THE CHALLENGING MANAGEMENT OF ACUTE MESENTERIC ISCHEMIA: A CASE REPORT OF A 45-YEAR-OLD WOMAN WITH SHORT BOWEL SYNDROME

Andreea Pușcașu[1], Dan Nicolae Păduraru[1,2], Alexandra Bolocan[1,2], Daniel Ion[1,2], Florentina Mușat[1,2], Octavian Andronic[1,2,3]

[1]Carol Davila University of Medicine and Pharmacy, Bucharest, Romania
[2]University Emergency Hospital of Bucharest, Romania
[3]Research Institute of the University of Bucharest, Bucharest, Romania

CASE REPORT

Doi: 10.33695/rojes.v5i2.74
Accepted: 11.12.2023

Corresponding author:
Alexandra Bolocan
alexandra.bolocan@umfcd.ro

Abstract

This case report explores the fatal prognosis of acute mesenteric ischemia leading to short bowel syndrome in a 45-year-old female patient. Acute mesenteric ischemia, a rare but critical condition, is caused by impaired blood flow to the intestines, necessitating prompt diagnosis and treatment to mitigate high mortality risks. The patient's case is complex, involving multiple surgeries for ischemic bowel resection, resulting in short bowel syndrome, characterized by a severe reduction in functional intestinal length. This syndrome presents significant challenges, including malnutrition and the necessity for long-term parenteral nutrition, which are difficult to manage and significantly impair quality of life. Despite surgical success in bowel resection, the patient's condition deteriorated due to complications from short bowel syndrome and resulted in her death six months post-surgery. This report underscores the need for early diagnosis and intervention in acute mesenteric ischemia, the limitations of current surgical treatments, and the dire consequences of short bowel syndrome. It also highlights the potential yet limited role of intestinal transplantation in managing these complex cases.

Keywords: acute mesenteric ischemia, short bowel syndrome, parenteral nutrition

Introduction

Mesenteric infarction is a surgical pathology caused by impaired blood perfusion in a segment of the small intestine, leading to intestinal ischemia and necrosis, with severe consequences and a fatal prognosis in the absence of proper treatment [1]. Acute mesenteric ischemia is associated with an extremely high mortality rate, between 60 and 80%, even if it affects only 0.09-0.2% of people in the global population [2]. The survival rate is correlated with the rapidity of the diagnosis because, the more time passes after the onset of symptoms, the intestinal necrosis extends. The mortality rate varies between 0 and 10% in the case of immediate treatment, increases to 50% when diagnosis and treatment are delayed by 6-12 hours, and reaches 80-100% if medical care is delayed for 24 hours or more [3].

Acute mesenteric ischemia is classified by the physiopathological mechanism into four distinct categories: arterial embolism, arterial

thrombosis, nonocclusive mesenteric ischemia (NOMI) and venous thrombosis [4]. The most common cause of mesenteric ischemia is arterial embolism, which is found in approximately 50% of all cases [5], and is associated with a cardiac pathology such as valvular dysfunction or atrial fibrillation [6]. The second cause of mesenteric ischemia is arterial thrombosis, in 25% of cases, and is related to an existing chronic atherosclerotic disease [7]. Due to the slow progression of atherosclerosis, the adaptation mechanisms improve, causing a delay in clinical presentation and worsening the prognosis, which leads to a perioperative mortality around 95% because of the extensive ischemia [8], [9]. NOMI occurs in approximately 20% of mesenteric infarction cases as a consequence of severe vasoconstriction of the superior mesenteric artery or decreased blood flow in the splanchnic circulation [10], caused by trauma or surgical stress [11]. It is very important to accurately diagnose this form because the correct treatment includes volume resuscitation and a vasodilator administration, not a surgical procedure [12], [13]. The rarest type of mesenteric ischemia is caused by venous thrombosis, found in less than 5-15% of cases [14]. It associates the lowest mortality rate, approximately 44%, due to the slower progression of ischemia induced by the venous thrombus [15].

The clinical presentation of acute mesenteric ischemia includes sudden onset of intense abdominal pain, nausea, vomiting and diarrhea. Other severe clinical signs such as fever and hemodynamic instability appear when the disease progresses with the peritoneum involvement [4]. Modified biological parameters show leukocytosis and metabolic acidosis with increased value of D-dimer and serum lactate, specific to ischemia [16]. The diagnosis of the mesenteric infarction is confirmed with the CT results, a gold standard for the diagnosis process [17], describing vascular calcifications, intramural hemorrhage and intravascular thrombi [5].

