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Case Report

"Abdominal cocoon" small bowel obstruction: a diagnostic challenge in adolescent female

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ABSTRACT

Primary sclerosing encapsulating peritonitis (SEP), also referred to as abdominal cocoon syndrome (ACS), is an exceedingly unusual etiology of intestinal obstruction. Morphologically, defined by the entire or partial encasement of the small intestine within a dense fibrocollagenous membrane. Historically it was prevalent among adolescent females in tropical regions. Latest evidence indicates its global distribution, with a higher incidence in males than females. ACS poses a significant diagnostic problem due to its non-specific clinical presentation. We report the case of a 15-year-old female presenting with acute on chronic intestinal obstruction with a history of recurrent similar episodes of abdominal distention with progressive constipation. Preoperative imaging (CECT abdomen) suggested small bowel obstruction with a transition point at the distal jejunum/proximal ileum, without pinpointing on the presence of cocooning. Exploratory laparotomy revealed that the ileal loops were entirely encased in a dense, avascular fibrous peritoneal sac, presenting a classic "cocoon" appearance. Adhesiolysis and excision of the thickened membrane were performed. Histopathology confirmed sclerosing peritonitis with chronic inflammatory infiltrate without evidence of granulomas or tuberculosis affirming that the disease is idiopathic (primary). This case highlights the importance of considering ACS in the differential diagnosis of intestinal obstruction in adolescent females, particularly in tropical regions and demonstrates the curative potential of surgical membrane excision.

Keywords: Peritoneal fibrosis, Intestinal obstruction, Tissue adhesions, Abdominal cocoon syndrome, Rare diseases, Case report

INTRODUCTION

Abdominal cocoon syndrome (ACS), also known as sclerosing encapsulating peritonitis (SEP), is a rare condition characterised by the partial or complete encasement of the small intestine within a thick fibrocollagenous membrane, causing recurrent or acute mechanical bowel obstruction.[1,2] The idiopathic form, often termed "abdominal cocoon syndrome," is predominantly observed in young adolescent females living in tropical or subtropical zones and was first described by Foo et al in 1978.[3]

SEP is the preferred academic nomenclature, recognising both the progressive fibrotic process at the peritoneal level and the encapsulating morphological consequence.[1,2] It is broadly divided into two clinico-pathological forms: primary (idiopathic) SEP—where no causative condition can be identified after thorough evaluation—and secondary SEP, where the peritoneal injury is attributable as a definable precipitant. The most common secondary causes include prolonged peritoneal dialysis (PD), which carries a cumulative incidence that rises steeply from 0.3% at three years to nearly 4% by eight years and abdominal tuberculosis, which remains a dominant aetiology in the

Indian subcontinent and other high-prevalence regions.[1,4-6] A heterogeneous group of causes are encompassing beta-blocker therapy (classically practolol), intraperitoneal chemotherapy, ventriculo-peritoneal shunts, liver cirrhosis, organ transplantation, and autoimmune conditions such as systemic lupus erythematosus and sarcoidosis.[1,7,8]

The pathological substrate of SEP is a progressive, low-grade peritoneal inflammatory reaction—often subclinical in its early stages—that ultimately culminates in the deposition of dense sheets of collagenous tissue over the serosal surface of the bowel.[1] Histologically, the membrane demonstrates fibroconnective tissue proliferation, mononuclear inflammatory infiltrates, and dilated lymphatics, without foreign body granulomas or birefringent material—findings that are characteristic but not pathognomonic.[1,2] At the molecular level, the fibrogenic process is mediated by cytokines, transforming growth factor-beta, and activated fibroblasts; an epithelial-to-mesenchymal transition of peritoneal mesothelial cells has been implicated in the generation of the thickened membrane.[4,9]

The syndrome is categorised into three types based on the anatomical extent of encasement: type I involves partial encasement of the small intestine; type II involves complete encasement of the entire small bowel; and type III extends to involve additional intra-abdominal organs, including the appendix, caecum, ascending colon, liver, stomach, and ovaries.[1,2,10] An alternative classification proposed by Li et al distinguishes between cases with and without a second enterocoelia—a fluid-filled accessory compartment within the cocoon that has operative and prognostic significance.[11]

