Chronic Myocarditis Mimicking Acute Coronary Syndrome due to Hypereosinophilia which was produced by Pancreatic Cancer

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Abstract
The present report describes chronic myocarditis induced by hypereosinophilia secondary to pancreatic cancer. A 58 year old man was admitted with a suspicion of acute coronary syndrome. He complained of recurrent resting chest pain 1 week ago. Electrocardiography showed T inversion at precordial leads. Cardiac enzyme was elevated. However, his coronary angiography showed no coronary artery stenosis. Because his laboratory test showed hypereosinophilia and we couldn’t find any evidence of infection or hematologic disease, we concluded that he suffered from eosinophilic myocarditis due to hypereosinophilic syndrome. After using prednisolone, his symptom was relieved, abnormal electrocardiographic finding was normalized, and eosinophil count was decreased, but not to normal range. Thereafter, because his clinical course showed repeated deterioration and improvement, we continued low dose of steroid therapy. One and half years later, he complained of abdominal pain and was diagnosed with pancreatic body cancer. He underwent subtotal pancreatectomy and splenectomy, due to pancreatic body cancer with invasion of splenic and mesocolon artery. Eosinophilia disappeared completely after the operation. Therefore, we suggest that eosinophilic myocarditis can be produced by pancreatic cancer. To the best of my knowledge, this is the first case of eosinophilic myocarditis induced by pancreatic cancer.

Keywords: Eosinophilic Myocarditis, Hypereosinophilia, Pancreatic Cancer

Introduction
The associations among eosinophilia, active carditis, and multiorgan involvement were first described by Loeffler in 1936 [1]. Hypereosinophilic state affects many organ at the same time and symptoms may be numerous. Symptoms of hypereosinophilic state include skin lesion, thrombo-embolic events, pulmonary disease, cardiomyopathy, neuropathy, hepatosplenomegaly and so on. Theoretically, all hypereosinophilic state can cause eosinophilic myocarditis. However, one retrospective multicenter study including 188 patients reported that only 5% of hypereosinophilia manifested eosinophilic myocarditis [2]. Known causes of hypereosinophilia, which may occur in association with myocarditis, include hypereosinophilic syndrome, eosinophilic leukemia, carcinoma, lymphoma, drug reactions or parasites, as reported in multiple case series. Carcinoma is known to be very rare cause of hypereosinophilia. We report the first case diagnosed with chronic myocarditis induced by hypereosinophilia secondary to pancreatic cancer.

Case Report
A 58 years old asian man was admitted with suspicion of an acute coronary syndrome because of resting chest pain, elevation of cardiac marker (Troponin T 1.37ng/ml; reference value: <0.0014) and T inversion at lead II, III, aVF and V4 ~ 6 on electrocardiography (Figure 1). He suffered from headache, sore throat, and myalgia for 7 days before admission. He was a current smoker, and was on antihypertensive medications due to hypertension and had no any other past medical history. On physical examination, he exhibited a blood pressure of 140/80mmHg, heart rate of 88 beats/min, and temperature of 36.7 °C. There were no abnormalities on physical examination. Laboratory examination showed elevated white blood cell count (12600/mm3) with elevated eosinophil count (28%) and cardiac marker elevation (CPK 80, CK-MB 4.5, Troponin 1.37).

Inflammation marker was not elevated. (ESR 15mm/h, CRP 0.56 mg/L). Transthoracic echocardiography showed slightly enlarged left atrium (44mm) and left ventricular hypertrophy (LV septum 14.4mm/posterior wall 14.4mm) with increased echo density and elevated EF (79%). There is no thrombus and mass at echocardiography. We thought that he had an acute coronary syndrome at first. So, we prescribed anti-anginal medications (Antiplatelet agent [Aspirin, Clopidogrel], B-blocker, angiotensin converting enzyme inhibitor, statin) and checked coronary angiography. However, coronary angiography showed no significant coronary artery stenosis. During hospitalization, he continuously complained of a headache and chest tightness. We rechecked the laboratory test, which demonstrated a more elevated white blood cell (WBC 24200/mm3) with eosinophil count (68.1%) and normal CRP (0.91 mg/L).

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One and half years later, he revisited our hospital for seeking previous medication. At that time, he said that he was admitted to another hospital for abdominal pain, and was diagnosed with pancreatic cancer. Therefore, he underwent subtotal pancreatectomy & splenectomy. His magnetic resonance image showed pancreatic body mass and splenic artery invasion (Figure 3), and his biopsy specimen showed atypical glandular cell (Figure 4A and 4B), which indicates adenocarcinoma. As such, his clinical course was mimicking acute coronary syndrome, but there was no abnormality of coronary artery, and echocardiography showed a thickened left ventricle and increased echo density. We performed a parasite test, peripheral blood smear and checked tumor marker, but there was no abnormal test except for hypereosinophilia on peripheral blood smear. Because we couldn’t find the cause of hypereosinophilia, we concluded that he had suffered from eosinophilic myocarditis by hypereosinophilic syndrome. We prescribed prednisolone 30 mg per day for the patient.

His symptom was improved, but eosinophil count was still high. We checked for the eosinophil count daily, and his eosinophil count decreased slightly (Figure 2A). We recommended regular outpatient clinic visits and the patient was discharged with a condition of maintaining a dose of prednisolone 30mg.

