Renal Replacement Therapy

CASE REPORT

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Pericardial effusion caused by viral pericarditis in a patient receiving peritoneal dialysis

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Abstract

Background: Acute pericarditis causes acute inflammation of the pericardium. Although most cases of pericarditis are idiopathic with no identifiable cause, its etiology can be infectious, such as viral, bacterial, mycotic, and tuberculous infections, or non-infectious, including post-pericardiotomy, metastatic malignant tumor, connective tissue disease, or uremia. However, there has been no report of pericarditis caused by adenoviral infection in patients undergoing peritoneal dialysis (PD). Herein, we report a case of pericarditis and pericardial effusion caused by adenoviral infection in a patient undergoing PD.

Case presentation: A 59-year-old man who had been undergoing PD in our department for 3 years had a bout of acute enteritis. He was later admitted to the emergency department of our hospital because of malaise and loss of consciousness due to pericardial effusion. Testing after admission revealed elevated adenovirus antibody titers. Pericardial effusion improved although no changes in his PD prescription were made. The patient was hospitalized and admitted to maintain hemodynamics and prevent hypotension. Since insufficient dialysis volume was ruled out by peritoneal equilibrium tests and dialysis volume assessment, the patient was kept under observation, and no changes were made regarding the method of dialysis. Pericardial effusion and the C-reactive protein level both gradually declined, and the patient's weight remained steady. The adenovirus antibody titer alone increased to 1:64 at approximately 2 weeks after hospitalization. The final diagnosis was acute pericarditis due to adenoviral infection rather than uremia or dialysis-associated pericarditis.

Conclusions: We treated a patient with a rare case of pericardial effusion caused by viral (adenoviral) pericarditis in a patient undergoing PD. In addition to testing for the usual causes, uremic and dialysis-associated pericarditis must always be ruled out in patients receiving dialysis. In cases of pericarditis with a viral origin, diagnosis and treatment must be comprehensive.

Keywords: Peritoneal dialysis, Pericarditis, Adenovirus, Pericardial effusion, Cardiac tamponade, Pericardiocentesis, Viral infection, Hemodialysis

Background

Acute pericarditis is a disease that causes acute inflammation of the pericardium, and its epidemiology has not been adequately studied. An autopsy study reported an incidence of pericarditis of approximately 1% [1]. It has also been reported that pericarditis occurs in approximately 5% of patients with non-ischemic chest pain [2].

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Most cases of pericarditis are idiopathic, as they have no identifiable cause. However, it can arise from infectious causes, such as viral, bacterial, mycotic, and tuberculous infections, or non-infectious causes, including post-pericardiotomy, metastatic malignant tumor, connective tissue disease, or uremia. Imazio et al. found that 83.2% of cases were idiopathic, with identifiable causes being malignant tumors in 5.1% of cases, tuberculous in 3.8%, autoimmunity in 7.3%, and pyogenic disorders in 0.7% [3]. Viral pericarditis is usually caused by Coxsackie B virus or echovirus. In patients receiving dialysis, uremic pericarditis must also be included in the differential diagnosis. However, there has been no report of pericarditis caused by an adenovirus in a patient undergoing peritoneal dialysis (PD). Herein, we report a case of pericarditis and pericardial effusion caused by adenoviral infection in a patient undergoing PD.

Case presentation

A 59-year-old Japanese man developed oliguria and ascites when he was 11 years old and was diagnosed with nephrotic syndrome, although a renal biopsy was not performed. After approximately 3 years of repeated hospitalization and discharge, he continued to undergo outpatient treatment with prednisolone and diuretics but stopped attending hospital appointments for about 40 years. In March 2009, he developed proteinuria and renal dysfunction and was referred to our department. He started PD in August 2013 and has been managed by our department since then. In late June 2016, he developed acute enteritis. In early July, he began to feel dizzy and ill. In late July, he lost consciousness at work and was brought to our hospital. The patient's father also had end-stage kidney disease, and had undergone hemodialysis (details, including the underlying condition, are unknown).

