Intraperitoneal pressure in PD patients: relationship to intraperitoneal volume, body size and PD-related complications

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Abstract

Background. The clinical determinants of intraperitoneal pressure (IPP) are ill defined, and the potential impact of elevated IPP on peritoneal dialysis (PD)-related complications is still a matter of debate. We measured IPP in newly started PD patients, assessed its clinical determinants and analysed the incidence of PD-related complications.

Method. IPP was measured in 61 consecutive patients [46 males and 15 females, 47 automated peritoneal dialysis (APD) and 14 continuous ambulatory peritoneal dialysis (CAPD), aged: 52 ± 17 years], an average of 2 months after PD onset, using increasing (from 0 to 3000 ml) dialysate volumes. The prescription of day and night dialysate infusion volumes was made to avoid IPP > 16 cm H₂O. We assessed the relationship between baseline clinical characteristics and IPP and the putative influence of IPP on subsequent PD-related complications, such as hernias, late leakage, gastro-oesophageal reflux (GOR) and enteric peritonitis (EP). IPP at the time of the complication was computed by linear interpolation across available couples of data (volume and IPP). Correlations were assessed using Pearson’s r; Kaplan–Meier survival curves with log-rank test were used for complication occurrence analysis.

Results. At baseline, mean IPP was 13.5 ± 3.3 (5–22.5) cm H₂O for 2000 ml inflow; IPP rose linearly as intraperitoneal volume (IPV) increased [R² = 0.96, 95% CI (0.88; 1.00)]. IPP was significantly higher in patients with a higher body mass index (BMI) (P = 0.03) but age, gender, weight, height, body surface area (BSA), diabetes mellitus or a past history of abdominal surgery did not correlate with IPP. Incidence of abdominal wall complications or GOR was not correlated with IPP. Patients with a night IPP > 14 cm H₂O had a higher incidence of EP (P = 0.039) and a worse survival free of EP (P = 0.03).

Conclusion. This study shows a strong linear correlation between IPP and IPV, a significant impact of BMI on IPP and a higher incidence of EP in patients with higher IPP. We recommend to measure IPP in PD patients to guide the prescription of intraperitoneal volumes.

Keywords: BMI; enteric peritonitis; gastro-oesophageal reflux; hernias; intraperitoneal pressure; peritoneal dialysis

Introduction

Measurement of intraperitoneal pressure (IPP) is not recommended by K/DOQI guidelines [1] and is rarely performed during current daily management of peritoneal dialysis (PD) patients. However, IPP measurement in children and adults has been reported to result in individualized dialysis prescriptions [2–5]. IPP has been shown to significantly influence ultrafiltration through increases in lymphatic reabsorption of fluid, modifications of transcapillary ultrafiltration rates and increases in fluid absorption to adjacent tissues [6–8]. Elevated IPP has also been correlated in paediatric PD patients with a higher incidence of hernia formation [9].

The aim of our study was first to evaluate the relationship between IPP and intraperitoneal fill volume (IPV), second, to assess the clinical determinants of high IPPs and third, to analyse the potential influence of an elevated IPP on PD-related complications such as abdominal wall complications, gastro-oesophageal reflux (GOR) and enteric peritonitis.

Subjects and methods

Patients

All 66 patients, 16 years or older, who started PD at Cliniques Universitaires St Luc, Brussels, between 1 January 1998 and 1 July 2003, were considered in this study. Five patients were excluded due to lack of IPP measurement (n = 4) or clinical information (n = 1). The causes of end-stage renal disease (ESRD) for the 61 included patients...
(46 males and 15 females) were chronic glomerulonephritis \( (n = 18) \), nephrosclerosis \( (n = 10) \), interstitial nephritis \( (n = 11) \), diabetic nephropathy \( (n = 7) \), autosomal dominant polycystic kidney disease (ADPKD) \( (n = 5) \), Alport’s syndrome \( (n = 5) \), scleroderma \( (n = 1) \), systemic lupus erythematosus \( (n = 1) \), Wilms’ tumour \( (n = 1) \), and unknown \( (n = 2) \). All patients had a pigtail swan-neck Missouri coiled catheter surgically inserted 1–3 weeks before dialysis onset in the pelvic position. None of the patients had outflow or inflow disturbances at the time of IPP measurement or had a peritonitis episode within 4 weeks prior to the IPP measurement.

