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Ileocecal Perforation with Tuberculous Sclerosing Encapsulating Peritonitis Complicated by Splenic Infarction and Enterocutaneous Fistula in a Young Immunocompetent Man: A Case Report

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ABSTRACT :

Abdominal tuberculosis most often involves the ileocecal region; free perforation is uncommon and highly lethal, and its coexistence with tuberculous sclerosing encapsulating peritonitis, splenic infarction and enterocutaneous fistula is rarely reported. A 26-year-old immunocompetent man with untreated interferon-gamma release assay–positive tuberculosis and 25 days of oral deflazacort presented with one day of abdominal pain, vomiting, fever and obstipation, in septic shock (blood pressure 80/60 mmHg, creatinine 2.1 mg/dL, procalcitonin 27.01 ng/mL). Computed tomography showed small-bowel perforation. At emergency laparotomy the abdomen was entirely plastered, with 1.5 L of faeculent fluid, a 5 × 3 cm ileocecal perforation and a separate 3 × 3 cm caecal perforation; segmental resection with end ileostomy was performed. Recovery was complicated by refractory shock, acute kidney injury, wound dehiscence, thrombocytosis peaking at 1,029,000/mm³, a splenic infarct involving 50–70% of the parenchyma with hilar arterial thrombus, and a low-output enterocutaneous fistula. Histopathology showed necrotising granulomatous inflammation at four sites. Antitubercular therapy began on postoperative day 17; the fistula and infarct were managed conservatively and the patient was discharged ambulant on day 24. Corticosteroids given for untreated tuberculous infection can precipitate perforation, and within an abdominal cocoon resection with proximal diversion is safest.

Keywords: *abdominal tuberculosis, ileocecal perforation, sclerosing encapsulating peritonitis, abdominal cocoon, splenic infarction, enterocutaneous fistula, septic shock*

INTRODUCTION

Tuberculosis remains one of the leading infectious causes of death worldwide, and India continues to carry the largest share of the global burden.[1] Extrapulmonary disease accounts for roughly one in five notified cases, and the abdomen is among the commonest extrapulmonary sites.[2,4] Within the abdomen, the ileocecal region is involved most frequently, a predilection attributed to physiological stasis, abundant lymphoid tissue in the Peyer's patches and the high rate of fluid and electrolyte absorption that favours bacillary contact with the mucosa.[3]

The clinical spectrum of intestinal tuberculosis is dominated by indolent presentations — chronic abdominal pain, subacute obstruction, weight loss and low-grade fever — and the diagnosis is frequently delayed because the symptoms mimic Crohn's disease, lymphoma and malignancy.[3] Free perforation is a comparatively uncommon complication, reported in a minority of patients with intestinal tuberculosis, but it carries a disproportionate mortality because it occurs in malnourished, often immunosuppressed hosts with friable, poorly vascularised bowel and a hostile peritoneal environment.[5,6]

Two further entities occasionally complicate abdominal tuberculosis and each is individually uncommon. Sclerosing encapsulating peritonitis, also termed the abdominal cocoon, is a fibro collagenous encasement of the viscera that transforms the peritoneal cavity into a plastered, surgically hostile field.[7,8] Splenic vascular events - arterial or venous thrombosis with infarction are described in disseminated tuberculosis but remain rare in the setting of localised intestinal disease.[9] Enterocutaneous fistula, on the other hand, is a well-recognised and greatly feared sequel of emergency laparotomy for perforation peritonitis, and it substantially prolongs hospitalisation and increases mortality.[10]

We report a young immunocompetent man in whom untreated, interferon-gamma release assay–positive tuberculosis, unmasked by a short course of oral corticosteroid, progressed to a double ileocecal perforation within a tuberculous abdominal cocoon, and whose postoperative course was complicated by refractory septic shock, an extensive splenic infarct with splenic arterial thrombus and a low-output enterocutaneous fistula. To our knowledge, this constellation of complications occurring together in a single immunocompetent patient who survived has not previously been described.

