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Encapsulating Peritoneal Sclerosis Mimicking Intra-abdominal Malignancy: A Case Report

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Abstract

Encapsulating peritoneal sclerosis (EPS) is an uncommon fibroinflammatory disorder characterized by extensive peritoneal fibrosis and, in characteristic cases, partial or complete encasement of bowel loops. We report a 62-year-old male with cirrhosis, hypertension, and type 2 diabetes mellitus who developed progressive severe abdominal pain, diarrhea, and reduced oral intake while undergoing investigation for weight loss and suspected gastrointestinal malignancy. Physical examination revealed a rigid, distended, and tender abdomen. Recovered laboratory findings included a C-reactive protein level of 62.2 mg/L, hemoglobin of 9.1 g/dL, platelet count of 85,000/µL, and serum creatinine of 2.9 mg/dL. Preoperative contrast-enhanced computed tomography demonstrated mesogastric peritoneal thickening, an adjacent 14.0 × 6.5 cm fluid collection, bowel distention, and indeterminate rectal wall thickening. Exploratory laparotomy revealed extensive dense adhesions, marked visceral and parietal peritoneal thickening, and severe fixation of intra-abdominal structures. Limited adhesiolysis and loop ileostomy were performed. Histopathological examination of tissue submitted as peritoneal thickening demonstrated nonspecific smooth-muscle fibrosis without neoplastic features in the sampled tissue. A distinct encapsulating membrane was not definitively documented, and the rectal abnormality remained incompletely evaluated in the available records. The integrated findings supported a working diagnosis of EPS-like fibroadhesive peritoneal disease. This case illustrates the diagnostic overlap between extensive peritoneal fibrosis and suspected intra-abdominal malignancy, emphasizing the importance of integrating clinical, radiological, operative, and histopathological findings while acknowledging diagnostic limitations.

Categories: General Surgery, Anatomy
Keywords: abdominal cocoon, acute abdomen, case report, cirrhosis, encapsulating peritoneal sclerosis, intestinal obstruction, sclerosing encapsulating peritonitis

Introduction

Encapsulating peritoneal sclerosis (EPS), historically also called sclerosing encapsulating peritonitis or abdominal cocoon syndrome, is a rare fibroinflammatory condition characterized by peritoneal fibrosis, dense adhesions, and, in characteristic cases, partial or complete encasement of bowel loops by a fibrocollagenous membrane. Patients may present with abdominal pain, distention, nutritional decline, and recurrent or acute obstructive symptoms. Unlike a single mechanical transition-point obstruction, EPS involves a more extensive process of peritoneal fixation and sometimes bowel encasement; however, diffuse adhesions or fibrosis alone do not establish the diagnosis [1,2].

Prolonged peritoneal dialysis is the most established risk factor. Other reported settings include tuberculous peritonitis, previous abdominal surgery, recurrent inflammation, selected medications, and systemic conditions [2-4]. An idiopathic designation requires a sufficiently thorough evaluation for secondary causes. CT may reveal peritoneal thickening, calcification, loculated fluid, clustered or tethered bowel loops, and signs of obstruction; a classic membrane or clustered-loop pattern is not present or documented in every case [1,3,5]. We report a patient with cirrhosis whose radiographic findings raised concern for abdominal malignancy but whose operation disclosed striking generalized fibroadhesive peritoneal disease. The educational objective is to illustrate the challenges of a clinically suspected EPS phenotype without overstating the specificity of limited imaging or histology.

This case was previously presented as a poster at the 24th Congresso Paulista de Cirurgia in São Paulo, Brazil, in August 2024.

Case Presentation

A 62-year-old male with hypertension, type 2 diabetes mellitus, cirrhotic liver disease, and a history of heavy alcohol use (reported abstinence for approximately one year) presented to the emergency department at the beginning of the reported clinical course (day 1). He had already been under investigation for possible gastrointestinal malignancy in the setting of poor appetite and weight loss; the duration and degree of

weight loss were not quantified in the recovered records. The emergency note reported approximately five days of progressively severe diffuse abdominal pain aggravated by movement and approximately three days of loose stools, together with markedly decreased oral intake. He also exhibited mild confusion. He denied recent operations or surgery involving the gastrointestinal tract, although his complete lifetime surgical history could not be reconstructed. No peritoneal dialysis was documented.

