Three Cases of Acute Massive Hydrothorax Complicating Continuous Ambulatory Peritoneal Dialysis (CAPD)

Takao SUGA, Yukio MATSUMOTO, Keiko NAKAJIMA, Masanobu MIYAZAKI, Takao KURAMOTO, Naohiro YANO, Masayuki ENDOH, Yasuo NOMOTO and Hideto SAKAI

Department of Internal Medicine, School of Medicine, Tokai University

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Acute hydrothorax is a rare complication of continuous ambulatory peritoneal dialysis (CAPD). We experienced three such cases among patients who started CAPD in our institute between April 1984 and April 1989. One was resolved with pleurodesis using autologous blood. The other two patients were switched to hemodialysis permanently because pleurodesis with autologous blood, tetracycline or OK432 failed. Acute hydrothorax is one important possible complication in CAPD.

(Key Words: continuous ambulatory peritoneal dialysis (CAPD), hydrothorax, pleurodesis, autologous blood, complication of CAPD)

INTRODUCTION

Continuous ambulatory peritoneal dialysis (CAPD) is an established effective method of maintenance dialysis in patients with end-stage renal disease. There are several problems to be overcome in CAPD. These include peritonitis, exit site infection, catheter failure, leakage, ultrafiltration failure and pleural effusion. Acute hydrothorax is a rare complication of intermittent peritoneal dialysis (3–5). Recently, the occurrence of acute hydrothorax has also been reported in CAPD patients (12, 13, 15). We describe here three patients with acute hydrothorax in our institute.

CASE 1

A 38-year old male was admitted because of dyspnea. He started CAPD 21 months before admission due to endstage renal failure secondary to focal glomerular sclerosis. He was well on regular CAPD with four daily 2-liter dialysis exchanges until one day before admission. He lifted a heavy barrel in the morning and experienced dyspnea in the afternoon. He also noticed decreased drainage volume and visited Tokai University Hospital the next day. A chest X ray revealed right sided massive hydrothorax (Fig. 1) and he was admitted on the same day. On physical examination, anemia in the eyelids and diminished breathing sounds in the right lung were noticed. No friction rub was audible. Laboratory data on admission revealed WBC 9.200/µl, RBC 246×10^4/µl, HGB 6.9 g/dl, glucose 90 mg/dl, urea nitrogen 56 mg/dl, creatinine 22.6 mg/dl, Na 142 mEq/l, K 4.2 mEq/l and LDH 20 U/l. Thoracentesis was performed and 2,800 ml of pleural effusion was obtained. The pleural effusion revealed a cell count of 40/µl, glucose 343 mg/dl, urea nitrogen 61 mg/dl, creatinine 21.8 mg/dl, Na 136 mEq/l, K 3.8 mEq/l and LDH 20 U/l. The peritoneal effluent revealed glucose 769 mg/dl, urea nitrogen 55 mg/dl, creatinine 15.8 mg/dl, Na 131 mEq/l, K 3.4 mEq/l and LDH 3 U/l. Gram stain and culture of both pleural effusion and peritoneal effluent were negative. Five milliliters of indigocarmine was injected into the peritoneal cavity. After 5 minutes the pleural effusion turned blue. Because of the

Takao SUGA, Department of Internal Medicine, School of Medicine, Tokai University, Bohseidai, Isehara, Kanagawa 259-11, Japan
very high glucose level in the pleural fluid and translation of indigocarmine from the peritoneal to pleural cavity, a diagnosis of hydrothorax secondary to pleuro-peritoneal communication was made. The dialysis schedule was changed to more frequent dialysis using smaller amounts of dialysate exchanges, i.e. five daily 500 ml dialysate exchanges. In spite of this, pleural effusion increased on the next day. We injected 100 ml of autologous blood after extraction of 2,000 ml of pleural fluid. Pleurodesis using autologous blood was attempted twice because pleural fluid reaccumulated after the first attempt. After the second attempt, the pleural fluid decreased gradually and did not reaccumulate even after instillation of 2 l of dialysate. On the 16th day of admission the dialysis schedule was returned to four daily 2 l dialysate exchanges, and he was discharged. Two months later right sided hydrothorax recurred but was resolved with pleurodesis using autologous blood. He is now well on CAPD without any recurrence of hydrothorax for 13 months.