Surgical treatment involves the restoration of blood perfusion to the segment of the ischemic bowel, the resection of the irreversible damaged part and the preservation of the viable regions of the intestine [5],[18]. Although large resection is indicated, it should be limited to a minimum remaining length of the small intestine that allows post-operative functionality because extensive resections can lead to short bowel syndrome associated with the need for permanent parenteral nutrition or small bowel transplantation [19]. Long-term parenteral nutrition involves significant complications in which case is required small bowel transplantation as the only solution for overcoming malnutrition. Unfortunately, this procedure implies an increased risk of graft rejection due to the intestine's immunogenicity and requires the administration of high doses of immunosuppressant drugs [17]. The postoperative prognosis is only 5 years after the surgical procedure, with 35-79% chances of complication occurrence [20], [21].

Case presentation

A 45-year-old female patient presented at the emergency room for intense, diffuse abdominal pain, nausea and vomiting, with a sudden onset six hours ago. Her medical history revealed an appendectomy and chronic treatment with contraceptives, along with smoking as a risk factor. The clinical examination presented diffuse and generalized abdominal pain, spontaneously and on palpation, with signs of peritoneal irritation. The modified biological parameters on admission were leukocytosis with a value of $19.7/10^3/\mu L$ (normal values=3.8-11.8/10^3/ microliter), percentual neutrophilia of 93.8% (normal values=42.7-76.8 %), APTT of 19.4 seconds (normal values=22-36/seconds) and a hydro-electrolytic imbalance with hypernatremia and hypochloremia. Abdominal X-ray and ultrasound did not indicate any changes, but the CT examination highlighted an incomplete opacification at the origin of the

superior mesenteric artery, with a length over 23 millimeters, describing a suggestive appearance of mesenteric infarction.

Following this diagnosis, surgery was executed on the same day, performing an exploratory laparotomy to determine the viability of the bowel. The surgeon found that the intestine starting from 30 cm after the duodenojejunal flexure up to the ileocecal valve was ischemic, with a typical appearance of entero-mesenteric infarction, without the presence of peristaltic movement. The patient was postoperatively transferred to the Intensive Care Unit and treated with heparin in order to obtain reperfusion of the bowel and restore a larger part of the small intestine.

The second surgery was performed after four days, discovering still viable, but dilated parts of the small intestine, with a thick wall and without peristalsis, from the duodenojejunal flexure, for a length of 1.5 meters. The rest of the bowel, up to 3 centimeters before the ileocecal valve, was necrotic, perforated and with stercoral perforation. Small intestine resection of the necrotic portion was performed with a jejunostomy on the right side of the abdomen. The postoperative evolution was favorable, but the leukocytosis was still increasing and a significant stasis was also observed on the nasogastric tube. The CT scan performed six days after the surgery showed a proximal stenosis on the superior mesenteric artery of 75%, with conglomerated intestines in the left iliac fossa, with the presence of a mixed liquid accumulation (Figure 1 and 2). In addition, liquid accumulations were observed close to the sigmoid colon and root of the mesentery, which required a third surgical intervention. During the laparotomy, cecal ischemia was observed, as a consequence of the extension of the mesenteric infarction, and required a right hemicolectomy. The postoperative evolution was favorable and the patient was discharged after another nine days of recovery, along with Clexane and Tonotil-N as treatment.

