Pediatr Nephrol (2008) 23:335–338
DOI 10.1007/s00467-007-0615-y

BRIEF REPORT

Dialysate leakage into pericardium in an infant on long-term peritoneal dialysis

Dagmara Borzych · Sebastain Ley · Franz Schaefer · Heiko Billing · Julia Ley-Zaporozhan · Jens Schenk · Claus Peter Schmitt

Received: 2 July 2007 / Revised: 7 August 2007 / Accepted: 7 August 2007 / Published online: 9 October 2007
© IPNA 2007

Abstract We report on a 2-year-old boy on automated peritoneal dialysis (PD) with a history of multiple hernias and dialysate leaks who developed pericardial effusion. Magnetic resonance imaging (MRI) demonstrated a peritoneo-pericardial fistula. Dialysis had to be discontinued, since head-down tilt reproducibly induced significant hypotension. In PD patients with pericardial effusion a peritoneo-pericardial leak should be considered.

Keywords MRI · Pericardial effusion · Intraperitoneal pressure · Fistula

Introduction

Dialysate leakage is a rare but potentially serious complication of peritoneal dialysis. In children on peritoneal dialysis (PD), peritoneal leaks occur at a frequency of 6.6 % [1]. While early leaks usually occur at the exit site, late leaks (beyond the first month of treatment) are less frequent and mostly drain into the abdominal wall and external genitalia [2]. Peritoneal leaks are related to intraperitoneal pressure (IPP), which is only 0.5–2 cm H_2O in a supine patient with an empty abdominal cavity, but increases linearly with dwell volumes of 600–1200 ml/m^2 body surface area, and exponentially thereafter [3, 4]. In infants the exponential increase in IPP already occurs at a lower fill volume [3, 4]. IPP markedly increases when an individual is in an upright position, and with coughing, and it is higher in patients with organomegaly, constipation and obesity [5]. In addition, surgical interventions and adhesions due to infections alter the abdominal wall structure and may predispose the patient to ruptures of the peritoneal membrane sheath.

Leaks into the pleural space via diaphragmatic defects are potentially life threatening [6]. The reported incidence is 1.6% in adults and 2–8% in children on PD [6–8]. Of pleural leaks, 40–50% occur within the first 2-to-4 weeks of PD and usually develop on the right side [4, 7].

Here, we report on a 2-year-old boy who had undergone postnatal initiation of PD, who repeatedly developed hernias and external leakage and, eventually, pleural effusion and a peritoneo-pericardial fistula.

Case report

The male twin was born at 35 weeks of gestation, weighting 2,200 g, by emergency caesarean section due to placental rupture. He suffered from severe perinatal asphyxia, multi-organ failure and left medial and anterior cerebral artery infarction. Since his renal function did not recover, PD was initiated on day 11, using a pigtail catheter. After 3 days, exit

D. Borzych
Department of Pediatric Nephrology,
Medical University of Gdansk,
Gdansk, Poland

S. Ley · J. Schenk
Radiology, University Hospital for Pediatric & Adolescent Medicine, University of Heidelberg,
Heidelberg, Germany

S. Ley · J. Ley-Zaporozhan
Department of Radiology, German Cancer Research Centre,
Heidelberg, Germany

F. Schaefer · H. Billing · C. P. Schmitt (✉)
Division of Pediatric Nephrology, University Hospital for Pediatric & Adolescent Medicine, University of Heidelberg,
Im Neuenheimer Feld 153,
69120 Heidelberg, Germany
e-mail: claus.peter.schmitt@med.uni-heidelberg.de

site leakage developed; PD was discontinued, and the patient was transferred to a paediatric nephrology centre. A single-cuff, curled Tenckhoff catheter was inserted, with a subcostal exit, and PD was resumed. PD was complicated by leakage from the incision site of the pigtail catheter and required surgical closure. Three days later, a leak from the Tenckhoff catheter exit site developed, and a second Tenckhoff catheter was placed contralaterally. Within 5 days, however, at the age of 4 weeks, the boy developed another leak along the catheter, and dialysis treatment was discontinued.