IPP was measured as previously described by Durand et al. [2]. Briefly, the patient was placed in a supine position on a horizontal plane. A graduated column (cm) was bound to the abdominal cavity through the PD catheter. The zero level of the column was placed on the medial axillary line. The peritoneal cavity was completely emptied before the test.IPP was measured on non-deep expiration (IPP exp) and non-deep inspiration (IPP insp) with a drained peritoneal cavity (time 0) and then for inflow volumes of 500, 1000, 1500, 2000, 2500 and 3000 ml, respectively with a 1.36% glucose dialysate solution (Dianeal®, Baxter Healthcare, Lessines, Belgium). For each inflow volume, the delay before IPP measurement is approximately 30 s. The results were read in centimetres of water (cm H\(_2\)O). IPP used in this study was the mean value between IPPs measured at expiration and inspiration: \( \text{IPP exp} + \text{IPP insp} / 2 \).

IPP was measured, on average 60 days (9–227 days) after PD onset.

**Characteristics of the patients at baseline**

Baseline clinical parameters [gender, age, weight, height, body mass index (BMI) \( \text{weight}/\text{height}^2 \) and body surface area (BSA) \( 0.001784 \times \text{weight exp} 0.425 \times \text{height exp} 0.725 \), incidence of diabetes mellitus] of the 61 patients were determined at the time of IPP measurement. Other parameters considered were past history of abdominal surgery, which were separated into the following categories: (i) ‘light surgery’ i.e. retroperitoneal or non-complicated, non-invasive’ (appendicectomy or cholecystectomy) interventions, (ii) ‘heavy surgery’ i.e. intra-abdominal surgery such as partial colectomy or repair of an abdominal aortic aneurysm and (iii) inguinal hernia repair.

Due to abdominal pain complaints, IPP measurements were not obtained for some patients at higher inflow volumes of 2500 \( (n = 4) \) and 3000 \( (n = 13) \) ml.

**Evaluation of the correlation between IPP and volume**

Previously [3,4], IPP has been shown to increase linearly with increases in dialysate volume. Therefore, we first verified this linearity in our population and then we interpolated IPP for the volume actually used in each patient at the time of a complication during day (IPP day) and night (IPP night) exchanges (see subsequent text).

**PD complications that might be related to abnormal IPP**

The influence of IPP on the incidence of PD-related complications such as (i) abdominal wall complication, (ii) enteric peritonitis (EP) and (iii) GOR was studied. Abdominal wall complications considered were hernias (inguinal, umbilical, incisional or diaphragmatic) and late leakage around the PD catheter (i.e. occurring more than 1 month after PD catheter implantation). EP was defined by the presence of a cloudy peritoneal effluent, with a white blood cell count greater than 100/mm\(^3\) with more than 50% of polymorphonuclear cells and a positive dialysate culture for one or several enteric microorganisms Only the first episode of EP was considered. In addition, EP associated with tunnel or exit-site infection was excluded, and none of the patients underwent a surgical intervention or colonoscopy/gastroscopy within the last 2 months before the peritonitis episode. As a control, incidence of EP due to cutaneous microorganisms was considered.

GOR was defined by pyrosis as epigastric pain, with endoscopic evidence of oesophagitis and/or improvement of symptoms with omeprazole, and/or improvement of symptoms with lower inflow volumes.

Estimated IPPs at the time of the complication were computed by linear interpolation across available couples of data (volume and IPP). Since the volume during the day and night were different in most of the patients, statistical analyses were done for both volumes (see subsequent text).

