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Purulent Pericarditis Caused by Group G Streptococcus as an Initial Presentation of Colon Cancer

Bacterial pericarditis has been recognized as a rare disease since the development of antibiotics. Usually, the disease is associated with underlying conditions or a seeding of infection elsewhere to the pericardium. Here we describe a case of group G streptococcal pericarditis as an initial presentation of colon cancer. A 52-yr-old man was admitted because of dyspnea. An electrocardiogram showed a diffuse ST-segment elevation and a two-dimensional echocardiogram showed a large amount of pericardial effusion. A pericardiocentesis was done and purulent fluid was drained. Group G streptococci was cultured in pericardial fluid. The patient was treated with antibiotics and pericardiostomy with saline irrigation. A colonoscopy revealed a small mass with moderately differentiated adenocarcinoma in rectosigmoid colon. He underwent a mucosectomy and was recovered without any complication.

Key Words : *Pericarditis; Group G Streptococcus; Colonic Neoplasms*

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INTRODUCTION

Pericarditis can be caused by a wide variety of infectious or noninfectious processes. It may be clinically silent or may result in a severe hemodynamic compromise and death (1-4).

Group G streptococcus may be found as a member of the normal microbial flora of the skin, pharynx, vagina, and gastrointestinal tract. Endocarditis, endovascular infections, and septic arthritis are the most common syndromes caused by group G Streptococcus (5-7). Here we report a case with a pericarditis caused by group G Streptococcus as an initial presentation of colon cancer.

CASE REPORT

A 52-yr-old man with a long-term alcohol abuse was admitted to the emergency room because of dizziness and dyspnea for the previous 10 days. His vital sign were as follows: blood pressure, 90/60 mmHg; pulse rate, 96 beats per minute; respiration rate, 20 per minute; and temperature, 36.0℃. Twenty years ago, the patient had been treated for pulmonary tuberculosis. Physical examination revealed regular heart beats without rub, diminished breath sounds at both basal lung fields, and hepatomegaly. Pulsus paradoxus was not assessed.

Initial laboratory data included a white blood cell count of $30.1 \times 10^3/\mu L$ (72% neutrophils, 19.5% monocytes, and 1.5% lymphocytes), platelet $304 \times 10^3/\mu L$, AST 131 IU/L (normal, 7-38 IU/L), ALT 127 IU/L (4-43 IU/L), albumin 2.9 g/dL (3.8-5.5 g/dL), BUN 64 mg/dL, creatinine 2.1 mg/dL, sodium 120 mEq/L (136-145 mEq/L), potassium 5.7 mEq/L (3.5-5.0 mEq/L), prothrombin time 18.0 sec, creatine kinase 189 IU/L (50-200 IU/L), T_4 3.73 μg/dL (5-13 μg/dL), free T_4 0.78 ng/dL (0.8-2.2 ng/dL), T_3 0.45 ng/mL (0.8-2.2 ng/mL), and TSH 4.62 μIU/mL (0.34-3.5 μIU/mL).

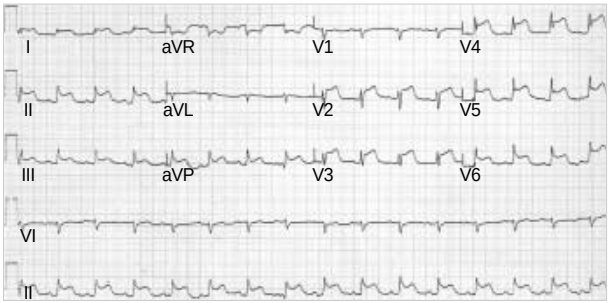

Fig. 1. Electrocardiogram shows a diffuse ST-segment elevation on arrival.

