
The Pan-Thames EPS study: treatment and outcomes of encapsulating peritoneal sclerosis

Gowrie Balasubramaniam1, Edwina A. Brown1, Andrew Davenport2, Hugh Cairns3, Barbara Cooper4, Stanley L. S. Fan5, Ken Farrington6, Hugh Gallagher7, Patrick Harnett8, Sally Krausze9 and Simon Steddon10

1West London Renal and Transplant Centre, 2Royal Free Hospital, 3King’s College Hospital, London, 4Basildon Hospital, Basildon, 5Royal London Hospital, London, 6Lister Hospital, Stevenage, 7St Helier Hospital, Carshalton, 8Southend Hospital, Southend, 9Canterbury Hospital, Canterbury and 10Guy’s Hospital, London, UK

Correspondence and offprint requests to: Gowrie Balasubramaniam; E-mail: gowrie@doctors.org.uk

Abstract

Background. Encapsulating peritoneal sclerosis (EPS) is a disease process that can occur as a complication of peritoneal dialysis (PD). The aim of this study was to make a general assessment of the clinical features, diagnosis, management and outcome of PD-related EPS cases from London and South-East England.

Methods. Questionnaires were sent to 11 PD units in March 2007; cases were identified retrospectively. Outcome data on surviving patients were collected in March 2008.

Results. A total of 111 patients were identified; the mean time on PD was 82 months (range 8–247). Mortality increased with length of time on PD, being 42% at <3 years (n = 12), 32% at 3–4 years (n = 19), 61% at 5–6 years (n = 31), 54% at 7–8 years (n = 24), 75% at 9–10 years (n = 8) and 59% at >10 years (n = 17). Twelve patients had no previous peritonitis episodes, 28 had one previous episode, 30 had two previous episodes and 33 had three or more previous episodes. Of the patients with PD details available, 41/63 were high (>0.81) transporters and 44/71 had ultrafiltration <1 l/24 h, but 7/63 were low average transporters (0.5–0.65) and 27/71 had ultrafiltration >1 l/24 h and a few had significant residual renal function. Sixty-five (59%) patients had their PD discontinued prior to diagnosis (51 HD; 14 transplanted). CT scans were performed on 91 patients and laparotomy on 47 patients. Drug treatment consisted of tamoxifen, immunosuppression or both. The median survival was 15 months in patients treated with tamoxifen (n = 17), 12 months in patients treated with immunosuppression (n = 24) and 21 months in patients who received both (n = 13), against 13 months (n = 46) in patients who received no specific treatment. Adhesiolysis was performed in 5 patients, and 39 patients were given parenteral nutrition. The overall mortality was 53% with a median survival of 14 months and a median time to death of 7 months.

Conclusion. This is one of the largest cohorts of patients with EPS in the literature. Long-term survival occurred in over 50%, regardless of the various treatments strategies undertaken by the centres.

Keywords: encapsulating peritoneal sclerosis; peritoneal dialysis; multicentre retrospective study; treatment

Introduction

Encapsulating peritoneal sclerosis (EPS) is an uncommon but serious complication of long-term peritoneal dialysis
(PD). It is has been associated with high morbidity and mortality since it was first described in 1980 [1]. Currently, the largest reported experience has been from Japan where patient survival on PD is longer than anywhere else [2]. A 4-year prospective multicentre study of almost 2000 patients in Japan showed strikingly that incidence and mortality correlated with PD duration, ranging from an incidence of 0.7–17% from 3 to >15 years PD duration, and an associated mortality from 17.2 to 100%.

