A CASE OF ACUTE PERI-MYOCARDITIS ASSOCIATED WITH DELAYED APPEARANCE OF EOSINOPHILIA

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A CASE OF ACUTE PERI-MYOCARDITIS ASSOCIATED WITH DELAYED APPEARANCE OF EOSINOPHILIA

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INDEXING WORDS

acute peri-myocarditis; eosinophilia

SYNOPSIS

A case, 34 year old male, of acute peri-myocarditis with eosinophilia was reported. His chest X-ray revealed enlarged heart and pulmonary edema. ECG showed elevations of ST segment initially and inversions of T wave in the late stage of clinical course.

In the course eosinophils gradually increased and reached to peak on the 14th hospital day when inflammatory signs had been normalized, and the eosinophilia had continued for 2 months.

It was considered that the eosinophilia was not related to the first manifestation of the disease but it was the secondary reaction in this case.

INTRODUCTION

Löffler reported the cases of endocarditis parietalis fibroplastica with eosinophilia 1936, [2] and recently endomyocardial fibrosis and eosinophilic leukemia had been discussed in the same category as the Löffler's endocarditis. However, these diseases are relatively rare and only 11

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cases of endomyocardial diseases associated with eosinophilia have been reported in Japan.[1,5,6,7] And the majority of cases were diagnosed by autopsy after long term illness due to congestive heart failure, therefore initial clinical feature and natural history of the disease still remained in obscure.

Recently authors had experienced a case of acute peri-myocarditis with eosinophilia who showed relatively benigh clinical course despite transient severe congestive heart failure. This report describes the detailed early clinical course of the disease, especially on appearance and disappearance of eosinophilia coincident with the manifestation of inflammatory signs and cardiac abnormalities.

CASE REPORT

Patient is a thirty four year-old-male, manager of a printing office. His family history and past history were not contributory. There was no history of allergic diseases such as asthma and ulticaria.

Patient had been well until early December of 1978, when he began to complain of general malaise and headache. In the evening of December 11, precordial pain was abruptly felt and it continued to the next morning with gradual change to the precordial oppression. When he was admitted on December 12 1978, blood pressure was 90/60 mmHg and cardiac rate was 104/min, regular. Temperature was 37.2°C. Chest was clear to auscultation. Systolic friction rub and fourth sound in the apex were audible, however there was no cyanosis, edema, hepatomegaly or splenomegaly.

Laboratory findings: Urine and stool were normal. The hematocrit was 46 per cent and Hb was 15.7 g/dl. WBC was 12.900 per cu mm with 6 per cent eosinophils, 61 per cent neutrophils, 20 per cent lymphocytes and 3 per cent monocytes and no immature or pathologic leucocyte was found. Platelet count was normal.

Examination of serum enzymes revealed GOT:59 K.U., GPT:20 K.U., CPK:262 mu/ml. LDH:749 W.U./ml.

Serum sodium was 132.8 mEq/L, potassium:4.8 mEq/L, chloride: 95.5 mEq/L, BUN:13.9 mg/dl, fasting blood sugar was 116 mg/dl, gamma globulin 13.5 per cent.

C-reactive protein was 2(+), IgG:1640 mg/dl, IgA:328 mg/dl, IgM: 130 mg/dl. Repeated study of the serological test on mycoplasma, toxoplasma, and various viruses such as Polio-myelitis, Para-influenza,

Influenza, Echo, Coxsackie, Mumps, RS virus, Herpes, Cytomegalo, Measles and Varicella were all negative.

Chest X-ray on admission showed cardio-thoracic ratio of 50 per cent and essentially clear lung as shown in Figure 1.

