

Acute Onset Sclerosing Peritonitis after Laparoscopic Hysterectomy- A Rare Case with Review of Literature

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Abstract

Sclerosing peritonitis (SP) is a rare condition in which peritoneal inflammation leads to sclerosis and fibrosis of peritoneum with ultimate encapsulation of the small bowels leading to mechanical obstruction. This entity is mostly described after chronic peritoneal dialysis and acute onset is very rare. Though many risk factors have been described, there are many idiopathic cases too. In this article, we describe a case of sclerosing peritonitis which develops within a span of 1 month after total laparoscopic hysterectomy and bilateral salpingo-oophorectomy. Previously only one case of sclerosing encapsulating peritonitis has been described after abdominal hysterectomy, but its course was chronic. Surgery with minimal adhesiolysis followed by use of steroids and immunosuppressants was the treatment given in our case, which resulted in good clinical outcome. SP is not common and literature is mostly confined to case reports and case series.

Keywords: sclerosing peritonitis, peritoneal dialysis, hysterectomy, adhesiolysis, steroids, immunosuppressants

INTRODUCTION

Sclerosing peritonitis(SP) is a rare but potentially life threatening condition in which the small bowel is encapsulated totally or partially by a fibrocollagenous membrane resulting in partial or complete mechanical bowel obstruction[1]. In this entity, peritoneal deterioration and intraperitoneal inflammation result in intestinal adhesions[2]. With progression of the fibrosing and sclerosing process, the bowel is ultimately invaded and encased in a 'fibrous cocoon', which is formed by collagen and myofibroblastic spindle cells[3,4]. SP was first described in 1907[5], with various terms being used since then like sclerosing encapsulating peritonitis(SEP), encapsulating peritoneal sclerosis(EPS), abdominal cocoon etc. Incidence may vary from 0.5-4.4%[6].

The etiology of SP remains unknown and presumably is multifactorial. The most common risk factor is prolonged history of peritoneal dialysis. Other risk factors are mentioned in table 1[2, 5-9]. A severe episode of pseudomonal or fungal peritonitis may precede the onset of encapsulating peritonitis[10]. In adolescent females, there may be an association with retrograde menstruation[11]. Due to low incidence and chronic course, there is no data comparing the incidence of SP related to various risk factors[12,13].

Symptoms include nausea, vomiting, abdominal pain, anorexia, constipation, ultimately leading to malnutrition[8,13]. In initial stages, there may be fever, increased levels of C-reactive protein(CRP), slight ileus symptoms and ascites[3]. Gradually patients have abdominal pain, fullness, abdominal mass and overt bowel obstruction[8]. Acute presentation includes features of acute sterile peritonitis[14] or a painful abdominal mass with bowel obstruction[15]. In many cases, patients have recurring symptoms over a period of months which gradually increases in severity and frequency and ultimately patient comes with complete bowel obstruction[2]. Complications include bowel ischemia, sepsis, bowel necrosis and enterocutaneous fistula[16].

Differential diagnosis includes congenital peritoneal encapsulation, pseudomyxoma peritonei, cirrhosis, pancreatitis, peritoneal carcinomatosis, peritoneal mesothelioma and tuberculous peritonitis[3,6].

CASE REPORT

A 42yr old lady came to the emergency department of our hospital with pain abdomen, excessive discharge per vagina and abdominal distension following total laparoscopic hysterectomy(TLH) with bilateral salpingo-oophorectomy(BSO) at a peripheral centre 1 month back, done due to abnormal uterine bleeding and fibroid uterus. She is para2, delivered vaginally with no other significant past or family history. She had heavy menstrual bleed around 3months back, which was not controlled by conservative management; and thus TLH was done. BSO was done as intraoperatively the ovaries were not healthy and one ovary had a solid cystic tumor. Histopathology examination(HPE) of her uterus showed leiomyoma with simple glandular endometrial hyperplasia with cystic changes. HPE of one ovary showed serous cystadenofibroma. However 1week post surgery patient developed pain abdomen and excessive vaginal discharge for which oral broad spectrum antibiotics were given. As her symptoms did not improve, she was admitted and injectable antibiotics were given at the same centre. Ultrasound(USG) abdomen done after 3weeks showed moderate septated ascites, bilateral pleural effusion with thickened omentum and mesentery. Blood investigations

showed leucocytosis with raised ESR and pro-calcitonin. Follow up USG showed gradual reduction in ascites. However abdominal pain, feeling of distension and vaginal discharge did not improve and patient was anorexic. She was treated as bacterial peritonitis and Meropenem, Amikacin and Metronidazole injectables were given on her 3rd admission in the peripheral centre at 4th week post surgery. As the condition of the patient did not improve, she was referred to our centre.