CASE DESCRIPTION

A 26-year-old man presented to the emergency department with diffuse abdominal pain of one day's duration, associated with progressive abdominal distension, seven episodes of non-bilious, non-blood-stained vomiting, and fever with chills and rigors. He had not passed stool for three days and had not passed flatus since the previous evening. He reported a marked reduction in urine output. There was no history of burning micturition, loose stools, haematemesis or breathlessness.

His past history was significant. He had been treated for low back pain for approximately three years; magnetic resonance imaging of the lumbosacral spine performed seven months earlier at another centre had reported multilevel disc desiccation, diffuse disc bulges from L2–L3 to L5–S1 with thecal sac indentation and bilateral neural foraminal narrowing, reported as lumbar disc disease. An interferon-gamma release assay performed during that evaluation was positive and spinal tuberculosis was suspected, but antitubercular therapy was never commenced. For the ten days preceding admission he had been taking salicylate 1000 mg, deflazacort 6 mg once daily (prescribed for 25 days), etoricoxib 90 mg and a tricyclic agent for back pain. He had a similar self-limiting episode of abdominal pain two months earlier for which he had been treated conservatively elsewhere.

The family history was striking: his father had been diagnosed with pulmonary tuberculosis in 2023 and had taken treatment for three months, and his sister had splenic tuberculosis and had completed six months of antitubercular therapy three months before this admission. He had no diabetes, hypertension, bronchial asthma, thyroid disease, epilepsy or coronary disease, no previous surgery, no known drug allergy and no history of alcohol or tobacco use.

EXAMINATION AND RESUSCITATION

On arrival he was conscious, oriented and febrile, with a Glasgow Coma Scale of 15/15 and evidence of severe dehydration. Blood pressure was 80/60 mmHg, pulse rate 135 beats/min, respiratory rate 16/min, oxygen saturation 97% on room air and temperature 98.6°F. The abdomen was distended with localised guarding over the right iliac fossa, diffuse tenderness and sluggish bowel sounds; the rectum was empty on digital examination. Cardiovascular and respiratory examinations were unremarkable and there was no focal neurological deficit. Bedside echocardiography showed a normal left ventricular ejection fraction of 59% with no regional wall motion abnormality, mild tricuspid regurgitation and a collapsing inferior vena cava measuring 1.2 cm, consistent with hypovolaemia rather than cardiogenic shock. He received 3 L of crystalloid in accordance with contemporary sepsis guidance but remained hypotensive with a urine output of only 10 mL, and a noradrenaline infusion was commenced for refractory septic shock.[11]

IMAGING

Plain computed tomography of the abdomen and pelvis demonstrated focal fluid-filled prominence of ileal loops in the hypogastric region with focal wall irregularity of the distal ileum at the right iliac fossa, a large loculated central mesenteric collection containing internal air pockets, diffuse mesenteric haziness, multiple enlarged right iliac fossa and central mesenteric nodes, mild diffuse fatty infiltration of the liver, bilateral perinephric stranding and minimal bilateral pleural effusion. The reported impression was small-bowel perforation complicated by intra-abdominal collection and diffuse peritonitis. Plain computed tomography of the thorax showed small centrilobular nodules in the right upper lobe and small bilateral pleural effusions, with no mediastinal or hilar adenopathy.

OPERATIVE FINDINGS AND PROCEDURE

After counselling the patient and his family regarding refractory septic shock, acute kidney injury, the possible need for dialysis, prolonged ventilation and a guarded prognosis, high-risk consent was obtained and an emergency laparotomy was performed on the day of admission under general anaesthesia with epidural analgesia.

Through a midline incision, the entire abdomen was found to be plastered. The liver, spleen, stomach, omentum and transverse colon could not be visualised as they were completely adherent to the anterior abdominal wall, the appearance being that of sclerosing encapsulating peritonitis. Approximately 1.5 L of peritoneal fluid mixed with faecal matter was evacuated. The perforation site could not be identified at diagnostic laparotomy and was located only after systematic interbowel adhesiolysis; the bowel was mobilised from the duodenojejunal flexure to 15 cm above the ileocecal junction. The terminal ileum was densely adherent to the bladder wall and to the peritoneal reflection, and these adhesions were released by combined blunt and sharp dissection. A large perforation measuring 5 × 3 cm was identified at the ileocecal junction together with a separate 3 × 3 cm perforation in the caecum, with completely sloughed-out surrounding tissue; the appendix could not be visualised.