One month later, white blood cell count 12000/mm³ with 36.4% eosinophils (Figure 2B), and Electrocardiography was normalized, except T wave inversion at lead III. Follow up echocardiography showed that normal left atrium size (37.3mm) decreased left ventricular hypertrophy (Septum 12mm/posterior wall 12mm) and normal EF (60%). Therefore, we tapered off a dose of prednisolone 30 mg to 5 mg, and regularly check echocardiography and white blood cells with eosinophil count, every 6 months.

Nine months later, he revisited the emergency medical center in our institute complaining of chest tightness and dyspnea, again. His laboratory test showed that white blood cell count was mildly elevated (WBC 12000/mm³ with 5.93 eosinophils) (Figure 2B), and normal electrocardiography. Because we thought chronic eosinophilic myocarditis was aggravated, we increased the dosage of prednisolone from 5 mg to 20 mg. Two days later, his symptom completely recovered and he was discharged with symptom-free state.

One and half years later, he revisited our hospital for seeking previous medication. At that time, he said that he was admitted to another hospital for abdominal pain, and was diagnosed with pancreatic cancer. Therefore, he underwent subtotal pancreatectomy & splenectomy. His magnetic resonance image showed pancreatic body mass and splenic artery invasion (Figure 3), and his biopsy specimen showed atypical glandular cell (Figure 4A and 4B), which indicates adenocarcinoma. As such,
his diagnosis was pancreatic body cancer with splenic artery invasion.

We received his medical record from the other hospital to understand his medical condition. His pancreatic cancer was at its terminal stage with small bowel obstruction by cancer mass. The medical staffs performed palliative operation to relieve his symptom.

After the palliative operation, his laboratory test showed normalized white blood cells with eosinophil count (WBC count 9070 with 0.1% eosinophil). Therefore, we thought his hypereosinophilia was produced by pancreatic cancer and we decreased dosage of prednisolone 20 mg to 5 mg.

He visited our hospital regularly on a six months basis and hypereosinophilia have not recurred, he no longer complained of angina symptoms. However, his pancreatic cancer progressed, and he died from pancreatic cancer. We report the first case of chronic myocarditis by hypereosinophilia secondary to pancreatic cancer. To the best of my knowledge, this is the first case until now.

Discussion

The clinical manifestation of eosinophilic myocarditis is nonspecific and has a wide spectrum. Patients may present with fever, dyspnea, chest pain, syncope, palpitation and critical arrhythmia [3,4]. If a first symptom of eosinophilic myocarditis is chest pain, it is hard to differentiate with acute coronary syndrome, and there are several case reports of eosinophilic myocarditis mimicking an acute coronary syndrome [5].

Electrocardiographic finding was varied as followings, ST segment elevation (45.8%), bundle branch blocks (6.3%), abnormal ST-T segment (45.8%) [3] and echocardiography shows a left ventricular systolic dysfunction, pericardial effusion (37.7%) and thrombus (18.0%) [3] in eosinophilic myocarditis. In our case, the patient complained of chest tightness, facial flushing, and generalized ache. Electrocardiography showed T wave inversion at lead II, III, aVF and V4~6, and cardiac marker was elevated (Troponin T 1.37ng/ml), as well as echocardiography indicated left atrium enlargement and left ventricular hypertrophy with increased echo density. His clinical manifestation did not mimic congestive heart failure, but acute coronary disease. Therefore, we performed coronary angiography, which showed no significant coronary stenosis. Because no other abnormalities were seen except hypereosinophilia and elevated cardiac markers, we diagnosed him with eosinophiliac myocarditis produced by hypereosinophilic syndrome initially.

In a retrospective study of 90 patients with eosinophilic myocarditis, the origin of idiopathic in 48% of patients, reactive to other disease in 28% of patients (periarteritis, asthma, a parasitic infection, Hodgkin’s disease, allergic reaction, or carcinoma), and in 24% of patients the underlying cause was an eosinophilic leukemia [6]. Eosinophilia can be aroused by hypereosinophilic syndrome, eosinophilic leukemia, carcinoma, lymphoma, drug reactions or parasites. Carcinoma is known as the unusual cause of eosinophilia. Hodgkin lymphoma often elicits severe eosinophilia, however, non-Hodgkin lymphoma and leukemia produce less marked eosinophilia [7]. Of solid tumor neoplasms, ovarian cancer is the most likely to provoke eosinophilia, though any other cancer can cause the condition [8]. There have been some case reports of eosinophilia-related tumors; pancreatic islet tumors, colon adenocarcinomas, non-small cell lung cancers, and metastatic renal cell carcinoma [9-12]. These articles suggest that solid tumors can lead to eosinophilia and may arouse eosinophilic myocarditis. However, there have been no articles or reports that pancreatic adenocarcinoma cause hypereosinophilia resulting in eosinophilic myocarditis.
The patient showed good response to steroid and eosinophilic count was decreased modestly but not to normal range. On second thought, hypereosinophilia by progressing pancreatic cancer was why normalization of eosinophilic count could not be achieved in spite of continuous steroid therapy. Therefore, we should have more carefully looked for potential causes for persistive hypereosinophilia in spite of steroid therapy.

In conclusion, we must seek a cause of eosinophil myocarditis more carefully and consider a pancreatic cancer as a cause of eosinophilic myocarditis.

References