On examination, patient had tachycardia, mild pallor. Per abdominal examination revealed mild distension with an irregular firm lump in the paraumbilical region. Patient was tolerating orally and passing stool and flatus. Per speculum and per vaginal examination showed moderate amount of dirty white discharge with an irregular and congested vault. Total leucocyte count was normal with hemoglobin of 8.8gm/dl, CRP of 88, ESR of 122 with normal pro-calcitonin, LFT, RFT and pancreatic enzymes. However serum albumin was very low(1.8gm/dl). Meropenem and Clindamycin injectables were started. USG and CECT abdomen showed loculated ascites, thickened enhancing peritoneum and thickened omentum(Fig1). Ascitic fluid analysis showed lymphocytic infiltrate(700cells/mm with 80% lymphocytes) and no evidence of bacterial or AFB infection(i.e.sterile ascites) with low serum ascites-albumin gradient(SAAG). Urine and vaginal culture also showed no organism. To rule out possibility of malignancy, CA-125, CA-19-9 and CEA were done, which were also normal. Gradually the abdominal discomfort decreased but patient was not able to tolerate solid foods. Thus liquid diet was started from day 4 of admission. USG guided ascitic fluid analysis for malignant cells was negative and omental biopsy showed fibrocollagenous and adipose tissue with lymphocytes, neutrophils and plasma cells infiltrate. Malignancy and tuberculosis was thus ruled out and decision to do exploratory laparotomy was taken. On day 9 of admission, laparotomy was done with a midline longitudinal incision. Extensive dense adhesions were seen between bowel, omentum and mesentery. The omentum was thickened into a firm, thick mass which was covering the bowel loops. The small bowel loops were encased together in a reddish membrane(Fig2). Minimal resection of the membrane and adhesiolysis was achieved as there was no proper plane of separation. Bowel had serosal tears which were repaired and the ascitic fluid was drained. Biopsy was taken from multiple sites. Large bowel was not seen to be affected. While doing adhesiolysis, the vault was seen to be open. However it could not be repaired as it was densely adherent to the bowels and the tissue was fragile. One intraperitoneal drain and indwelling Ryle's tube were given.

Postoperatively patient was monitored in ICU. She was kept nil per orally. IV fluids and albumin infusion were given. The vitals were maintained, but on post-operative day(POD) 3 she had breathing discomfort, her oxygen saturation dropped minimally and she had abdominal distension. Chest X-ray(CXR) showed redistributed pleural effusion(R>L). Strict input-output monitoring was done and diuretics given. As sepsis markers were slightly rising, Inj. Tigecycline was started from day 4. From POD5, orally sips of water were started and gradually liquid and semisolid diet started. Patient improved symptomatically. From POD9, oral antibiotics were started. Patient was passing flatus and stool. HPE showed fibrocollagenous and adipose tissue with areas of hemorrhage and necrosis. Elongated spindle shaped cells were seen with infiltration by lymphocytes, plasma cells and macrophage. Thus depending on laparotomy and HPE findings, we diagnosed this case as sclerosing peritonitis in stage 2. Prednisone 30mg was started and after 2weeks follow up, azathioprine was started with gradual tapering of steroids.

Follow up upto 6months showed the patient was doing better symptomatically with the lump becoming softer.

DISCUSSION

Though there is a history of recent surgery in this case, it is very difficult to find the definite cause of the sclerosing peritonitis here. The most possible mechanism may be presence of subclinical peritonitis with a genetic predisposition, which flared up after the surgery. There is literature suggesting that low grade or subclinical peritonitis in which patient does not develop any symptoms, may ultimately lead to sclerosis, membrane formation and development of abdominal cocoon[17-19]. Though SP has been described as a chronic phenomenon, but there are reports of rapid development of membrane within 12weeks[18,20]. Acute SP after surgery has been described in cases of liver transplantation. In most of these cases the patients had perioperative acute peritonitis and ultimately developed SP[21]. There is only one case report of SP after abdominal hysterectomy done for gestational trophoblastic disease, in which features developed after 3years. Here they have attributed SP to chronic and recurrent low grade peritonitis after the surgery, which manifested as chronic abdominal pain[22].

The exact etiology and pathophysiology is not known. Inflammation or infection in presence of peritoneal deterioration, that is mostly seen after peritoneal dialysis or chronic peritoneal irritation leads to formation of the capsule. Thus SP development depends on the balance between the severity of peritoneal deterioration and that of inflammation[2,23]. However complications of intense bacterial peritonitis can readily lead to SP[24].