**Statistical analysis**

In order to assess the relationship between IPP and dialysate volumes, a second order polynomial component was fitted for each patient’s data points. Since likelihood ratio tests were not significant, quadratic terms were removed and a linear relationship was kept for interpolation (mean \( R^2 = 0.96 \) across the 61 patients). IPP at the time of the complication was then computed by linear interpolation across available couples of volume and IPP data points for each patient.

Correlations between IPP at baseline and clinical variables were then assessed using the Pearson cross-product correlation coefficient for continuous variables, the Student t-test for binary variables, and the ANOVA F test for discrete variables with more than two levels. A cut-off point was set on night IPP and on day IPP to define normal and abnormal IPP values. Incidences of complications were recorded within each subgroup and compared using a Fisher exact \( P \)-value.

To account for the time to occurrence of a complication when assessing the effect of IPP during follow-up, we used survival analysis. Survival free of complication curves were established using Kaplan–Meier product limit method, and the log-rank test was used to compare curves. Analyses were performed with Splus6.0 and SPSS11.0 statistical software. All tests were two-tailed and the statistical significance level was set to 0.05.

**Results**

**Relationship between IPP and IPV**

IPP of the drained peritoneal cavity (time 0) averaged \( 8.3 \pm 3.3 \) (range 5.0–15.75) cm H\(_2\)O, and \( 13.5 \pm 3.3 \) (range 5.0–22.5) cm H\(_2\)O for a fill volume of 2000 ml. IPP increased linearly by \( 8.3 \) cm H\(_2\)O for each 500 ml.

\( R^2 = 0.96 \) across the 61 patients. IPP at the time of the complication was then computed by linear interpolation across available couples of volume and IPP data points for each patient.

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Intraperitoneal pressure in PD patients

In the whole population, mean IPP for an inflow volume of 0 ml was $8.8 \pm 3.3$ (range 0.5–15.8) cm H₂O whereas mean IPP for an inflow volume of 2000 ml was $13.5 \pm 3.3$ (range 5.0 to 22.5) cm H₂O. IPP increased linearly by $1.3 \pm 0.4$ cm H₂O for each additional 500 ml infused, independently of IPP of the empty cavity. Mean coefficient correlation was 0.96 ± 0.04.

500 ml of additional infused volume; this occurred independently of the initial IPP ($R^2 = 0.96 \pm 0.04$) (Figure 1A). Correlation between IPP and dialysate fill volume reported to BSA is shown in Figure 1B.

**Relationship between IPP and baseline patients’ characteristics**

Analysis of the relationship between IPP (inflow volume 2000 ml) with gender, weight, height, BMI and BSA are presented in Table 1. IPP was significantly higher in patients with higher BMI ($R = 0.276$, $P = 0.031$); age, weight, height and BSA did not significantly affect IPP. No differences in IPP were observed between males and females ($13.5 \pm 3.1$ cm H₂O and $13.5 \pm 4.0$ cm H₂O, respectively, $P = NS$), nor in diabetic vs non-diabetic ($13.1 \pm 2.6$ cm H₂O and $13.5 \pm 3.5$ cm H₂O, $P = NS$) patients. Patients who underwent light or heavy abdominal surgery or hernia repair before initiation of PD did not have an IPP higher than those who had not ($P = NS$).

### Table 1. Relationship between IPP and patient characteristics as evaluated for an inflow volume of 2000 ml (Data are mean ± SD or $R$)