A limited segmental resection was performed 5 cm proximal to the unhealthy ileum and 5 cm distal to the perforated caecal segment. Given the degree of peritoneal contamination, the friability of the bowel and haemodynamic instability, restoration of continuity was not attempted. The distal colonic loop

was closed in two layers and fixed to the lateral peritoneal wall, and the proximal ileal loop was brought out as an end ileostomy. Thorough peritoneal lavage was performed, haemostasis secured, a pelvic drain placed, the fascia closed with 1-0 polypropylene and the skin approximated with staples.[4,6]

POSTOPERATIVE COURSE

The patient was transferred to the intensive care unit in view of persistent refractory hypotension. He was maintained on mechanical ventilation and extubated on the first postoperative day. Management comprised intravenous fluids, broad-spectrum antibiotics, vasopressor support, analgesia, proton pump inhibitors, anticoagulation, nebulisation and chest physiotherapy, with early stoma care.

The stoma was noted to be flush with the skin on postoperative day 2 and a surgical gastroenterology opinion was sought regarding possible stoma revision. Hypokalaemia (serum potassium 2.9 mmol/L) was identified on day 3 and corrected with an internal medicine opinion on day 4. The lower half of the laparotomy wound was seen to gape on day 5, and daily dressings were instituted.

On postoperative day 8, non-contrast followed by contrast-enhanced computed tomography of the abdomen was performed for persistent sepsis. This demonstrated a large, irregular hypodense area measuring 12.5 × 7.2 × 11.4 cm involving 50–70% of the splenic parenchyma, predominantly in the upper and mid pole with subcapsular extension, together with abrupt luminal narrowing of the proximal third of the superior hilar branch of the splenic artery — reported as a splenic infarct with superior hilar division thrombus. Postoperative changes were seen in the surgical bed with small loculated collections in both paracolic gutters extending into the hypogastrium and pelvis, diffuse mesenteric fat stranding with multiple enlarged central mesenteric nodes, basal left lower lobe consolidation, a small left pleural effusion and fatty liver. The collections were small, peripherally enhancing and did not require percutaneous drainage. A blood transfusion was given on day 7 for a haemoglobin nadir of 7.1 g/dL.

Sputum culture obtained on day 3 grew a carbapenem-resistant, extended-spectrum beta-lactamase–producing Klebsiella pneumoniae, resistant to all agents on the standard panel including meropenem, imipenem, ertapenem, piperacillin–tazobactam, amikacin and ciprofloxacin. Extended testing showed susceptibility only to ceftazidime–avibactam, with colistin intermediate and tigecycline, minocycline and aztreonam resistant; ceftazidime–avibactam plus aztreonam synergy testing was positive. A pulmonology opinion was obtained to exclude active pulmonary tuberculosis. Blood and urine cultures showed no growth.

Histopathological examination of the resected specimen reported necrotising granulomatous inflammation with features of perforation in the perforated segment of the ileocecal region, with the possibility of tuberculosis to be considered. Five lymph nodes showed necrotising granulomas, the peritoneal biopsy showed necrotising granulomas and a mesenteric nodule showed a necrotising granuloma. On the basis of these findings, together with the positive interferon-gamma release assay and the family history, four-drug antitubercular therapy was commenced for abdominal tuberculosis on postoperative day 17, with pyridoxine supplementation.

On the same day an enterocutaneous fistula was noted in the lower third of the midline laparotomy wound. Contrast-enhanced computed tomography on postoperative day 18 confirmed a small, poorly walled linear extravasation of ingested oral contrast from the medial wall of the ileostomy loop tracking along the linea alba to the midline laparotomy incision, with a cluster of non-dilated clumped mid-ileal loops in the right iliac fossa, persistent interbowel adhesions, a thin-walled peripherally enhancing hypogastric collection of approximately 50–60 mL extending into the pelvis and a smaller left paracolic collection. The splenic infarct was unchanged.

