Increased peritoneal function has been suggested to be a risk factor for developing encapsulating peritoneal sclerosis (EPS); however, clinical evidence is scarce. The present study aimed to clarify the specific character of peritoneal function in patients who developed EPS after withdrawal from peritoneal dialysis (PD).

We studied 12 patients who developed EPS after PD withdrawal [(EPS group) mean PD duration: 109 months; mean period of EPS development: 7.0 months after withdrawal] and 128 patients who did not develop EPS (non EPS group). All 140 patients were withdrawn from continuous ambulatory peritoneal dialysis (CAPD) and were observed for the following 24 months. Based on the records of the annual peritoneal equilibration tests (PETs), we analyzed (1) the patients’ dialysate-to-plasma (D/P) creatinine at various durations on PD, and (2) the accumulative appearance incidence of high-transport (HT) state of peritoneal membrane.

The mean D/P creatinine in EPS group was significantly higher than that in the non EPS group in the course of PD from the 6th to the 10th year. The accumulative incidence of HT was significantly higher in the EPS group than in the non EPS group, indicating early development of HT membrane in EPS group.

Early development of increased D/P creatinine, classified as HT state, was observed during certain periods on PD in patients who developed EPS after PD withdrawal. That finding may indicate that HT state of peritoneal membrane is an early marker for EPS, and that the PET is useful to detect patients at high risk of EPS.

**Key words**
Encapsulating peritoneal sclerosis (EPS), high-transport state, peritoneal equilibration test, long-term CAPD

**Introduction**
Long-term continuous ambulatory peritoneal dialysis (CAPD) treatment has been revealed to be one of the risk factors for developing encapsulating peritoneal sclerosis (EPS) (1). Peritoneal solute transport is reported to increase in parallel with the duration of CAPD treatment (2). Thus, it is supposed that patients with EPS might have characteristic properties of peritoneal function. However, the issue remains unclear and controversial, probably partly because of the various stages of EPS pathology found in examinations performed for previous reports. In addition, no data are available regarding peritoneal function before the development of EPS. Clarification concerning EPS and peritoneal function could lead to detection of risk factors for EPS in the clinical setting.

The aim of the present study was to use dialysate-to-plasma (D/P) creatinine to retrospectively examine the sequential changes of peritoneal function in the course of CAPD treatment, and to compare results between the patients who developed EPS and those who did not.

**Patients and methods**
We enrolled 140 patients into the study. All patients were withdrawn from CAPD in the period preceding December 1998. Thereafter, they were followed up for 24 months. After being transferred to regular hemodialysis, 12 patients developed EPS (EPS group); the other 128 patients did not (non EPS group). Table I shows the profiles of the patients.

All patients performed a peritoneal equilibration test (PET) annually until the end of CAPD treatment. The diagnosis of EPS was primarily based on the recommendations of the Ad Hoc Committee of the International Society for Peritoneal Dialysis.

At the start of PD, the EPS group was significantly younger than the non EPS group. Although PD was conducted over a long period of years, no significant
We used D/P creatinine to classify peritoneal function into the four categories of high, high-average, low-average, and low as reported elsewhere.

**Analysis 2**

We used the Kaplan—Meier method to examine the cumulative incidence of high transport based on D/P creatinine. Because all of the patients with EPS had performed CAPD for more than 60 months, we excluded cases in which the term of PD was less than 60 months from the analysis. Consequently, 63 patients (long-term PD group) remained as controls for the examination (Table I). The point in time at which high transport appeared in each case was set as the discontinuation time of the treatment.

**Results**

**Result 1**

In the cross-sectional data on D/P creatinine (based on annual PET results), the EPS group tended to show a higher value as compared with the non EPS group. Significant differences were seen between the groups at the 6th, 7th, 9th years (Figure 1). However, after the 10th year, no significant differences were observed between the groups.

In the analysis of peritoneal function, the same trends were observed. In the non EPS group, the incidence of high transport increased in parallel with the duration of PD (8.0% at 5th year, reaching 80.0% at the 12th year of PD). On the other hand, the EPS group difference was observed between the groups concerning the frequency of peritonitis. In the EPS group, the primary disease was chronic glomerulonephritis (9 of 12 cases). Average age at the start of PD was 35.1 years; average duration of PD was 109 months; average time from discontinuation of PD to onset of EPS was 7.0 months; and frequency of onset of peritonitis was 0.16 episodes per year. The reason for PD withdrawal was ultrafiltration failure in all cases (Table II). In regard to the incidence of EPS and its relationship to the duration of CAPD, we observed EPS in no patient where PD was conducted for less than 60 months.

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We compared D/P creatinine between the EPS group and the non EPS group on the basis of annual data obtained in the course of CAPD treatment. In addition, we used D/P creatinine to classify peritoneal function into the four categories of high, high-average, low-average, and low as reported elsewhere.

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showed a 40.0% incidence of high transport even at the 5th year of PD, and that incidence increased to 80.0% thereafter (Figure 2).

Result 2
Figure 3 shows the cumulative incidence of high transport. A significant difference was seen between the groups. The EPS group showed a greater incidence of high transport in the early stage as compared with the long-PD group.

Discussion
The results of the present study can be summarized as follows: the cross-sectional data showed that the inci-
dence of increased solute transport state rose with the duration of PD in patients who did not develop EPS. The incidence of increased transport state was higher in patients who developed EPS than in patients who did not, which is also reflected in the incidence of the high peritoneal membrane classification. The cumulative incidence of high transport was apparently higher in EPS group in the course of CAPD treatment.