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>n</th>
<th>IPP 2000ml</th>
<th>$P$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>61</td>
<td>0.10</td>
<td>0.44</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>61</td>
<td>0.18</td>
<td>0.16</td>
</tr>
<tr>
<td>Height (CM)</td>
<td>61</td>
<td>–0.07</td>
<td>0.57</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>61</td>
<td>0.28</td>
<td>0.03</td>
</tr>
<tr>
<td>BSA (m²)</td>
<td>61</td>
<td>0.11</td>
<td>0.38</td>
</tr>
<tr>
<td>Gender</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M</td>
<td>46</td>
<td>13.5 ± 3.1</td>
<td>0.9</td>
</tr>
<tr>
<td>F</td>
<td>15</td>
<td>13.5 ± 4.0</td>
<td></td>
</tr>
<tr>
<td>Diabetes</td>
<td>9</td>
<td>13.1 ± 2.6</td>
<td>0.68</td>
</tr>
<tr>
<td>+</td>
<td>52</td>
<td>13.5 ± 3.5</td>
<td></td>
</tr>
<tr>
<td>Surgery</td>
<td>23</td>
<td>13.2 ± 3.8</td>
<td></td>
</tr>
<tr>
<td>Light</td>
<td>31</td>
<td>13.7 ± 3.3</td>
<td></td>
</tr>
<tr>
<td>Heavy</td>
<td>2</td>
<td>12.5 ± 2.1</td>
<td></td>
</tr>
<tr>
<td>Hernia</td>
<td>5</td>
<td>14.2 ± 1.9</td>
<td>0.87</td>
</tr>
</tbody>
</table>

The same analysis was made between IPP for a drained peritoneal cavity (inflow volume 0 ml); only BMI was found to statistically affect IPP ($P < 0.001$) by multivariate analysis.

**Relationship between IPP and PD complications**

Abdominal wall complications (Table 2). Thirty-one and 30 patients had an IPP day < and ≥13 cm H₂O, respectively; and 34 and 27 patients had IPP night < and ≥14 cm H₂O, respectively.

During the whole follow-up, a total of 14 abdominal wall complications (parietal event) occurred in 14 patients: seven inguinal, four umbilical, one incisional and one diaphragmatic hernia, and one late leakage around the PD catheter. Mean follow-up of patients was 592 ± 413 days given a global incidence of 0.14 abdominal wall complications per patient-year.

No differences in the occurrence of parietal events in patients with IPP day <13 cm H₂O [eight events in 31 patients (25.8%)] compared with IPP day ≥13 cm H₂O [six events in 30 patients (20.0%) ($P = NS$)] were observed even though the day’s inflow volumes were significantly higher in the latter category ($1726 ± 301$ ml vs $1877 ± 264$ ml, respectively, $P = 0.04$). There was no difference in day inflow volume when reported to BSA ($987 ± 154$ ml/m² vs $1039 ± 139$ ml/m², respectively, $P = NS$). Patients who presented a parietal event had significantly lower day inflow volume ($1635 ± 310$ ml) than patients who did not ($1849 ± 269$ ml), ($P = 0.03$); this difference remained statistically significant when inflow volume was reported to BSA ($P = 0.018$).

No differences were observed in the occurrence of parietal events in patients with IPP night <14 cm H₂O [10 events in 34 patients (29.4%)] compared with IPP night ≥14 cm H₂O [4 events in 27 patients (14.8%), $P = NS$]. Mean night volume was significantly higher in the latter category ($1897 ± 361$ ml vs $2122 ± 414$ ml, respectively, $P = 0.03$); however, this difference was not
Mean inflow volume/BSA (ml/m²) 988
BMI (kg/m²) 22.4
Gender 7/24 8/22 0.77 8/26 7/20
Mean inflow volume (ml) 1726
/C6
Age (years) 50.0
0.15
/C6
BMI (kg/m²) 22.43
Gender 7/24 8/22 0.77 8/26 7/20 1
Mean inflow volume (ml) 1710
/C6
H2O than in patients with day IPP this difference remained statistically significant when inflow volume was reported to BSA (P = 0.001). BMI was higher in patients with a day IPP ≥ 13 cm H2O than in patients with day IPP < 13 cm H2O (24.7 ± 4.4 vs 22.5 ± 3.2, respectively, P = 0.03); this trend was not observed for night IPP. Age and BMI did not significantly affect day or night IPPs. The proportion of patients treated by CAPD or APD did not differ according to IPP day or night.