At 8 months of age, the patient was transferred to our centre. He presented with severe psychomotor retardation and severe growth failure, with a weight of 5.3 kg [standard deviation score (SDS) of −4] and a length of 64 cm (−3 SDS). Glomerular filtration rate (GFR) was below 10 ml/min per 1.73 m^2 body surface area. At 9 months of age, the child underwent the insertion of a two-cuff Tenckhoff catheter and a gastrostomy; the right testis was mobilised into the scrotum and the right processus vaginalis was closed. PD was resumed 2 weeks after surgery, using a fill volume of 700 ml/m^2 body surface area. Repeated measurements of intraperitoneal pressure showed 10 to 12 cm H_2O at a dwell volume of 600 to 1,000 ml/m^2. The boy showed remarkable catch-up growth and weight gain and an improved psychomotor development, with gradually improving right-sided hemiparesis. Growth hormone was initiated when he was 19 months old. However, he developed recurrent inguinal, umbilical and ventral abdominal hernias, requiring laparoscopic and open repairs, four times within 11 months. Each time, PD was discontinued postoperatively for 4–6 weeks, resulting in transient failure to thrive until PD was resumed. Fill volume was limited to 440 to 540 ml/m^2. In the course of his treatment he had an episode of peritonitis, caused by a coagulase-negative *Staphylococcus* sp., which relapsed twice. When the child was aged 2 years, routine echocardiography revealed a clinically and haemodynamically insignificant pericardial effusion. Weekly echocardiographic monitoring reconfirmed a persistent small effusion of 2–4 mm in diameter. No infectious cause, connective tissue disorder or hypothyroidism could be identified by serological analyses; the patient appeared euvolaemic. Uraemic pericarditis was considered unlikely in the face of a total creatinine clearance of 15 ml/min per 1.73 m^2 body surface area (BSA) and a Kt/V of 4.8. After 4 weeks, the pericardial effusion increased. Chest X-ray revealed an extensive, right-sided, pleural effusion; the patient remained asymptomatic. Magnetic resonance imaging (MRI) (1.5 T Magnetom Symphony, Siemens) was performed with T1- and T2-weighted sequences before and after intraperitoneal administration of gadolinium (Omniscan), with the boy under sedation during free respiration. The contrast medium was applied at a

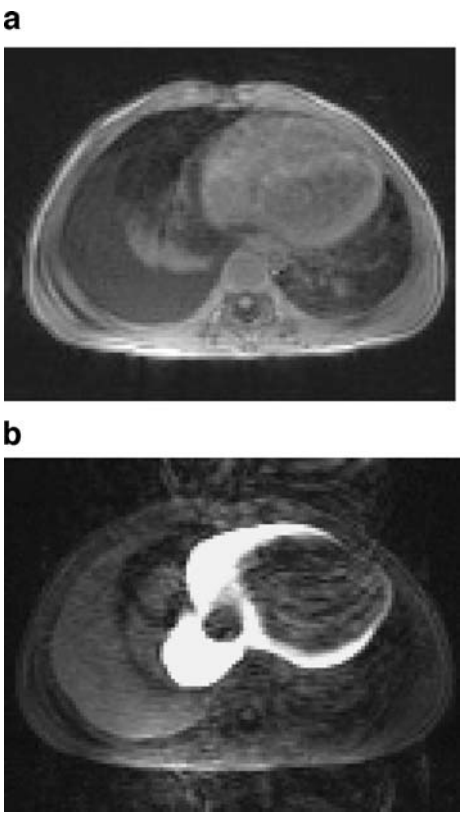

Fig. 1 **a** Transversal T1-weighted gradient echo (GRE) image before infusion of PD fluid, showing a large pleural effusion in the right dorsal lung. **b** After infusion of PD fluid containing gadolinium, selective enhancement of the pericardial effusion was demonstrated, whereas the pleural effusion in the right dorsal lung was not enhanced

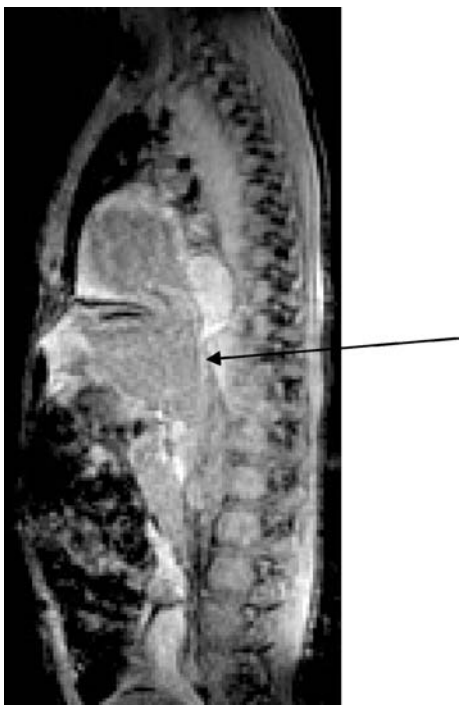

Fig. 2 ECG and respiratory gated T1-weighted gradient echo sequence with a spatial resolution of 1.4×1.4×2 mm^3 revealed a dorsal peritoneo-pericardial fistula (*arrow*)