PD before hospitalization included three cycles of 2000 mL of DIANEAL® N PD-4 1.5 PD solution (Baxter, Deerfield, IL; 7 h) and 1500 mL of EXTRANEAL® PD solution (Baxter; 12-h dwell period). Further, peritoneal equilibration tests in 2016 revealed a dialysate to plasma ratio of solute concentration of creatinine (Cr) of 0.64 (low average) and the dialysate glucose at 4 h versus the dialysate glucose at time zero of 0.36 (low average). Previously, dialysis efficiency and volume, as measured in 2015, were a total Kt/V of 2.5 (peritoneal Kt/V, 1.73 + renal Kt/V, 0.76) and a total weekly creatinine clearance (CCr) of 89.3 L/week/1.73 m^2 (peritoneal CCr, 48.7 L/week/1.73 m^2 + renal CCr, 40.7 L/week/1.73 m^2).

Physical examination findings on arrival were as follows: height, 162.8 cm; weight, 58.8 kg; temperature, 36.5 °C; blood pressure, 104/79 mmHg; heart rate 97 bpm; respiratory rate, 22 breaths/min; and percutaneous arterial oxygen saturation, 98% (on room air). Mild pallor of the palpebral conjunctiva was observed with no jaundice of the bulbar conjunctiva. Respiratory sounds were clear, but pericardial rub and a systolic murmur (Levine II/IV) that were loudest at the apex were present. The abdomen was mildly distended and soft; there were no areas of tenderness, and bowel sounds were normal. The PD catheter exit site was normal, and there was no pedal edema.

Electrocardiography (ECG) revealed a low voltage compared to those of previous tests (Fig. 1). A chest radiograph showed an increased cardiothoracic ratio (Fig. 2a), and pericardial effusion was visible on computed tomography (Fig. 2b). Echocardiography showed no abnormal movement of the left ventricular wall; the ejection fraction (EF) was 70%, left ventricular end diastolic diameter/left ventricular end systolic diameter was 39/22 (mm), interventricular septum/posterior wall thickness was 14/10 (mm), aorta/left atrium diameter was 34/33 (mm), early diastolic wave/atrial systolic wave was 100/116, deceleration time was 201 ms, early filling velocity on transmitral Doppler (E)/early relaxation velocity on tissue Doppler (E′) (E/E′) was 12.6, and the inferior vena cava diameter was 22 mm (no respiratory variation). No pulmonary hypertension, tricuspid valve regurgitation, aortic regurgitation, or mitral valve stenosis or regurgitation were observed, except a mild aortic stenosis; however, pericardial effusion was present (8 mm anterior to the right ventricle and 13.5 mm posterior to the left ventricle). Peritoneal dialysate analysis, urinalysis, and blood biochemistry tests did not reveal any significant infection, such as PD-associated peritonitis, urinary tract infection, or pneumonia (Tables 1 and 2). Viral antibody measurement patterns were consistent with those of previous cytomegalovirus and Epstein-Barr viral infections, and antibody titers to adenovirus type-2, Coxsackie virus type A2, and echovirus type 9 were elevated.

Figure 3 shows the patient's clinical course. As the patient had recently developed acute enteritis and there was a possibility of a previous viral infection, the provisional diagnosis was viral pericarditis. Pericardiocentesis was not due to lack of storage; therefore, the properties of pericardial fluid could not be confirmed. Although the diameter of the inferior vena cava was enlarged, there was no tricuspid valve regurgitation, and intravenous saline was administered for several days after the patient was admitted to maintain hemodynamics and prevent hypotension due to the reduced cardiac EF resulting from pericardial effusion. Compared to past data, there were no signs of fluid overload and no major changes in blood urea nitrogen or serum creatinine levels were observed. Since insufficient dialysis volume was ruled out by peritoneal equilibrium tests and dialysis volume assessment,