The fistula was of low output and was managed conservatively. A wound bag was applied on postoperative day 22 alongside the stoma appliance to protect the skin, quantify effluent and allow granulation, and the patient was supported with a high-protein diet, correction of electrolyte and albumin deficits, control of sepsis and daily wound care in line with established principles of fistula management.[10] He improved symptomatically and clinically; the total leucocyte count fell from a peak of 41,220/mm³ on postoperative day 10 to 16,680/mm³, renal function normalised and he became ambulant and tolerant of oral feeds.

At discharge on postoperative day 24 he was afebrile and haemodynamically stable, with a blood pressure of 120/80 mmHg, pulse 73/min, oxygen saturation 96% on room air and temperature 96.9°F. The laparotomy wound was gaping with healthy granulation tissue and no purulent discharge, the enterocutaneous fistula persisted in the lower third of the wound, and the stoma was healthy and functioning well with both the stoma bag and the wound bag in situ. He was discharged on four-drug antitubercular therapy, pyridoxine, a proton pump inhibitor, paracetamol and low-dose bisoprolol, with advice on a high-protein diet, generous oral fluids, stoma and wound bag care, daily dressings and ambulation, and was reviewed in the surgical outpatient department one week later.

LABORATORY INVESTIGATIONS

Investigations on admission were consistent with septic shock and acute kidney injury. The total leucocyte count was 16,900/mm³ with 86.9% neutrophils,

haemoglobin 12.1 g/dL, platelet count 571,000/mm³, blood urea 38 mg/dL and serum creatinine 2.1 mg/dL. Serum albumin was 2.8 g/dL with a reversed albumin–globulin ratio of 0.8, and transaminases were normal. Procalcitonin was markedly elevated at 27.01 ng/mL (reference 0–0.5 ng/mL) and serum cortisol was 783.74 nmol/L, appropriate for the degree of physiological stress and excluding adrenal insufficiency despite recent steroid use. The prothrombin time was 15.7 s with an international normalised ratio of 1.29 and the activated partial thromboplastin time was normal at 28.0 s. Venous blood gas analysis showed a pH of 7.359, bicarbonate 21.6 mmol/L and a mildly raised anion gap. Serology for human immunodeficiency virus, hepatitis B surface antigen and hepatitis C virus was non-reactive, confirming an immunocompetent host.[11]

The subsequent trajectory of the haematological and biochemical parameters is summarised in Tables 1 and 2. Three features merit emphasis. First, the leucocyte count displayed a biphasic course, falling transiently to 11,000/mm³ on postoperative day 3 before rising to a peak of 41,220/mm³ on day 10 in association with the splenic infarct, the residual intra-abdominal collections and nosocomial respiratory infection, and settling thereafter. Second, the platelet count fell to 110,000/mm³ on day 3 during the consumptive phase of sepsis and subsequently rose progressively to an extreme reactive thrombocytosis of 1,029,000/mm³ on day 16, in keeping with the inflammatory state and functional hyposplenism after infarction. Third, acute kidney injury was rapidly reversible, with serum creatinine falling from 2.1 mg/dL on admission to 0.9 mg/dL by day 3 and 0.4–0.7 mg/dL thereafter, confirming a haemodynamic rather than intrinsic renal insult.

Serum albumin fell to a nadir of 1.6 g/dL on postoperative day 3, reflecting the combined effect of sepsis, capillary leak and the catabolic state, and recovered only partially to 2.3 g/dL by day 12 despite nutritional support a finding of prognostic relevance in the context of a developing enterocutaneous fistula. Serum potassium fell to 2.9 mmol/L on day 3 and calcium to 6.8 mg/dL; both were corrected, with magnesium measured at 1.8 mg/dL. Transient hyponatraemia (131 mmol/L on day 9) and a late transient hyperkalaemia (5.3 mmol/L on day 17) were managed conservatively. A summary of the microbiological, serological and histopathological findings is provided in Table 3.

