Acute viral myocarditis, four cases in three weeks

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Summary

Four cases of acute viral myocarditis were diagnosed within three weeks. The clinical features, electrocardiography, cardiac enzymes and other laboratory investigations are described.

Key words: Viral fever, Myocarditis.

Introduction

Viral illness is the most frequent infectious disease. Inflammation of the myocardium causing myocarditis is quite common in a variety of viral infections, especially those due to Coxsackie A and B, adenoviruses and influenza viruses.[1] The incidence of specific types of viral myocarditis varies with the age of patients surveyed, geographical location, endemic or epidemic occurrence of viral illnesses. It is not possible to ascertain the true incidence of myocarditis as the majority of patients recover spontaneously. We describe here four cases of viral myocarditis occurring within three weeks in October 1988. All the four cases were admitted to an acute female medical ward in the General Hospital Kuala Lumpur.

Case One

A 35 year old Malay woman presented with four days' history of high fever associated with chills and rigors. She also complained of generalised bodyache, sore throat and tiredness. She vomited several times and had nonspecific chest pain one day before admission. All her four children also had fever and sore throat at the same time.

On examination the temperature was 39.5°C, pulse rate 100/min and blood pressure 120/90 mmHg. The eyes and throat were inflammed. The heart and respiratory systems were clinically normal. Electrocardiography (ECG) showed sinus tachycardia with widespread T wave inversion (V1–V6). (Figure 1).

Cardiac enzymes were markedly elevated. The creatinine phosphokinase (CPK) 1355 iu/l (normal 24–170 iu/L), lactate dehydrogenase (LDH) 1480 iu/L (normal 230–460 iu/L), aspartate transaminase (AST) 86 iu/L (up to 40 iu/L), alanine transaminase (ALT) 161 iu/L (up to 37 iu/L). Total white blood cells (TWBC) 3500 x 10^9/L, polymorphs (PMN) 57% lymphocytes (L) 40%. Atypical lymphocytes and target cells were seen. Platelets count was 9,000 x 10^9/L. Serology tests for viral antibodies were non-conclusive.

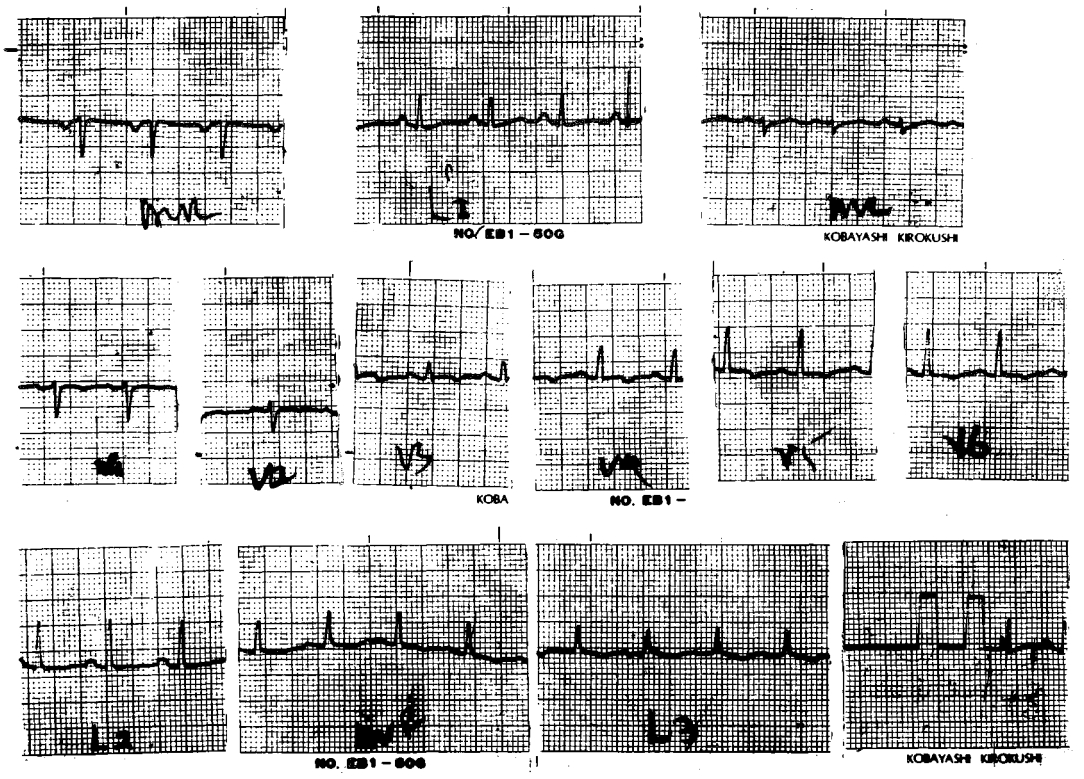

Figure 1: Electrocardiographic Tracing Case One

The patient was treated with paracetamol and thymol gargles. Her recovery was uneventful. The cardiac enzymes and TWBC returned to normal within four days but her ECG remained unchanged on the day of discharge. On review four weeks later, the ECG was normal.

Case Two

A 34 year old Malay woman presented with a history of high continuous fever associated with generalised bodyache and nonspecific chest pains. One day prior to admission she developed generalised body rashes, but there was no joint or body pains and no gum bleeding.