Five patients had a past history of hernias prior to PD onset; two experienced a recurrence while on PD. Only one patient had a late leakage around the PD catheter (i.e. 674 days after catheter insertion).

**Gastro-oesophageal reflux.** Thirteen patients had GOR during the whole follow-up (i.e. incidence of GOR was 0.13 per patient-year). Mean time elapsed between the beginning of PD and the diagnosis of GOR was 450 ± 443 days. Occurrence of GOR was not different for patients with elevated day and night IPP; no risk factor for GOR development was identified. Five patients had a past history of oesophagitis, but none were treated at PD onset; one experienced a relapse while on PD.

BMI of patients who developed GOR (25.2 ± 3.5 kg/m²) was slightly higher (P = 0.07) than those with no GOR development (23.0 ± 3.9 kg/m²).

**Enteric peritonitis (Table 3).** Sixteen EP cases occurred during the study period, giving an incidence of 0.16 episodes per patient-year. Microorganisms responsible for the EP were *Escherichia coli* (n = 4), *Enterococcus faecalis* (n = 3), *Enterobacter spp* (n = 2), *Morganella morgani* (n = 1), *Klebsiella sp* (n = 1), *Hafnia alvei* (n = 1); four patients had a polymicrobial peritonitis of whom two had intestinal micro-perforation that eventually resulted in PD catheter removal and transfer to haemodialysis. Eighteen peritonitis episodes occurred in 61 patients in the control group, giving an incidence of 0.18 episodes per patient-year.

Microorganisms implicated in peritonitis of the control group were *Staphylococcus aureus* (n = 2), *Staphylococcus epidermidis* (n = 11), *Streptococcus viridans* (n = 1) and *Acinetobacter* (n = 4). Mean time elapsed between PD onset and the first EP episode was 312 days ± 280 SD. A significant difference in EP

### Table 2. Relationship between IPP and abdominal peritonitis (EP): sixteen patients presented with EP during follow-up, giving an overall incidence of 0.14 events/patient-year

<table>
<thead>
<tr>
<th>IPP day</th>
<th>P</th>
<th>IPP night</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean IPP (cm H2O)</td>
<td>&lt;13 cm H2O &gt;13 cm H2O</td>
<td>&lt;14 cm H2O &gt;14 cm H2O</td>
<td></td>
</tr>
<tr>
<td>Number of events</td>
<td>8 (25.8%) 6 (20.0%) 0.41</td>
<td>10 (29.4%) 4 (14.8%) 0.15</td>
<td></td>
</tr>
<tr>
<td>Age (years)</td>
<td>50.0 53.0 0.47</td>
<td>49.8 53.6 0.38</td>
<td></td>
</tr>
<tr>
<td>Gender</td>
<td>7/24 8/22 0.77 8/26 7/20 1</td>
<td>8/26 7/20 1</td>
<td></td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>22.4 ± 3.2 24.6 ± 4.4 0.03</td>
<td>23.1 ± 4.0 24.0 ± 3.9 0.40</td>
<td></td>
</tr>
<tr>
<td>BSA (m²)</td>
<td>1.76 ± 0.21 1.81 ± 0.17 0.24</td>
<td>1.76 ± 0.21 1.81 ± 0.17 0.36</td>
<td></td>
</tr>
<tr>
<td>CAPD</td>
<td>5 (16.1%) 9 (30.0%) 0.23</td>
<td>7 (20.5%) 7 (25.9%) 0.76</td>
<td></td>
</tr>
<tr>
<td>APD</td>
<td>26 (83.9%) 21 (70.0%)</td>
<td>27 (79.5%) 20 (74.1%)</td>
<td></td>
</tr>
<tr>
<td>Mean follow-up (days)</td>
<td>539 ± 359 648 ± 462 0.31</td>
<td>526 ± 348 677 ± 476 0.17</td>
<td></td>
</tr>
<tr>
<td>Mean inflow volume (ml)</td>
<td>1726 ± 301 1877 ± 263 0.04</td>
<td>1897 ± 360 2122 ± 414 0.03</td>
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<td>Mean inflow volume/BSA (ml/m²)</td>
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<td>1083 ± 207 1178 ± 228 0.10</td>
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Intraperitoneal pressure in PD patients