There is little literature about the clinical course of EPS. Most centres will see only a few patients; the largest well-described cohorts have been from Japan consisting of 124 patients [3] and 48 patients [2]. An earlier Australian multicentre retrospective study reported on 54 cases [4]. All showed that development of EPS correlated with PD duration, high membrane permeability [5] and loss of ultrafiltration. Recurrent peritonitis and use of chlorhexidine were also suggested to be risk factors [6]. Evidence-based treatment is lacking and there is no uniformly accepted management. Steroids have been shown to be beneficial especially in the early ‘inflammatory’ phase of the disease process that precedes the diffuse scarring of the peritoneum [2]. Use of immunosuppressive treatment has been reported in small patient series [4,7] and case reports [8,9] and has been suggested to be beneficial. Tamoxifen has also been used in some cases [10–13]. Early surgery with bowel resection and anastomosis had a high mortality rate; surgical management now mainly focuses on enterolysis with some centres showing good results [13–15]. However, this procedure is still associated with significant mortality and has a high recurrence rate [16].

There is concern that more cases of EPS are being diagnosed in the UK, particularly after transplantation [17,18]. As a result, the Pan-Thames Audit Group, which encompasses all the renal units in London and South-East England, decided to carry out a survey of their patients with a diagnosis of EPS. The aims of the study were to determine how clinicians were making the diagnosis, its clinical features, investigations, management. We hoped to collect data on a large cohort of patients and gain data on incidence and outcome.

Methods

Physicians in charge of the 11 PD units in the Pan-Thames region were contacted in February 2007 and asked to obtain information on patients who had been diagnosed with EPS since 1997. The diagnosis of EPS was made by clinicians from the various centres based on clinic-pathological criteria as outlined by International Society for Peritoneal Dialysis ad hoc committee on ultrafiltration management in PD [19]. The peritoneal biopsy material would have been reviewed by pathologists at the various centres. Previously described changes consistent with EPS including interstitial fibrosis, fibrin deposition and microvascular sclerosis [19,20] would have been used to confirm the diagnosis of EPS. Data from each patient were entered on to a proforma; this included date of diagnosis, duration of PD, frequency and type of peritonitis, clinical features, investigations, management and outcome. Details on dialysis adequacy, ultrafiltration, residual renal function and membrane transport status were also requested. Data sheets were compiled and information was entered into a standard database program, Microsoft Access (Microsoft). A further questionnaire was sent out in March 2008 to determine status (treatment modality or date of death) of patients who were alive at the time of the initial survey.

Results

There were 111 cases of EPS identified from 11 units; the earliest case was from 1997 and 77 cases were diagnosed since 2004. A variable number of cases were reported from individual units (Table 1). Mortality from each unit is also shown in Table 1.

Demographics

The mean age was 52.0 ± 14.3 years with a range of 21–79 years; 52 were male and 59 were female. The cause of kidney disease was reported as chronic glomerulonephritis in 24 patients (22%), diabetic nephropathy in 18 (16%), hypertension in 12 (11%), polycystic kidney disease in 8 (7%), reflux in 9 (7%) and other causes in 40 patients (37%).

PD details

The mean duration of PD was 83.7 ± 3.9 months, with a range of 8–243 months (Figure 1). Seventy-nine (71%) patients diagnosed with EPS had been on PD for more than 5 years. PD details are shown in Table 2.

Peritonitis history was available in 100 patients (Table 2). Ten patients had no peritonitis, 28 had one previous episode, 29 had two previous episodes and 33 had three or more previous episodes. Seventeen patients developed EPS within
3 months after transferring to haemodialysis (HD) because of peritonitis.

**Modality at diagnosis of EPS**

Forty-six patients were on PD at the time of diagnosis, 51 patients were on HD and 14 had a functioning transplant. Peritonitis was the cause of transfer onto HD in 20 patients and ultrafiltration failure in 17 patients, the remainder were not specified. The average time on HD prior to diagnosis of EPS was 5.5 months (range 0.5–37 months), with 38 (75%) patients having the diagnosis within 6 months. Twenty-nine of the 51 patients transferred to HD developed EPS within 3 months; peritonitis accounted for 17 of these cases. The average time post-transplant before diagnosis of EPS was 5.4 months (range 1–19 months).