In the present study, the patients with EPS had characteristic clinical features, such as very long-term CAPD treatment and onset of EPS after withdrawal from CAPD. The patients with EPS had characteristic peritoneal function properties in the course of CAPD treatment as compared with patients who did not develop EPS. An increased HT state that is found relatively early in the course of PD may be a characteristic property for patients at high risk of EPS. The mechanism of increased solute transport remains undetermined. However, it was previously demonstrated that microvessel walls are the main barrier to solute transport, and thus HT-state membrane (reflected in an increased D/P creatinine) might be related to an increase in microvessels in the peritoneum (3). A recent study revealed that there is neoangiogenesis in patients with peritoneal sclerosis (4). In that respect, knowing more about the state of stimulated neoangiogenesis in the peritoneum in those cases may be very interesting. That stimulated state may be involved in the mechanism of EPS development. The hypothesis awaits further study in future.

**Conclusion**

Early-onset HT state in the course of PD may be a risk factor for EPS after withdrawal from long-term PD treatment.

**References**


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To evaluate the incidence and characteristics of encapsulating peritoneal sclerosis (EPS), we analyzed 111 patients who had been transferred to hemodialysis and had been periodically monitored using the peritoneal equilibration test. Encapsulating peritoneal sclerosis was diagnosed in 11 patients. All patients had high-transport peritoneum, and 10 patients had been treated with continuous ambulatory peritoneal dialysis (CAPD) for more than 72 months. Incidence of EPS increased according to prolongation of CAPD.

To evaluate outcome, we analyzed 15 cases of EPS. All patients were treated with total parenteral nutrition (TPN). Five patients improved with TPN alone. However, the remaining 10 patients showed deterioration. Of those 10, 4 patients died. They had not been treated with corticosteroids (CSs). The remaining 6 patients were treated with CSs, but they did not improve. Those cases required surgical treatment. The postoperative course in 4 patients was satisfactory, but 2 patients died.

In our center, the EPS survival rate was 60%. In patients treated with TPN alone, the remission rate was only 33.3%. Administration of CSs could not improve that rate. In 6 patients treated with surgery, the mortality rate was 33.3%. When EPS symptoms are not ameliorated by CS and TPN, surgical treatment should be considered. To prevent EPS, high-risk patients who have more than 72 months on CAPD and who have a high-transport peritoneum should discontinue CAPD.

**Key words**
Encapsulating peritoneal sclerosis, outcomes, corticosteroid, total parenteral nutrition

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**Introduction**
Encapsulating peritoneal sclerosis (EPS) is a serious complication of continuous ambulatory peritoneal dialysis (CAPD) because EPS influences not only the continuation of CAPD treatment, but also patient survival (1—4). It is therefore a matter of urgency to establish an effective therapeutic strategy for EPS.

We previously reported that total parenteral nutrition (TPN) with gastric drainage was an effective treatment in 8 patients with EPS, and could improve the prognosis (5). However, many cases were not improved by TPN alone. Therefore, corticosteroids (CSs) were administered to patients with noninfectious inflammation (6). When EPS symptoms were not improved by CS administration or TPN or both, then total intestinal enterolysis was performed (7).

Several investigators have evaluated the mechanism and prevention of EPS. However, their analyses of the outcome are not persuasive, because treatment is not performed according to a uniform strategy. In analyzing the patients with EPS in our center, we propose a timing for CAPD discontinuation and a beneficial effect of surgical treatment.

**Methods**

**Classification of EPS**
We classify EPS into four periods. The first is the pre-symptomatic period, in which the peritoneal thickening and sclerosis appear alone, without intestinal adhesions. The second is the inflammatory period (not bacterial infection), in which peritoneal inflammation, intestinal edema, and ascites are present. The third is the encapsulating period, in which the inflammation has settled, and intestinal adhesions and encapsulation are present. The fourth is the ileus period, in which obstruction of the small bowel occurs because of widespread adhesions and encapsulation. However, the boundaries between the periods are indistinct.
We then analyzed patients who showed typical EPS in the encapsulating and ileus periods (the most serious phases clinically), because most physicians cannot undeniably diagnose EPS. Our definition of typical EPS is this: Continuous or intermittent presence of ileus symptoms owing to small-bowel obstruction, and diffuse peritoneal thickening and widely distributed intestinal adhesions confirmed by computed tomography or laparoscopy (8).

Incidence of typical EPS

The incidence of EPS in Europe and the United States is about 0.5%—2.5% (1). In 1998, a Japanese EPS study group reported that 1.7% of 7343 patients (124 patients) were identified as having EPS. In a recent re-evaluation, Kawanishi et al reported that 2.8% of 3760 patients (106 cases) were diagnosed with EPS (9). Those incidences were calculated for all patients. In other words, many ongoing patients with short-term CAPD durations were included. However, 84.6% of the patients with EPS were diagnosed after transfer to hemodialysis (HD) (9). Moreover, many EPS patients lacked a previous history of bacterial peritonitis, but underwent CAPD treatment for long durations. Those facts suggest that it is not adequate to calculate frequency by including ongoing CAPD patients. Therefore, to evaluate the incidence of typical EPS, we analyzed patients who were transferred to HD from CAPD for reasons such as ultrafiltration failure and refractory peritonitis.