standard dose of 0.1 mmol/kg body weight in 1,000 ml of peritoneal dialysis fluid. For precise visualisation of the pericardial contrast enhancement, a dedicated electrocardiogram (ECG) and respiration-gated, T1-weighted, gradient echo sequence was applied ($1.4\times1.4\times2$ mm^3). This examination revealed rapid drainage of contrast agent containing PD fluid via a fistula into the pericardial cavity but not into the pleural space (Figs. 1 and 2). Head-down tilt resulted in acute bradycardia and hypotension, necessitating discontinuation of PD. After the MRI, the peritoneum was purged several times. The effusions regressed within few days after cessation of PD; a pericardiocentesis was not performed.

The child was listed as high urgency and received a renal allograft within 3 weeks.

Discussion

To the best of our knowledge, this is the first report of spontaneous leakage of dialysate fluid from the peritoneal cavity into the pericardium. This complication has hitherto been described only in two adult PD patients who had undergone cardiac surgery and pericardiocentesis [9, 10] and who may have had insufficient tissue repair and local adhesions, resulting in tear stress.

Our patient had suffered beforehand from multiple external leaks and hernias. Intraperitoneal pressure was measured repeatedly and showed usual values reported for this age group [11] far below the tolerable upper limit of 15–18 cm H_2O [12, 13]. After surgery, PD was discontinued for 4 to 6 weeks to allow optimal wound healing, and dwell volume was gradually reduced to less than 500 ml/m^2 BSA. Nonetheless, multiple hernias and, eventually, the peritoneo-pericardial leak had developed, suggesting a constitutive connective tissue weakness in this child, which may have been aggravated by malnutrition. Whether the hemipaesis has contributed to the development of the fistula remains uncertain, hernias and leaks occured bilaterally.

MR peritoneography provides detailed information about the anatomical distribution of dialysate in experimental dialysis [14] and patients receiving PD [15]. Peritoneal leakage can also be detected by MRI in patients without clinical symptoms [16]. In this patient we performed MRI with intraperitoneal administration of gadolinium contrast agent to visualise directly the PD fluid distribution. This imaging approach revealed a pericardial effusion that corresponded with the abdomen and, hence, demonstrated precisely the peritoneo-pericardial fistula. A communication with the large pleural effusion on the right side, first identified by chest X-ray, could not be demonstrated, although intermittent drainage of dialysate fluid into the pleural cavity was likely. Of note, an increasing number of dialysis patients who have developed nephrogenic systemic fibrosis in association with exposure to gadolinium-containing contrast agents have been reported [17]. This risk needs to be counterbalanced against the imaging benefits when gadolinium is used instead of pure dialysate fluid as contrast agent; families must be informed accordingly.

Aortic and oesophageal apertures are potential predilection sites for mediastinal hernias and subsequent leakage. Transdiaphragmatic lymphatics may channel leaks. The dorsal leak demonstrated in our MRI study could not clearly be allocated to an anatomic predilection site. Other factors that could have caused the peritoneo-pericardial fistula, such as infections, connective tissue disease, haematologic or metabolic disorders, were ruled out. Uraemic pericarditis due to insufficient PD clearance was unlikely, owing to the considerable residual renal function present when the complication occurred.

Moreover, the three episodes of *Staphylococcus* epidermidis peritonitis, experienced in the 6 months before the pericardiac fistula had developed, may have contributed by leading to local adhesions and tear stress. An increased incidence of hernias has been associated with peritonitis episodes [7].

Patients with pericardial effusions may present with minor clinical symptoms, such as cough, but also with abdominal and chest pain, dyspnoea and life-threatening cardiac tamponade. Our patient was largely asymptomatic, mostly likely due to the small amounts of fluid leaking into the pericardium. However, provocation by head-down tilting reproducibly resulted in significant cardiac symptoms, forcing us to discontinue PD. In the face of the small size of the fistula and the repeated leaks experienced in the past, a surgical closure was precluded. Spontaneous closure of a pericardiocentesis-related peritoneo-pericardial fistula occurred in one patient [10], while a surgery-related fistula has been reported in a second patient. He developed life-threatening cardiac tamponade after resumption of PD [9].