The pathologic features of SP are peritoneal thickening and encapsulation. The capsule consists of mainly fibrin exudation from peritoneal microvessels[25] which undergo hyperplasia[26]. The fibrocytes are transformed into fibroblasts by the transforming growth factor(TGF β)[9]. These invading fibroblasts then lyse or remodel the fibrin leading to fibrosis and adhesion formation[27]. However degree of microvascular hyperplasia varies among patients. Patients with mild microvascular hyperplasia may develop SP after a strong, traumatic, superimposed inflammation; whereas, those with marked microvascular hyperplasia may develop SP after mild stimuli[25,26]. Thus the development of SP is a complex process and involves various growth factors.

Garosi has divided the idiopathic cases into 2 categories- first, which is associated with retroperitoneal fibrosis or peritoneal sclerosis, i.e. a systemic connective tissue disorder and second, which has a genetic predisposition[15]. Nakamoto categorized SP into 4groups depending on clinical presentation[28]. In Stage1 patient is usually asymptomatic with mild ascites and no inflammation. In Stage2, inflammatory stage, patients have nausea and altered bowel habits due to partial encapsulation of bowel and intestinal swelling. Mild inflammation with fibrin exudation is present. In Stage3, there is formation of the cocoon with symptoms of bowel obstruction. There may be associated mild to severe inflammation. Stage4 is the chronic stage of ileus. There is absolute bowel obstruction due to thickening of the encapsulating cocoon with little inflammation. Based on this, our patient can be put up in stage2 or in early stage3.

Diagnosis of SP is very difficult. Symptoms are vague and non-localizing. Abdominal X ray findings are non-specific. CE-CT is an useful tool and helps in pre-operative diagnosis[29] and

also helps to monitor disease progression[8]. Ultrasound may show trilaminar appearance of bowel, tethering of bowel to posterior abdominal wall, ascites and dilatation and fixation of bowel loops[30]. Peritoneal enhancement, peritoneal thickening, calcification, bowel tethering, bowel wall thickening, thick fibrous enhancing membrane, signs of bowel obstruction, peritoneal or mesenteric thickening and loculated collections are the most common CT findings[1,31]. But CT findings are not so specific. A high index of suspicion is required to make a diagnosis preoperatively based on clinical and imaging findings. Diagnosis is confirmed by surgical exploration and histopathology(HPE) of peritoneal biopsy[15]. Diagnostic laparoscopy can help in diagnosis[32], but management requires laparotomy. Dynamic cine-MR scanning with advanced image analysis may be useful in early detection of SP. However, more studies are required to find the efficacy of this modality[33].

Intraoperative findings may show a thickened brownish or a whitish, opaque peritoneum encapsulating the entire intestine[8,9]. The intestinal loops are adherent to one another. Adhesions between the visceral and parietal peritoneum are rare, except in cases of severe inflammation[34].

Histopathology of the peritoneum shows proliferation of fibro-connective tissue, inflammatory infiltrates, and dilated lymphatics, with no evidence of foreign body granulomas, giant cells, or birefringent material[34,35]. Various HPE features have been described- submesothelial cell thickening, arterial occlusion, tissue and arterial calcification, fibrin deposition, fibroblasts hypertrophy, capillary angiogenesis, mononuclear cell infiltrate, positive iron staining, positive immunohistochemical staining of podoplanin, upregulation of VEGF and down regulation of mast cells[8].

Medical management using steroids, tamoxifen, immunosuppressants like azathioprine, methotrexate, rapamycin, mycophenolate mofetil, sirolimus, and cyclosporine etc.[8,36] have been proposed in early cases of the disease to reduce the ascitic fluid, inflammation and also to prevent fibrin deposition and collagen synthesis[14,23,37]. Steroids with or without total parenteral nutrition(TPN) have shown to improve symptoms. Tamoxifen has shown beneficial effects which acts by upregulating transforming growth factor(TGF) β , which stimulates metalloproteinase-9 to remove degenerated collagen, thereby preventing damage to mesothelial cells[38,39]. However some studies have not shown any benefit[40]. Apart from steroids, evidence regarding the efficacy of any other immunosuppressive therapy in peritoneal sclerosis remains weak due to lack of robust randomized clinical trials[41].

