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# TUBERCULOSIS IN DISGUISE: JEJUNAL TUBERCULOSIS PRESENTING AS PROTEIN-LOSING ENTEROPATHY WITH ANASARCA — A RARE CASE REPORT

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## ABSTRACT

**Background:** Intestinal tuberculosis accounts for a substantial share of extrapulmonary tuberculosis, but its distribution across the gastrointestinal tract is uneven and isolated jejunal disease is uncommon. Protein-losing enteropathy, defined by excessive enteric loss of serum protein leading to hypoalbuminemia, is a rare and easily overlooked mode of presentation of intestinal tuberculosis.

**Case presentation:** A 63-year-old man presented with a six-month history of abdominal pain, recurrent vomiting, bilateral lower-limb swelling and weight loss, without fever, cough, dyspnea or diarrhea. Examination revealed pitting pedal edema without abdominal distension. Investigations showed severe hypoalbuminemia (1.5 g/dL), low serum-ascites albumin gradient (0.5), and markedly elevated ascitic adenosine deaminase (65.4 U/L). Ultrasonography demonstrated free intraperitoneal fluid, and contrast-enhanced computed tomography showed diffuse mural thickening and mucosal edema of the jejunum and proximal ileum with mesenteric haziness. Ascitic cytology was negative for malignancy. Mini-laparotomy with mesenteric lymph node biopsy revealed caseating granulomatous inflammation, confirming a tuberculous etiology.

**Conclusion:** Jejunal tuberculosis presenting as protein-losing enteropathy is rare and can mimic cardiac, hepatic or renal causes of edema. A high index of suspicion, supported by ascitic fluid SAAG and adenosine deaminase, cross-sectional imaging, and tissue biopsy, allows timely diagnosis. Anti-tubercular therapy combined with nutritional rehabilitation resulted in resolution of edema and normalization of albumin.

**KEYWORDS:** Jejunal tuberculosis; Protein-losing enteropathy; Hypoalbuminemia; Ascites; Extrapulmonary tuberculosis; Serum-ascites albumin gradient

## 1. INTRODUCTION

Tuberculosis remains a major cause of morbidity in endemic regions, and extrapulmonary disease constitutes a considerable proportion of the overall burden. Abdominal tuberculosis — encompassing peritoneal, intestinal, and nodal disease — is reported in roughly 6% of extrapulmonary cases, and within the gastrointestinal tract the ileocecal region is affected in the large majority of cases because of its relative stasis, abundant lymphoid tissue, and increased absorptive activity.[1,2] Involvement confined to the jejunum is comparatively infrequent, and its clinical presentation is often nonspecific, ranging from chronic abdominal pain and subacute obstruction to bleeding or perforation.[2,3]

Protein-losing enteropathy is an uncommon and frequently under-recognized complication of gastrointestinal disease in which excessive loss of serum protein through the enteric mucosa produces hypoalbuminemia, generalized edema, and ascites, sometimes in the absence of overt gastrointestinal symptoms.[4,5] Because the resulting clinical picture — bilateral limb edema, low-SAAG ascites, and hypoalbuminemia — overlaps with cardiac, hepatic, renal, and nutritional causes, protein-losing enteropathy secondary to jejunal tuberculosis is easily mistaken for these more common conditions and the diagnosis is often delayed. We report a case in which jejunal tuberculosis presented almost exclusively through protein-losing enteropathy and anasarca, without the classical constitutional or luminal symptoms of intestinal tuberculosis, and discuss the diagnostic pathway that established the true etiology.

### Case Presentation

A 63-year-old man presented with a six-month history of dull aching abdominal pain, recurrent vomiting, progressive bilateral lower-limb swelling, and unquantified weight loss. There was no history of fever, cough, dyspnea, or diarrhea. He was a known case of systemic hypertension, hypothyroidism, and an old cerebrovascular accident, with no past surgical history, and was neither a smoker nor consumed alcohol. On examination, he had bilateral pitting pedal edema with no abdominal distension, organomegaly, or lymphadenopathy.

