症例報告

A CASE OF PLEUROPERITONEAL COMMUNICATION IN CONTINUOUS AMBULATORY PERITONEAL DIALYSIS

NORIHIKO MATSUMURA, TADATOSHI KIKAWA, MASAKAZU HANATANI
and HIDEO NONAKA
Department of Internal Medicine, Nara Prefectural Mimuro Hospital
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Abstract : We present a patient who developed a massive right-sided hydrothorax two months after starting CAPD treatment, a 41-year-old female with IgA nephropathy who was followed up at our outpatient clinic. Due to deteriorating renal function, a peritoneal catheter was inserted on March 29, 1997. CAPD was implemented on the same day. At the time when CAPD was initiated, the patient was experiencing repeated episodes of nausea and vomiting, probably due to uremia. On May 27, she developed dyspnea. A chest X-ray showed massive right-sided hydrothorax. After extracorporeal ultrafiltration was performed, the patient's respiratory distress was rapidly relieved. Two days later, CAPD was resumed, but this resulted in almost immediate recurrence of massive right-sided hydrothorax. The property of the drained fluid from the right pleural cavity and scintigraphy using Technetium-99m macroaggregated albumin confirmed pleuroperitoneal communication. Increased intra-abdominal pressure due to frequent vomiting may be responsible for hydrothorax due to pleuroperitoneal communication.

Key words : pleuroperitoneal communication, hydrothorax, intra-abdominal pressure, continuous ambulatory peritoneal dialysis

INTRODUCTION

Continuous ambulatory peritoneal dialysis (CAPD) is an established and effective renal replacement therapy for patients with end-stage renal disease. Up to 2 % of CAPD patients tend to have right hydrothorax due to pleuroperitoneal communication (PPC)[1]. We present a patient who developed a massive right-sided hydrothorax (MRH) two months after starting CAPD treatment. The causes responsible for the occurrence of PPC are discussed.

CASE REPORT

A 41-year-old female was diagnosed with IgA nephropathy in December, 1995. She was subsequently followed up at our outpatient clinic. Due to deteriorating renal function, a peritoneal catheter was inserted on March 29, 1997. CAPD was commenced on the same day. At the time she was experiencing repeated episodes of nausea and vomiting, probably due to

uremia. Initially the CAPD involved 4 daily exchanges of 1 L dialysate containing 1.5% dextrose. One week later, the CAPD menu was changed to 4 daily exchanges of 1.5 L dialysate containing 1.5% dextrose. On May 27, she developed dyspnea with diminished air entry in the base of the right lung. She did not have lung disease, cardiac failure, or water overload. A chest X-ray showed MRH (Fig. 1). She was admitted to the hospital on that day. Relevant laboratory data on admission indicated C-reactive protein of 0.5 mg/dl and a leukocyte count of 8,700 /mm^3(Table 1). A flexible double lumen catheter was inserted and extracorporeal ultrafiltration (ECUM) was performed. The patient's respiratory distress was rapidly relieved. Post-ECUM film of the chest showed almost complete clearing of the effusion (Fig. 2). Two days later CAPD was resumed, but this resulted in almost immediate recurrence of the MRH. Thoracocentesis was undertaken, enabling a transparent fluid to be drained from the pleural cavity. Cultures for bacteria were negative and microscopic examination of the fluid revealed no malignant cells. High glucose (267 mg/dl) and low protein concentrations (0.4 g/dl) suggested that it was dialysate. Technetium-99m macroaggregated albumin (Tc-99m MAA) was then employed in an attempt to demonstrate a PPC. Five mCi of Tc-99m MAA in 1.5 liter of 1.5 % dialysate were administered into the peritoneal cavity through the PD catheter. Images were taken with a large-view Gamma camera. Three hours after instillation, the isotope was detected in the right hemithorax confirming the presence of PPC (Fig. 3). Neither treatment with thoracoscopy and pleurodesis nor surgical repair were undertaken because the patient did not want to stay on PD. CAPD was discontinued and she received hemodialysis thereafter. She is doing well on hemodialysis.

DISCUSSION

We describe a CAPD patient with hydrothorax. Massive hydrothorax is an infrequent but

Table 1. Laboratory Data on Admission

Urinalysis			Creatine phosphokinase	1,007	IU/L
Protein	(2+)		Amylase	106	IU/L
Sugar	(-)		γ-Glutamyl transpeptidase	14	IU/L
Occult blood	(-)		Total cholesterol	231	mg/dl
Fecal examination			Triglyceride	178	mg/dl
Occult blood	(-)		Total protein	5.2	g/dl
Hematology			Albumin	3.2	g/dl
Red blood cell count	155×10^4	/μl	Blood urea nitrogen	43.8	mg/dl
Hemoglobin	4.9	g/dl	Creatinine	12.3	mg/dl
Hematocrit	14.1	%	Uric acid	7.9	mg/dl
White blood cell count	8,700	/μl	Sodium	138	mEq/L
Platelet count	10.5×10^4	/μl	Potassium	4.2	mEq/L
Erythrocyte sedimentation rate	130	mm/h	Chloride	94	mEq/L
Blood chemistry			Calcium	7.7	mg/dl
Total bilirubin	0.2	mg/dl	Phosphorus	6.3	mg/dl
Aspartate aminotransferase	23	IU/L	Plasma glucose	90	mg/dl
Alanine aminotransferase	15	IU/L	Serum iron	135	μg/dl
Lactate dehydrogenase	882	IU/L	Ferritin	370	ng/ml
Alkaline phosphatase	453	IU/L	C-reactive protein	0.5	mg/dl

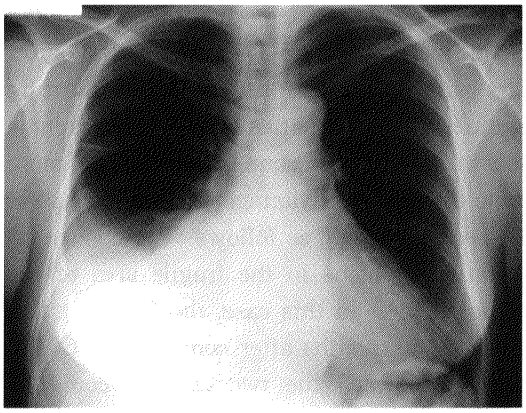

Fig. 1. Chest radiograph on admission showing massive right-sided pleural effusion.