In state of bowel obstruction, surgery is the choice. Treatment is excision of the membrane and releasing the adhesions around bowel loops. Bowel resection is required only in non-viable areas[42,43]. Lysing the encapsulating membrane may be a technical challenge[1,18]. If it is difficult to lyse the encapsulated intestine, a longitudinal incision of the capsules on the intestinal surface may be made; but the actual site of bowel obstruction must be relieved. However, because the fibrin membranes are continuous with the altered peritoneum, the serosa may not be demarcated, making it difficult to identify the plane of separation. Severe peritoneal calcification makes the adhesiolysis even more difficult. Inappropriate adhesiolysis or careless intestinal traction can lead to intestinal perforation. Intense difficulties during the removal process may result in gut perforation[1,18,44]. Also there is possibility of intestinal fistula in a thinned portion of the intestine because of increased peristalsis 2 – 3 days after

surgery. In cases where there is severe damage and bowel is difficult to suture, jejunostomy may be considered[2].

Surgery relieves the state of intestinal obstruction, but it does not improve the peritoneal deterioration. Thus, re-encapsulation can occur and SP may recur after 6-12months[24] with 25% recurrence rate post surgery[8]. Adhesions also occur due to surgical injury to the intestinal wall and serosa. Failure to perform a complete adhesiolysis leads to persistence of symptoms in some cases. In recurrent patients or in those where complete adhesiolysis is difficult, bypass anastomosis between the oral site jejunum and large intestine may be required. Mortality post-surgery ranges from 19 to 34.5%[45], and is mainly seen in patients with advanced SP presenting as surgical emergencies[46]. The mortality of SP can be as high as 24% and may be even 60% in cases managed operatively[3] due to complications like anastomosis insufficiency, intraabdominal infections, and enterocutaneous fistulas. However studies have shown excellent postoperative prognosis in which bowel loops were not injured[47]. In Japan, introduction of Noble placcation (suturing of the intestines to each other to prevent obstruction)along with enterolysis has reduced the recurrence rate to12.3%[45].

The current literature supports surgery as the curative treatment of established SP[24,42]. Recent research suggests a good outcome following surgical intervention with either no or minimal perioperative mortality with survival rate of 94% after three years of follow-up[47,48].

A course of postoperative steroids with or without immunosuppressants may help in avoiding the stress from surgical stimuli, reducing inflammation, reducing relapses and improving symptoms. In our case, prednisolone and azathioprine have been used. This intervention has shown to improve the survival rate in some studies[47,49]. Drugs are also found to be successful in cases where complete adhesiolysis or excision could not be done[39,50].

CONCLUSION

Sclerosing peritonitis is a benign and a rare cause of mechanical bowel obstruction and needs a high index of suspicion for diagnosis when no other cause is found. Exact cause is not known. CECT mostly aids in the diagnosis and planning elective surgery in experienced hands. In early stages, steroids help in reducing inflammation and progression of the disease. In later stages, dissection of the membrane from the gut to release the entrapped intestine and separation of the inter loop adhesions is the treatment of choice. Iatrogenic injury to bowel and consequent bowel resections are to be avoided as these lead to high morbidity. However, there is no consensus on whether surgery or medical management is preferred. Due to the low incidence of SP, there are no clinical trials comparing the different therapeutic approaches, and experience is limited to case reports. However best outcomes are reported in early surgical intervention cases, followed by course of steroids and immunosuppressants.