**Membrane permeability and ultrafiltration**

Data on the last peritoneal equilibration test (PET) were available for 63 patients (Table 2). The length of time between the test and the development of EPS was not known. The mean D/P was high at 0.84 ± 0.02 (range 0.54–0.99) though seven patients were in the low average range (0.5–0.65). The last D/P was available on 14 of the 17 patients who had ultrafiltration failure and were transferred onto HD prior to diagnosis of EPS; 13 out of the 14 were high transporters. Ultrafiltration details were available for 68 patients; the mean ultrafiltration was 936 ± 70.4 ml (range 100–3300 ml). Urine output was available in 14 patients (range 0–1500 ml); 11 patients had residual renal function with urine output in the range of 200–1500 ml.

**Clinical features**

All patients had symptoms suggestive of EPS to prompt investigation to confirm diagnosis. Abdominal pain and vomiting were the commonest symptoms, being reported in 74 (67%) and 66 (59%) patients, respectively. A large proportion, 57 (51%) patients, had both symptoms and a clinical diagnosis of bowel obstruction was made in 37 (33%) of these patients. Ascites was reported in 43 (39%) patients and weight loss was noted as a significant problem in 21 (20%) patients. Other symptoms reported were diarrhoea, abdominal distention and loss of appetite.

**Making the diagnosis**

CT scanning was the commonest mode of investigation that led to the confirmation of a diagnosis of EPS. Scan results were available in 96 patients. Common CT findings reported to support the diagnosis of EPS were peritoneal thickening (n = 60, 58%), matted bowel (n = 30, 31%) and calcification (n = 27, 28%). Other findings were dilated small bowel and ascites. The CT scan was reported as normal in 14 patients; of these, eight went on to have a laparotomy and six had their diagnosis made entirely on clinical symptoms. Laparotomy was performed in 47 patients in total; 16 of these had a peritoneal biopsy with histological features reported as consistent with a diagnosis of EPS. Biopsy material was not available. Barium meal and follow-through were performed in 10 patients with 9 showing slow transit.

**Treatment**

A total 105 patients survived for further management. We cannot comment on the timing and varying doses of drug treatment used in these patients. Drug treatment consisting of tamoxifen, immunosuppression and steroids were used in 54 patients in total; the remaining 46 patients had no specified treatment. Five patients underwent surgical treatment with adhesionolysis, two of who had various drug treatments as well, one as a transplant regime (indicated on Table 3, excluded from analysis). Of the five patients who underwent adhesionolysis, four received parenteral nutritional support. A further 30 patients also received parenteral nutrition (PN). The various drug regimes used are shown in Table 3.

Tamoxifen was given to 17 patients as a single agent and in combination with other drugs to 14 patients. Steroids were used in 29 patients; this included 4 patients as a single agent and 13 patients in association with a transplantation regime. Immunosuppression drugs were used in 26 patients, 21 associated with transplantation and 5 as an additional treatment with steroids. The immunosuppression drugs used were azathioprine, cyclosporin (CsA), tacrolimus, mycophenolate mofetil (MMF) and sirolimus.

**Overall survival (Figures 1 and 2)**

One-year overall survival was 56%. The overall median survival was 14 months (range 0–119). Six patients died around the time of diagnosis. The overall mortality was 53% with 59 deaths in the group of 111 patients. Mortality increased with length of time on PD being 42% at <3 years, 32% at 3–4 years, 61% at 5–6 years, 54% at 7–8 years, 75% at 9–10 years and 59% at >10 years. The median time to death from diagnosis was 7 months (range 0–64) (Figure 2).
Table 3. Treatment regimes used, transplanted patients shown in shaded boxes (changes made after transplantation or diagnosis shown in italics)