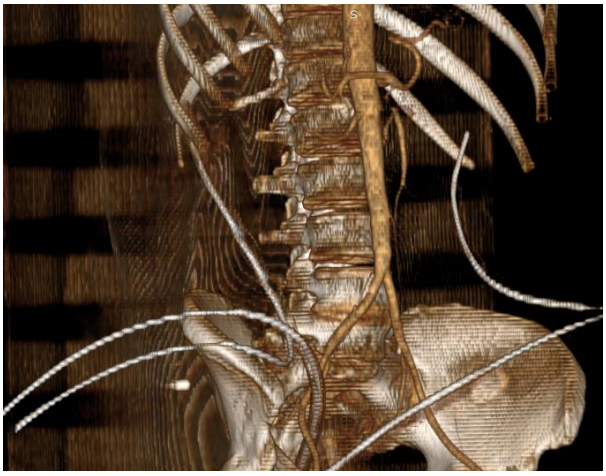

Figure 1 – 3D reconstruction showing the occlusion of superior mesenteric artery

The patient returned to the emergency room one day after the previous discharge with intense epigastric pain, with modified biological parameters such as leukocytosis ($19000/mm^3$), AST and ALT elevated as a sign of hepatocytolysis, increased amylase and lipase. The radiological examination showed multiple air-fluid levels of the remnant small intestine in the umbilical region, presacral space and left side of the abdomen, while the CT scan described circumferential edema in the proximal jejunal loops with hydro-distended and hypotonic segments. A surgical reintervention was decided, where a significant adhesion process was discovered, performing viscerolysis in order to restore the local anatomy. After 20 centimeters from Treitz's angle, there was observed an area of 30 centimeters of ischemic jejunum, followed by approximately 15 centimeters of viable bowel. The jejunostomy on the right side of the abdomen was abolished and a partial enterectomy was performed on 45-50 centimeters of the jejunum, followed by a jejunostomy on the left side of the abdomen. The postoperative evolution was slowly favorable and the patient was discharged after 14 days after the last surgery, following a home treatment with Clexane, Tonotil-N and Ciprofloxacin.