Surgical management remains the definitive treatment for symptomatic ACS, and the operative principles are demanding in their precision. Careful, meticulous excision of the encasing membrane—peeling it free from the serosal surface of the bowel in a plane analogous to capsulectomy—combined with complete adhesiolysis, constitutes the procedure of choice.[1,2,12] The surgical axiom is unambiguous: bowel resection is not required and should be avoided unless gangrenous or perforated segments are encountered, as unnecessary resection dramatically increases morbidity and mortality and risks the catastrophic sequela of short bowel syndrome.[1,2] When the membrane is completely excised, the risk of recurrence is low and long-term outcomes are excellent.[2,12] Medical therapy—comprising corticosteroids, tamoxifen, colchicine, and immunosuppressants—plays an established role in secondary SEP, particularly the peritoneal dialysis-related form, and is reserved for primary SEP in cases where conservative management is appropriate or when surgical risk is prohibitive.[2,13]

The following report presents our operative and clinical experience with abdominal cocoon syndrome, followed by a comprehensive discussion synthesising contemporary literature. We seek to provide the practising surgeon with a granular, clinically actionable understanding of this condition—from its first whisper in the preoperative imaging studies to the decisive moment of membrane excision in the operating theatre.

The condition is characterized by the encasement of small bowel loops within a dense, whitish fibrous sac, leading to acute or recurrent subacute intestinal obstruction. The exact etiology of the idiopathic form remains unclear, though several theories have been proposed including retrograde menstruation, subclinical viral peritonitis, and chemical peritonitis. Secondary causes include prolonged peritoneal dialysis, abdominal tuberculosis, prior abdominal surgery, systemic lupus erythematosus, sarcoidosis, and malignancy.

CASE REPORT

A 15-year-old female, presented with complaints of sudden onset lower abdominal pain in the infraumbilical region, colicky type, gradually progressive, around the umbilicus and radiating to back, continuous and severe in nature, interfering with the daily activity and sleep and associated with abdominal distension.

There was no history of fever, vomiting, menstrual irregularities, jaundice. There was no significant family history.

Significantly, the patient reported a history of similar self-limiting episodes of recurrent abdominal pain. She had experienced two episodes approximately one year ago in her native place, and one episode two months prior to the current presentation. None of these previous episodes required surgical intervention, and symptoms of progressive constipation had resolved with conservative management, with enemas. The patient had no known history of hypertension, diabetes mellitus, hypothyroidism, or tuberculosis contact.

On general physical examination, patient was well built and the vital parameters were within normal limits.

On abdominal examination, abdomen was mildly distended. A small umbilical hernia noted with a positive cough impulse. No visible peristalsis. All regions moving equally with respiration. Abdomen was soft. Tenderness present in the right iliac region, left iliac region, suprapubic region, and umbilical region. A palpable lump noted in left iliac fossa. No guarding or rigidity was present. The umbilical hernia was reducible and non-tender with a defect measuring 1×1 cm. Tympanic note was present throughout the abdomen. Bowel sounds were sluggish, consistent with partial intestinal obstruction. Per rectal examination suggested no ballooning and rectum was empty.

Laboratory tests were within normal limits.

Ultrasonography (USG) abdomen revealed mild free fluid in the peritoneal cavity (ascites), umbilical hernia with bowel contents, and no evidence of organomegaly or focal lesions.

Erect abdomen X-ray showed loaded bowel loops (Figure 1).

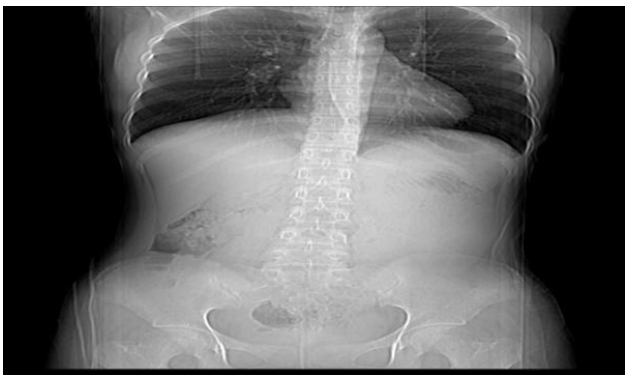

Figure 1: Erect abdomen X ray showing loaded bowel.

Contrast-enhanced computed tomography (CECT) abdomen showed evidence of dilated jejunal loops with maximum caliber measuring-4.0-4.5 cm with fecal matter within the distal jejunal loops, there was smooth narrowing at the level of distal jejunum/proximal ileum. However, no obvious evidence of wall thickening noted (likely the transition point). Rest of the distal small bowel loops appeared collapsed. The bowel loops showed normal wall enhancement on post contrast study and no evidence of pneumoperitoneum seen. The large bowel loops were normal in caliber loaded with fecal matter. No evidence of dilatation and normal wall enhancement noted on post contrast study. Mild to moderate ascites in peritoneal cavity and multiple subcentrimentric and few enlarged mesenteric lymph nodes largest measuring 1.3×0.9 cm noted in right iliac fossa (Figure 2).