The subjects of the present analysis were 111 patients in our hospital who had been transferred to HD from CAPD, and who had been periodically monitored for peritoneal function by peritoneal equilibration test (PET). Among the 111 patients, 11 (9.9%) were identified as having typical EPS; 10 of the 11 had been on CAPD for long durations (more than 72 months). All patients with EPS had a high-transport peritoneal membrane (PET category). Figure 1 shows the frequency of EPS by duration of CAPD. Incidence increased in accordance with prolonged duration of CAPD. In the patients with more than 120 months CAPD duration, 33.3% (5 of 15 cases) of the patients were diagnosed as having typical EPS.

Outcome of typical EPS

Another 4 patients were added to the above-mentioned cases. Consequently, 15 patients with EPS were treated in our hospital. Figure 2 shows the outcomes of those 15 patients.

All patients were treated with TPN. Five of the patients improved after TPN alone; however, 10 patients did not. Four of the 10 patients died of sepsis or...
pneumonia. Those 4 patients were not treated with CSs because, at the time, the usefulness of CS treatment for EPS was not yet supported. The other 6 patients received CSs. Dosage of prednisolone was 0.5—1.0 mg/kg body weight daily in the first 2—4 weeks. Afterwards, it was decreased gradually over 2—6 months. Steroid pulse therapy was used to treat 2 of the 5 patients. Steroid administration improved intestinal edema and ileus symptoms temporarily. However, TPN was not, in the end, able to relieve the 6 patients.

Recognizing that conservative treatment, including CS administration, was not improving the 6 patients, we decided to proceed to surgical treatment. In 4 of the 6 patients, laparotomy and total intestinal enterolysis were performed according to protocol. In those 4 patients, the intra-abdominal organs were covered with thickened, fibrous, membrane-like capsules that were severely adherent to the intestine. We did not observe necrotic segments of the intestine. After the enterolysis, movement of the intestines improved remarkably. The postoperative course in 3 of the 4 patients was satisfactory. Administration of CSs to those 4 patients had discontinued more than 6 months earlier. At the 7th day post operation, 1 patient died of septic multi-organ failure owing to intestinal perforation. He had discontinued steroid administration 1 month before the operation.

In the other 2 patients, emergency operations were performed owing to development of enterorrhexis during the wait for elective surgery. Those 2 patients had been treated by TPN for a long time. The operative findings were that the intra-abdominal organs showed the characteristics described earlier and, in addition, that necrotic intestine was present in both cases. Short-segment intestinal necrosis was observed in one case. That patient did well postoperatively after resection of the necrotic segment and ileostomy. However, in the second case, a patient who was maintained on TPN for 37 months finally died of sepsis. Macroscopic findings at the time of urgent surgery revealed several necrotic segments of intestine owing to non occlusive mesenteric artery infarction (NOMI). Although wide-ranging resection of the intestine was performed, she died.

**Discussion**

Encapsulating peritoneal sclerosis is an extremely serious morbidity with a high mortality rate. We could not find a definite management protocol for EPS. The outcome of typical EPS in our hospital was a remission rate of 60% (9 of 15 cases) and a mortality rate of 40% (6 of 15 cases). Only 33.3% (5 of 15 cases) of the patients were improved by TPN alone. Recently, CS administration was reported to be an effective treatment for EPS patients with inflammation. We administered CS to our EPS patients with inflammation. The patients treated with steroids did not die, but we did not observe an improvement in the remission rate owing to CS administration. On the other hand, patients not treated with steroids had died from sepsis or aspiration pneumonia. Those facts suggested that CS administration could not cure EPS. However, CS administration might have the effect of regressing the inflammatory process, contributing to an improved clinical course. In addition, steroid administration might be effective at the pre-EPS stage. In the future, we need to clarify the use and indications for steroids.

Patients who did not improve by conventional treatment required long-term TPN management. However, in 2 patients on long-term TPN, intestinal rupture occurred before surgery could be performed. Ohmori et al (10) reported that NOMI was found at autopsy in an EPS patient. In our 2 patients, the cause of intestinal rupture might be related to NOMI. In management by TPN alone, a good prognosis was hard to secure.

In the patients who had not improved by TPN alone, the mortality rate of 60% (6 of 10 cases) was extremely high. Therefore, when TPN and CS administration cannot effect an improvement within several months, surgical treatment should be considered. However, we could not be satisfied with the outcome of surgical management in EPS because of the high mortality rate (33.3% (2 of 6 cases)). In patients who were treated with CSs, the suitable time at which to perform the operation is still unclear. Administration of CSs had several adverse effects, such as infection. Surgical treatment should therefore be performed as late as possible after CS discontinuation.

Treatment of typical EPS is extremely difficult. Preventing EPS and managing the pre-EPS period are therefore important.

In the present study, we analyzed patients transferred to HD from CAPD. The incidence of typical EPS was 9.9% in those subjects. The incidence of EPS increased in parallel with prolonged duration of CAPD. In patients with more than 120 months on CAPD, the incidence was 33.3%. Moreover, 90.9%
of the patients with EPS had undergone CAPD for more than 72 months. All of the patients with EPS had a high-transport peritoneum (PET category). Those facts indicate a high risk of EPS for patients who have undergone CAPD for more than 72 months and who have a high-transport peritoneal membrane. Such patients should be withdrawn from CAPD to prevent EPS.