<table>
<thead>
<tr>
<th>Immunosuppressive regime without tamoxifen</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Steroids</td>
<td>3</td>
</tr>
<tr>
<td>Steroids, azathioprine</td>
<td>3</td>
</tr>
<tr>
<td>Steroids, azathioprine (for SLE)</td>
<td>1</td>
</tr>
<tr>
<td>Steroids (for previous failed transplant)</td>
<td>1</td>
</tr>
<tr>
<td>Steroids, CsA, MMF*</td>
<td>1</td>
</tr>
<tr>
<td>Steroids, CsA, MMF (changed to Sirolimus single agent after diagnosis)</td>
<td>1</td>
</tr>
<tr>
<td>Steroids, CsA, azathioprine</td>
<td>1</td>
</tr>
<tr>
<td>Steroids, tacrolimus, MMF</td>
<td>1</td>
</tr>
<tr>
<td>Tacrolimus</td>
<td>3</td>
</tr>
<tr>
<td>Tacrolimus, MMF (changed to Sirolimus single agent after diagnosis)</td>
<td>2</td>
</tr>
<tr>
<td>Tacrolimus, MMF</td>
<td>1</td>
</tr>
<tr>
<td>Steroids, CsA, azathioprine</td>
<td>1</td>
</tr>
<tr>
<td>Steroids, CsA, MMF</td>
<td>4</td>
</tr>
<tr>
<td>Tacrolimus, MMF</td>
<td>1</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Immunosuppression with tamoxifen</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Steroids, tamoxifen</td>
<td>6</td>
</tr>
<tr>
<td>Steroids, tamoxifen*</td>
<td>1</td>
</tr>
<tr>
<td>Steroids, MMF, sirolimus, tamoxifen</td>
<td>1</td>
</tr>
<tr>
<td>Steroids, azathioprine, tamoxifen</td>
<td>1</td>
</tr>
<tr>
<td>Steroids, tacrolimus, MMF (tamoxifen added after diagnosis)</td>
<td>1</td>
</tr>
<tr>
<td>Tacrolimus, azathioprine (tamoxifen added after diagnosis)</td>
<td>1</td>
</tr>
<tr>
<td>Tacrolimus (tamoxifen added after diagnosis)</td>
<td>1</td>
</tr>
<tr>
<td>Steroids, tamoxifen (tacrolimus and MMF added after transplant)</td>
<td>1</td>
</tr>
<tr>
<td>Steroids, azathioprine, tamoxifen (tacrolimus and MMF added after transplant)</td>
<td>1</td>
</tr>
<tr>
<td>Tamoxifen alone</td>
<td>17</td>
</tr>
</tbody>
</table>

*Patients who had adhesionolysis from these groups (excluded from analysis).

Fig. 2. Kaplan–Maier survival curve showing the overall outcome. Most patients die within 2 years. Patients surviving beyond 2 years have good survival.

eight people died within a month of diagnosis and remaining deaths occurred within 30 months, apart from one at 64 months.

Modality and survival (Figure 3)

Fourteen patients had functioning transplants at the time of diagnosis and a further eight patients received a transplant after diagnosis. Patients with functioning transplants prior to a diagnosis of EPS had a median survival of 14 months (range 2–101). The eight patients transplanted after diagnosis had a mean age of 43.8 ± 5 years, with a mean PD duration of 45.0 ± 11 months and a median survival of 34.5 months (range 9–108). Overall survival in transplanted patients appeared to be better (median 20.5 months, n = 22, range 2–108) compared to patients who were maintained on HD (median 13.5 months, n = 74, range 1–119). Of the 46 patients on PD at diagnosis, 1 died around diagnosis, 33 patients were maintained on HD and 5 were eventually transplanted. The seven patients remaining on PD had a median survival of 19 months (range 1–26).

Drug treatment and survival (Figure 4)

Survival time was reviewed in various groups of patients who had medical treatment. This excluded patients who died around diagnosis, underwent adhesionolysis or were transplanted. The median survival for patients not given any specific drug treatment was 13 months (range 1–84, n = 46). The median survival of patients given tamoxifen was 15 months (range 1–58, n = 17), those given steroids ± immunosuppression drugs was 7 months (range 1–119, n = 8) and those give tamoxifen plus steroids ± immunosuppression was 14 months (range 6–38, n = 8).

