Dear Sir,

Hydrothorax related to ascites has sometimes been noted in patients with hepatic cirrhosis [1] or solid ovarian tumors [2] and the genesis of its pleural effusion has been considered to involve direct transfer of ascites from the peritoneal cavity to the pleural space through diaphragmatic defects or transdiaphragmatic lymphatics [3]. In recent years, reports of hydrothorax related to continuous ambulatory peritoneal dialysis (CAPD) have increased [4-10]. Although intraperitoneal fluid is continuously drained by lymphatics and the peritoneal lymphatic absorption rate in CAPD patients is consistent with that in patients with hepatic ascites [5], the appearance rate of hydrothorax in CAPD patients has been reported to be 1.6% in Japan [4]. The reason why its incidence is low compared to the incidence of hydrothorax due to hepatic cirrhosis remains unclear.

Concerning the treatment of hydrothorax due to CAPD-induced diaphragmatic communication, brief interruption of CAPD [6], combined use of small exchange volume in a semisitting position [4], pleurodesis [7-9] and surgical procedures [10] have been found to be effective. Although treatment with pleurodesis employing adhesive agents such as iodized talc [7], fibrin adhesive [8] and tetracycline [9] has been successfully performed, side effects such as pyrexia and chest pain often occur. In addition, there is a possibility that fibrotic changes may be induced in the peritoneum when a strong adhesive agent passes through the diaphragma. We therefore attempted pleurodesis with autoblood which is thought to be a weak adhesive agent.

A 54-year-old male on CAPD (schedule: 4 × 1,500 ml of a 1.5 g% glucose dialysate) was admitted in November 1989, because of right pleural effusion. We had diagnosed hydrothorax due to CAPD-induced diaphragmatic communication by using technetium-99m-labeled macroaggregated albumin. Since the hydrothorax was not improved by combined application of a small exchange volume in a semisitting position, the patient was subjected to pleurodesis with 100 ml of autovenous blood in a sitting position after draining the dialysate and pleural effusion as completely as possible. CAPD was discontinued for 24 h after the pleurodesis, but was subsequently reinitiated on a daily schedule of 6 × 500 ml of a 1.5 g% glucose dialysate for 2 days and then 6 × 1,000 ml for 2 days with restriction of protein,
potassium, sodium and water. Since examination of a chest radiograph on the 6th day after the pleurodesis revealed complete disappearance of the pleural effusion, the CAPD schedule was returned to the original condition, and the patient was discharged in December 1989. During and after the pleurodesis, side effects such as pyrexia and chest pain were never recognized. The patient continues on CAPD at present (August 1992).

Treatment of hydrothorax due to CAPD-induced diaphragmatic communication with pleurodesis using autoblood thus proved to be effective, safe and inexpensive, and this technique provided a cure over a period of 2.5 years. We therefore propose that autoblood should be the first choice among adhesive agents when treatment by pleurodesis for hydrothorax due to CAPD-induced diaphragmatic communication is performed.

References


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Okada/Takahashi/Kinoshita Therapy of CAPD-Induced Diaphragmatic Communication