Several investigators reported that most patients who were diagnosed with EPS after removal from CAPD had experienced no bacterial peritonitis. Those reports suggest that the intraperitoneal inflammatory reaction was accelerated after CAPD withdrawal. From the clinical point of view, intraperitoneal lavage to remove possible biologic factors that cause or facilitate the development of EPS can be performed through the CAPD catheter remaining in place after transfer to HD. However, we have not yet confirmed the mechanism and efficacy of peritoneal lavage.

**Conclusion**
In our single-center experience, the EPS survival rate was 60% (9 of 15 cases). However, in patients treated with TPN alone, the remission rate was only 33.3% (5 of 15 cases), and therapy with CSs did not improve that rate. When EPS symptoms are not improved by TPN and CS administration, surgical intervention should be considered. However, we could not be satisfied with the consequences of surgical treatment.

The most important management at present is to prevent EPS. High-risk patients (more than 72 months duration of CAPD and high peritoneal transport) should be withdrawn from CAPD to prevent EPS. More studies will be required before EPS a serious and fatal complication of long-term CAPD treatment can be overcome.

**References**


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Hiroyasu Yamamoto, MD, Department of Nephrology and Hypertension, Jikei University School of Medicine, 3-25-8 Nishi-Shinbashi, Minato-ku, Tokyo 105-8461 Japan.
Although encapsulating peritoneal sclerosis (EPS) is feared as a fatal complication of peritoneal dialysis (PD), several recent studies have reported that many cases of EPS can be completely cured by appropriate methods of prevention and treatment. In this study, we describe therapeutic tactics for EPS and discuss methods of prevention.

To begin, appropriate management of peritonitis and use of a biocompatible dialysis fluid are important to prevent the development of EPS. After the development of EPS is confirmed, the basic therapeutic tactics should be chosen according to the disease stage. That is, immediately after onset (inflammatory stage), EPS should be initially treated by steroid administration. If steroid therapy is ineffective, the steroid dose should be decreased immediately, and followed (encapsulating stage) by management with total parenteral nutrition (TPN). If ileus symptoms remain (ileus stage), active laparotomy and total intestinal enterolysis should be performed. During surgical treatment of EPS, it is important to perform total enterolysis without damaging the capsule-covered intestine.

We believe that EPS is no longer an incurable disease, and that it can be completely cured by active diagnosis and treatment.

Key words
Encapsulating peritoneal sclerosis (EPS), sclerosing encapsulating peritonitis (SEP), surgical treatment, enterolysis

Introduction
Encapsulating peritoneal sclerosis (EPS) is feared as a final and fatal complication of long-term peritoneal dialysis (PD) (1—3). Recently the developmental mechanism of EPS has gradually been elucidated by detailed study of its pathologic conditions. Furthermore, novel therapeutic tactics for EPS have also recently been developed (4). Because the pathologic conditions of EPS are gradually being elucidated, the condition is no longer a fatal complication of PD.

Previously, EPS was called sclerosing encapsulating peritonitis (SEP). However, the appropriateness of categorizing as peritonitis pathologic conditions of the peritoneum without inflammatory findings, even when a definitive diagnosis of SEP had been established, was doubtful. The Ad Hoc Committee of the International Society for Peritoneal Dialysis therefore proposed that this pathologic condition was appropriately called EPS (5). However, as described in the present study, the involvement of inflammation is essential to the development of EPS, and inflammatory cell infiltration is observed in peritoneal tissue in EPS. Further discussions of terminology, including a definition of EPS, are therefore needed in the future. Temporarily, the term SEP/EPS is used in the present report.

Discussion

Definition and developmental mechanism of SEP/EPS
To classify the pathologic conditions that constitute SEP/EPS, a full understanding of the definition of the condition is most important. Table I shows the current definition of SEP/EPS, which adequately reflects the actual pathologic conditions (6). That is, long-term PD deteriorates the peritoneum, resulting in a state of peritoneal hypertrophy. Peritoneal thickness including the state of peritoneal fibrosis does not always include peritoneal sclerosis. In addition, peritoneal thickness accompanied by infiltration by certain inflammatory cells is called sclerosing peritonitis. Thus, adhesion and encapsulation of the intestine with peritoneum causes the development of SEP/EPS.

Although long-term PD causes peritoneal hypertrophy to develop, the developmental mechanism of the subsequent intestinal adhesion and encapsulation...
remains unclear. As Figure 1 shows, inflammation is essential to the development of intestinal adhesion and encapsulation in the currently proposed developmental mechanism of SEP/EPS.

The inflammation involved in the proposed developmental mechanism is caused by the following problems: (A) bacterial peritonitis; (B) chronic peritonitis (persistence of sclerosing peritonitis and inflammation owing to residual inflammatory cell infiltration inside the peritoneum despite the improvement of peritonitis and the elimination of bacteria); and (C) excessive reaction during the process of peritoneal regeneration after PD is discontinued.

Inflammation caused by those problems induces adhesion of the peritoneum lacking peritoneal mesothelial cells, resulting in encapsulation after inflammatory products such as fibrin accumulate. Although ileus symptoms do not appear during the encapsulating stage, they appear when further accumulations of inflammatory products strangle the intestine, resulting in the development of SEP/EPS.

**Causative factors and SEP/EPS prevention**

Based on the classification that follows of SEP/EPS causative factors, prevention of SEP/EPS development should address those factors.