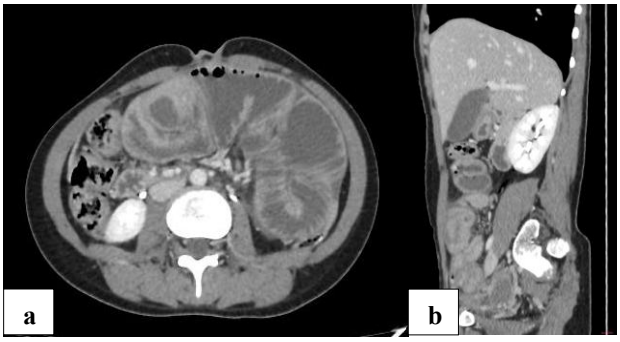

Figure 2: (a) Axial images showing dilated jejunal loops and narrowing at the level of distal jejunum and proximal ileum (likely the transition point), and (b) sagittal CECT image showing collapsed distal small bowel loops.

The patient was taken up for exploratory laparotomy and proceed under general anesthesia. The exploratory laparotomy revealed grossly dilated cocooned small bowel loops. Multiple loops of small bowel (ileum and jejunum) were found completely encased within a thick, dense, whitish-grey fibrous peritoneal sac, creating the pathognomonic "cocoon" appearance. The membrane had a smooth, shiny surface and was relatively avascular. The ileum, starting from the ileocaecal junction and extending proximally, showed complete encasement with dense adhesions within the peritoneal sac. The bowel loops were collapsed and matted together, due to multiple bands and adhesions (Figure 3). Approximately 50-100 ml of straw-colored peritoneal fluid was present. Ileocecal junction was traced by appendix, retrograde tracing showed complete ileum cocooned within the peritoneal sac. A finding of a cystic pedunculated swelling left of the umbilicus, adherent to inner wall of peritoneum was noted. Ileum was separated and found to be collapsed and healthy and extensively adherent to inner wall of sac. Blunt dissection and separation of complete ileum from the sac done. Specimens of sac sent for further evaluation (Figure 4).

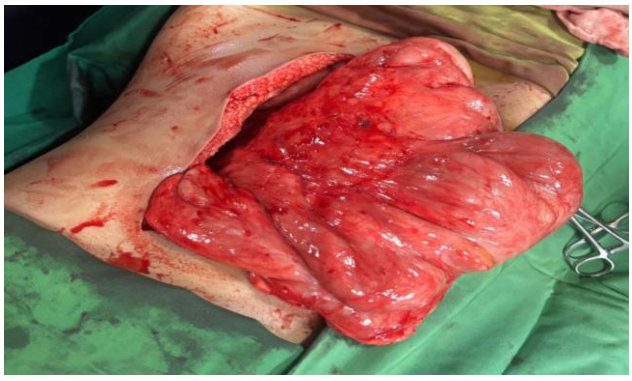

Figure 3: Grossly dilated cocooned small bowel loops- "cocoon abdomen".

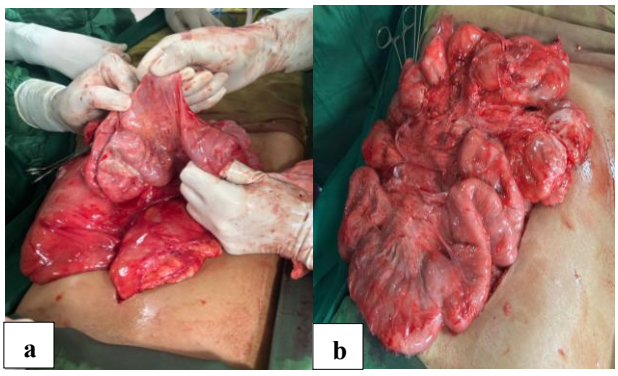

Figure 4: (a) Separation of complete ileum from the sac, and (b) released ileum loops from the sac.

On histopathological examination of peritoneal membrane grossly consisted of multiple irregular pieces of greyish-white tissue measuring 3×2×1 cm in aggregate. Pieces of sac were also sent for ruling out Abdominal Koch's. Features were consistent with sclerosing peritonitis (chronic inflammatory type) (Figure 5).