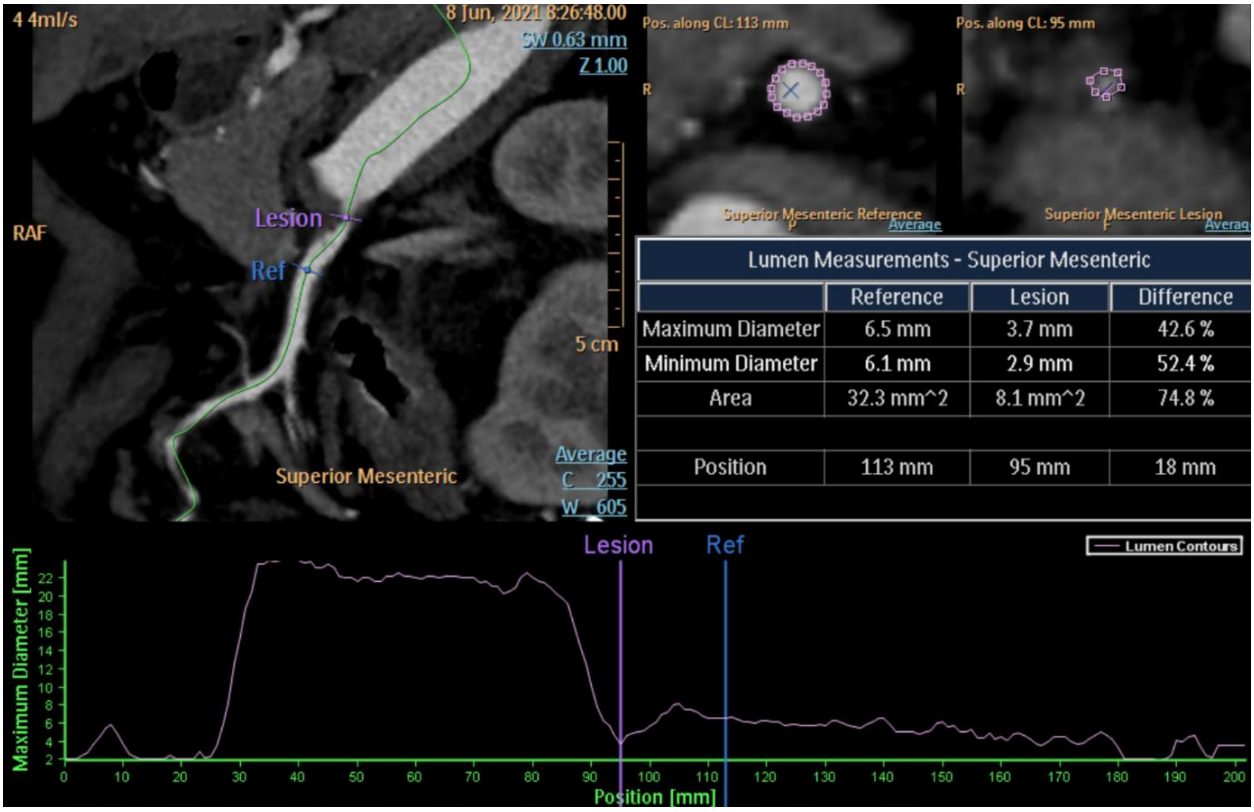

Figure 2 – CT image showing the occlusion of superior mesenteric artery

Although the 45-year-old patient was considered surgically cured when she was discharged, the complications of massive intestine resection occurred. The removal of a significant part of the jejunum, the entire ileum and the right part of the colon led to a short bowel syndrome, related to an unfavorable prognosis. The patient had progressively deteriorated despite all the medical efforts to maintain nutritional support and she died six months after her last surgery.

Discussions

This case report highlights a less common complication of acute mesenteric ischemia discussed in the medical literature, the short bowel syndrome, responsible for the fatal prognosis of the 45-year-old patient. The occurrence of this syndrome in patients with mesenteric infarction is almost inevitable because the surgical treatment involves resection of the irreversible ischemic areas of the bowel to prevent peritonitis and bacterial translocation [22]. The study by Thompson et al. demonstrated that mesenteric infarction is associated with a larger resection of the small intestine, up to a remaining length of less than 60 centimeters, compared to other causes that lead to short bowel syndrome such as the presence of adhesions (57% versus 23%, $p< 0.05$) [23]. Another study carried out on a group of 83 patients with mesenteric infarction described an average remnant small bowel of 40-60 centimeters. Moreover, there was an association between the length of the viable intestine and the survival rate, showing an average of 37-64 centimeters in the patients who survived, respectively of 33-57 centimeters in deceased patients [24]. This information emphasizes the fact that acute mesenteric ischemia is related to a decreased length of the remnant viable bowel, which involves a higher mortality rate.

The short bowel syndrome produced by the surgical treatment of acute mesenteric ischemia is a serious medical issue, accompanied by an impairment of the quality of life and an increased mortality rate. The survival rate associated with short bowel syndrome varies according to age, counting as 84% for adults younger than 41 years and 53% for older adults [25]. The prognosis is better when the duodenum and colon remain intact and the jejunum is longer than 200 centimeters [26], which explains the poor prognosis of the 45-year-old patient with a right hemicolectomy, total ileum resection and jejunostomy with a viable jejunum of approximately 20 centimeters.

In order to explain the unfavorable progress of the treatment in the studied case, considering the young age and the lack of comorbidities, we must reflect on the implicit complications of the short bowel syndrome and how it affects the postoperative evolution and mortality rate. The earliest complications of this syndrome are dehydration, found in 18.1% of patients and hydro-electrolytic imbalance in 52.5%, especially for the electrolytes such as magnesium, calcium and potassium [27]. These imbalances are difficult to correct because oral supplementation can cause osmotic diarrhea and rapid intravenous supplementation can increase the renal excretion of these electrolytes, without correcting the imbalance [26].

Long-term complications of short bowel syndrome include malnutrition [28] and the need for long-term parenteral nutrition [29]. In the case of the 45-year-old patient, malnutrition should have been managed by maintaining a hypercaloric and hyperproteic diet, respectively by following a treatment with Tonotil-N, an oral nutritional supplementation with glucose, amino acids and vitamins. Although nutrient supplementation was attempted, jejunostomy associated with a very short length of viable jejunum leads to an accelerated transit and rapid emptying of the stomach by decreasing the hormonal secretion of polypeptide YY and enteroglucagon, which diminishes the nutrient absorption by reducing the time of interaction with the intestinal villi. This increased transit leads to an excretion of a high amount of water and electrolytes into the lumen to regulate endoluminal hyperosmolarity, which promotes dehydration and hydro-electrolyte imbalance [30], [31]. In addition, the terminal ileum is responsible for the reabsorption of bile acids, which means that its absence leads to a decreased level of these substances, affecting the absorption of lipids, an imbalance that cannot be corrected by hypercaloric nutrition [32].