The incidence of EP was similar for patients on CAPD or APD [4/14, 28% and 12/47, 21%, respectively; \( P = 0.20 \)] were not statistically different. There was no difference in the mean night inflow volume between patients with IPP day < 13 cm H\(_2\)O \( (P = 0.08) \) and their night IPP reported to BSA \( (1102 \pm 202 \text{ ml/m}^2 \text{ and } 1176 \pm 231 \text{ ml/m}^2, P = 0.20) \) were not statistically different. There was no difference in the mean night inflow volume between patients with \( 2063 \pm 476 \text{ ml} \) or without \( 2000 \pm 383 \text{ ml} \) EP \( (P = \text{NS}) \).

The incidence of EP was similar for patients on CAPD or APD [4/14, 28% and 12/47, 21%, respectively; \( P = \text{NS} \)].

Mean follow-up of patients was similar in both groups \( (525 \pm 348 \text{ days } \text{and } 676 \pm 476 \text{ days when IPP were } < 14 \text{ cm H}_2\text{O and } \geq 14 \text{ cm H}_2\text{O, respectively; } P = \text{NS}) \).

A trend of increased incidence of EP, though not statistically significant, was also observed for patients with the highest day IPP (i.e. \( \geq 13 \text{ cm H}_2\text{O} \)) \( (P = 0.09) \).

In the control group, there was no significant difference in the occurrence of peritonitis according to IPP day and IPP night. Twelve (35%) vs 6 (22%) peritonitis episodes occurred in the control group when IPP night was \( < \) and \( \geq 14 \text{ cm H}_2\text{O respectively} \) \( (P = \text{NS}) \). Proportions were also similar when IPP day was \( < \) or \( \geq 13 \text{ cm H}_2\text{O} \) (35% vs 23%, respectively; \( P = \text{NS} \)).

Survival times free of EP according to IPP night and IPP day are shown in Figure 2. Patients who had an IPP night \( \geq 14 \text{ cm H}_2\text{O} \) had a worse survival free of EP than patients who had an IPP night lower than 14 cm H\(_2\)O \( (P = 0.03) \). When IPP day was considered, the results were similar though not significant \( (P = 0.06) \).

### Discussion

Our study, performed on 61 consecutive PD patients from one-centre, documents a close relationship between IPP and IPV, a positive correlation between IPP and BMI, and a higher incidence of EP in patients with elevated IPPs.

#### Relationship between fill volume and intraperitoneal pressure

IPP was strongly correlated to IPV but had a high variability amongst patients for the same dialysate fill volume. In this study, the lowest IPP for a fill volume of 2000 ml was 5 cm H\(_2\)O while the highest was 22.5 cm H\(_2\)O. Despite those variations, IPP increased linearly with increasing dialysate volumes in a similar manner, in all patients (i.e. for each 500 ml infused, IPP increased by 1.33 cm H\(_2\)O). The same trends in IPP were observed when intraperitoneal volumes were normalized for BSA.

Mean IPP observed in our population was similar to values obtained in other studies for the same inflow volume [2–4].

