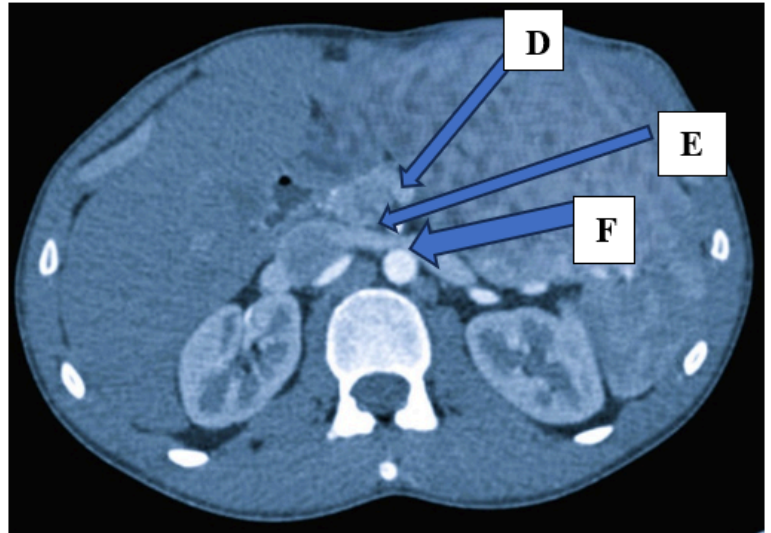


Figure 1

Sagittal view of the superior mesenteric artery and the aorta. **(A)** Normal aortomesenteric angle is 38 to 56 degrees and aortomesenteric distance is 10–25 mm. **(B)** In SMA syndrome the aortomesenteric angle is less than 25 degrees and distance less than 8 mm, leading to compression of the third portion of the duodenum between the aorta and the SMA. (16)



a



b

Figure 2

Computed tomography (CT) of the abdomen **(a)**Computed tomography (CT) of the abdomen with contrast, sagittal view, showing narrowing of the aortomesenteric angle between the SMA **(A)** and aorta **(C)**compressing the third portion of the duodenum **(B)**; **(b)** Axial CT scan of the upper abdomen demonstrating the superior mesenteric artery **(D)** and aorta **(F)** with the third portion of the duodenum compressed between them **(E)**.