Short bowel syndrome in those with required jejunostomy after acute mesenteric ischemia resulted in a long-term need for parenteral nutrition. A retrospective study on 113 patients described that parenteral nutrition in short-term management was stopped after one year in only 20 patients, after 2 years in 29 patients and after 5 years in 44 patients, the rest of them continuing for a longer period of time [33]. Long-term parenteral nutrition, although essential for the metabolic needs of the patient with short bowel syndrome, implies complications that affect the survival rate. It decreases progressively with the duration of the parenteral nutrition administration, being 86% after 1 year, 77% after 2 years, 73% after 5 years and 71% after 10 years [34]. Among the complications of parenteral nutrition, there are listed septic events in the vascular approach area, hyperglycemia, hepatobiliary disease and bone disease [35].

The only potential solution to redress malnutrition caused by short bowel syndrome after mesenteric infarction is intestinal transplantation. This procedure is hard to obtain due to the small number of patients eligible for intestinal transplantation. A study conducted on 711 patients with acute mesenteric ischemia concluded that only 6 persons were eligible for this procedure [36]. The most common complications of intestinal transplantations are postoperative infections

found in 97% of patients, acute cellular rejection in 50-75% of cases and chronic kidney disease in 25% of patients, caused by the the intraluminal microbiota and the large lymphoid tissue found in the structure of the bowel [37]. The survival rate in patients with intestinal transplantation seems to be similar to those who continue parenteral nutrition, emphasizing the fact that intestinal transplantation is not the ideal solution for patients with short bowel syndrome [38].

Conclusions

The case of this 45-year-old patient highlights a medical problem less common in literature- the fatal outcome of mesenteric infarction, a pathology with a lack of efficient treatment. Even if acute mesenteric ischemia involves a poor prognosis itself, the patients who survive continue to have a high mortality rate due to the unavoidable short bowel syndrome. Many patients considered surgically cured are affected by the complications of the short bowel syndrome, especially the untreatable malnutrition or long-term parenteral nutrition and its disadvantages.

The only promising and lasting solution for managing the short bowel syndrome acquired from mesenteric ischemia may be intestinal transplantation, but the procedure is limited by this unfavorable area of the bowel, notable for its immunogenicity and various microbiota.

References

[1] A. Patel, R. N. Kaleya, and R. J. Sammartano, "Pathophysiology of Mesenteric Ischemia," Surgical Clinics of North America, vol. 72, no. 1, pp. 31–41, Feb. 1992, doi: 10.1016/S0039-6109(16)45626-4.

[2] A. Gragossian, M. E. Shaydakov, and P. Dacquel, "Mesenteric Artery Ischemia," StatPearls, Aug. 2022. Available: https://www.ncbi.nlm.nih.gov/books/NBK513354

[3] "Acute mesenteric ischemia natural history, complications and prognosis - wikidoc.". Available: https://www.wikidoc.org/index.php/Acute_mesenteric_ischemia_natural_history,_complications_and_prognosis

[4] W. A. Oldenburg, L. L. Lau, T. J. Rodenberg, H. J. Edmonds, and C. D. Burger, "Acute mesenteric ischemia: a clinical review," Arch Intern Med, vol. 164, no. 10, pp. 1054–1062, May 2004, doi: 10.1001/ARCHINTE.164.10.1054.

[5] M. Bala et al., "Acute mesenteric ischemia: Guidelines of the World Society of Emergency Surgery," World Journal of Emergency Surgery, vol. 12, no. 1, pp. 1–11, Aug. 2017, doi: 10.1186/S13017-017-0150-5/FIGURES/3.

[6] K. S. Choi et al., "Percutaneous Aspiration Embolectomy Using Guiding Catheter for the Superior Mesenteric Artery Embolism," Korean J Radiol, vol. 16, no. 4, pp. 736–743, Aug. 2015, doi: 10.3348/KJR.2015.16.4.736.

[7] M. Bala et al., "Acute mesenteric ischemia: guidelines of the World Society of Emergency Surgery", doi: 10.1186/s13017-017-0150-5.

[8] S. Florim, A. Almeida, D. Rocha, and P. Portugal, "Acute mesenteric ischaemia: a pictorial review," Insights Imaging, vol. 9, no. 5, pp. 673–682, Oct. 2018, doi: 10.1007/S13244-018-0641-2/FIGURES/12.

[9] M. A. Mansour, "Management of acute mesenteric ischemia," Arch Surg, vol. 134, no. 3, pp. 328–331, 1999, doi: 10.1001/ARCHSURG.134.3.328.