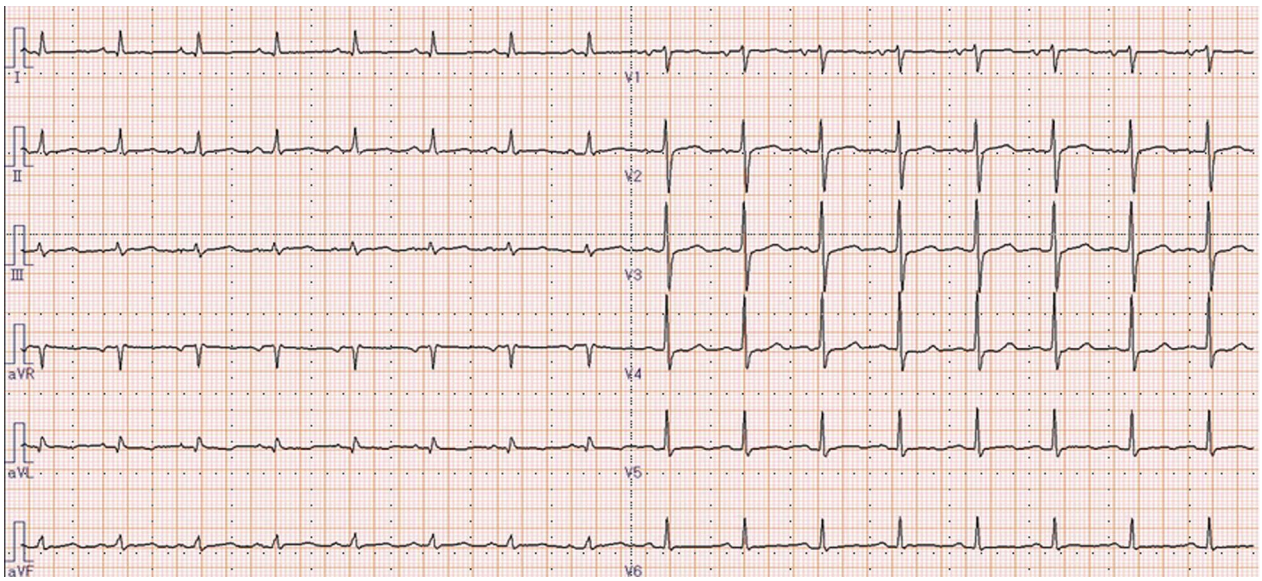

Fig. 1 Electrocardiogram at hospitalization. The electrocardiogram illustrates a heart rate of 95 bpm, sinus rhythm, normal axis, and no ST change. It also illustrates a low potential trend compared to pre-hospitalization electrocardiograms

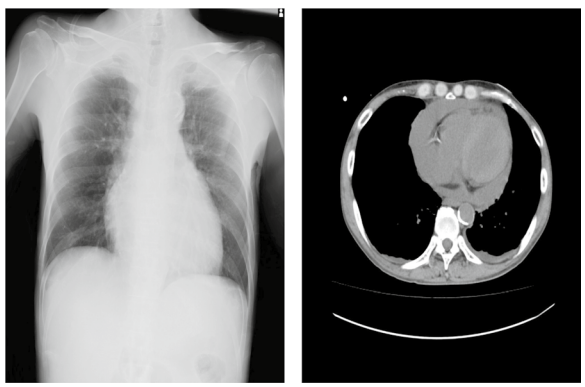

Fig. 2 Chest radiograph and chest computed tomography at hospitalization. **a** The chest radiograph reveals cardiomegaly **b** Chest computed tomography reveals bilateral pericardial effusion. Nodule shadow, interstitial shadow, and invasion shadow of the lung field are not recognized