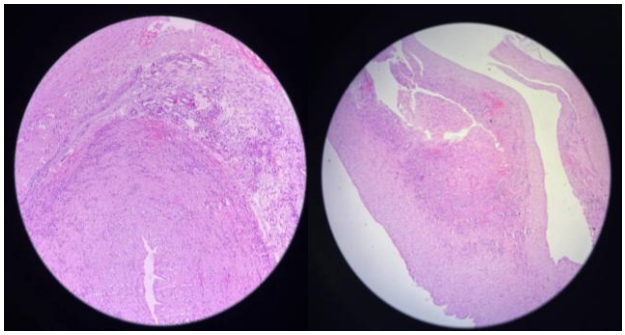

Figure 5: Slide of peritoneal membrane which shows thickened fibrocollagenous tissue with areas of hyalinization, and focal areas of fibrosis with collagen deposition-sclerosing peritonitis.

The cystic structure measuring 3×2 cm, filled with clear fluid was also sent for histopathological examination, sections showed cystic structure lined by tubal epithelium, features were consistent with fallopian tube tissue and luminal dilatation was consistent with hydrosalpinx (Figure 6).

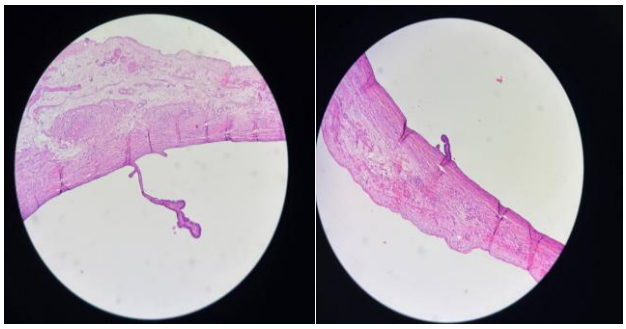

Figure 6: Slide of cystic structure showing lining of tubal epithelium features consistent with fallopian tissue and luminal dilatation consistent with hydrosalpinx.

Peritoneal fluid analysis (Ziehl-Neelsen stain for acid-fast bacilli), cartridge based nucleic acid amplification test (CBNAAT), and interferon gamma release assay (IGRA) were all negative.

DISCUSSION

The diagnostic paradox: a condition that hides in plain sight

The central challenge of abdominal cocoon syndrome is not its rarity—it is its invisibility. The condition pursues its fibrotic agenda silently, behind a clinical facade indistinguishable from the commonplace diagnoses of adhesive small bowel obstruction, internal hernia, or even inflammatory bowel disease. Our case exemplifies this diagnostic paradox: a patient presenting with recurrent subacute obstruction whose imaging, on initial appraisal, could have been ascribed to adhesive disease or intraperitoneal malignancy. It was only the characteristic CT constellation—centrally congregated bowel loops enveloped within a soft-tissue density mantle, the absence of a discrete transition point, and the presence of encapsulated ascites—that generated the preoperative suspicion of ACS.

While the etiology is often multifaceted, Jagdale et al highlight the significance of distinguishing between primary and secondary forms. In the primary variant, particularly among the adolescent female demographic, retrograde menstruation is frequently cited as a causative factor. This hypothesis suggests that the reflux of menstrual debris into the pelvic cavity incites a chronic inflammatory subacute peritonitis. The subsequent organization of inflammatory exudate results in the development of a thick peritoneal membrane.[8]

The literature documents that between 56–71% of cases are still diagnosed intraoperatively, despite the widespread availability of modern cross-sectional imaging.[2,10] This alarming figure reflects not a failure of CT technology, but a failure of clinical awareness. Tannoury and Abboud emphasise that a preoperative diagnosis is achievable but demands a high index of suspicion—a cognitive trigger that most clinicians, who will encounter this condition perhaps once in an entire career, are ill-equipped to have spontaneously activated.[1] The systematic review by Chorti et al demonstrated with statistical significance ($p<0.001$) that when CT was employed preoperatively, the rate of preoperative diagnosis rose substantially—a finding with direct implications for case planning, consent, and operative preparation.[10]