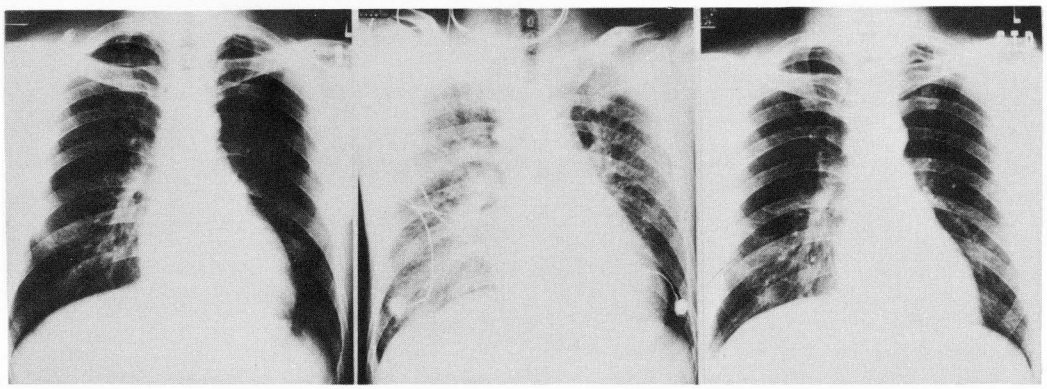

Fig. 1 Chest X-ray on admission of December 12 (left), December 15 (middle) 1978 and March 1 1979 (right). The middle figure shows increased CTR and pulmonary edema.

ECG revealed sinus tachycardia, right axis deviation, QS pattern in lead I, aVL, and elevation of ST segment in lead II, III, aVF, V4, 5, 6, as shown in Figure 2.

M-mode echocardiography of the 3rd hospital day displayed pericardial effusion, shoulder formation indicating B-B' step and increased A/E ratio (0.88) of the anterior mitral leaflet, and decreased ejection fraction of left ventricle (56 per cent). The echo pattern of the pulmonary valve suggested pulmonary hypertension by decreased E-F slope and disappearance of the "a" wave. Cavity of the left ventricle and left atrium, thickness of the ventricular septum and posterior wall were normal as shown in Figure 3.

Phonocardiogram showed fourth heart sound. Apexcardiogram indicated the increased "a" wave ratio (21 per cent) and decreased ET/PEP ratio (1.67).

Clinical course (Figure 4 and 5): In the afternoon of the first hospital day temperature elevated and the fever continued for next 5 days. C-reactive protein showed 7(+) on the 5th hospital day and returned to negative on the 10th hospital day. Serum enzymes which was elevated after the admission was normalized on the 14th hospital day. However, eosinophils gradually increased and reached to peak on

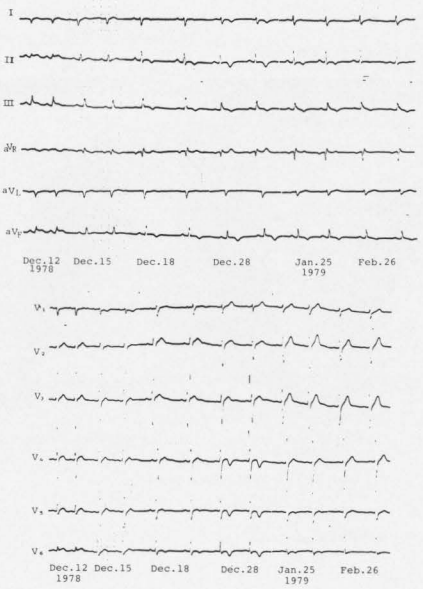

Fig. 2
Serial changes of ECG from December 12, 1978 to February 26, 1979.

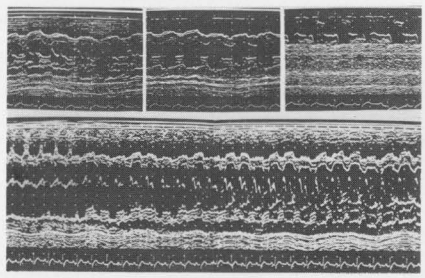

Fig. 3
M-mode echocardiogram on the 3rd hospital day. Upper left shows shoulder formation and dominant "a" wave of the anterior leaflet echo of the mitral valve. Upper middle shows decreased wall motion of the left ventricle and pericardial effusion. Upper right shows pulmonary valve echo suggesting pulmonary hypertension by decreased E-F slope and "a" wave. Lower figure shows M-mode scanning of the left ventricle from base (left) to the apex (right). Pericardial effusion is cleary demonstrated.