the patient was kept under observation and no changes were made regarding the method of dialysis. Pericardial effusion and the C-reactive protein (CRP) level both gradually declined, and there was almost no weight change during hospitalization, as the patient's weight remained steady at approximately 59 kg. The adenovirus type-2 antibody titer was later found to be elevated at 1:32 after hospitalization. Although Coxsackie virus and echovirus antibody titers were increased, the adenovirus type-2 antibody titer alone increased further to 1:64 at approximately 2 weeks after hospitalization. The final diagnosis was acute pericarditis due to adenoviral infection. The patient's subsequent course was uneventful, and he was discharged in early August. At an outpatient appointment in late August, both the cardiothoracic ratio and pericardial effusion had improved, and the adenovirus type-2 antibody titer decreased to 1:16.

Discussion and conclusions

In the present case, a patient receiving PD developed pericardial effusion due to viral pericarditis. Acute pericarditis is a condition in which fluid accumulates in the pericardial space as a result of pericardial inflammation. Its cause may be idiopathic, infectious, or non-infectious, post-myocardial infarction (Dressler syndrome), metastatic malignant tumors, trauma, and amyloidosis. According to Imazio et al., the idiopathic type generally accounts for most cases [4, 5].

Diagnosis is based on clinical signs and investigation findings, including ECG and echocardiography. Clinical symptoms include chest pain, fever, and dyspnea. Pericarditis can be differentiated from myocardial infarction as the chest pain is worsened by lying flat and taking a deep breath and is relieved by sitting or leaning forward. In idiopathic or viral infection cases, precursor symptoms, such as upper respiratory and gastrointestinal symptoms, and general malaise may be evident 1–2 weeks before the onset of pericarditis. The tests performed include ECG,

Table 1 Laboratory findings at hospitalization

CBC•coagulation					
WBC	10,470	/μL	CEA	3.1	ng/mL
Neutro	81.4	%	CA19-9	33	U/mL
Lymph	11.6	%	CRP	4.64	mg/dL
RBC	252×10^4	/μL	PCT	0.71	ng/mL
Hb	8.4	g/dL	β-D-glucan	2.8	pg/mL
MCV	100.8	fL	Cryoglobulin	negative	
Plt	421×10^3	/μL	IL-2R	1875	U/mL
INR	1.13		IgG	560	mg/dL
APTT	27.9	sec	IgA	239	mg/dL
			IgM	34	mg/dL
Biochemistry•immunity			IgE	518	IU/mL
TP	5	g/dL	C3	97	mg/dL
Alb	1.9	g/dL	C4	41	mg/dL
T-Bil	0.2	mg/dL	CH50	54	Unit/mL
AST	21	U/L	RF	4.5	IU/mL
ALT	26	U/L	ANA	1:40	
LDH	225	U/L	PR3-ANCA	< 1.0	U/mL
ALP	379	U/L	MPO-ANCA	< 1.0	U/mL
γGPT	36	U/L	anti-GBM antibody	< 2.0	U/mL
CK	265	U/L	C1q-CIC	< 1.5	μg/mL
CK-MB	6.4	ng/mL	Anti-ds DNA antibody	≦1.7	IU/mL
Amy	5	U/L	Anti-Sm antibody	< 1.0	U/mL
BUN	67.8	mg/dL	Anti-RNP antibody	< 1.0	U/mL
Cre	12.44	mg/dL	Anti-SS-A antibody	1.0	U/mL
Na	129	mmol/L	Anti-SS-B antibody	< 1.0	U/mL
K	4.2	mmol/L	Anti-Scl-70 antibody	< 1.0	U/mL
Cl	89	mmol/L	Anti-centromere antibody	< 5.0	U/mL
cCa	10	mg/dL	interferon-gamma release assay	negative	
IP	6.5	mg/dL			
Mg	2.1	mg/dL	*Blood gas analysis (Arterial blood)*		
BS	112	mg/dL	pH	7.498	
BNP	285.7	pg/dL	pCO_2	28.9	mmHg
FT3	1.5	pg/mL	pO_2	65.4	mmHg
FT4	1.4	mg/dL	HCO_3^-	22.3	mmol/L
TSH	2.13	μIU/mL	Glu	118	mg/dL
ACE	9.7	IU/L	Lac	6	mg/dL

