Dear Sir,

A subclavian catheter is used as a rapid means of temporary access for dialysis patients who have no ready access. However, a fatal complication of subclavian hemodialysis catheter insertion is often reported and this event is sudden and may lead to a fatal clinical course [1-4]. We experienced four cases of catheter-induced cardiac tamponade, and all cases improved after pericardiocentesis. Of them, we would like to report an unusual case who showed early and late onset of cardiac tamponade.

A 44-year-old man with chronic renal failure was admitted to the Kangnam St. Mary’s Hospital for hemodialysis therapy. On admission, he was in good condition and blood pressure was 160/110 mm Hg. On the second hospital day, a double-lumen subclavian catheter was placed through a right infraclavicular approach without any difficulties. A chest x-ray film showed that the catheter tip was in the right atrium. Just after insertion of the catheter, the patient began his first dialysis treatment without a hypotensive episode. Six hours after hemodialysis, blood pressure dropped to 80/60 mm Hg and he complained of anterior chest pain. A chest x-ray revealed no pneumothorax but the heart shadow was slightly larger than on the first film. The two-dimensional echocardiogram revealed a small amount of pericardial fluid. Normal saline was given and blood pressure increased to 130/80 mm Hg and maintained thereafter. Therefore, we de-
Hospital Day

Fig. 1. Clinical course. Early (A) and delayed (B) onset of cardiac tamponade is shown. PCC = Pericardiocentesis; SBP = systolic blood pressure; PR = pulse rate.

ment and hepatomegaly. With the provisional diagnosis of cardiac tamponade due to accumulation of blood during the observation period, percutaneous pericardiocentesis was performed. Pericardiocentesis yielded 1,400 ml of dark red blood and the patient’s blood pressure dramatically improved to

cided to observe blood pressure changes and did not perform pericardiocentesis. During the observation period heparin-free hemodialysis was performed 3 times per week (fig. 1). On the 10th hospital day, he became hypotensive (100/60 mm Hg), and a physical examination revealed neck vein engorge-

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140/90 mm Hg. Repeated echocardiography 2 weeks later revealed no reaccumulation of pericardial fluid.

The patient in this report showed two patterns of cardiac tamponade. The first event seemed to be caused by acute accumulation of blood in the pericardial cavity but which was well compensated by fluid therapy. The second event, concerning a large amount of drained blood with dark red color and no hemodynamic change after initial insult, seemed to be associated with a gradual accumulation of blood in the pericardial space during the observation period.

In conclusion, although a small amount of pericardial fluid is detected on echocardiography, pericardiocentesis must be performed if cardiac tamponade is clinically suspected. In addition, a follow-up echocardiography must be considered with a short interval because gradual accumulation of blood in the pericardial space may occur without any evidence of hemodynamic change as in our case.

References