At the initial assessment, he was described as hemodynamically stable, awake, and mildly disoriented (Glasgow Coma Scale score of 14). His abdomen was distended and rigid, with diffuse tympany, generalized superficial tenderness, and reduced bowel sounds. The initial record noted a heart rate of 71 beats/min and room-air oxygen saturation of 91%; a subsequent examination recorded a blood pressure of 126/94 mmHg, heart rate of 73 beats/min, respiratory rate of 12 breaths/min, and oxygen saturation of 92% on room air. Point-of-care ultrasound described a heterogeneous liver, perihepatic free fluid, and a small right pleural effusion. The recovered records did not establish whether the fluid represented chronic ascites, superimposed peritonitis, or a combination, and did not identify an established cause for the altered mental status. The admission medication list included losartan, neutral protamine Hagedorn (NPH) insulin, furosemide, oral iron, and vitamin supplementation; no beta-blocker was listed. Prior long-term beta-blocker exposure could not be determined. Empiric ceftriaxone and metronidazole were initiated during the emergency assessment.

The recovered laboratory results from the initial emergency evaluation (day 1) are listed in Table *1*. They showed C-reactive protein (CRP) of 62.2 mg/L, hemoglobin of 9.1 g/dL, platelet count of 85,000/µL, creatinine of 2.9 mg/dL, urea of 105 mg/dL, sodium of 127 mEq/L, leukocyte count of 10,610/µL, and international normalized ratio of 1.21; total bilirubin was 0.55 mg/dL. These abnormalities indicate substantial systemic illness but are not specific for EPS. The absence of values for other tests in the recovered record must not be interpreted as normal results.

Laboratory parameter	Result	Unit
C-reactive protein (CRP)	62.2	mg/L
Hemoglobin	9.1	g/dL
Hematocrit	26	%
White blood cell count	10,610	cells/µL
Platelet count	85,000	platelets/µL
Serum creatinine	2.9	mg/dL
Blood urea	105	mg/dL
Sodium	127	mEq/L
Potassium	4.3	mEq/L
Prothrombin time	13.9	s
International normalized ratio (INR)	1.21	ratio
Activated partial thromboplastin time	28	s
Total bilirubin	0.55	mg/dL
Direct bilirubin	0.28	mg/dL
Indirect bilirubin	0.27	mg/dL

TABLE 1: Laboratory findings at initial evaluation.

Laboratory findings recovered from the original hospital records during the patient's initial evaluation. The results demonstrate elevated C-reactive protein, anemia, thrombocytopenia, renal dysfunction, and hyponatremia. Individual specimen collection times could not be fully verified in the recovered documentation.

Postoperative quantitative CRP values were unavailable and were not estimated. These findings are nonspecific and should be interpreted in the context of the patient's underlying cirrhosis and acute clinical presentation.

Values were transcribed from recovered hospital laboratory documentation linked to the initial hospitalization. Individual specimen collection times were not consistently available; a quantitative

postoperative CRP value was not recovered. A CRP reference range of <5.0 mg/L was shown on the recovered report.

Contrast-enhanced abdominopelvic CT before surgery documented central mesogastric peritoneal thickening and an adjacent fluid collection measuring approximately 14.0 × 6.5 cm, in close contact with intestinal loops. Gas and fluid bowel distention with coprostasis were noted. The mid-to-upper rectum showed concentric wall thickening with luminal narrowing, described as indeterminate. Portal-hypertensive features, including splenomegaly and abdominal varices, were also noted. The available radiology report did not specify a distinct encasing membrane, characteristic clustering of bowel loops, or peritoneal calcification. A precisely localized mechanical transition point was not recorded. Original DICOM images were not available for a retrospective diagnostic interpretation of these features.

Because of severe abdominal pain, a rigid tender abdomen, substantial inflammatory abnormalities, and concern for a significant obstructive or other acute intra-abdominal process, exploratory laparotomy was undertaken. The operative account in the source case material described extensive dense intestinal adhesions, marked thickening of both visceral and parietal peritoneal surfaces, and near-complete fixation of visible intra-abdominal structures. The structures that could be safely evaluated showed no gross neoplastic implants. A large amount of abdominal fluid was reported and described as inflammatory, but objective fluid-cell counts, cytology, and routine culture findings were not recoverable; therefore, its inflammatory or infectious nature cannot be independently confirmed. The current source material did not establish that this fluid was identical to the 14.0 × 6.5 cm CT collection.

Because extensive enterolysis was considered unsafe, only selected adhesions were released, and a loop ileostomy was created. The historical operative summary does not document a specific site of bowel transition, the exact extent of bowel involvement, bowel viability findings, the anatomic level of the ileostomy, or its intended physiological relationship to an identified distal obstruction. Likewise, no explicitly described or photographically validated discrete fibrocollagenous membrane could be confirmed in the retrieved records. These gaps limit the specificity of the operative diagnosis. The original operative photograph (Figure *1*) shows visibly abnormal conglomerated fibroadhesive tissue; reliable labeling of specific bowel segments or a distinct encapsulating membrane is not possible from the available copy.