Table 1. Serial haematological parameters

Parameter (reference range)	Day 0	POD 1	POD 3	POD 7	POD 10	POD 16	POD 24
Haemoglobin, g/dL (13–17)	12.1	11.6	7.1	8.8	8.7	8.8	11.4
Total leucocyte count, /mm³ (4,000–11,000)	16,900	28,300	11,000	20,670	41,220	15,960	16,680
Neutrophils, % (40–80)	86.9	94.9	93.8	93.6	94.3	78.4	80.5
Lymphocytes, % (20–40)	8.7	3.5	5.0	4.1	2.2	8.0	6.5
Platelet count, /mm³ (140,000–450,000)	571,000	309,000	110,000	323,000	809,000	1,029,000	822,000
Haematocrit, % (40–50)	37.5	36.1	22.3	26.1	25.8	25.3	31.6

POD, postoperative day. Day 0 denotes the day of admission and emergency laparotomy. The platelet nadir on POD 3 corresponds to the consumptive phase of sepsis; the subsequent rise to 1,029,000/mm³ represents reactive thrombocytosis following splenic infarction.

Table 2. Serial biochemical parameters

Parameter (reference range)	Day 0	POD 2	POD 3	POD 7	POD 9	POD 17
Blood urea, mg/dL (19–43)	38	72	57	27	14	15
Creatinine, mg/dL (0.66–1.25)	2.1	1.4	0.9	0.5	0.4	0.7
Sodium, mmol/L (135–145)	140	142	142	137	131	134
Potassium, mmol/L (3.5–5.0)	4.5	4.4	2.9	3.7	3.6	5.3
Calcium, mg/dL (8.5–10.5)	9.0	7.5	6.8	7.1	7.9	9.0
Total protein, g/dL (6.3–8.2)	6.2	4.4	4.1	–	5.3	–
Albumin, g/dL (3.5–5.0)	2.8	1.8	1.6	2.0	2.2	–
Total bilirubin, mg/dL (0.2–1.3)	1.18	1.27	0.84	–	0.70	–

Parameter (reference range)	Day 0	POD 2	POD 3	POD 7	POD 9	POD 17
AST, U/L (up to 40)	14	28	28	–	24	–
ALT, U/L (up to 65)	14	12	14	–	15	–

ALT, alanine aminotransferase; AST, aspartate aminotransferase; POD, postoperative day; –, not performed on that day.

Table 3. Key microbiological, serological and histopathological findings

Investigation	Timing	Result
HIV, HBsAg, anti-HCV (CLIA)	Day 0	Non-reactive
Procalcitonin	Day 0	27.01 ng/mL (0–0.5)
Serum cortisol	Day 0	783.74 nmol/L (morning 151.6–793.3)
Interferon-gamma release assay	7 months before admission	Positive
Blood culture	POD 1	Sterile after 5 days of incubation
Urine culture	POD 1	No growth
Sputum culture and sensitivity	POD 3	Klebsiella pneumoniae; carbapenem-resistant ESBL producer; susceptible only to ceftazidime–avibactam; colistin intermediate; ceftazidime–avibactam + aztreonam synergy positive
Histopathology – resected ileocaecal segment	POD 5 (report)	Necrotising granulomatous inflammation with perforation; tuberculosis to be considered
Histopathology – lymph nodes (n = 5)	POD 5 (report)	Necrotising granulomas
Histopathology – peritoneal biopsy	POD 5 (report)	Necrotising granulomas
Histopathology – mesenteric nodule	POD 5 (report)	Necrotising granuloma

CLIA, chemiluminescent immunoassay; ESBL, extended-spectrum beta-lactamase; HBsAg, hepatitis B surface antigen; HCV, hepatitis C virus; HIV, human immunodeficiency virus; POD, postoperative day.