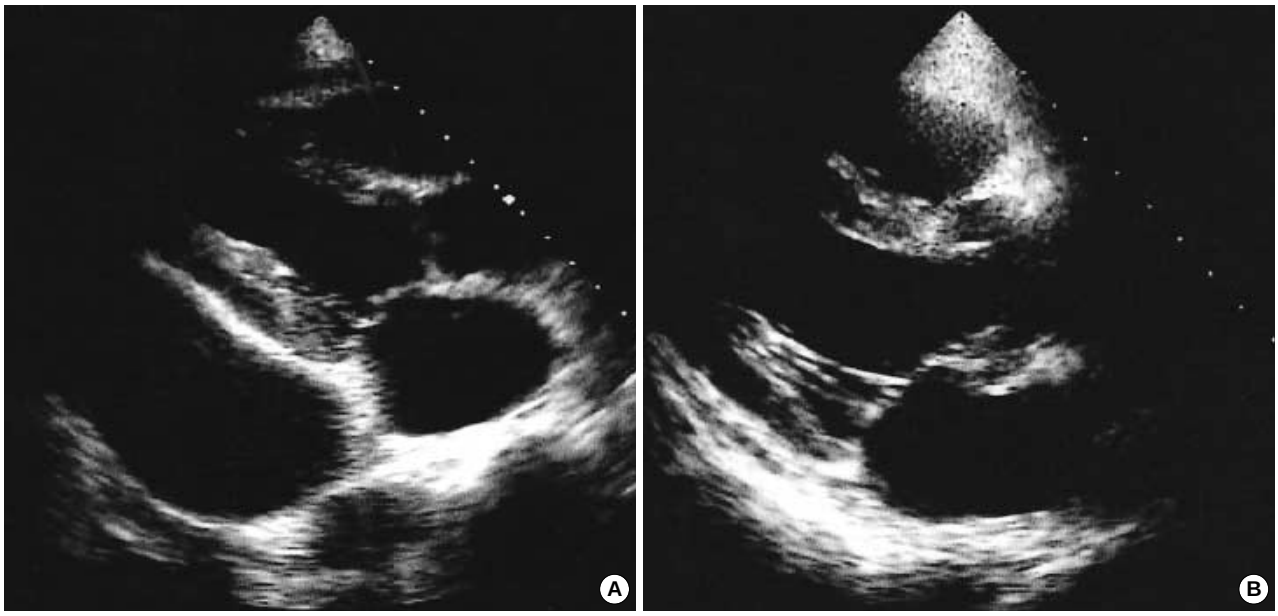

Fig. 2. (A) Two-dimensional echocardiography on admission shows a large amount of pericardial effusion. (B) Follow-up echocardiography shows the nearly absorbed pericardial effusion and thickened pericardium.

An electrocardiogram showed a diffuse ST-segment elevation (Fig. 1). A chest radiograph revealed a large cardiac silhouette with an obliteration of the right costophrenic angle and fibrostreaky lesions in the right upper lung field. Computed tomography (CT) of the chest and abdomen revealed fibrostreaky lesions in the right upper lobe of the lung, pericardial and both pleural effusions, and liver cirrhosis with ascites. A two-dimensional echocardiogram showed a large pericardial effusion (Fig. 2A). Immediately, a subxiphoid pericardiocentesis was done and 700 mL of fluid was drained. Pericardiocentesis showed turbid and cloudy pericardial fluid with total white blood cell count of $27.3 \times 10^3/\mu L$ (91% neutrophils, 9% lymphocytes), pH 7.0, protein 5.5 g/dL, glucose 795 mg/dL, LDH 1,613 IU/L, and adenosine deaminase 122 IU/L (normal, 4-21 IU/L). Gram's stain of the pericardial fluid showed Gram-positive cocci and cultures were positive for group G β-hemolytic Streptococcus. The organism was susceptible to ampicillin, clindamycin, cefotaxime, and vancomycin. Blood, urine, and sputum cultures were subsequently negative.

Initially the patient was treated with cephalothin, which was later changed to ampicillin 1.0 g every 8 hr and amikacin 500 mg every day for 4 weeks. Also, thyroid hormone (Synthroid®, 50 μg) replacement therapy and empirical antituberculosis treatment with isoniazid 300 mg, rifampin 600 mg, pyrazinamide 1.5 g, and ethambutol 800 mg were started. On the fourth hospital day, a thoracentesis was performed in the right pleural space and 300 mL of serosanguinous fluid was drained. The nature of the pleural fluid was transudate. On the seventh hospital day, a pericardiostomy was performed because of reaccumulation of fluid and a saline irrigation through the catheter was done. On the 25th hospital day, the catheter was removed. On the 26th hospital day, a colonoscopy revealed a 1×1 cm-sized exophytic tumor mass in the rectosigmoid colon. Biopsies of the mass confirmed that the mass was moderately differentiated adenocarcinoma. He underwent a mucosectomy on the 41st hospital day. He was discharged without any complication, but Doppler echocardiography revealed a slightly thickened pericardium (Fig. 2B). He is free of disease and well 6 months after discharge.

DISCUSSION

A wide variety of bacterial organisms have been reported as causative agents of pericarditis. Although some reports suggested an increased incidence of Gram-negative organisms, the recent series reported that the most commonly isolated organisms were Gram-positive cocci (1-4).

Group G streptococci may be found in the nasopharynx, skin, and genital tract; intestinal colonization has also been reported. Infection due to group G streptococci is relatively rare. However, a number of severe infections, including infective endocarditis, septicemia, cellulitis and wound infection, ascending cholangitis, pneumonia, empyema, and peritonitis, have been reported (5-7). Only two cases of purulent pericarditis have been reported previously (8, 9).

The infection can be endogenous or exogenous. The endogenous infection often occurs in hosts predisposed by age (neonates or the elderly), alcoholism, injection drug abuse, diabetes mellitus, immunosuppressive therapy with corticosteroids or cytotoxic drugs, or underlying malignancy (5-7).

Colonic carcinoma has been identified as one potential portal of entry into the blood stream for group G Streptococcus. However, there has been no report in the pericarditis caused by group G Streptococcus as a presentation of colon cancer. The patient had underlying debilitation conditions with alcohol abuse, hypothyroidism, liver cirrhosis, and colon cancer. However, he did not complain about any symptoms other than fatigue. The portal of entry of the organism in this patient was not determined but most likely was the intestinal tract.

Although group G streptococci are uniformly sensitive to penicillin, it has been reported that the infections of the organisms respond poorly to penicillin alone and improved by an addition of aminoglycosides (10). Our patient appeared to benefit by the combination therapy. Also, we administered an empirical anti-tuberculosis medicine in spite of negative results of cultures and acid fast staining of pericardial fluid because of a high value of adenosine deaminase activity (11).

In conclusion, group G Streptococcus has rarely been described as an important cause of purulent pericarditis in humans. In the setting of pericarditis caused by group G Streptococcus, the possibility of occult colon cancer should be considered.

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