**FREQUENT AND PERSISTENT BACTERIAL PERITONITIS**

The pathologic conditions of frequent and persistent bacterial peritonitis occurring during the early stage after the introduction of PD consist of intestinal adhesion and encapsulation caused by residual infection and abscess formation in the abdomen after insufficient treatment of severe peritonitis. Prevention and control of peritonitis are therefore essential countermeasures against frequent and persistent bacterial peritonitis.

**NONSPECIFIC ANGITIS ACCOMPANYING PERITONEAL THICKNESS (CHRONIC PERITONITIS)**

In some patients undergoing medium- or long-term PD, fibrous changes in the peritoneum and infiltration of inflammatory cells (consisting mainly of neutrophils) are observed under the peritoneal tissue or around microvessels during peritoneal biopsy after catheter extubation. Patients with pathologic conditions of that kind may develop SEP/EPS in the future. Inflammatory cell infiltration is most likely to be caused by unapparent bacterial peritonitis, accompanied by residual infection in the peritoneal tissue despite seemingly cured mild peritonitis. Because the inflammation persists even after PD is discontinued, patients with pathologic conditions of that type may develop SEP/EPS in the future. If peritoneal biopsy findings of that kind are obtained, administration of steroids should be considered after the possibility of other infectious diseases has been rejected.

**INTRACTABLE BACTERIAL PERITONITIS ACCOMPANYING PERITONEAL SCLEROSIS**

Patients undergoing long-term PD are prone to SEP/EPS when they develop peritoneal sclerosis accompanied by intractable bacterial peritonitis. In long-term PD patients with peritoneal sclerosis, peritoneal mesothelial cells disappear, and the peritoneal tissue is completely replaced by fibrous tissue, resulting in decreased defense against bacterial infection. Such

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**TABLE 1** Definition for encapsulating peritoneal sclerosis (EPS)

<table>
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<tr>
<th>Description</th>
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<tbody>
<tr>
<td>EPS is a syndrome involving persistent, intermittent, and/or recurrent adhesive bowel obstruction (ileus) caused by wide ranging adhesion of the diffusely hypertrophied peritoneum.</td>
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<tr>
<td>Morphologically, peritoneal thickening and/or sclerosing peritonitis are observed.</td>
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<tr>
<td>- Peritoneal thickening: the state of peritoneal fibrosis and sclerosis (does not always include peritoneal sclerosis).</td>
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<tr>
<td>- Sclerosing peritonitis: peritoneal thickness accompanied by infiltration by certain inflammatory cells.</td>
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patients are therefore prone to intractable peritonitis. In addition, intestinal adhesions also occur in such patients, resulting in the development of SEP/EPS. Basically, such patients are treated by controlling their peritonitis. However, potent antibacterial therapy and steroid administration should also be considered when the inflammatory state persists.

PERITONEAL SCLEROSIS

Even when long-term PD patients are not complicated by peritonitis, those who are complicated by peritoneal sclerosis may develop SEP/EPS. Most previously reported cases of SEP/EPS developed after discontinuation of PD and subsequent catheter extubation. Although the developmental mechanism remains unclear, the speculation is that certain excessive reactions occur during the healing process in the damaged peritoneum. To prevent the occurrence of excessive reactions and intestinal adhesions, leaving a peritoneal catheter in place even after discontinuation of PD is recommended, as is periodical peritoneal irrigation (7).

Therapeutic approaches for SEP/EPS

Encapsulating peritoneal sclerosis develops with peritoneal sclerosis and encapsulation, plus intestinal adhesion. From the therapeutic perspective, the diagnosis should be established before SEP/EPS develops. Early diagnosis of SEP/EPS is important. Determining the therapeutic tactics for SEP/EPS according to the disease stage is also important (4).

Table II shows the staged classification and therapeutic tactics that we propose for SEP/EPS. Most cases of SEP/EPS develop several weeks or months after removal of the peritoneal catheter, with manifestations of fever, increased C-reactive protein level, and slight ileus symptoms accompanied by increased ascites (inflammatory stage). Upon precise identification of the onset of the inflammatory stage, steroid administration should be initiated immediately. Methylprednisolone pulse therapy is recommended during the early stage.

If the SEP/EPS is not relieved or if it recurs within 1 month (encapsulating stage), the dose of steroids should be decreased, and the patient should be managed by total parenteral nutrition (TPN). If ileus symptoms remain despite the absence of inflammatory findings and decreased ascites, laparotomy and enterolysis should be considered within 6 months (ileus stage). It is important that the enterolysis be performed without damaging the capsule-covered intestine.

Surgical cases of SEP/EPS

Previously, surgical treatment for SEP/EPS was contraindicated. In particular, the intestinal wall in patients undergoing long-term PD shows marked serous degeneration, which causes anastomotic failure (1,2,8). Because the pathologic conditions of SEP/EPS were not sufficiently understood, resection of the ileus-producing lesion and intestinal anastomosis were performed to relieve ileus symptoms. Our nationwide study of SEP/EPS revealed that 82% of patients undergoing intestinal anastomosis died postoperatively (Kawanishi H. Unpublished data). Based on those findings, we developed a novel surgical technique basically consisting of acute enterolysis.

We have performed laparotomy and enterolysis in 27 patients with SEP/EPS (Figure 2). Although steroid therapy was initially performed in 20 of those patients after SEP/EPS developed, laparotomy was performed owing to residual ileus symptoms. The interval between the onset of SEP/EPS and the laparotomy ranged from 1 month to 33 months (mean: 10.2 months).