CASE 2

A 50-year old female was admitted because of facial edema. She regularly visited Tokai University Hospital with chronic renal failure secondary to polycystic kidney disease. On physical examination, anemia in eyelids and large kidneys were noted. On the 20th day of admission, she started CAPD because her renal function deteriorated. After one month of CAPD, she was on a dialysis schedule of four daily 2 l dialysate exchanges, and an endoscopic cutting biopsy of a gastric submucosal tumor was performed. After the biopsy, bloody and cloudy dialysate developed. At the same time she experienced severe nausea and vomiting. The next day, she complained of dyspnea. Physical examination revealed decreased breathing sounds in the right lung. No friction rub was audible. Chest X ray showed massive right sided hydrothorax (Fig. 2). Laboratory data revealed WBC 5,100/μl, RBC 305 × 10^4/μl, HGB 9.1 g/dl, Hct 27.2%, glucose 98 mg/dl, urea nitrogen 60 mg/dl, creatinine 9.5 g/dl, Na 142 mEq/l, K 4.0 mEq/l and LDH 190 U/l. Pleural effusion revealed a cell count of 6,800/μl with 98% neutrophils, glucose 310 mg/dl, urea nitrogen 20 mg/dl, creatinine 6.0 mg/dl, Na 100 mEq/l, K 2.5 mEq/l and LDH 75 U/l. Peritoneal effluent revealed a cell count of
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Fig. 2 Chest radiograph of case 2.

6,600/μl with 95% neutrophils, glucose 699 mg/dl, urea nitrogen 30 mg/dl, creatinine 4.4 mg/dl, Na 133 mEq/l, K 2.3 mEq/l and LDH 8 U/l. Cultures of both pleural and peritoneal effluent showed streptococci. After four days of intraabdominal gentamicin and intravenous vancomycin administration, her pleuropertitonitis improved. In spite of this, the hydrothorax was not resolved with a small amount of dialysate instillation (500 ml) and pleurodesis with autologous blood or tetracycline. She was permanently transferred to hemodialysis.

CASE 3

A 60-year old male was admitted because of the emergence of hydrothorax. He started CAPD 5 months before admission due to end stage renal failure secondary to glomerulonephritis. He was on a dialysis schedule of four daily 2 l dialysate exchanges. He was well until one day before admission when he experienced productive cough and noticed a decreased drainage volume. On physical examination anemia in the eyelids and diminished breathing sounds in the right lung were noted. No friction rub was audible. Chest X ray showed massive right sided hydrothorax (Fig. 3) and he was admitted on the same day. Laboratory data revealed WBC 6,600/μl, RBC 2 × 10⁴/μl, HGB 8.9 g/dl, glucose 98 mg/dl, urea nitrogen 51 mg/dl, creatinine 12.5 mg/dl, Na 143 mEq/l, K 3.7 mEq/l and LDH 199 U/l. Pleural effusion revealed glucose 218 mg/dl, urea nitrogen 48 mg/dl, creatinine 10.7 mg/dl, Na 141 mEq/l, K 3.5 mEq/l and LDH 34 U/l. Because of the very high glucose level in the pleural fluid, a diagnosis of hydrothorax secondary to pleuroperitoneal communication was made. The hydrothorax did not decrease by using a small amount of dialysate (500 ml) instillation and pleurodesis with autologous blood, tetracycline or OK432: After two months, he was permanently transferred to hemodialysis.

DISCUSSION

Hydrothorax is a rare complication of CAPD. Nomoto et al (9) reported in a large series in Japan that the complication rate was 1.6% in 3,195 patients undergoing CAPD. Although this complication is rare, it can cause permanent discontinuation of peritoneal dialysis (7). The mechanism leading to formation of acute hydrothorax is unknown. Demonstration of
anatomical defects in the diaphragm by injection of dyes, radiopaque media or aggregated radioisotopes and by direct inspection on surgery or autopsy has failed (3, 8, 11, 14). Increased intraabdominal pressure can cause this complication. Because intrathoracic pressure is negative, an increase results in a large pressure gradient between the peritoneal and pleural spaces. The peritoneal fluid might move through peritoneal diaphragmatic defects such as those around the major vessels and esophagus or through the diaphragmatic foramina. Two of our three cases developed this complication after increased intraabdominal pressure. Drainage volumes of the effluent in these patients were the same as those of the other patients, but the hydrothorax developed in one patient after lifting a heavy barrel and the other after severe vomiting. It was suggested that increased intraabdominal pressure is one of the major reasons for this complication. Eighty eight percent of CAPD related hydrothorax develops on the right side (9). This has been explained by the fact that the right diaphragm has more anatomical defects or a more abundant supply of lymphatics (6) than the left side. Nomoto et al reported full resolution of this complication in 27 (54%) out of 50 patients with hydrothorax, and the remaining 23 patients (46%) were switched to hemodialysis permanently. Treatments include brief interruption of CAPD (1) or brief interruption with pleurodesis with tetracycline (2), autologous blood (16), OK 432 (10) or N-CWS (Nocardia rubra cell wall skeleton) (12). One of the three patients in this report recovered with 100ml of autologous blood instillation. This method appeared worth attempting because it is easy and without serious complications. In the first patient, hydrothorax recurred. There have been several reports of recurrence (4, 14) but no statistical data were available. Nomoto et al noted only one case of recurrence out of 27 patients who were resolved. In conclusion, hydrothorax is very rare but important possible complication because it can cause permanent discontinuation of CAPD.

REFERENCES