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References

- [1] Machado NO. Sclerosing Encapsulating Peritonitis: Review. *Sultan Qaboos Univ Med J*. 2016;16(2):e142-e151. doi:10.18295/squmj.2016.16.02.003
- [2] Kawanishi H, Moriishi M, Ide K, Dohi K. Recommendation of the surgical option for treatment of encapsulating peritoneal sclerosis. *Perit Dial Int*. 2008 Jun;28 Suppl 3:S205-10. PMID: 18552257.
- [3] Pepels MJ, Peters FP, Mebis JJ, Ceelen TL, Hoofwijk AG, Erdkamp FL. Sclerosing peritonitis: an unusual cause of ascites in a patient with systemic lupus erythematosus. *Neth J Med*. 2006 Oct;64(9):346-9. PMID: 17057274.
- [4] Odama UO, Shih DJ, Korbet SM. Sclerosing peritonitis and systemic lupus erythematosus: a report of two cases. *Perit Dial Int*. 1999 Mar-Apr;19(2):160-4. PMID: 10357188
- [5] Owtschinnikow PJ, "Peritonitis chronica fibrosa incapsulata," *Archiv fur Klinische Chirurgie*, 1907. vol. 83, pp. 623-634, 1907.
- [6] Caldwell J, Dyer A. Sclerosing encapsulating peritonitis (cocoon bowel) presenting after laparotomy for splenic abscess. *J Radiol Case Rep*. 2013 Oct 1;7(10):17-23. doi: 10.3941/jrcr.v7i10.1508. PMID: 24421919; PMCID: PMC3888340.
- [7] Foo KT, Ng KC, Rauff A, Foong WC, Sinniah R. Unusual small intestinal obstruction in adolescent girls: the abdominal cocoon. *Br J Surg*. 1978 Jun;65(6):427-30. doi: 10.1002/bjs.1800650617. PMID: 656764.
- [8] Moinuddin Z, Summers A, Van Dellen D, Augustine T, Herrick SE. Encapsulating peritoneal sclerosis-a rare but devastating peritoneal disease. *Front Physiol*. 2015 Jan 5;5:470. doi: 10.3389/fphys.2014.00470. PMID: 25601836; PMCID: PMC4283512.
- [9] Singhal M, Krishna S, Lal A, Narayanasamy S, Bal A, Yadav TD et al. Encapsulating Peritoneal Sclerosis: The Abdominal Cocoon. *Radiographics*. 2019 Jan-Feb;39(1):62-77. doi: 10.1148/rg.2019180108. Epub 2018 Dec 7. PMID: 30526331.
- [10] Kawanishi H, Kawaguchi Y, Fukui H, Hara S, Imada A, Kubo H et al. Encapsulating peritoneal sclerosis in Japan: a prospective, controlled, multicenter study. *Am J Kidney Dis*. 2004 Oct;44(4):729-37. PMID: 15384025.
- [11] Minutolo V, Gagliano G, Angirillo G, Minutolo O, Morello A, Rinzi C. Intestinal obstruction due to idiopathic sclerosing encapsulating peritonitis. Clinical report and review of literature. *G Chir*. 2008 Apr;29(4):173-6. PMID: 18419984.
- [12] Keating JP, Neill M, Hill GL. Sclerosing encapsulating peritonitis after intraperitoneal use of povidone iodine. *Aust N Z J Surg*. 1997 Oct;67(10):742-4. doi: 10.1111/j.1445-2197.1997.tb07126.x. PMID: 9322732.
- [13] Merkle M, Wörnle M. Sclerosing Peritonitis: A Rare but Fatal Complication of Peritoneal Inflammation. *Mediators of Inflammation*. Volume 2012, Article ID 709673. <https://doi.org/10.1155/2012/709673>
- [14] Jung JY, Cho JT. A case of fulminant sclerosing peritonitis presented like acute culture-negative peritonitis and successfully treated with corticosteroid therapy. *J Korean Med Sci*. 2013;28(4):620-623. doi:10.3346/jkms.2013.28.4.620
- [15] Garosi G, Di Paolo N. Peritoneal sclerosis: one or two nosological entities? *Semin Dial*. 2000 Sep-Oct;13(5):297-308. doi: 10.1046/j.1525-139x.2000.00080.x. PMID: 11014691.
- [16] Kawaguchi Y, Kawanishi H, Mujais S, Topley N, Oreopoulos DG. Encapsulating peritoneal sclerosis: definition, etiology, diagnosis, and treatment. *International Society for Peritoneal Dialysis Ad Hoc*