WBC, white blood cell count; RBC, red blood cell count; Hb, hemoglobin; CRP, C-reactive protein; MCV, Mean corpuscular volume; Plt, platelet; INR, international normalized ratio; APTT, activated partial thromboplastin time; neutro, neutrophils; lympho, lymphocytes; TP, total protein; Cl, chloride; cCa, corrected calcium; IP, inorganic phosphorus; Mg, magnesium; BS, blood sugar; BNP, B-type natriuretic peptide; FT3, triiodothyronine; FT4, thyroxin; TSH, thyroid stimulating hormone; ACE, angiotensin converting enzyme; Cre, creatinine;Na, sodium; K, potassium; ALT, alanine transaminase; LDH, lactate dehydrogenase; ALP, alkaline phosphatase; γGPT, gannma-glutamyl transpeptidase; CK, creatine kinase; CK-MB, creatine kinase-muscle and brain; Amy, amylase; BUN, blood urea nitrogen; CEA, carcinoembryonic antigen; CA19-9, carbohydrate antigen 19–9; PCT, procalcitonin; IL-2R, interleukin-2 receptor; IgG, immunoglobulin G; IgA, immunoglobulin A; IgM, immunoglobulin M; IgE, immunoglobulin E; C3, complement 3; C4, complement 4; CH50, 50% hemolytic unit of complement; RF, rheumatoid factor; ANA, antinuclear antibody; PR3, proteinase3; ANCA, anti-neutrophil cytoplasmic antibody; MPO, myeloperoxidase; GBM, glomerular basement membrane; C1q-CIC complement 1q circulating immune complexes; DNA, deoxyribonucleic acid; Anti-Sm, anti-sm antibody; Anti-RNP, anti-rebonucleoprotein antibody; Anti-SS-A, anti sjogren's syndrome-A antibody; Anti-SS-B, anti sjogren's syndrome-B antibody; Anti-Scl-70, anti-scleroderma-70 antibody; pH, power of hydrogen; pCO_2, partial pressure of arterial carbon dioxide; pO_2, partial pressure of arterial oxygen; HCO_3^-, bicarbonate ion; Glu, glucose; Lac, lactate

echocardiography, chest radiography, and blood tests. Characteristic ECG findings are convex upward ST elevation on all leads other than aVR and inversions that do not coincide with the regions supplied by the coronary artery. These features change over time, with ST elevation normalizing after a few days, and only a negative T-wave remains. The trace is completely normalized after 1–2 months. Severe pericardial effusion causes QRS

Table 2 Viral antibody at hospitalization

CMV IgG	17.6(+)
CMV IgM	0.27(−)
EBV anti-VCA IgM	1:< 10
EBV anti-VCA IgG	1:40
EBNA antibody	1:40
Adenovirus type 1	1:4
Adenovirus type 2	1:32
Adenovirus type 3	1:< 4
Adenovirus type 4	1:< 4
Adenovirus type 5	1:4
Adenovirus type 6	1:< 4
Adenovirus type 7	1:< 4
Coxsackie virus type A2	1:8
Coxsackie virus type A9	1:< 4
Coxsackie virus type B3	1:< 4
Coxsackie virus type B5	1:< 4
Echovirus type 3	1:< 8
Echovirus type 6	1:< 4
Echovirus type 7	1:< 8
Echovirus type 9	1:8

CMV, cytomegalovirus; EBV, Epstein-Barr virus; VCA, virus capsid antigen; EBNA, EBV nuclear antigen

waves to decay to a constant potential. ECG reveals an echo-free space in the pericardial cavity. Chest radiography reveals enlargement of the heart due to pericardial effusion. Blood test results often include elevated CRP levels and white blood cell count (WBC count), which are suggestive of an inflammatory process. If myocarditis is also present, cardiac enzymes are mildly elevated.