DISCUSSION

This case illustrates, in a single patient, several of the most difficult problems posed by abdominal tuberculosis: a fulminant perforation in a host previously regarded as having only latent infection, a surgically hostile encapsulated peritoneal cavity, an uncommon splenic vascular complication and a postoperative enterocutaneous fistula. The ileocecal region accounts for the majority of intestinal tuberculosis, and transmural caseation with obliterative endarteritis produces the friable, poorly vascularised bowel that ultimately gives way.[3] Free perforation complicates only a small proportion of cases but is associated with mortality that has historically approached one third, because it presents as faeculent peritonitis in a chronically ill, hypoproteinaemia patient.[5,6]

CORTICOSTEROID EXPOSURE AND PROGRESSION OF UNTREATED TUBERCULOSIS

The most instructive element of this case is the sequence of events preceding presentation. A positive interferon-gamma release assay with suspected spinal tuberculosis had been documented seven months earlier, yet antitubercular therapy was never started. The patient then received deflazacort for 25 days for presumed mechanical back pain. Corticosteroids impair macrophage activation, granuloma integrity and T-cell–mediated containment of Mycobacterium tuberculosis, and their use in a person with untreated infection risks progression to active, disseminated disease. Indian guidance on extrapulmonary tuberculosis emphasises that patients with evidence of tuberculous infection who are to receive immunosuppression should be evaluated and treated before, or concurrently with, that therapy.[4] The strong household exposure - pulmonary tuberculosis in the father and splenic tuberculosis in the sister, further raised the pre-test probability of active disease and should, in retrospect, have prompted a more complete evaluation of the earlier abdominal pain episode. In a tuberculosis-endemic setting, a positive interferon-gamma release assay in a symptomatic young adult about to receive

steroids is not a finding that can be safely deferred.

SCLEROSING ENCAPSULATING PERITONITIS AND THE OPERATIVE STRATEGY

The operative finding of a completely plastered abdomen, with solid viscera and omentum adherent to the anterior abdominal wall, is characteristic of sclerosing encapsulating peritonitis. In its secondary form this condition is most often associated with long-term peritoneal dialysis, beta-blocker use, previous abdominal surgery and, in endemic regions, tuberculous peritonitis, in which chronic inflammation drives fibrin deposition and progressive fibro collagenous encasement of the bowel.[7,8] The entity is rarely diagnosed preoperatively; cross-sectional imaging may show clumped, tethered loops with a surrounding membrane and mesenteric thickening, but the diagnosis is usually made at laparotomy, as it was here.[8]

The practical consequence of the cocoon is that it forecloses the simpler surgical options. Identification of the perforation required systematic adhesiolysis from the duodenojejunal flexure distally, and the visceral serosa in such fields is easily injured, generating the very enterotomies that later present as fistulae. Published experience with tuberculous small-bowel perforation is consistent in advising against simple closure, which is associated with high rates of leak and reperforation because the adjacent bowel is already diseased; resection of the involved segment with either anastomosis in a stable patient or proximal diversion in the presence of gross contamination is preferred.[4,6] In this patient, with two perforations at and adjacent to the ileocecal junction, sloughed surrounding tissue, 1.5 L of faeculent contamination and vasopressor-dependent shock, limited resection with an end ileostomy and closure of the distal colonic stump was the only defensible option. Excessive resection was deliberately avoided in a young patient in whom subsequent restoration of continuity is planned.

SPLENIC INFARCTION WITH SPLENIC ARTERIAL THROMBUS

Splenic infarction involving 50–70% of the parenchyma, with abrupt narrowing and thrombus in the superior hilar division of the splenic artery, was an unexpected finding on postoperative day 8. Splenic vascular complications of tuberculosis are described predominantly in disseminated disease and more commonly involve the splenic vein, where periportal or peripancreatic nodal masses compress or inflame the vessel.[9] Several mechanisms plausibly converged in this patient: tuberculous endarteritis and periarterial inflammation extending from the adjacent plastered omentum and mesenteric nodal mass; the profound sepsis-associated hypercoagulable state, with endothelial injury and a prothrombotic milieu; hypoperfusion during refractory shock; and progressive reactive thrombocytosis, which reached 1,029,000/mm³. Notably, the plain computed tomogram on the day of admission described a normal spleen, establishing that the infarct developed during the postoperative sepsis rather than as part of the initial disease.