Two of the 27 patients died of postoperative intestinal perforation. One of those 2 patients had SEP/EPS caused by tuberculous peritonitis, and severe intestinal adhesion was observed after 85.3 months of PD. Although two small intestinal perforations that occurred intra-operatively were suc-
cessfully closed by suturing, another perforation occurred in the small intestine on the third postoperative day, and the patient died of sepsis complications despite a second operation. The other patient had been undergoing PD for 166 months and was complicated by severe calcification. Perforation of the small intestine occurred at a point other than in the calcified region. That patient died of sepsis 1 month after surgery.

Ileus symptoms recurred in 2 other patients. Owing to severe calcification, the initial enterolysis was not sufficiently performed in one patient who had been undergoing PD for 152 months. Because ileus symptoms recurred 2 months postoperatively, a second laparotomy was performed 8 months after the initial surgery. Although the region ablated by the initial enterolysis adhered severely, the second enterolysis was successfully performed because there were no capsules. Because residual calcification caused the ileus symptoms, the calcified region was completely removed. The other patient, who had been undergoing PD for 166 months, was complicated by strong intestinal encapsulation and severe degeneration of the visceral peritoneum, which were almost completely ablated during the initial surgery. However, the patient complained of persistent fever immediately after surgery, and ileus symptoms recurred 1 month later. Although a second operation was performed 12 months later, recurrent SEP/EPS was suspected because re-encapsulation was observed. The ileus symptoms in that patient were relieved after complete enterolysis.

In the remaining 22 patients, the postoperative course was stable and ileus symptoms completely disappeared after surgery. They were all able return to their previous social activities.

Timing of the indication of surgery
As described earlier, surgery should be performed after the inflammation has subsided. The duration of PD and the presence or absence of peritoneal calcification are important factors for determining the indication for surgical treatment.

In patients who have received long-term PD, the intestinal wall degenerates: the result is an ill-defined boundary between the capsule and the intestinal wall. And because peritoneal calcification extends from the serosa to the muscular layer, enterolysis can easily induce intestinal perforation in such patients.

If ileus symptoms are suspected in patients who have received PD for less than 10 years without complication by peritoneal calcification, surgery should be performed. Surgery may also be indicated in those whose ileus symptoms are induced by increased oral food intake.

Patients who have received PD for more than 10 years without complication by peritoneal calcification should be treated surgically even when peritoneal degeneration is suspected. Surgery may be indicated for patients with local peritoneal calcification. However, surgery should be carefully performed in those with peritoneal calcification extending to the entire intestine.

The indication for surgery in patients with SEP/EPS should be determined by the surgeons in consultation with physicians who specialize in PD.

Surgical techniques
Surgical techniques for SEP/EPS consist simply of acute ablation of capsules and intestinal adhesion. However, it is important to determine the area of ablation, because the severity of the intestinal wall degeneration varies among patients. Generally, the entire intestine is encapsulated, and fibrous intestinal adhesion is observed in patients with SEP/EPS. Therefore, the basic surgical techniques for SEP/EPS consist of excision and ablation of the capsules, beginning from the region where enterolysis is easily performed to relieve the intestine from strangulation. At a minimum, the ileus-producing lesion should be ablated completely.

Ablation of the stomach and duodenum is not required in most cases of SEP/EPS.

If encapsulation-induced stenosis or loss of peristalsis is observed in the large intestine, ablation may be required, particularly in the sigmoid colon. How-
ever, complete ablation is not required in the large intestine; linear excision of the capsule is sufficient for recovering peristalsis. Moreover, linear excision of the capsules is often performed at several regions of the small intestine if complete enterolysis is difficult to perform.

When, in patients who have received long-term PD, the capsule is not well demarcated from the intestinal serosae owing to degeneration of the intestinal wall, enterolysis should be performed at the muscular layer. However, enterolysis at the muscular layer should be limited to the minimum level in such patients.

Because enterolysis at the calcified region easily causes intestinal perforation, careful ablation is required. If the intestinal wall is perforated, the perforation should be closed by suturing, using reinforcing materials such as an absorbing sheet.

Conclusions

Today, SEP/EPS is no longer a fatal complication of long-term PD. It can be completely cured by appropriate diagnosis and treatment. In the future, rapid development of a biocompatible dialysis fluid that does not cause SEP/EPS is expected.

References


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A growing incidence of encapsulating peritoneal sclerosis (EPS) has recently been reported in Japan. It is now urgent to establish preventive measures against EPS development. In the present paper, we describe our observational results regarding the risk of EPS development and the characteristic features of patients with EPS, in terms of peritoneal morphology and peritoneal function as determined by peritoneal equilibration test. The ongoing working protocol for EPS prevention at Jikei University Hospital is also discussed.

Our findings have revealed that long-term continuous ambulatory peritoneal dialysis (CAPD) is a risk factor for EPS development after transfer to hemodialysis from peritoneal dialysis (PD), and that, in most patients with EPS, peritoneal function is characterized by a longstanding high-transport state. The striking alterations in peritoneal morphology between patients with EPS and those with simple long-term CAPD hyperplasia include, in EPS patients, a prominent thickening of the collagenous layer of the peritoneum with neoangiogenesis and myofibroblastic transformation.