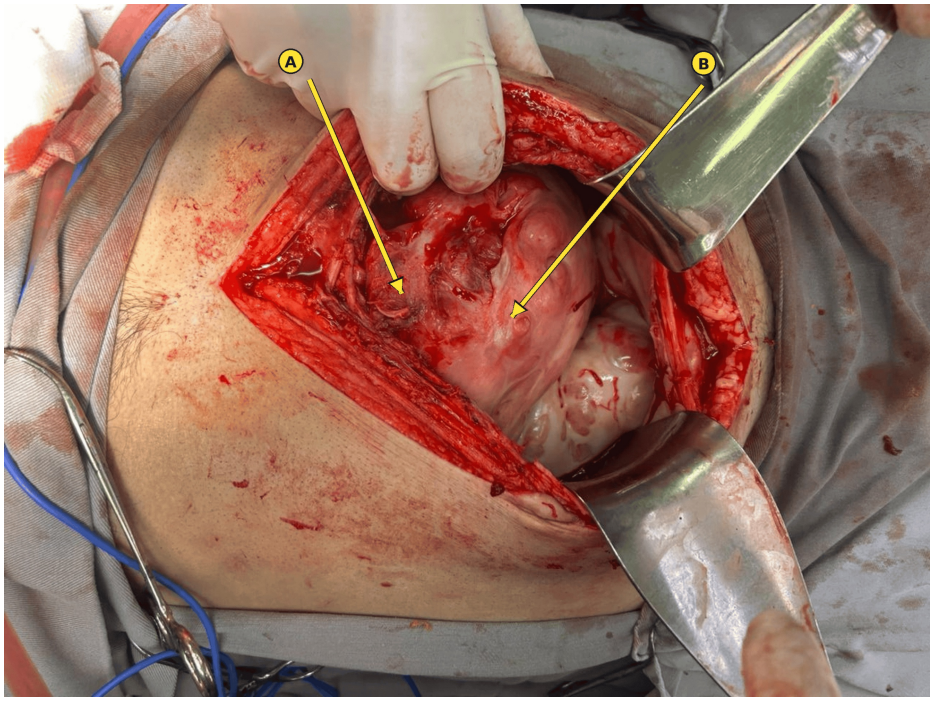
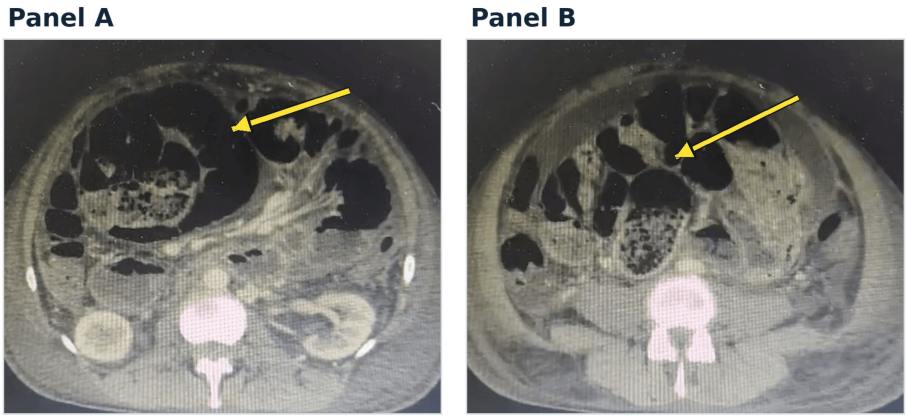


FIGURE 1: Intraoperative appearance of extensive fibroadhesive peritoneal disease.

Intraoperative photograph showing markedly abnormal, adherent intra-abdominal tissues. Arrow A highlights an irregular fibroadhesive-appearing region, and arrow B indicates a thickened-appearing surface within the conglomerated operative field. The image demonstrates macroscopic fibroadhesive changes but does not independently establish a discrete bowel-encapsulating membrane.

Representative axial CT frames illustrating bowel distention are presented in Figure *2*. These images do not independently demonstrate the characteristic encapsulating membrane of EPS.



Yellow arrows: gas-distended bowel loops. Source: filmed CT display (not DICOM).

FIGURE 2: Representative axial abdominal CT frames showing gas-distended bowel loops.