The course and prognosis of pericarditis vary depending on the etiology. Idiopathic and viral cases generally have good prognoses, as patients recover spontaneously within 2–6 weeks. However, its recurrence rate is 18.3%, cardiac tamponade occurs in 3.1% of cases, and 1.5% of patients progress to constrictive pericarditis [3].

If there is a clear cause for treatment, the cause is treated. Treatment of idiopathic or viral pericarditis is mainly symptomatic, using non-steroidal anti-inflammatory drugs. Combination therapy or monotherapy with corticosteroids is effective in first-onset cases and in preventing recurrence. Steroids are used if the cause is a connective tissue disease or an autoimmune condition. Uremic pericarditis is treated by commencing dialysis or improving its efficiency. However, if there is no improvement, steroid use should be considered. If cardiac tamponade is present, pericardial drainage is performed.

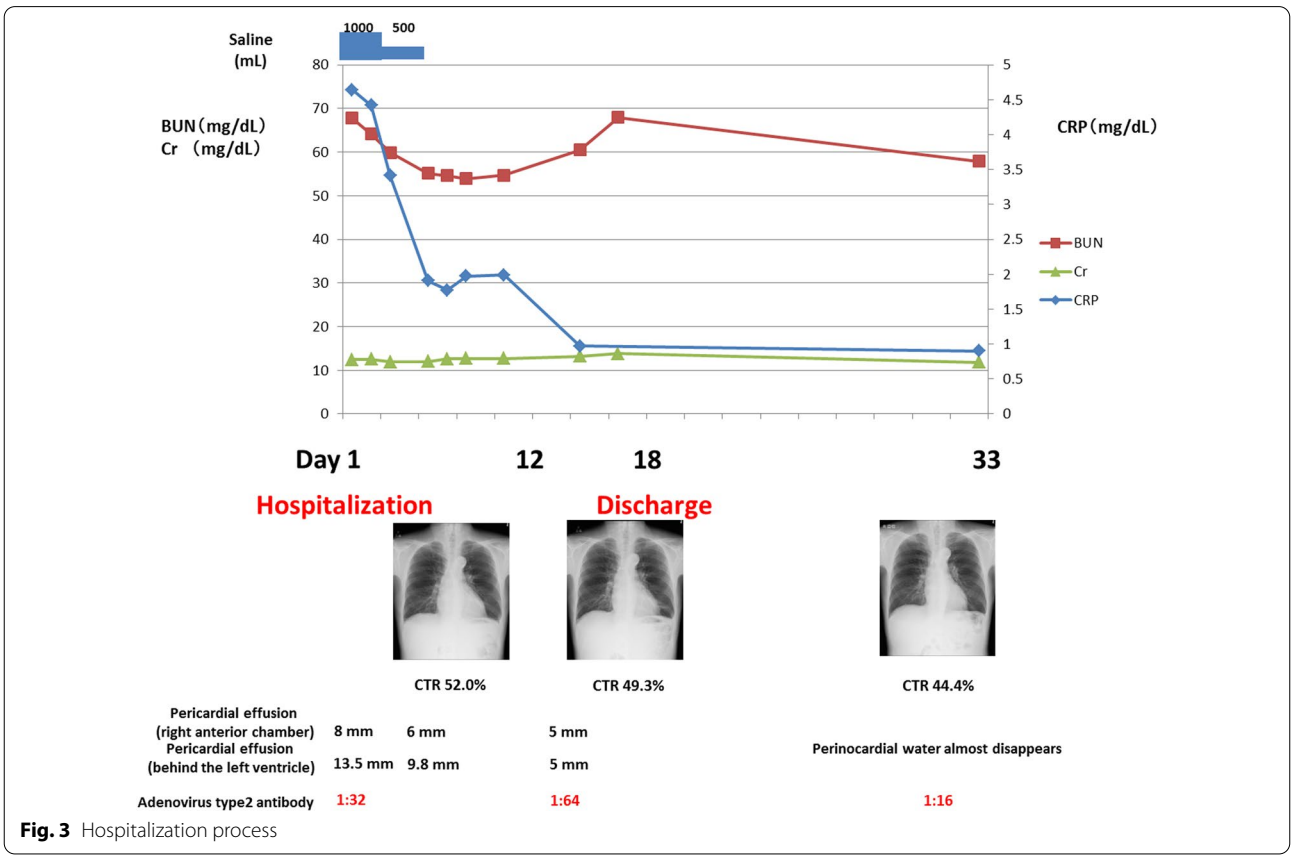

Fig. 3 Hospitalization process

Pericardial fenestration is performed in the event of repeated pleural effusion leading to the development of cardiac tamponade or heart failure.

In the present case, some form of infection was suggested by the elevated WBC count, elevated CRP level, and mildly elevated procalcitonin levels. However, there were no signs suggestive of serious bacterial infection, and mycotic and tuberculous infections were ruled out by negative βD-glucan elevation and interferon-gamma release assay. Connective tissue disease-related autoantibody tests were negative; there was no myocardial infarction, and there were no positive signs that were suggestive of malignant tumors on imaging or tumor markers. The patient had no history of trauma. Amyloidosis was not seriously considered because of the short duration of dialysis, and M proteins were not detected. Therefore, the most likely differential diagnoses were either an infection, particularly a viral infection, or uremic or dialysis-related pericarditis.

Pericarditis in dialysis patients may be uremic or dialysis-related [6]. Pericarditis occurs in patients with end-stage kidney disease, either before or immediately after commencing dialysis. Although the details of its pathogenesis are unknown, it may involve factors including increased vascular permeability due to the accumulation of uremic toxins, immune system dysfunction, and fluid overload. Waker et al. reported that 41% of patients with acute and chronic renal failure were diagnosed with uremic pericarditis on autopsy, which is a very high rate [7]. However, Hakim et al. reported that 7% of patients developed pericarditis within 6 months of commencing dialysis [8]. It is conceivable