Based on our findings, we established a withdrawal protocol for long-term CAPD patients, with the goal of preventing EPS. Those who have been on PD treatment for more than 72 months with high-transport state are candidates for withdrawal from PD, with performance of peritoneal lavage thereafter for a certain period of time. The clinical benefit of post-PD lavage has not yet been determined; however, the maneuver could be precluded in patients at low risk of EPS, because it was found that some patients can recover to average transport state during the period of PD withdrawal. Patients who remain high transporters with inflammatory reaction might require pharmacologic intervention, including prednisolone therapy. Further observations are required to validate our approach.

Key words
Encapsulating peritoneal sclerosis (EPS), long-term PD, high-transport state, prevention, peritoneal lavage

Introduction
A growing incidence of encapsulating peritoneal sclerosis (EPS) has recently been reported in Japan. It is now urgent to establish a treatment for EPS.

The first survey on EPS was conducted in 1994. From 1980 to 1994, 62 cases of EPS (0.9%) were reported among 6923 peritoneal dialysis (PD) patients (1). In 1997, a multicenter survey was conducted and among 3760 patients, 106 cases of EPS were reported in 159 centers, the incidence being 2.8% (2). Although recent therapeutic measures such as immunosuppressant therapy with prednisolone and a surgical approach have proved to be greatly successful, EPS is still associated with a poor prognosis, the 2-year survival rate being as low as 58% (2). For that reason, it really is an urgent matter to develop a measure to detect, at an early stage, patients at risk for EPS and to establish a means to prevent the illness.

Discussion
Risk factors for EPS development
Bacterial or fungal peritonitis has been noted to be an important factor in the development of EPS; however, recent surveys have confirmed that long-term PD may be a major factor contributing to EPS. In addition, the distinctive feature of EPS reported in Japan was that most of the cases are not related to peritonitis: the condition occurs after patients are transferred to hemodialysis. The same trend was reported in Australia (3) which suggests that long-term PD treatment is a...
critical risk factor for EPS development, independent of racial differences. Accordingly, clarifying the characteristics of the peritoneum during long-term continuous ambulatory peritoneal dialysis (CAPD) treatment is a necessity.

In the present paper, the results of an analysis of the clinical background of patients with EPS at Jikei University Hospital are introduced, together with the outcome of a clinical trial to discover how to prevent EPS from developing.

Functional and morphologic aspects associated with long-term CAPD treatment

Reports indicate that the peritoneal solute transport as reflected in the dialysate-to-plasma creatinine ratio (D/P Cr) increases with time on CAPD. However, the D/P Cr does not necessarily increase linearly from the start of CAPD treatment. The D/P Cr was noted to increase in patients kept longer on PD treatment. The number of patients with a high-transport state increased among the population on long-term CAPD. Regarding changes in morphology, the term peritoneal sclerosis is used to describe a peritoneum that has been subjected to long-term PD treatment. The condition is characterized by findings such as loss of mesothelium, proliferation of fibroblasts, an increase in the collagen layers and hyalinosis, vascular thickening or obliteration (or a combination of the two), microvascular proliferation, and expansion of adipose tissues. The expression tanned peritoneum is used to describe the macroscopic change. However, peritoneal thickening with a brown coloration is not necessarily observed uniformly even in patients on long-term CAPD. The changes are seen only sporadically and locally in some cases. The same changes are found on the serosa of the intestinal tract, and a sweet-corn appearance owing to the expansion of subserosal adipose tissue can be observed locally. In general, and according to our observations, those changes in morphology do not interfere with intestinal motility.

Functional and morphologic aspects of EPS

Reported data on the peritoneal function of patients with EPS have been contradictory so far; there is no fixed pattern to describe the condition. The problem is partly that the functional data may be readily affected by the stage of the EPS pathology.

We analyzed D/P Cr profiles in 12 patients with EPS who were treated at Jikei University Hospital. The common features were a history of PD for more than 60 months and development of EPS after PD withdrawal without apparent bacterial peritonitis (Table I). To clarify the risk factors for EPS, those clinical profiles were compared to the profiles of patients who did not show symptoms of EPS up to 24 months after PD withdrawal (n = 63). In most of the patients who developed EPS, D/P Cr was significantly higher at withdrawal from CAPD and a high-transport state occurred significantly earlier during CAPD treatment (Figure 1). Those observations suggest that the earlier development of a high-transport membrane may be one of the risk factors for EPS, in addition to the longer duration of PD treatment.

At present, the implications of a high-transport state of the membrane for the development of EPS are unknown. However, the increase in the microvascular bed of the peritoneum caused by a high-transport state results in ready exposure of the peritoneal tissue to the endotoxins released from the intestines, which could accelerate the inflammatory reaction of the peritoneum. At any rate, early development of a high-transport membrane may be a clinical marker by which patients who are at high risk of developing EPS can be detected in the clinical setting.

The macroscopic morphology in EPS may be altered, depending on the stage, from the early to the terminal phase. At the terminal phase, the intestinal tract is covered by a thick collagenous layer. On the other hand, in the relatively early phase, the parietal peritoneum as well as the serosal side show prominent exudative lesions with angioneogenesis. Interestingly, in the earlier phase, the lesion does not extend to the entire peritoneal cavity; it is located only in the pelvic area in some patients (Figure 2).