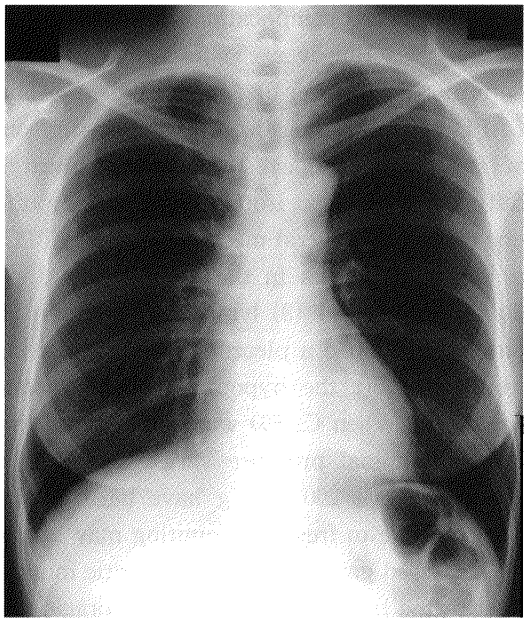

Fig. 2. Chest radiograph after extracorporeal ultrafiltration showing the absence of right pleural effusion.

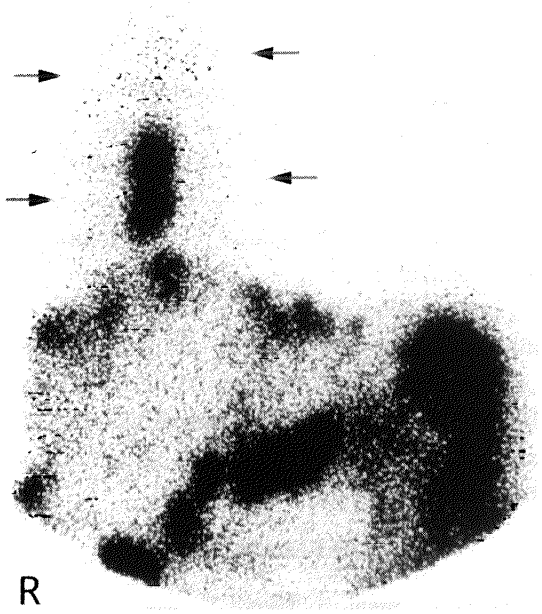

Fig. 3. Isotopic activity (arrow) was detected over the right hemithorax three hours after intraperitoneal administration of 5 mCi of Technetium-99m macroaggregated albumin.

well-recognized complication of CAPD[1]. CAPD-related pleural effusion has been accounted for by PPC. However, PPC is a mere conceptual name, and its physiological and pathological definition has never been established. The diagnosis of PPC is difficult before starting CAPD treatment, because the abnormality only manifests when a patient has a large volume of free intraperitoneal fluid. Anatomical demonstration of the connection can be more difficult. Peritoneal isotopic studies may offer some diagnostic advantage, but this is as yet unconfirmed. Several mechanisms as to PPC have been proposed, as follows : (1) congenital or acquired defects in the diaphragm[2, 3]; (2) laceration of blebs in the fragile area of the diaphragm[4-6]; and (3) lymphatic transport along the aorta[7]. In this case, the fact that this patient developed a pleuroperitoneal communication at two months after commencing CAPD would support the hypothesis that a communication between the two cavities could be acquired. When CAPD was commenced, the patient vomited frequently because of uremia. Intra-abdominal pressure varies according to various postural changes[8], indeed, pressure changes above 200 mmHg have been recorded during lifting[9]. Increased intra-abdominal pressure due to frequent vomiting may be responsible for either rupture of the blebs or even an increased gap in the diaphragmatic muscle. Once a defect appears, leakage will be further aggravated by the negative intra-thoracic pressure on inspiration. A large pressure gradient between the peritoneal and pleural spaces could be the driving force causing dissection through potential diaphragmatic defects. Although we did not perform thoracoscopy, multiple blebs or defects in the diaphragm will be likely observed.

Previous stretching of the diaphragm during pregnancy could be a factor. Our patient had had three full-term pregnancies.

The prognosis in patients with PPC is good. In 1989, Nomoto et al.[1] reported that among the 3,195 patients undergoing CAPD from 161 centers, 50 patients (1.6%) developed acute massive pleuroperitoneal communicating hydrothorax at some time during the study period. In their study, the successful long-term resolution of this problem was obtained in 27 (54%) out of 50 patients following a brief interruption of CAPD only or a brief interruption with pleural instillation of tetracycline or other agents. The remaining 23 patients (46%) were switched to hemodialysis permanently.

In conclusion, we had a patient who developed a massive right-sided hydrothorax due to pleuroperitoneal communication two months after starting CAPD treatment. Especially when uremic symptoms are severe, when CAPD would be traditionally commenced it should be postponed without hesitation and, instead, hemodialysis be applied temporarily. We would stress the importance of temporary hemodialysis as an aid for successful continuation of CAPD therapy through prevention of an acquired PPC

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