Parenteral nutrition (Figure 5)

The median survival of the 39 patients who received PN was 10 months (range 0–101, n = 30), compared to 15 months (range 0–119, n = 53) for those maintained on oral nutrition. There were 20 deaths in the PN group with a median time to death of 5 months (range 0–16) compared to 28 deaths with a median time to death of 9 months (range 0–64) in those remaining on oral nutrition. Patients who received PN, therefore, had a worse initial mortality.
Treatments and outcomes of EPS

![Graph showing proportion survival vs months](Image)

Fig. 5. Kaplan-Meier curve showing survival of patients who received parenteral nutrition (PN) and those who did not. Patients who received PN had a worse survival at 1 year.

**Surgical adhesionolysis**

Five patients underwent surgical adhesionolysis, four received PN support. Two died at 6 and 21 months (patient without PN), 3 are alive at 25 and 59 months (additional steroid and tamoxifen treatment) and 89 months (transplanted, steroid, MMF, tacrolimus treatment). Three patients had other surgical procedures with PN support; jejunostomy, bowel resection and ileal-transverse colon bypass; survival was 9 (deceased), 2 (deceased) and 17 months (alive) respectively.

**Discussion**

EPS is mostly diagnosed by the presence of clinical symptoms of bowel obstruction, weight loss and in some cases, ascites. Confirmation of the diagnosis can be made using radiological investigations such as CT scanning, ultrasound, barium studies and/or finding the characteristic cocoon at surgery [19]. The current understanding is that EPS mostly occurs in patients on long-term PD and is related to changes in the peritoneal membrane, that peritonitis is sometimes involved in its development and that many cases occur after discontinuation of PD [21].

Most information about EPS comes from Japan where, for various reasons, there are more long-term PD patients than elsewhere. This survey of EPS by the Pan-Thames Audit Group provides information about a large case series in the UK. It is limited by being retrospective and relies on clinical recall. Not all cases were reported, e.g. one unit had lost all the notes of their deceased patients because of a flood in the basement where the notes were stored. In particular, there is limited information about PD details for individual patients. The strength of this study lies in the information about mode of diagnosis and outcome with regard to treatment and mortality.

As in previous studies, EPS is predominantly a complication of long-term PD with 75 (71%) patients having been on PD for more than 5 years, though there were 12 (11%) patients who had been on PD for less than 3 years. As in the prospective study of Kawanishi [2], mortality increased with duration on PD from 33% at 3–5 years to 63% for patients on PD for more than 10 years. The majority of patients (68%) were diagnosed after 2004, but as this is a retrospective study relying on physician recall, we cannot conclude that this represents an increase in the incidence of EPS. There did not appear to be any difference in outcome in patients diagnosed before or after 2004. There was variation in the frequency of EPS in different centres; this can be partly explained by the age of the centre with newer centres having fewer long-term PD patients. Increased physician awareness of EPS will also prompt more diagnostic investigations and therefore more cases being detected.

Not surprisingly, the symptoms of EPS were predominantly nausea, vomiting, abdominal distension and pain. CT scanning was generally the investigation of choice. The decision to undertake surgical exploration and biopsy was centre dependent; 25 patients had a laparotomy even though they had features suggestive of EPS on CT scans. Of note, eight patients with CT scans reported as normal went on to have their diagnosis made at laparotomy. This may represent the lack of knowledge by local radiologists about EPS or may suggest that some cases may occur without any obvious CT findings. There were six patients whose diagnoses were made by their clinicians on entirely clinical grounds. We cannot postulate on circumstances that may have caused this; the patient may have been too unwell or management may not have been felt to have altered due to the result of the scan. Either way, both patients who had a normal CT scan and those who did not have a scan appear to have had just as poor survival as those with CT findings of EPS.