Representative axial CT frames extracted from a video recording of the imaging workstation. Yellow arrows in panels A and B point to gas-filled, distended bowel loops. The recovered CT report additionally described mesogastric peritoneal thickening, an adjacent approximately 14.0 × 6.5 cm collection, and indeterminate rectal thickening. Their precise locations cannot be reliably marked on the available low-resolution frames. A discrete encapsulating membrane, clustered-loop pattern, or peritoneal calcification cannot be confirmed from these images.

The surgical pathology report described three irregular tissue fragments submitted as "peritoneal thickening." Histology identified smooth-muscle tissue with fibrosis; the pathologist explicitly stated that the finding was nonspecific and that no neoplastic features were identified in the specimen. The report did not specifically characterize granulomatous inflammation or caseous necrosis. Thus, the result supports the presence of fibrosis at the sampled site, but neither proves EPS in isolation nor excludes malignancy in unexamined sites. Although the original case narrative described negative mycobacterial culture, polymerase chain reaction, and acid-fast bacilli results, the primary laboratory reports and specimen descriptions were not present in the newly recovered documentation. These negative results should therefore be treated as historical report entries, rather than definitive exclusion of abdominal tuberculosis.

Five days after the initial emergency presentation (day 6), a noncontrast postoperative abdominopelvic CT described small foci of pneumoperitoneum, moderate abdominal free fluid, reduction of the mesogastric collection, and a left-sided drain terminating near it. An additional suspected perisplenic collection measured approximately 5.3 × 5.1 × 5.5 cm (estimated 77 mL), and associated fecal content could not be ruled out in the radiology report. The report did not identify which intraoperative fluid compartment corresponded to the preoperative collection or document a fluid specimen analysis.

The earlier postoperative case summary described initial improvement and functioning ileostomy by postoperative day 6, accompanied by a qualitative decline in CRP; no subsequent quantitative CRP value was available for verification. Management in an intensive care setting was described, but the clinical indication, organ-support measures, and a complete record of nutrition and medications given after surgery were not recovered. There is no source documentation establishing that corticosteroids or tamoxifen were administered. A recovered progress note recorded 16 days after the initial emergency presentation (day 17) discussed disposition planning but did not establish a completed transfer, subsequent treatment, or long-term outcome. No transfer to palliative care is established by the available record. The actual longer-term course and stoma outcome remain unknown.

Discussion

This case is clinically notable for an extensive fibroadhesive peritoneal process encountered during investigation for malignancy in a patient with cirrhosis and acute abdominal symptoms. EPS was considered after the combination of diffuse visceral and parietal peritoneal thickening, severe adhesions and fixation of abdominal structures at surgery, and nonspecific fibrosis in the operative sample. However, the defining feature of classic EPS, i.e., a distinct fibrous membrane partly or entirely encasing bowel, was not explicitly documented in the recovered operative record or preoperative CT report. The diagnosis must therefore be regarded as a clinically supported working diagnosis rather than unequivocally proven encapsulation. Important alternatives include extensive nonspecific peritoneal fibrosis and adhesions, chronic inflammatory peritonitis, tuberculous peritonitis, peritoneal malignancy, and changes associated with cirrhosis and portal hypertension [1-4]. Previous reports have described EPS mimicking other acute surgical

conditions [6], occurring in patients with advanced liver cirrhosis [7], and in association with propranolol exposure [8]. However, the latter association cannot be established in our patient because his longitudinal medication history was unavailable.

The preoperative CT established mesogastric peritoneal thickening, an associated fluid collection, bowel distention, and an indeterminate rectal abnormality, but did not describe clustered loops, peritoneal calcification, or a discernible encapsulating membrane. Failure to mention a finding in a report is not equivalent to confirming its absence on original images. The previously published case-control CT study by Vlijm et al. compared 15 patients with EPS against 16 patients receiving long-term peritoneal dialysis without EPS; its diagnostic estimates cannot be directly extrapolated to this nondialysis patient with cirrhosis or used to conclude that CT established the diagnosis [5]. Prolonged peritoneal dialysis is a well-established clinical setting for EPS, as emphasized in the International Society for Peritoneal Dialysis position paper [9]. Published reviews also discuss the complex pathophysiology of EPS and the available treatment approaches [10].