Laboratory evaluation revealed severe hypoalbuminemia (serum albumin 1.5 g/dL). Diagnostic ascitic tap showed a serum-ascites albumin gradient of 0.5 (consistent with an exudative, low-SAAG ascites) and a markedly elevated

ascitic fluid adenosine deaminase of 65.4 U/L. Ascitic fluid cytology was negative for malignant cells. Abdominal ultrasonography showed free fluid in the perihepatic, pericholecystic, and pelvic regions with normal liver and kidney echotexture, effectively excluding hepatic and renal causes of anasarca. Contrast-enhanced computed tomography of the abdomen demonstrated diffuse mural thickening and mucosal edema involving the jejunum and proximal ileum, with adjacent mesenteric fat haziness, findings suggestive of an inflammatory small-bowel process.

Given the combination of low-SAAG exudative ascites, a markedly raised ascitic ADA, and imaging evidence of jejunoileal wall thickening, tuberculosis was suspected and tissue diagnosis was pursued. A mini-laparotomy with regional mesenteric lymph node biopsy was performed; the exteriorized jejunal segment appeared thickened, edematous, and congested (Figures 1 and 2). Histopathological examination of the lymph node biopsy showed caseating granulomatous inflammation, confirming a tuberculous etiology. The patient was started on standard first-line anti-tubercular therapy (isoniazid, rifampicin, pyrazinamide, and ethambutol) under

[weight-band dosing per national TB programme / DOTS protocol — confirm regimen, duration, and any modification for renal/hepatic status], together with high-protein nutritional supplementation and diuretics for edema.

Outcome and follow-up: The patient tolerated anti-tubercular therapy well, with no hepatotoxicity or other drug-related adverse effects on serial monitoring. Pedal edema began to regress within the first two weeks of therapy, paralleling a gradual rise in serum albumin. At the [6-week] follow-up visit, serum albumin had improved to [2.9] g/dL, pedal edema had resolved completely, and the patient reported improved appetite with weight gain of [2] kg. Repeat ultrasonography confirmed resolution of ascites. By [3 months], the patient had completed the intensive phase of anti-tubercular therapy and remained asymptomatic, with serum albumin within normal limits ([3.8] g/dL) and no recurrence of abdominal pain, vomiting, or edema. He was continued on the continuation phase of anti-tubercular therapy to complete a total treatment duration of [6 months], with planned clinical and biochemical review at completion of therapy.

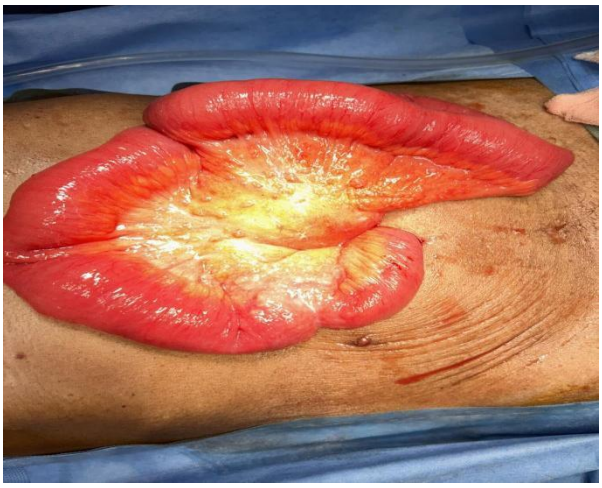

**Figure 1. Intraoperative photograph at mini-laparotomy showing an edematous, thickened, and congested segment of jejunum with prominent serosal vascularity, exteriorized for assessment and mesenteric lymph node biopsy.**

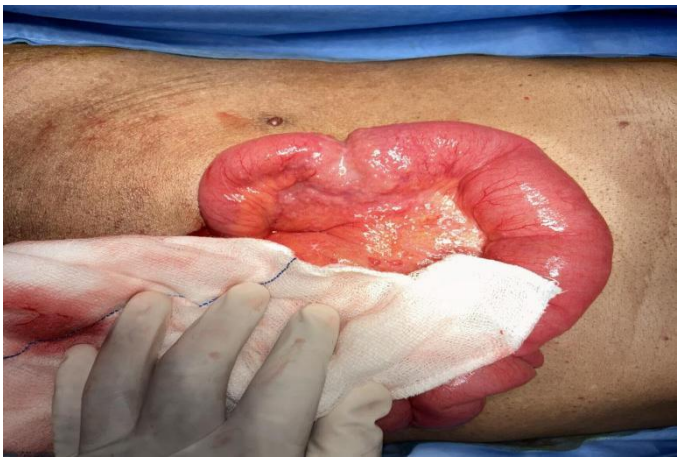

**Figure 2. Intraoperative photograph showing the same exteriorized jejunal segment with mesenteric fat wrapping and hyperemic serosa; a gauze swab supports the loop during handling.**