that advances in dialysis therapy and the introduction of erythropoietin may have influenced this shift. Diabetes-associated pericarditis develops in patients with diabetes undergoing dialysis and is usually caused by insufficient dialysis or inadequate fluid removal (fluid overload). Therefore, it is necessary to optimize dialysis therapy. Furthermore, Tseng et al. found that 85.1% of patients with diabetes and 82.9% of those without diabetes showed improvement after hemodialysis was intensified [9]. Conversely, Connors et al. reported that the improvement rate was as low as 57% after the commencement of dialysis [10]. A study on pericarditis in patients receiving PD found that 4.3% of these patients developed pericarditis more than 4 months after starting dialysis, which is comparable to the rate among patients receiving hemodialysis [11]. As our patient had been undergoing PD for more than 3 years, dialysis-associated pericarditis was considered as a differential diagnosis. However, this was ultimately ruled out because the results of the dialysis efficiency and dialysis volume tests from the previous year did not reveal any major problems. There was also no subcutaneous edema, the pericarditis resolved with no change in the PD prescription, and there was almost no change in body weight when the patient was hospitalized.

Adenoviral infections occur throughout the year in all age groups. Although approximately half are subclinical infections, they account for 5–10% of pediatric respiratory diseases, and may also cause acute gastroenteritis and conjunctivitis. There have also been recent cases of fatal infections, including hepatitis, pneumonia, and meningitis, in immunocompromised individuals, such as organ transplant recipients and patients with acquired immune deficiency syndrome. With regards to the relationship between adenovirus and pericarditis, Ivanova et al. reported that among 235 patients with myocarditis or pericarditis, 63 were negative for Coxsackie type B virus immunoglobulin M (IgM) antibodies, and in the above-mentioned 63 patients, six were positive for adenovirus IgM antibodies, including four patients with pericarditis [12]. Prodromal symptoms such as upper respiratory and gastrointestinal symptoms and general malaise are often present before the onset of pericarditis. Since the patient had pericardial effusion with inflammatory response and was preceded by enteritis, viral pericarditis was suspected. Viral tests were performed for Coxsackie virus and other viruses that cause pericarditis, as well as adenovirus, which causes enteritis, and a diagnosis of adenovirus-induced pericarditis was made based on elevated adenovirus antibodies.