[10] A. J. Aschoff et al., "Evaluation of acute mesenteric ischemia: accuracy of biphasic mesenteric multi-detector CT angiography," Abdominal Imaging 2008 34:3, vol. 34, no. 3, pp. 345–357, Apr. 2008, doi: 10.1007/S00261-008-9392-8.

[11] G. C. Hunter and J. M. Guernsey, "Mesenteric Ischemia," Medical Clinics of North America, vol. 72, no. 5, pp. 1091–1115, Sep. 1988, doi: 10.1016/S0025-7125(16)30731-3.

[12] G. Salamone et al., "Non-occlusive mesenteric Ischemia (NOMI) in Parkinson's disease: Case report," Giornale di Chirurgia, vol. 38, no. 2, pp. 71–76, 2017, doi: 10.11138/gchir/2017.38.2.071.

[13] R. N. Kaleya, R. J. Sammartano, and S. J. Boley, "Aggressive approach to acute mesenteric ischemia," Surg Clin North Am, vol. 72, no. 1, pp.

157–182, 1992, doi: 10.1016/S0039-6109(16)45633-1.
[14] E. Sulger, H. S. Dhaliwal, A. Goyal, and L. Gonzalez, "Mesenteric Venous Thrombosis," StatPearls, Jul. 2022. Available: https://www.ncbi.nlm.nih.gov/books/NBK459184
[15] I. G. Schoots, G. I. Koffeman, D. A. Legemate, M. Levi, and T. M. Van Gulik, "Systematic review of survival after acute mesenteric ischaemia according to disease aetiology," Br J Surg, vol. 91, no. 1, pp. 17–27, Jan. 2004, doi: 10.1002/BJS.4459.
[16] P. Kougias, D. Lau, H. F. el Sayed, W. Zhou, T. T. Huynh, and P. H. Lin, "Determinants of mortality and treatment outcome following surgical interventions for acute mesenteric ischemia," J Vasc Surg, vol. 46, no. 3, pp. 467–474, Sep. 2007, doi: 10.1016/J.JVS.2007.04.045.
[17] F. Kühn, T. S. Schiergens, and E. Klar, "Acute Mesenteric Ischemia," Visc Med, vol. 36, no. 4, pp. 256–263, Aug. 2020, doi: 10.1159/000508739.
[18] M. C. Wyers, "Acute mesenteric ischemia: diagnostic approach and surgical treatment," Semin Vasc Surg, vol. 23, no. 1, pp. 9–20, Mar. 2010, doi: 10.1053/J.SEMVASCSURG.2009.12.002.
[19] M. Bala et al., "Acute mesenteric ischemia: Guidelines of the World Society of Emergency Surgery," World Journal of Emergency Surgery, vol. 12, no. 1, pp. 1–11, Aug. 2017, doi: 10.1186/S13017-017-0150-5/FIGURES/3.
[20] T. W. Carver, R. S. Vora, and A. Taneja, "Mesenteric Ischemia," Crit Care Clin, vol. 32, no. 2, pp. 155–171, Apr. 2016, doi: 10.1016/J.CCC.2015.11.001.
[21] A. L. Menges, K. Stoklasa, L. Meuli, B. Reutersberg, and A. Zimmermann, "Chronic Mesenteric Ischemia," Gefasschirurgie, vol. 27, no. 6, pp. 435–443, Aug. 2022, doi: 10.1007/s00772-022-00931-z.
[22] D. Yildirim, A. Hut, C. Tatar, T. Donmez, M. Akinci, and M. Toptas, "Prognostic factors in patients with acute mesenteric ischemia," Turk J Surg, vol. 33, no. 2, p. 104, 2017, doi: 10.5152/UCD.2016.3534.
[23] J. S. Thompson, J. K. DiBaise, K. R. Iyer, M. Yeats, and D. L. Sudan, "Postoperative Short Bowel Syndrome," J Am Coll Surg, vol. 201, no. 1, pp. 85–89, Jul. 2005, doi: 10.1016/j.jamcollsurg.2005.02.034.
[24] A. Sitges-Serra, X. Mas, F. Roqueta, J. Figueras, and F. Sanz, "Mesenteric infarction: an analysis of 83 patients with prognostic studies in 44 cases undergoing a massive small-bowel resection," British Journal of Surgery, vol. 75, no. 6, pp. 544–548, Dec. 2005, doi: 10.1002/bjs.1800750614.
[25] M. Cavicchi, P. Beau, P. Crenn, C. Degott, and B. Messing, "Prevalence of Liver Disease and Contributing Factors in Patients Receiving Home Parenteral Nutrition for Permanent Intestinal Failure," Ann Intern Med, vol. 132, no. 7, p. 525, Apr. 2000, doi: 10.7326/0003-4819-132-7-200004040-00003.
[26] C. L. Donohoe and J. V. Reynolds, "Short bowel syndrome," The Surgeon, vol. 8, no. 5, pp. 270–279, Oct. 2010, doi: 10.1016/j.surge.2010.06.004.
[27] M. T. Siddiqui, W. Al-Yaman, A. Singh, and D. F. Kirby, "Short-Bowel Syndrome: Epidemiology, Hospitalization Trends, In-Hospital Mortality, and Healthcare Utilization," Journal of Parenteral and Enteral Nutrition, vol. 45, no. 7, pp. 1441–1455, Sep. 2021, doi: 10.1002/jpen.2051.
[28] L. E. Matarese, S. J. O'Keefe, H. M. Kandil, G. Bond, G. Costa, and K. Abu-Elmagd, "Short Bowel Syndrome: Clinical Guidelines for Nutrition Management," Nutrition in Clinical Practice, vol. 20, no. 5, pp. 493–502, Oct. 2005, doi: 10.1177/0115426505020005493.
[29] C. F. Brandt et al., "Home Parenteral Nutrition in Adult Patients With Chronic Intestinal Failure: The Evolution Over 4 Decades in a Tertiary Referral Center," Journal of Parenteral and Enteral Nutrition, vol. 41, no. 7, pp. 1178–1187, Sep. 2017, doi: 10.1177/0148607116655449.
[30] J. Nightingale, "Guidelines for management of patients with a short bowel," Gut, vol. 55, no. suppl_4, pp. iv1–iv12, Aug. 2006, doi: 10.1136/gut.2006.091108.
[31] S. Massironi, F. Cavalcoli, E. Rausa, P. Invernizzi, M. Braga, and M. Vecchi, "Understanding short bowel syndrome: Current status and future perspectives," Digestive and Liver Disease, vol. 52, no. 3, pp. 253–261, Mar. 2020, doi: 10.1016/j.dld.2019.11.013.
[32] B. Bielawska and J. Allard, "Parenteral Nutrition and Intestinal Failure," Nutrients, vol. 9, no. 5, p. 466, May 2017, doi: 10.3390/nu9050466.