The differential diagnosis is wide and must be systematically excluded. Internal hernias are the most clinically proximate differential, sharing with ACS the CT features of centrally clustered bowel loops and evidence of obstruction; however, the defining CT signature of ACS—the membrane-like sac—is absent in internal hernias.[1,14] Congenital peritoneal encapsulation, a developmental anomaly in which the small bowel lies posterior to an accessory peritoneal membrane derived embryologically from the yolk sac, is usually asymptomatic and distinguishable by its thin, smooth membrane and absence of inflammatory changes.[2,15] Voluminous intussusception, intestinal malrotation, and chronic idiopathic intestinal pseudo-obstruction round out the differential, each distinguishable by specific imaging characteristics.[1] In tuberculosis-endemic regions such as ours, peritoneal tuberculosis represents a critical and eminently treatable differential that must be actively excluded through Mantoux testing, ascitic fluid analysis, and histopathological examination.[5,6]

The pathological architecture: understanding what you are dissecting

When the surgeon incises the peritoneum in a case of ACS and is confronted by the cocoon, it is essential to understand precisely what that structure represents—

because this understanding governs every surgical decision that follows. The membrane is not the peritoneum itself; it is a new, pathological structure deposited upon it.[2] It is composed of densely hyalinised, collagen-rich fibrous tissue with a chronic lymphocytic and mononuclear inflammatory infiltrate, representing the culmination of progressive peritoneal fibrogenesis.[1,2,4] There are no foreign body granulomas, no giant cells, and no birefringent material—their presence would mandate an immediate revision of the diagnosis toward tuberculosis or foreign body reaction.[1]

The membrane exists in two layers in many cases: an outer fibrous shell, tough and relatively avascular, and an inner thinner membrane that is more intimately adherent to the bowel serosa. Our operative findings paralleled those described by Mehmood et al and Lasheen and ElKorety, who similarly documented this bilayered architecture.[4,15] The surgical importance of this structure is paramount: the correct plane of dissection lies between the outer fibrous layer and the serosal surface of the bowel, and the temptation to incise the bowel wall instead of the cocoon—particularly in the presence of dense adhesions obscuring natural tissue planes—represents the most consequential intraoperative hazard.[2,12]

The fibrotic process also involves the root of the mesentery in advanced cases, producing retraction and shortening that draws the bowel loops into compacted clusters. This mesenteric fibrosis generates the pathognomonic "gingerbread man" sign on CT, and intraoperatively manifests as a fixed, tethered mesentery that limits the mobility of released bowel loops even after successful membrane excision.[1] In type II and III cases, the encasement extends to other viscera, demanding a broader operative strategy and heightened vigilance against injury to the liver, stomach, colon, and pelvic organs.[1,2,10]

Surgical strategy: the art of meticulous liberation

The operative management of ACS is elegant in concept but technically exacting in execution. The overarching surgical goal is the complete excision of the encasing membrane and the total liberation of the entrapped bowel—preserving every centimetre of viable intestine and avoiding resection with the same discipline that guides the surgeon away from unnecessary hepatic resection in benign disease.

The preferred approach remains open laparotomy via a midline incision, providing unobstructed access to the entire peritoneal cavity.[1,2,10] Laparoscopic approaches have been described in selected cases, and a recent series by Aziz et al reported successful laparoscopic management in two patients; however, the laparoscopic route carries a significantly elevated risk of iatrogenic bowel injury at the time of trocar insertion, particularly when adhesions distort the normal relationship of the anterior abdominal wall and underlying viscera.[16-18] If laparoscopy is contemplated, the open Hasson technique of port insertion is mandatory, and conversion to open surgery at the first sign of difficulty must be accepted without hesitation.[2]

Once the cocoon is visualised, the operative sequence follows a well-defined progression. An incision is made into the outer fibrous shell, taking care to identify the plane between membrane and serosa. The membrane is then systematically stripped circumferentially—analogous to the technique of decortication in empyema surgery—beginning at a point of relative mobility and progressing toward the fixed mesenteric root. The entire small bowel, from the duodenojejunal flexure to the ileocaecal valve, must be inspected, freed, and evaluated for viability. Adhesions between individual bowel loops within the cocoon are lysed under direct vision, with sharp dissection preferred to minimise inadvertent enterotomies.[2,12]

Bowel resection is the operative decision that demands the greatest discipline in ACS. The literature is unequivocal: resection is indicated only for frankly non-viable—gangrenous or perforated—bowel segments, and never for the cocoon membrane itself.[1,2,10,12] Singh and Gupta's series of 18 cases documented that ileostomy creation was required in only two of 17 operated patients—both presenting with ileal perforation—while the remainder were managed with adhesiolysis alone, confirming that the vast majority of bowel, however severely compressed, recovers full viability once the constricting membrane is removed.[19] Wei et al's series of 24 cases similarly demonstrated that resection without clear indication carries a disproportionate contribution to postoperative morbidity and mortality.[20]