In conclusion, peritoneal-pericardial leakage is a potential cause of pericardial effusion and should be considered, particularly in patients with a history of leaks and hernias. MRI with intraperitoneal administration of contrast agent permits definitive diagnostic proof of this lesion.

References

1. Rinaldi S, Sera F, Verrina E, Edefonti A, Perfumo F, Sorino P, Zacchello G, Andreetta B, Ardissino G, Bassi S, Capasso G, Caringella DA, Gianoglio B, Gusmano R, Rizzoni G (1998) The Italian Registry of Pediatric Chronic Peritoneal Dialysis: a ten-year experience with chronic peritoneal dialysis catheters. Perit Dial Int 18:71–75
2. Leblanc M, Quimet D, Pichette V (2001) Dialysate leaks in peritoneal dialysis. Semin Dial 14:50–54
3. Fischbach M, Terzic J, Becmeur F, Lahlou A, Desprez P, Battouche D, Geisert J (1996) Relationship between intraperitoneal

hydrostatic pressure and dialysate volume in children on PD. Adv Perit Dial 12:330–334
4. Warady A, Schaefer F, Fine R, Alexander S (2004) Pediatric dialysis. Kluwer, London, pp 417
5. Fischbach M, Terzic J, Dangelser C, Schneider P, Roger ML, Geisert J (1998) Effect of posture on intraperitoneal pressure and peritoneal permeability in children. Pediatr Nephrol 12:311–314
6. Stone MM, Fonkalsrud EW, Salusky IB, Takiff H, Hall T, Fine RN (1986) Surgical management of peritoneal dialysis catheters in children: five-year experience with 1,800 patient-month follow-up. J Pediatr Surg 21:1177–1181
7. Nomoto Y, Suga T, Nakajima K, Sakai H, Osawa G, Ota K, Kawaguchi Y, Sakai T, Sakai S, Shibata M, Takahashi S (1989) Acute hydrothorax in continuous ambulatory peritoneal dialysis—a collaborative study of 161 centers. Am J Nephrol 9:363–367
8. Krishnan RG, Ognjanovic MV, Crosier J, Coulthard MG (2007) Acute hydrothorax complicating peritoneal dialysis. Perit Dial Int 27:296–299
9. Nather S, Anger H, Koall W, Reis A, Mukhtar B, Grimm C, Drescher J, Osten B (1996) Peritoneal leak and chronic pericardial effusion in a CAPD patient. Nephrol Dial Transplant 11:1155–1158
10. Hou CH, Tsai TJ, Hsu KL (1994) Peritoneopericardial communication after pericardiocentesis in a patient on continuous ambulatory peritoneal dialysis with dialysis pericarditis. Nephron 68:125–127
11. Fischbach M, Terzic J, Laugel V, Escande B, Dangelser C, Helmstetter A (2003) Measurement of hydrostatic intraperitoneal pressure: a useful tool for the improvement of dialysis dose prescription. Pediatr Nephrol 18:976–980
12. Fischbach M, Terzic J, Menouer S, Bergere V, Ferjani L, Haraldsson B (2000) Impact of fill volume changes on peritoneal dialysis tolerance and effectiveness in children. Adv Perit Dial 16:321–323
13. Durand PY, Chanliau J, Gamberoni J, Hestin D, Kessler M (1994) APD: clinical measurement of the maximal acceptable intraperitoneal volume. Adv Perit Dial 10:63–67
14. Fischbach M, Michallat AC, Zollner G, Dheu C, Barthelmebs M, Helwig JJ, Loichot C, Escande B, Schmitt KP, Schaefer F, Haraldsson B, Jacques C (2005) Measurement by magnetic resonance imaging of the peritoneal membrane in contact with dialysate in rats. Adv Perit Dial 21:17–20
15. Prischl FC, Muhr T, Seiringer EM, Funk S, Kronabethleitner G, Wallner M, Artmann W, Kramar R (2002) Magnetic resonance imaging of the peritoneal cavity among peritoneal dialysis patients, using the dialysate as “contrast medium”. J Am Soc Nephrol 13:197–203
16. Yavuz K, Erden A, Ates K, Erden I (2005) MR peritoneography in complications of continuous ambulatory peritoneal dialysis. Abdom Imaging 30:361–368
17. Grobner T, Prischl FC (2007) Gadolinium and nephrogenic systemic fibrosis. Kidney Int 72:260–264