The majority of patients (58%) developed EPS after discontinuation of PD. This is well recognized in the Japanese literature [2] with 68% in Kawanishi’s prospective study developing EPS on stopping PD. This may represent previous peritoneal injury that becomes more progressive due to failure to remove pro-fibrotic cytokines by PD effluent [21]. The development of EPS shortly after transplantation is of particular interest in that these patients will have been on potent immunosuppression drugs. Some patients may have had subclinical EPS associated with poor ultrafiltration and weight loss while still on PD. This does pose a significant management dilemma with regard to transferring modality in patients who may be detected early.

Although data about the actual PD was limited, our study does confirm that EPS predominantly occurs in patients with the high membrane transport status and in those with poor ultrafiltration. It is important to note, though, that finding the low membrane transport status on a peritoneal equilibration test or having significant ultrafiltration do not preclude the diagnosis. Almost all the patients who had ultrafiltration failure prior to a diagnosis of EPS were high transporters. This goes against conventional teaching that ultrafiltration failure in EPS is associated with low transport status. Changes in the membrane transport status and/or ultrafiltration rates may be more informative as predictors, but this information is not available from this study.

Peritonitis data were available for almost all patients (103/111). As in other studies, peritonitis did precede EPS in some patients, with 17 developing the syndrome within 3 months of transferring to HD because of peritonitis. A history of peritonitis, however, is not a prerequisite for developing EPS; 38 patients had no or only one previous episode. This is not surprising as peritonitis is a major cause of technique failure in PD so patients with a high peritonitis rate would not remain on PD for long term. Other series also report
that EPS is associated with peritonitis in some but not all patients [3].

The principal advantage of collecting data on this large cohort of patients with EPS relates to survival and the effects of treatment in comparison with the many anecdotal reports based on small numbers or even single patients. Such reports often suggest beneficial effects of corticosteroids [2], immunosuppressive drugs such as CsA, azathioprine [4, 7], tacrolimus, sirolimus [8], MMF [9] and tamoxifen [10, 11, 12, 13]. Physicians, of course, do not write papers on individual patients who do not respond to such therapies and there could also be a bias towards selecting fitter patients for immunosuppression therapy. Overall, 53% patients died and the median survival was 14 months. Death occurred early in the course of the disease with the median time to death being 7 months. There are numerous treatment strategies utilized by the various centres and information from the data is difficult to interpret. Numbers in our groups were small and treatment was very varied. Furthermore, we do not have information on when and for how long treatment would have been used in particular. However, we could not see better survival between patients getting no drug treatment and the groups being given tamoxifen and/or immunosuppression (including steroids). Interestingly, a recent study in rats has shown that CsA had a profibrotic action in the peritoneum suggesting that it may in fact contribute to the pathogenesis of EPS post-transplantation [22]. All of the 21 transplant patients in our cohort had a calcineurin inhibitor as part of their treatment regime; two patients were switched to sirolimus monotherapy after the diagnosis. The group would have a strong selection bias towards better survival, and treatment subgroups were too small, so any negating effect from the continued use of calcineurin inhibitors would be difficult to observe.

Nutritional support is vital for the management of these patients. About one-third of the patients in this study were on PN. There was no information about the nature of oral nutritional supplements, nutritional status of patients or why patients were selected for PN. Median time to death was shorter in this group compared to patients maintained on oral nutrition. This could be due to many factors such as infectious complications of administering PN, failure to provide adequate nutrition in HD patients with fluid restrictions and/or selecting sicker malnourished patients for PN.

The modality outcome appears to have a significant impact on survival. Patients who are transplanted, both before and after the diagnosis of EPS have the best survival. A strong positive selection bias would favour better survival in this group, but it is useful to highlight that EPS should not preclude patients from transplantation, and all patients should be optimized to receive a transplant as this provides the best survival outcome. The observation that patients on long-term PD can develop EPS shortly after transplantation while on potent immunosuppression treatment may support the data that such treatment does not have a role in the management of patients with EPS.