Some authors advocate prophylactic appendectomy at the time of cocoon excision, reasoning that a future acute appendicitis would require re-entry into a hostile, post-operative abdomen.[2,12] We concur with this approach in principle, provided operative conditions permit. The instillation of anti-adhesive agents between liberated bowel loops prior to abdominal closure has been proposed as a strategy to reduce early postoperative small bowel obstruction (EPSBO)—the most common postoperative complication, occurring in approximately 5–6% of cases—though the evidence base for this intervention remains limited.[5,12,19]

Postoperative course and complications

The immediate postoperative period following ACS surgery is characterised by the spectre of EPSBO, which results from extensive bowel manipulation, prolonged operative duration, intestinal oedema, and serosal tears encountered during dissection.[2,12] Li et al demonstrated in their 65-patient series that perioperative nutritional optimisation—enteral where possible, parenteral where necessary—is an independent predictor of favourable postoperative outcomes.[12] Their institutional protocol of somatostatin combined with low-dose corticosteroids in patients developing EPSBO resulted in resolution without

reoperation in the majority, and this conservative approach to EPSBO is now broadly endorsed.[2,12]

Long-term outcomes following complete membrane excision and adhesiolysis are uniformly favourable in primary SEP, with low recurrence rates and excellent functional recovery documented across multiple series.[1,2,10,12] Secondary SEP carries a more guarded prognosis, particularly in the dialysis-associated form, where mortality following surgery has been reported between 45–82%—a figure that reflects the severity of underlying renal disease, the systemic complications of long-term dialysis, and the advanced stage at which surgical intervention is often necessitated.[1,2,13]

Reflections on surgical awareness and institutional learning

The global literature on ACS is composed almost entirely of case reports and small series—a reflection of the condition's rarity and the absence of any single institution's capacity to accumulate statistically powered patient numbers. The systematic review by Akbulut (193 patients, 73 articles, 23 countries) and the meta-analysis by Chorti et al (240 cases, 97 articles) represent the current zenith of evidence aggregation in this field.[2,10] Both unambiguously establish surgical management as the treatment of choice in symptomatic cases, CT as the preoperative imaging cornerstone, and membrane excision with adhesiolysis as the operative gold standard.

We present this case to the surgical community not merely as an institutional record, but as a contribution to the global exercise of pattern recognition that will, case by case and report by report, push the rate of preoperative diagnosis above its current benchmark of less than 30–45%. Every additional case documented with rigour—its imaging, its operative findings, its postoperative course, its histopathology—adds a pixel to the collective clinical picture of a condition that deserves far greater recognition than its numerical incidence alone would suggest.

CONCLUSION

ACS is a mysterious and potentially fatal cause of mechanical obstruction of intestine. It necessitates a heightened level of preoperative apprehension especially when faced with recurring instances of subacute small intestinal blockage in adolescent patients lacking a history of previous abdominal surgery. Although modern imaging techniques like CECT are exceptionally sensitive in identifying the distinctive "cauliflower" encasement of intestinal loops, a conclusive diagnosis is often attained solely through exploratory laparotomy. In tropical and endemic areas, thorough histopathological and microbiological assessment is essential to rule out secondary causes, including abdominal tuberculosis. Comprehensive surgical intervention via careful membrane excision and complete adhesiolysis – while deliberately preventing unwarranted intestinal resection—yields a definite cure, reinstates normal bowel motility, and assures a remarkable long-term prognosis.

This case report presents a classic example of idiopathic abdominal cocoon syndrome (primary sclerosing encapsulating peritonitis) in a 15-year-old female with recurrent episodes of subacute intestinal obstruction. The case highlights the importance of considering ACS in the differential diagnosis of intestinal obstruction in adolescent females, particularly those from tropical regions.

The concurrent finding of cystic dilatation of fallopian tube known as hydrosalpinx can occur as a complication of endometriosis which causes pelvic adhesion and inflammation this supports the theory of retrograde menstruation as a possible etiopathogenic mechanism. The absence of granulomas and negative tuberculosis markers confirmed the idiopathic nature of the condition. Surgical membrane excision with adhesiolysis provided definitive cure, demonstrating the excellent prognosis of this condition when promptly diagnosed and appropriately managed.

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