[33] F. Adaba et al., “Mesenteric Infarction,” Ann Surg, vol. 262, no. 6, pp. 1059–1064, Dec. 2015, doi: 10.1097/SLA.0000000000001100.
[34] D. A. J. Lloyd, R. Vega, P. Bassett, A. Forbes, and S. M. Gabe, “Survival and dependence on home parenteral nutrition: experience over a 25-year period in a UK referral centre,” Aliment Pharmacol Ther, vol. 24, no. 8, pp. 1231–1240, Oct. 2006, doi: 10.1111/J.1365-2036.2006.03106.X.
[35] A. Ukleja and M. M. Romano, “Complications of Parenteral Nutrition,” Gastroenterol Clin North Am, vol. 36, no. 1, pp. 23–46, Mar. 2007, doi: 10.1016/j.gtc.2007.01.009.
[36] A. Lemma et al., “Potential for intestinal transplantation after acute mesenteric ischemia in patients aged less than 70 years: A population-based study,” Scandinavian Journal of Surgery, vol. 112, no. 2, pp. 77–85, Jun. 2023, doi: 10.1177/14574969231151374/ASSET/IMAGES/LARGE/10.1177_14574969231151374-FIG2.JPEG.
[37] S. Kesseli and D. Sudan, “Small Bowel Transplantation,” Surgical Clinics of North America, vol. 99, no. 1, pp. 103–116, Feb. 2019, doi: 10.1016/j.suc.2018.09.008.
[38] D. Sudan, “Long-term outcomes and quality of life after intestine transplantation,” Curr Opin Organ Transplant, vol. 15, no. 3, pp. 357–360, Jun. 2010, doi: 10.1097/MOT.0b013e3283398565.