The pathology sample was submitted as peritoneal thickening, although microscopically it consisted of smooth-muscle tissue with fibrosis and nonspecific features. Histopathology neither provides independent proof of EPS nor directly resolves the separate rectal mural abnormality. No malignancy was identified in the sampled tissue or surgically assessable field. The rectosigmoid was incompletely evaluated intraoperatively, and the recovered chart did not document definitive endoscopic assessment or a rectal biopsy. This should be interpreted as an unresolved radiologic finding in the available records, not as a diagnosis of neoplasia [1,2].

The recovered microbiologic records are similarly incomplete. The original case narrative described negative culture, PCR, and acid-fast testing for tuberculosis without specifying the submitted material. Even negative results from appropriately selected extrapulmonary samples do not independently exclude tuberculosis because diagnostic sensitivity varies and false-negative results occur. Accordingly, the available evaluation did not identify evidence of tuberculosis, but a categorical exclusion is not justified [11]. Likewise, the admission list did not include a beta-blocker; although propranolol has been reported in association with EPS, no duration of exposure can be assigned to this patient [8]. A definitive idiopathic classification is therefore not warranted.

Intraoperative management was constrained by severe adhesions and concern about iatrogenic bowel injury. The record states that limited adhesiolysis and loop ileostomy were performed, but lacks sufficient anatomic detail to attribute the diversion to a confirmed obstruction point or a particular involved segment. The preoperative fluid collection and the subsequently observed abdominal fluid may have represented related processes, but no correspondence can be demonstrated from the available records. Empiric ceftriaxone and metronidazole were initiated during the initial emergency assessment; the complete postoperative antimicrobial regimen and other specific ICU, nutritional, antifibrotic, and long-term treatment details could not be reconstructed from the available records. Corticosteroids and tamoxifen are discussed as options in the EPS literature, not as established therapies received by this patient [2,4,10].

Table *2* distinguishes findings documented in the available records from limitations of ascertainment. Negative tissue findings do not establish the absence of malignancy at unsampled sites; reported negative tests do not categorically exclude tuberculosis.

Potential secondary cause or associated factor	Documented findings and interpretation
Long-term peritoneal dialysis	No history of peritoneal dialysis was documented.
Previous abdominal surgery	No previous abdominal surgery was reported; complete lifetime surgical records were unavailable.
Tuberculous peritonitis	The previous case account reported negative mycobacterial culture, polymerase chain reaction, and acid-fast bacilli testing. Source reports and specimen types were unavailable; these findings do not definitively exclude tuberculosis.
Intra-abdominal malignancy	No malignancy was identified in sampled tissue or the assessable operative field. The CT finding of rectal wall thickening remained incompletely characterized in the recovered records.
Medication-associated EPS	No beta-blocker appeared on the admission medication list. Longitudinal medication exposure, including propranolol, could not be confirmed.
Cirrhosis	Present with radiologic features of portal hypertension; an association does not establish causality.

TABLE 2: Evaluation of potential secondary causes and associated factors.

Summary of potential secondary causes and associated factors evaluated in this patient. No history of peritoneal dialysis or previous abdominal surgery was documented. Previously reported negative microbiological investigations did not identify evidence of tuberculosis; however, the specimen details were unavailable, and negative results do not definitively exclude extrapulmonary tuberculosis. Histopathological examination showed no malignancy in the sampled tissue, although the indeterminate rectal abnormality was not conclusively evaluated in the recovered records. Long-term medication exposure, including beta-blocker use, could not be reliably established.

EPS: encapsulating peritoneal sclerosis.

The principal limitations are the absence of a detailed original operation record documenting a distinct encapsulating membrane, unavailability of source DICOM data for independent retrospective radiologic review, nonspecific histology from a limited sample, inability to identify the submitted specimens for mycobacterial testing, lack of documentation resolving the rectal abnormality, incomplete longitudinal drug history, and uncertain longer-term outcome. The case remains useful because it shows how an extensive fibroadhesive intra-abdominal condition can mimic malignancy and complicate emergency operative decisions, while highlighting the need to avoid equating negative sampling or nonspecific fibrosis with proof of a definitive diagnosis.

Conclusions

In patients with unexplained peritoneal thickening, loculated intra-abdominal fluid, and extensive adhesions, EPS should be considered within a broader differential diagnosis. In this patient, the integrated clinical, radiologic, and operative findings supported an EPS-like fibroadhesive peritoneal process, but a distinct encasing membrane was not definitively documented. The examined tissue showed nonspecific fibrosis without neoplastic features, and no gross malignancy was identified in surgically assessable areas; the indeterminate rectal finding remained incompletely characterized in the available records. Accurate documentation of operative anatomy, characterization of secondary causes, and explicit reporting of evidentiary limitations are central to interpreting similar cases.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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Disclosures

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