The histologic findings are primarily based on the changes related to long-term CAPD as noted above; however, the prominent widening of the collagenous layer that accompanies hyalinosis is one of the characteristic features of EPS (Figure 3). Immunohistochemical studies have revealed positive staining for vascular endothelial growth factor (VEGF) and α-SMA (myofibroblasts) in the interstitial cells. In some cases, CD64-positive mononuclear cells (macrophages) could be found. Radical oxygen species have been shown to have an important role in the production of VEGF as well as in the transformation of fibroblasts; thus the peritoneal changes in EPS could be related to increased oxidative stress. The findings
concerning infiltrating macrophages suggest that an immunologic response may have occurred in some patients.

Clinical trial of post-PD peritoneal lavage to prevent development of EPS

Our epidemiologic study led to the conclusion that most patients in Japan with EPS developed the condition after withdrawal from PD. That finding provided us with an important key to planning for the prevention of EPS. We suggest that, in patients with EPS, an inflammatory reaction is accelerated after withdrawal from PD. Given that clinical point of view, peritoneal lavage after PD withdrawal could be performed as a mechanism to prevent EPS by eliminating the possible factors that cause or facilitate its development.

At Jikei University Hospital, a clinical trial of peritoneal lavage after PD withdrawal was started in 1997. The method is this: A catheter is left in place for up to 12 months after PD withdrawal, and patients are instructed to exchange a solution bag once daily (Figure 4). In the early days of the trial, a standard PD solution with low glucose content (1.36%) was used as the lavage solution; more recently, a saline solution has been tried. Twelve patients at high risk for EPS (on PD for more than 60 months and with a state of high-transport membrane) were enrolled in the trial. At the end of the study, 8 of those patients showed a decrease in D/P Cr, indicating improvement in peritoneal function. In 4 cases, the patients showed a sustained high level of D/P Cr at the end of the study. Of those 4 patients, 2 developed EPS: one at 3 months after the catheter removal, and the other at 8 months after. Those results suggest that peritoneal lavage is effective in averting full-blown EPS development in high-risk patients, but that it does not eliminate the primary pathologic process that leads to the development of EPS.

Prednisolone offers a benefit to patients by suppressing the systemic inflammatory reaction that often

<table>
<thead>
<tr>
<th>Sex</th>
<th>Age at PD initiation (years)</th>
<th>Renal disease</th>
<th>Duration of PD (months)</th>
<th>Onset of EPS after withdrawal from PD (months)</th>
<th>Episodes of peritonitis (n)</th>
<th>Reason for withdrawal from PD</th>
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<tr>
<td>M</td>
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<td>2</td>
<td>3</td>
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<td>0</td>
<td>5</td>
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<td>M</td>
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<td>CGN</td>
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<td>10</td>
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<td>UF loss</td>
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<tr>
<td>F</td>
<td>33</td>
<td>CGN</td>
<td>120</td>
<td>0</td>
<td>1</td>
<td>UF loss, invagination</td>
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<tr>
<td>M</td>
<td>53</td>
<td>Gouty kidney</td>
<td>128</td>
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<tr>
<td>M</td>
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<td>CGN</td>
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<td>6</td>
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<td>139</td>
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<td>0</td>
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</tr>
<tr>
<td></td>
<td>35.1±3.3</td>
<td></td>
<td>109±6.8</td>
<td>7.0±1.7</td>
<td>0.16±0.05</td>
<td>(per year)</td>
</tr>
</tbody>
</table>

EPS Jikei Experience 2002

TABLE I: Profiles of patients with encapsulating peritoneal sclerosis (EPS)

<table>
<thead>
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<th>Sex</th>
<th>Age at Onset of EPS</th>
<th>Reason for withdrawal from PD</th>
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<td>M</td>
<td>Renal disease</td>
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</tr>
<tr>
<td>F</td>
<td>Renal disease</td>
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<tr>
<td>M</td>
<td>Renal disease</td>
<td></td>
</tr>
<tr>
<td>M</td>
<td>Renal disease</td>
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<td>F</td>
<td>Renal disease</td>
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</tr>
<tr>
<td>M</td>
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<tr>
<td>F</td>
<td>Renal disease</td>
<td></td>
</tr>
<tr>
<td>M</td>
<td>Renal disease</td>
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</tr>
</tbody>
</table>

PD = peritoneal dialysis; M = male; F = female; CGN = chronic glomerulonephritis; UF = ultrafiltration.
accompanies the progressive phase of EPS. It can prolong the patient’s survival, at least from the viewpoint of short-term observation. However, whether the administration of prednisolone and other immunosuppressive agents can suppress the adhesive process in the course of EPS is still uncertain. In our experience, prednisolone cannot eradicate the exudative lesions with neovascularization in the serosa and the parietal peritoneum. That problem should be clarified by additional long-term observations.

**Conclusion**

To prevent the development and acceleration of EPS, it is necessary to preserve the cellular components in the peritoneum, including the mesothelium, as much as possible during the course of CAPD, and to eliminate factors that may possibly...
cause oxidative stress. According to recently published reports, a neutral solution in a two-chambered bag with fewer glucose degradation products (GDPs) may be beneficial in preserving mesothelial viability. The administration of antioxidative agents should be taken into consideration from the start of CAPD. The blockage of the rennin—angiotensin system may offer some benefit to the peritoneum under CAPD through the antifibrogenic and antioxidative effects of angiotensin-converting enzyme (ACE) inhibitors as well as of angiotensin receptor blockade action.

In future, solutions that are more biocompatible should be made available, and the primary mechanism by which oxidative stress is triggered during CAPD treatment should be clarified, with special reference to the formation of advanced glycation end-products in the peritoneum.

References

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