## DISCUSSION

Gastrointestinal tuberculosis most often localizes to the ileocecal region, reported in up to 80–90% of cases owing to relative stasis, dense lymphoid tissue, and efficient absorption at that site; isolated jejunal or jejunoileal disease is comparatively uncommon.[2,3,6] When the jejunum is involved, the clinical picture is typically dominated by subacute or chronic abdominal pain, weight loss, anorexia, and constitutional symptoms, and complications such as stricture-related subacute obstruction, bleeding, or perforation are the usual triggers for further work-up.[2,3] Presentation as protein-losing enteropathy, as in the present case, is distinctly rare and can occur with minimal or no luminal symptoms, which delays clinical suspicion of tuberculosis.

Protein-losing enteropathy should be considered whenever hypoalbuminemia and edema occur without proteinuria or a clear hepatic synthetic defect, since a wide range of inflammatory, infective, infiltrative, and lymphatic disorders of the gut can produce this picture.[4,5] In our patient, normal renal and hepatic imaging, together with the absence of proteinuria, pointed toward an enteric source of protein loss, and the subsequent finding of jejunoileal wall thickening on cross-sectional imaging directed attention to the small bowel.

Ascitic fluid analysis is a valuable early step when abdominal tuberculosis is suspected. A low serum-ascites albumin gradient (<1.1 g/dL) indicates an exudative process, and ascitic adenosine deaminase has consistently shown high diagnostic accuracy for tuberculous peritonitis and abdominal tuberculosis more broadly, with pooled sensitivity and specificity both exceeding 90% in meta-analyses, although optimal cut-off values vary between studies.[7,8,9] In this case, a low SAAG together with a markedly elevated ascitic ADA (65.4 U/L) provided strong indirect biochemical support for tuberculosis well before histological confirmation was available. Nonetheless, because ascitic smear and culture for acid-fast bacilli have low sensitivity, tissue biopsy — obtained here by mini-laparotomy with lymph node sampling — remains the reference standard for definitive diagnosis.[9,10]

Contrast-enhanced computed tomography is useful for localizing the affected bowel segment and characterizing mural thickening, mesenteric changes, and lymphadenopathy, and helps to narrow the differential diagnosis, which for jejunal wall thickening includes Crohn's disease, lymphoma, and other infective enteritides.[3,6] In our patient, imaging findings of diffuse jejunoileal mural thickening with mesenteric haziness were nonspecific in isolation but, combined with the biochemical picture, were highly suggestive of an inflammatory/infective process and supported the decision to proceed to tissue sampling rather than prolonged observation.

Management of intestinal tuberculosis centers on standard multidrug anti-tubercular therapy, reserving surgery for complications such as obstruction, perforation, or fistula, or for diagnostic tissue acquisition as in this case.[3,10] Protein-losing enteropathy associated with tuberculosis has been reported to improve with anti-tubercular therapy and nutritional support once the underlying inflammatory process is controlled, paralleling the resolution described in other causes of enteric protein loss once the primary disease is treated.[4,5] This case reinforces that protein-losing enteropathy, though rare, deserves consideration as a presenting feature of intestinal tuberculosis, particularly in tuberculosis-endemic settings, even when classical gastrointestinal symptoms such as diarrhea, fever, or abdominal distension are absent.

## CONCLUSION

Jejunal tuberculosis is an uncommon form of abdominal tuberculosis, and protein-losing enteropathy is an even rarer mode of presentation. Clinical suspicion should be high when persistent hypoalbuminemia, low-SAAG ascites, and constitutional symptoms coexist, particularly in tuberculosis-endemic populations. Simple ascitic fluid tests such as SAAG and adenosine deaminase provide early direction, cross-sectional imaging helps localize disease, and tissue biopsy remains essential for definitive diagnosis. Timely initiation of anti-tubercular therapy combined with nutritional rehabilitation can achieve progressive resolution of edema and restoration of serum albumin, underscoring the importance of early recognition and a targeted diagnostic approach in this rare but treatable condition.

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