There have been few reports on viral pericarditis in patients receiving dialysis [13, 14] (Table 3). However, all reported cases have been in patients undergoing hemodialysis. The causative viruses in those reports were influenza type A and Coxsackie type B viruses. There have been no previously reported cases of adenoviral infection leading to pericarditis in patients receiving dialysis. The main treatment was continuation of hemodialysis and pericardial pericardiectomy, but some patients improved after hemodialysis was changed to PD [13]. There have been no reports of pericarditis and pericardial effusion caused by adenovirus in patients receiving PD. Hence, this is an extremely valuable case report, although it has some limitations, including the fact that we were unable to perform pericardiocentesis to test the pericardial fluid.

We treated a patient receiving PD who had pericardial effusion caused by adenoviral pericarditis. In addition to testing for the usual causes, uremic and dialysis-associated pericarditis must always be excluded in patients receiving dialysis. Furthermore, in patients receiving PD, pericarditis due to viral infections should be kept in mind.

Table 3 Comparison of present case with previously reported viral pericarditis in patients on dialysis

References	Year	Sex	Age	RRT	Cause of ESKD	Duration of dialysis	Viral infection	Therapy	Clinical outcome
[13]	1970	M	33	HD	CGN	about 84 months	Coxsackievirus B	Converted to peritoneal dialysis	Recovered
[14]	1979	M	51	HD	DGN	about 36 months	Influenza virus A	Partial pericardiectomy	Recovered
[14]	1979	M	68	HD	NS	about 60 months	Influenza virus A	Continue to hemodialysis (from two to three times a week)	Recovered
[14]	1979	M	41	HD	MNS	About 84 months	Influenza virus A	Partial pericardiectomy	Recovered
[14]	1979	M	42	HD	CGN	About 108 months	Coxsackievirus B	Continue to hemodialysis, pericardiocentesis	Recovered
The present case	2016	M	59	PD	Unknown	About 26 months	Adenovirus type 2	Continue to peritoneal dialysis	Recovered

M, male; RRT, renal replacement therapy; ESKD, end stage kidney disease; HD, hemodialysis; PD, peritoneal dialysis; CGN, chronic glomerulonephritis; NS, nephrosclerosis; DGN, diabetic glomerulosclerosis; MNS, malignant nephrosclerosis;

Abbreviations
PD: Peritoneal dialysis; CCr: Creatinine clearance; ECG: Electrocardiography; EF: Ejection fraction; E: Early filling velocity on transmitral Doppler; E′: Early relaxation velocity on tissue Doppler; CRP: C-reactive protein; WBC: White blood cell; IgM: Immunoglobulin M.

Acknowledgments
The authors are grateful to the medical staff of Shimane University Hospital, Izumo, Japan.

Author contributions
ME was responsible for this manuscript. SF, KT, RY, AY, KY, KT, HS, and TI provided information on the discussion and treatment of the patient. ME collected the data and drafted the manuscript. All authors have read and approved the final manuscript.

Funding
The authors received no funding for this case report.

Availability of data and material
All data generated or analyzed during this study are included in this published article.

Declarations

Ethics approval and consent to participate
Written informed consent was obtained from the patient. For case reports, a formal approval from a local ethics committee was not required.

Consent for publication
Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

Competing interests
The authors declare that they have no competing interests.

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Received: 30 May 2021 Accepted: 10 April 2022
Published online: 18 April 2022

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