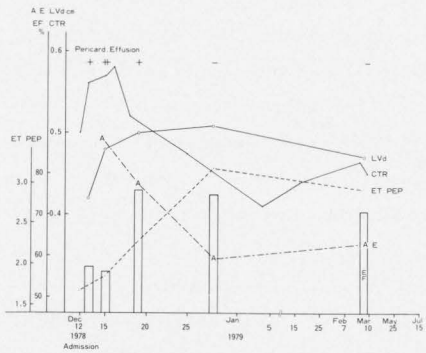

Fig. 4
Change of inflammatory sings, serum enzymes and eosinophilia throughout the clinical course. BTc: body temperature, BSR: blood sedimentation rate, WBC: white blood cell count, Eo: eosinophils (per cent in peripheral leucocytes), CRP: C-reactive protein, LDH: in wu, CPK: in mu.

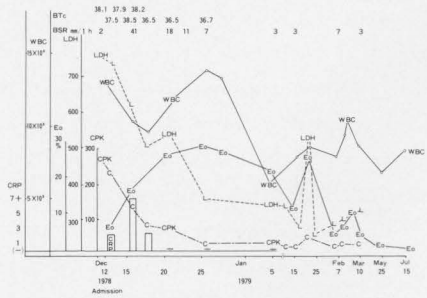

Fig. 5
Changes of cardio-thoracic ratio and the left ventricular function detected by echocardiography and apexcardiography in the course. A/E: ratio of A and E amplitude of anterior mitral leaflet echo, LVD: transverse dimension of left ventricle in diastole, EF: ejection fraction, ET/PET: ratio of ejection time and pre-ejection period.

the 14th hospital day when c-reactive protein and serum enzymes had been normalized, and the eosinophilia had continued for 2 months. Thus the phase of blood eosinophilia was delayed, if compared with inflammatory signs and release of cardiac enzymes.

On the 3rd hospital day, pulmonary edema was noted with enlarged cardiac silhouette with evidence of pericardial effusion.

CTR and ventricular function observed by mechanocardiography and echocardiography were aggravated just after admission and recovered within next one or two weeks.

ECG abnormalities such as QS pattern and elevated ST segment was gone on December 18 and maximal T wave inversions were noted on December 28 in lead I, II, III, aVF and V4, 5, 6.

DISCUSSION

On the endomyocardial diseases associated with eosinophilia Roberts discussed that tropical eosinophilia with ECG changes,[8] eosinophilic leukemia, Loffler's fibroplastic parietal endocarditis and endomyocardial fibrosis of Davies were same disease at different stage of development.[3,4]

Japan is not in the tropical area and there has been no report on the tropical eosinophilia with ECG changes in Japan. And reported cases of acute myocarditis with eosinophilia were also quite rare in Japan.

If this reported case locates in the left column of the Roberts's spectrum, observation of this reported case could reveal a clinical feature of the early stage of these disease entity.

In this report, the comparative observation during the clinical course of the inflammatory signs, serum enzymes, left ventricular function and eosinophilia from early to recovery phase were done. ECG revealed diffuse myocardial damage not only in the subendocardial but also transmural region of the anterior and inferior wall of the left ventricle. Acute pericarditis was also suspected by friction rub and pericardial effusion.

The eosinophilic phase had been delayed to those of inflammatory signs, releases of enzymes and suppression of left ventricular function. Delayed appearance of the eosinophilia in the course suggested that the eosinophilia was not related to the first manifestation of the inflammation directly but the secondary reactive phenomenon on the recovery stage of the disease.

Although this case showed benign prognosis after acute pancarditis,

it might be possible that chronic congestive heart failure associated with remarkable thickening of the endocardium would develope in the Roberts's spectrum if the eosinophilia continues.

SUMMARY

A case of acute peri-myocarditis associated with eosinophilia who showed benign prognosis after acute pulmonary edema in the early clinical course was reported.

During course of the disease the change of the left ventricular dysfunction observed by echocardiography was parallel with the inflammatory signs and increased serum enzymes, however appearance of the eosinophilia had been significantly delayed to these changes.

Relation between this case and Löffler's fibroplastic parietal endocarditis was also discussed.

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