- Committee on Ultrafiltration Management in Peritoneal Dialysis. *Perit Dial Int.* 2000;20 Suppl 4:S43-55. PMID: 11098928.
- [17] Tannoury JN, Abboud BN. Idiopathic sclerosing encapsulating peritonitis: abdominal cocoon. *World J Gastroenterol.* 2012;18(17):1999-2004. doi:10.3748/wjg.v18.i17.1999
- [18] Akbulut S. Accurate definition and management of idiopathic sclerosing encapsulating peritonitis. *World J Gastroenterol.* 2015 Jan 14;21(2):675-87. doi: 10.3748/wjg.v21.i2.675. PMID: 25593498; PMCID: PMC4292304.
- [19] Serter A, Kocakoç E, Çipe G. Supposed to be rare cause of intestinal obstruction; abdominal cocoon: report of two cases. *Clin Imaging.* 2013 May-Jun;37(3):586-9. doi: 10.1016/j.clinimag.2012.08.010. Epub 2012 Oct 3. PMID: 23041158.
- [20] Candido Pde C, Werner Ade F, Pereira IM, Matos BA, Pfeilsticker RM, Silva R Filho. Sclerosing encapsulating peritonitis: a case report. *Radiol Bras.* 2015 Jan-Feb;48(1):56-8. doi: 10.1590/0100-3984.2013.1909. PMID: 25798009; PMCID: PMC4366030.
- [21] Mekeel K, Moss A, Reddy KS, Douglas D, Mulligan D. Sclerosing peritonitis and mortality after liver transplantation. *Liver Transpl.* 2009 Apr;15(4):435-9. doi: 10.1002/lt.21702. PMID: 19326414.
- [22] Suh WN, Lee SK, Chang H, Hwang HJ, Hyung WJ, Park YN et al. Sclerosing encapsulating peritonitis (abdominal cocoon) after abdominal hysterectomy. *Korean J Intern Med.* 2007 Jun;22(2):125-9. doi: 10.3904/kjim.2007.22.2.125. PMID: 17616031; PMCID: PMC2687622.
- [23] Kawanishi H, Harada Y, Noriyuki T, Kawai T, Takahashi S, Moriishi M et al. Treatment options for encapsulating peritoneal sclerosis based on progressive stage. *Adv Perit Dial.* 2001;17:200-4. PMID: 11510276.
- [24] Kawanishi H, Moriishi M, Tsuchiya S. Experience of 100 surgical cases of encapsulating peritoneal sclerosis: investigation of recurrent cases after surgery. *Adv Perit Dial.* 2006;22:60-4. PMID: 16983941.
- [25] Dobbie JW. Pathogenesis of peritoneal fibrosing syndromes (sclerosing peritonitis) in peritoneal dialysis. *Perit Dial Int.* 1992;12(1):14-27. PMID: 1347465.
- [26] Numata M, Nakayama M, Nimura S, Kawakami M, Lindholm B, Kawaguchi Y. Association between an increased surface area of peritoneal microvessels and a high peritoneal solute transport rate. *Perit Dial Int.* 2003 Mar-Apr;23(2):116-22. PMID: 12713076.
- [27] Sulaiman H, Dawson L, Laurent GJ, Bellington GJ, Herrick SE. Role of plasminogen activators in peritoneal adhesion formation. *Biochem Soc Trans.* 2002 Apr;30(2):126-31. PMID: 12023839
- [28] Nakamoto H. Encapsulating peritoneal sclerosis--a clinician's approach to diagnosis and medical treatment. *Perit Dial Int.* 2005 Apr;25 Suppl 4:S30-8. PMID: 16300270
- [29] Hur J, Kim KW, Park MS, Yu JS. Abdominal cocoon: preoperative diagnostic clues from radiologic imaging with pathologic correlation. *AJR Am J Roentgenol.* 2004 Mar;182(3):639-41. doi: 10.2214/ajr.182.3.1820639. PMID: 14975962.
- [30] Mohanty D, Jain BK, Agrawal J, Gupta A, Agrawal V. Abdominal cocoon: clinical presentation, diagnosis, and management. *J Gastrointest Surg.* 2009 Jun;13(6):1160-2. doi: 10.1007/s11605-008-0595-7. Epub 2008 Jul 23. PMID: 18649113.
- [31] Vlijm A, Stoker J, Bipat S, Spijkerboer AM, Phoa SS, Maes R et al. Computed tomographic findings characteristic for encapsulating peritoneal sclerosis: a case-control study. *Perit Dial Int.* 2009 Sep-Oct;29(5):517-22. PMID: 19776044.