This study confirms the previous correlation of incidence and mortality with prolonged PD duration. Diagnosis is based on clinical symptoms, supported mostly by CT findings. Available drug treatments and parenteral nutritional support do not appear to offer a clear survival advantage, but PN may be indicated in more severely affected patients with intestinal failure. Overall, long-term survival is possible, particularly after transplantation. With the presence of increasing numbers of patients with prolonged PD, prospective studies are required to investigate predictive factors and any benefit from treatment.

Conflict of interest statement. E. Brown in receipt of speaker fees from Baxter Healthcare.

References

Intermittent peritoneal dialysis (IPD): an old but still effective modality for severely disabled ESRD patients

Costas Fourtounas, Andreas Hardalias, Periklis Dousdampanis, Eirini Savidaki and Jannis G. Vlachojannis

Department of Internal Medicine-Nephrology, University Hospital of Patras, Patras, Greece

Correspondence and offprint requests to: Costas Fourtounas; E-mail: cfourt@usa.net

Abstract

Background. Hospital-based intermittent peritoneal dialysis (IPD) is an old PD modality applied for as long as 40 h per week using high volumes of PD fluid, but it has almost been abandoned due to its low solute clearances. However, IPD might be the only option for elderly dialysis patients with significant comorbidities, unable to undergo haemodialysis (HD) or PD at home without any assistance, for various reasons.

Methods. We describe our experience with 25 patients aged 71.2 ± 7.5 years with a previous history of HD for 55.4 ± 54 months, dialysed with IPD for more than 3 months. IPD was performed three times weekly for 8–10 h.

Results. Mean values for haematocrit, serum urea, creatinine, sodium, potassium and calcium were comparable with other ESRD populations, whereas there were significantly lower values for albumin (3.2 ± 0.3 mg/dL) and significantly higher values for phosphorus (7.1 ± 1.7 mg/dL) despite the use of phosphate binders. The patients survived for a mean of 16.8 ± 11.5 (3–43) months despite very low solute clearances, as expressed by Kt/V urea (1 ± 0.26) and weekly creatinine clearance (27.2 ± 7.6 L/week). However, by using 22.9 ± 4.5 L of various combinations of isotonic and hypertonic PD fluids, the mean ultrafiltrate was 1854 ± 326 mL per session. There were only two cases of peritonitis, unrelated to IPD per se.

Conclusions. Considering the underlying comorbidities, IPD remains a valuable and effective option with acceptable survival rates, for a special population of ESRD patients not able for various reasons to undergo HD, neither PD at home.

Keywords: peritoneal dialysis; peritoneal dialysis adequacy; peritoneal dialysis modalities; peritoneal dialysis outcomes; ultrafiltration

Introduction

Hospital-based intermittent chronic peritoneal dialysis (IPD) is the oldest PD modality, applied for as long as 40 h per week, using high volumes of PD fluids [1]. By using the ancient cyclers in the early 1980s, patients were undergoing IPD in cycles lasting for 1 or more hours, for as long as 24 h once a week, 12 h two or more times per week, or even shorter treatments lasting 6–8 h performed as many as five times per week [2]. However, with the advent and improvements of continuous ambulatory (CAPD) and automated peritoneal dialysis (APD), the method has been criticized for its long duration and low adequacy regarding solute clearances and has almost been abandoned in western countries [3]. Nevertheless, the dialysis population is ageing and carries a significant burden of comorbidities [4,5]. The number and extent of comorbid illnesses in the average patient initiating dialysis have increased over the past two decades highlighting the need for more attention not only for prognostic reasons, but mainly for the day-to-day care of these patients [6,7]. There are not few haemodialysis (HD) patients with vascular access exhaustion, unable to undergo PD by themselves and without any partners to assist them at home. The recently introduced concept of assisted PD, where patients can be assisted in performing their PD exchanges at home by private nurses, is a real solution for...