#### Relationship between body size, clinical parameters and intraperitoneal pressure

A significant relationship between IPP and BMI had previously been documented in adults and children [4,6,10]. In this study, we showed that patients with a higher BMI had a significantly higher IPP while height, weight and BSA had no influence on IPP. These results might be a consequence of lower abdominal muscle tone and/or higher abdominal wall depth in patients with higher BMIs. Interestingly, intra-abdominal pressure (IAP), which has recently been measured [average IAP 6.5 mm H\(_g\) (8.8 cm H\(_2\)O)] through a bladder catheter in a population of non-dialysed patients to identify factors predicting its normal variation, has also been shown to exhibit a significant positive relationship to BMI [11]. In addition, similar to our PD patients, age, gender, height and weight did not affect IAP. Corpulence of the patients, as reflected by BMI, but not BSA, is probably, thus, the main clinical determinant of IPP.
The identification of a relationship between BMI and IPP is not without interest. Indeed, the progressive loss in residual renal function and the change in peritoneal membrane permeability over time frequently observed in PD patients usually directly results in the prescription of higher exchange volumes to achieve Kt/V and ultrafiltration targets. Limitations in increasing volumes in patients with the highest BMI, because of clinical intolerance, could require a transfer to haemodialysis due to inadequate diffusion of uraemic toxins and/or an inadequate ultrafiltration.

In this study, we did not observe any effect of age on IPP and, to the best of our knowledge, such a relationship has not been previously documented in adults.

A relationship between IPP and gender was not observed in our study. This matter is, however, controversial as De Jesús et al. [12] previously documented that males had a higher IPP than females but Scanziani et al. [4], just like in our population, did not observe any significant differences between genders.

Finally, the presence of diabetes or a pre-PD history of abdominal surgery did not influence IPP, an observation also found by De Jesús et al. [12].

Relationship between peritoneal dialysis-related complications and intraperitoneal pressure

Development of hernias (inguinal, umbilical, incisional or diaphragmatic). The influence of IPP on the development of hernias has not been thoroughly studied in PD patients. Parietal complications related to the PD procedure are numerous, with hernias being the most frequent. The prevalence of hernias in adults and paediatric PD patients ranges from 7 to 27%, and up to 40%, respectively [13,14–16]; therefore, the prevalence of 19.6% observed in our study was not unexpected. Predisposing factors commonly mentioned for hernia development in PD patients are (i) ADPKD as original nephropathy [13,16], (ii) CAPD, by opposition to APD [13], (iii) body size [14,15] and (iv) gender, with a higher proportion of hernias in males [16]. In the majority of these studies, patients with smaller body size were found to have more occurrences of hernias than others. Some authors attributed this finding to possibly higher IPPs even though (i) IPP was not measured, and (ii) fill volume was not reported [13,15]. In the present study, we were unable to identify a predisposing factor (such as diabetes, gender, mode of PD therapy) for the development of hernias. We only observed that patients who developed hernias had significantly lower day and night fill volumes as compared with those who did not, whereas mean IPPs were equivalent. One might thus say that if fill volumes had been equivalent in both groups of patients, IPP would have probably been higher in patients with hernias. Another possible explanation for our results is that dialysate volume was made to avoid high IPPs, especially in patients who had a past history of repaired hernias.

Three previous studies have evaluated the occurrence of hernias in relation to IPP. Durand et al. [17] could not find any correlation in adults, while Aranda et al. [9] found correlations in children. In our study, and in all the aforementioned studies, IPP was measured at rest and in a supine position. Since IPP varies greatly from patient to patient according to their daily physical activities [18], the current methodology of IPP determination might be inadequate to detect patients at risk. However, until the existence of a putative relation between IPP and the development of hernias in PD patients has been thoroughly studied in a large number of patients, it is prudent to continue teaching our PD patients to refrain from important physical activities without draining the peritoneal cavity. The discrepancy between studies over the influence of IPP on hernia formation and the higher rate of hernias in patients on PD compared with that of patients on haemodialysis, suggests that IPP might have a deleterious influence but also that another intrinsic predisposing factor of parietal weakness may exist in PD patients.

Finally, late peri-exit site leakage (i.e. occurring more than 1 month after PD catheter insertion), an uncommon (<0.05%) and unpredictable complication of PD [19], does not seem to be clearly associated with fill volume or IPP, neither in the literature nor in the present study.