- [32] Qasaimeh GR, Amarin Z, Rawshdeh BN, El-Radaideh KM. Laparoscopic diagnosis and management of an abdominal cocoon: a case report and literature review. *Surg Laparosc Endosc Percutan Tech*. 2010 Oct;20(5):e169-71. doi: 10.1097/SLE.0b013e3181f193ec. PMID: 20975494.
- [33] Wright B, Summers A, Fenner J, Gillott R, Hutchinson CE, Spencer PA et al. Initial observations using a novel "cine" magnetic resonance imaging technique to detect changes in abdominal motion caused by encapsulating peritoneal sclerosis. *Perit Dial Int*. 2011 May-Jun;31(3):287-90. doi: 10.3747/pdi.2010.00078. Epub 2011 Mar 31. PMID: 21454394.
- [34] Honda K, Oda H. Pathology of encapsulating peritoneal sclerosis. *Perit Dial Int*. 2005 Apr;25 Suppl 4:S19-29. PMID: 16300269.
- [35] Okada K, Onishi Y, Oinuma T, Nagura Y, Soma M, Saito S et al. Sclerosing encapsulating peritonitis: regional changes of peritoneum. *Nephron*. 2002 Oct;92(2):481-3. doi: 10.1159/000063290. PMID: 12218337.
- [36] Lafrance JP, Létourneau I, Ouimet D, Bonnardeaux A, Leblanc M, Mathieu N et al. Successful treatment of encapsulating peritoneal sclerosis with immunosuppressive therapy. *Am J Kidney Dis*. 2008 Feb;51(2):e7-10. doi: 10.1053/j.ajkd.2007.07.036. PMID: 18215692.
- [37] Habib SM, Betjes MG, Fieren MW, Boeschoten EW, Abrahams AC, Boer WH et al; Eps Registry. Management of encapsulating peritoneal sclerosis: a guideline on optimal and uniform treatment. *Neth J Med*. 2011 Nov-Dec;69(11):500-7. PMID: 22173363.
- [38] Allaria PM, Giangrande A, Gandini E, Pisoni IB. Continuous ambulatory peritoneal dialysis and sclerosing encapsulating peritonitis: tamoxifen as a new therapeutic agent? *J Nephrol*. 1999 Nov-Dec;12(6):395-7. PMID: 10626830.
- [39] Cornelis T, Oreopoulos DG. Update on potential medical treatments for encapsulating peritoneal sclerosis; human and experimental data. *Int Urol Nephrol*. 2011 Mar;43(1):147-56. doi: 10.1007/s11255-010-9744-5. Epub 2010 May 7. PMID: 20449655; PMCID: PMC3061214.
- [40] Evrenkaya TR, Atasoyu EM, Unver S, Basekim C, Baloglu H, Tulbek MY. Corticosteroid and tamoxifen therapy in sclerosing encapsulating peritonitis in a patient on continuous ambulatory peritoneal dialysis. *Nephrol Dial Transplant*. 2004 Sep;19(9):2423-4. doi: 10.1093/ndt/gfh150. PMID: 15299115.
- [41] Bozkurt D, Sipahi S, Cetin P, Hur E, Ozdemir O, Ertilav M et al. Does immunosuppressive treatment ameliorate morphology changes in encapsulating peritoneal sclerosis? *Perit Dial Int*. 2009 Feb;29 Suppl 2:S206-10. PMID: 19270219.
- [42] Kawanishi H, Watanabe H, Moriishi M, Tsuchiya S. Successful surgical management of encapsulating peritoneal sclerosis. *Perit Dial Int*. 2005 Apr;25 Suppl 4:S39-47. PMID: 16300271.
- [43] Taylor M, Clarke MG, Jarvis J, Booth M. A mystery wrapped in an enigma: the abdominal cocoon syndrome. *N Z Med J*. 2012 Nov 23;125(1366):77-80. PMID: 23254530.
- [44] Oran E, Seyit H, Besleyici C, Ünsal A, Aliş H. Encapsulating peritoneal sclerosis as a late complication of peritoneal dialysis. *Ann Med Surg (Lond)*. 2015 Apr 2;4(3):205-7. doi: 10.1016/j.amsu.2015.03.006. PMID: 26150908; PMCID: PMC4486463.
- [45] Kawanishi H. Surgical and medical treatments of encapsulation peritoneal sclerosis. *Contrib Nephrol*. 2012;177:38-47. doi: 10.1159/000336934. Epub 2012 May 8. PMID: 22613913.
- [46] Augustine T, Brown PW, Davies SD, Summers AM, Wilkie ME. Encapsulating peritoneal sclerosis: clinical significance and implications. *Nephron Clin Pract*. 2009;111(2):c149-54; discussion c154. doi: 10.1159/000191214. Epub 2009 Jan 16. PMID: 19147997.

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- [47] Célécout B, Levard H, Hay J, Msika S, Fingerhut A, Pelissier E. Sclerosing encapsulating peritonitis: early and late results of surgical management in 32 cases. French Associations for Surgical Research. Dig Surg. 1998;15(6):697-702. doi: 10.1159/000018681. PMID: 9845640.
- [48] Singh B, Gupta S. Abdominal cocoon: a case series. Int J Surg. 2013;11(4):325-8. doi: 10.1016/j.ijsu.2013.02.011. Epub 2013 Feb 28. PMID: 23459185.
- [49] Junor BJ, McMillan MA. Immunosuppression in sclerosing peritonitis. Adv Perit Dial. 1993;9:187-9. PMID: 8105920.
- [50] Solak A, Solak İ. Abdominal cocoon syndrome: preoperative diagnostic criteria, good clinical outcome with medical treatment and review of the literature. Turk J Gastroenterol. 2012;23(6):776-9. doi: 10.4318/tjg.2012.0500. PMID: 23864454.