Management was conservative. In the absence of splenic rupture, abscess formation, uncontrolled haemorrhage or haemodynamic instability attributable to the spleen, non-operative treatment with analgesia, anticoagulation and treatment of the underlying cause is appropriate, and splenectomy in a contaminated, cocooned abdomen would have carried prohibitive risk. The patient was maintained on anticoagulation, and serial imaging showed no progression. Functional hyposplenism after extensive infarction warrants counselling regarding pneumococcal, meningococcal and Haemophilus influenzae type b vaccination and awareness of overwhelming post-splenectomy-type infection, which was addressed at follow-up.

ENTEROCUTANEOUS FISTULA AND ITS CONSERVATIVE MANAGEMENT

An enterocutaneous fistula arising from the medial wall of the ileostomy loop and tracking along the linea alba to the dehisced midline wound was confirmed radiologically on postoperative day 18. The recognised risk factors were all present: emergency surgery for faeculent peritonitis, extensive adhesiolysis through diseased bowel, hypoalbuminemia with a nadir albumin of 1.6 g/dL, sepsis, vasopressor use and wound dehiscence. Contemporary management follows a staged, sepsis-first approach - resuscitation, control of sepsis and drainage of collections, protection of the skin and quantification of effluent, nutritional optimisation, and only then consideration of definitive surgery.[10] Spontaneous closure is more likely in low-output, long-tract fistulae without distal obstruction, malignancy, radiation or active infection at the fistula site, and a period of at least six to twelve weeks of optimisation is generally recommended before reoperation.[10]

This patient's fistula was low output and was managed with a dedicated wound bag alongside the stoma appliance, a high-protein diet, correction of electrolyte and protein deficits, control of intra-abdominal sepsis and daily wound care. An important additional consideration in tuberculous fistulae is that effective antitubercular therapy is itself a determinant of healing; persistent mycobacterial activity in the bowel wall will prevent closure regardless of nutritional measures.[3,4] Antitubercular therapy was commenced on postoperative day 17, once the histopathological picture of necrotising granulomatous inflammation in the bowel, five lymph nodes, the peritoneum and a mesenteric nodule had been reported, and treatment for a minimum of six months in accordance with national guidance was planned with continued surgical follow-up.[4]

ANTIMICROBIAL CONSIDERATIONS

The isolation of a carbapenem-resistant, extended-spectrum beta-lactamase–producing Klebsiella pneumoniae from sputum on postoperative day 3, susceptible only to ceftazidime–avibactam with positive ceftazidime–avibactam plus aztreonam synergy, illustrates the additional hazard faced by patients with prolonged intensive care stays in high-endemicity settings. Distinguishing colonisation from true ventilator-associated infection is essential to avoid unnecessary escalation, and the decision to treat was guided by the clinical picture, the leucocyte trend and the radiological finding of left basal consolidation. Blood and urine cultures remained sterile throughout, which supports the interpretation that the dominant driver of the initial shock state was the faeculent peritonitis itself rather than bacteraemia.

CONCLUSION

Perforated ileocecal tuberculosis remains a life-threatening emergency in endemic regions and should be considered in any young adult presenting with peritonitis, particularly where there is untreated latent infection, a household contact history or recent corticosteroid exposure. This case demonstrates that a documented positive interferon-gamma release assay in a symptomatic patient must not be left untreated before immunosuppressive therapy is prescribed. When perforation occurs within a tuberculous abdominal cocoon, resection with proximal diversion rather than primary repair or anastomosis offers the safest path through a hostile peritoneal field. Rare vascular complications such as extensive splenic infarction with arterial thrombus may develop during the postoperative septic phase and can usually be managed conservatively. Finally, a postoperative enterocutaneous fistula in this setting should be approached with a structured, sepsis-first, nutrition-focused conservative strategy, with early and complete antitubercular therapy as an indispensable component of healing. Survival with functional recovery is achievable, as in this patient, when these principles are applied in concert.[4,6,10]

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