Development of gastro-oesophageal reflux. Upper GI symptoms are not infrequent in PD patients and are mainly due to GOR [20]. GOR is more common in PD than in haemodialysis (HD) patients as well as in the general population [21] which strongly suggests that PD treatment is a putative cause of GOR: the presence of a large amount of dialysate in the peritoneal cavity could indeed favour the occurrence of acid reflux from the stomach. In the present study we could not find any influence of IPP on the occurrence of GOR. Our patients with GOR had a higher (though not significant) BMI. This is not surprising as, in the general population, obesity is an independent risk factor for the development of GOR [22]. IPP and GOR interconnection merits, therefore, further evaluation in a larger population of obese PD patients.

Development of enteric peritonitis. One of the most important findings of our study is the observation of a significantly higher incidence of EP, but not of gram-positive peritonitis, as well as a worse survival time free of EP, in patients with IPP night >14 cm H₂O. A similar trend, though not significant, was observed for patients with IPP day >13 cm H₂O. Because EP entails a high risk of morbidity and mortality, increased incidence of catheter removal, and the development of peritoneal lesions which preclude further PD therapy [23,24], the identification of predisposing factors is of major importance.
Currently, the most recognized predisposing factor for peritonitis is related to exchange connectology: the incidence rate of gram-positive peritonitis has clearly decreased with the advent of the flush-before-fill double-bag system, while that of gram-negative did not decrease [23,25] which suggests other predisposing factors. EP is thought to be due to a translocation of microorganisms from the intestinal lumen or from urogenital organs to the peritoneal cavity or due to visceral microperforation (that can be seen in diverticulitis, duodenal or gastric ulcer, cholecystitis or ischaemic bowel disease). The existence of intestinal abnormalities such as diverticulosis has also been thought to predispose to EP [26] but this issue is still debated [27–28]. In addition, Caravaca et al. [27], but not Del Peso et al. [28], found that gastric acid inhibitor prescription was an independent risk factor for EP because their use might favour microorganism overgrowth within the upper gastrointestinal tract.

Since EP prevalence is far higher in patients on PD therapy than in patients on haemodialysis or after transplantation [29], other predisposing factors directly related to the PD procedure might exist. Translocation of microorganisms from the intestine has been recognized in the abdominal compartment syndrome. This syndrome, which includes an increased IAP [as defined by an IAP >15 mmHg (20.4 cm H2O)] and intra-abdominal organ dysfunctions, leads to translocation of microorganisms via intestinal and peritoneal hypoperfusion and ischaemia [30]. Likewise a progressive increase in IAP (from 10 to 25 mmHg) in rats leads to bacterial translocation and to structural alterations of the terminal ileum [31]. Of course, the clinical condition of PD patients differs from that of patients with abdominal compartment syndrome, mainly because of the degree of IAP. This contention, which is certainly true when PD patients are at rest, should be viewed in conditions of increased IPP, such as those observed during physical activities [18,32]. Therefore, our finding that higher IPP might be associated with an increased incidence of EP is thus of great interest, and merits evaluation in a larger cohort of patients.

The main limitation of our study is the small number of patients. Another point of concern is the absence of longitudinal measurement of IPP, as IPP was measured on average of 2 months after PD onset, allowing a peritoneal accommodation to the infused volumes. In this context, Durand et al. [33] found that IPP was significantly higher during the first 3 days following peritoneal catheter implantation than in the next 12 days; a finding that supports the need for regular measurement of IPP to correlate its evolution with clinical events. Finally, we did not evaluate serum albumin concentration or other comorbid factors in the interpretation of PD-related complications. We recommend including these factors in future studies investigating IPP-related complications.

Conclusion

Elevated IPP, mainly observed in corpulent patients, is associated with an increased incidence of EP; therefore, its determination should become an important step in the management of PD patients. Since a linear correlation exists between IPP and IPV, clinicians could thus determine the IPP of their PD patients at any time for any inflow or outflow volumes in order to detect patients at risk for EP. Until a large prospective study evaluates all PD technical complications in relation with IPP, it seems reasonable to avoid excessively high IPP.

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