Table 1: Risk factors of SP

Infections	Recurrent peritonitis Tuberculosis Cytomegalovirus or parasitic infections Acute fungal or pseudomonal peritonitis
Autoimmune/ inflammatory	SLE Sarcoidosis Familial Mediterranean fever Protein S deficiency Fibrogenic foreign materials
Surgery related	Renal or liver transplant Ventriculoperitoneal or peritoneovenous shunts Use of povidone iodine
Peritoneal irritation	Rupture of dermoid cyst Trauma ⁶ Endometriosis Intraperitoneal chemotherapy Intraperitoneal exposure to particulate matter
Possible Paraneoplastic phenomenon in various tumors-	Gastric cancer Ovarian thecoma Ovarian teratoma Pancreas carcinoma Renal carcinoma
Drugs	Beta blockers Vancomycin Asbestos exposure

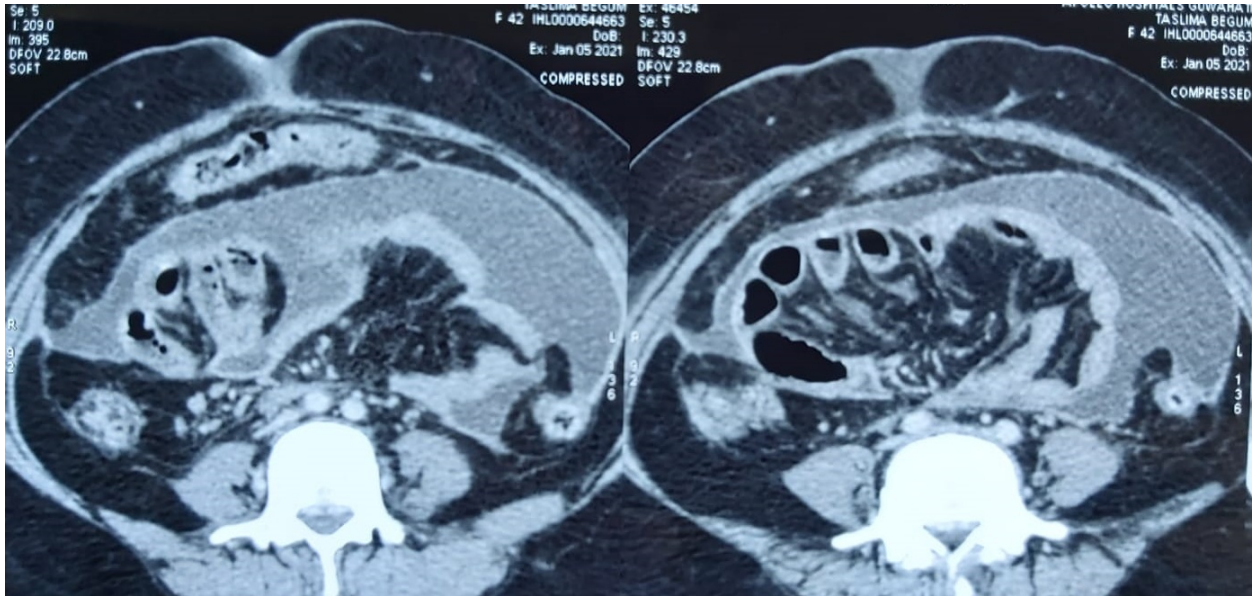
FIGURES

Fig 1: CT scan abdomen showing thickening of omentum, mesentery and peritoneal enhancement.

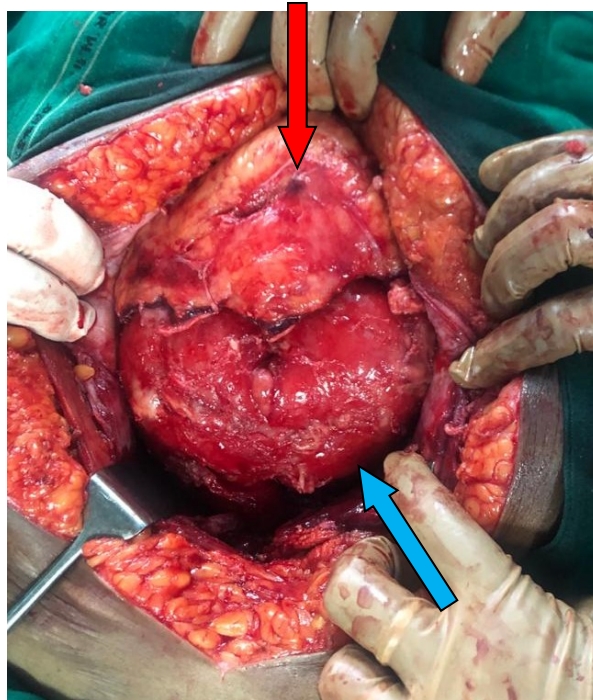


Fig 2: Thickened leather like omentum(red) and small bowel loops encapsulated together in a membrane(blue)