On examination, she was febrile 38°C. Her pulse was regularly irregular with a rate of 80/min. Her throat was injected. Generalised cutaneous macula-papular rashes were noted. Other systems were normal. ECG showed prolonged PR interval and ventricular ectopics (Figure 2). Cardiac enzymes were elevated; CPK 293 iu/L, LDH 1031 iu/L, ALT 298 iu/L and ALT 224 iu/L. Total white blood cell count was 3600 x 10^9/L with PMN 32%, L 60% and platelets 120,000 x 10^9/L.

Serology tests for measles, mumps, varicella, smallpox, Herpes simplex, cytomegalovirus, Influenza A, B and advenovirus were non-conclusive, but her rubella titer was 256 and IgM was positive. The cardiac enzymes and blood investigations returned to normal on discharge, but the ECG only returned to normal two weeks later.

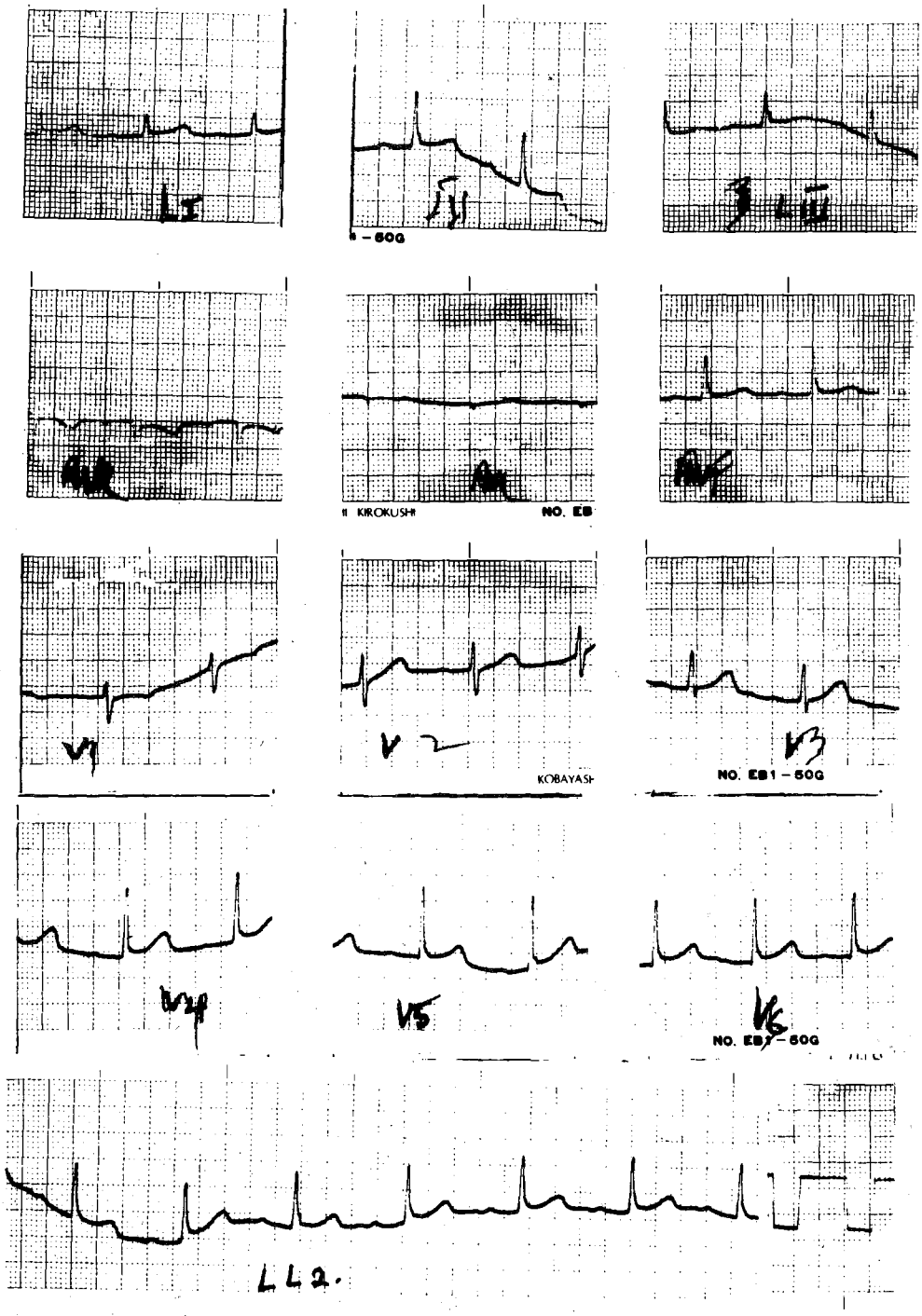

Figure 2: Electrocardiographic Tracing Case Two

Case Three

A 21 year old Chinese woman presented with three weeks' history of cough, coryza and intermittent low grade fever. Her fever apparently subsided five days prior to admission but

her cough persisted as a dry cough. Three days prior to admission, she had chest pains associated with palpitations and shortness of breath.

On examination she was afebrile, bracycardia with a heart rate of 60/min and irregular rhythm. Blood pressure was 110/80 mmHg. ECG showed sinus bradycardia with unifocal ventricular ectopic (1:5) (Figure 3). The cardiac enzymes were normal CPK 80 iu/L, LDH 337 iu/L, AST 11 iu/L ALT 30 iu/L. TWBC 10,000 x 10^9/L PMN 60%, lymphocytes 31%. Serology viral tests for Influenza A, B, Respiratory syncytial virus, parainfluenza I, II, III, mumps and Herpes were non-conclusive.

She was discharged home after six days hospitalization and her ECG showed occasional unifocal ventricular ectopic. Subsequent ECG's were normal when reviewed at outpatients.

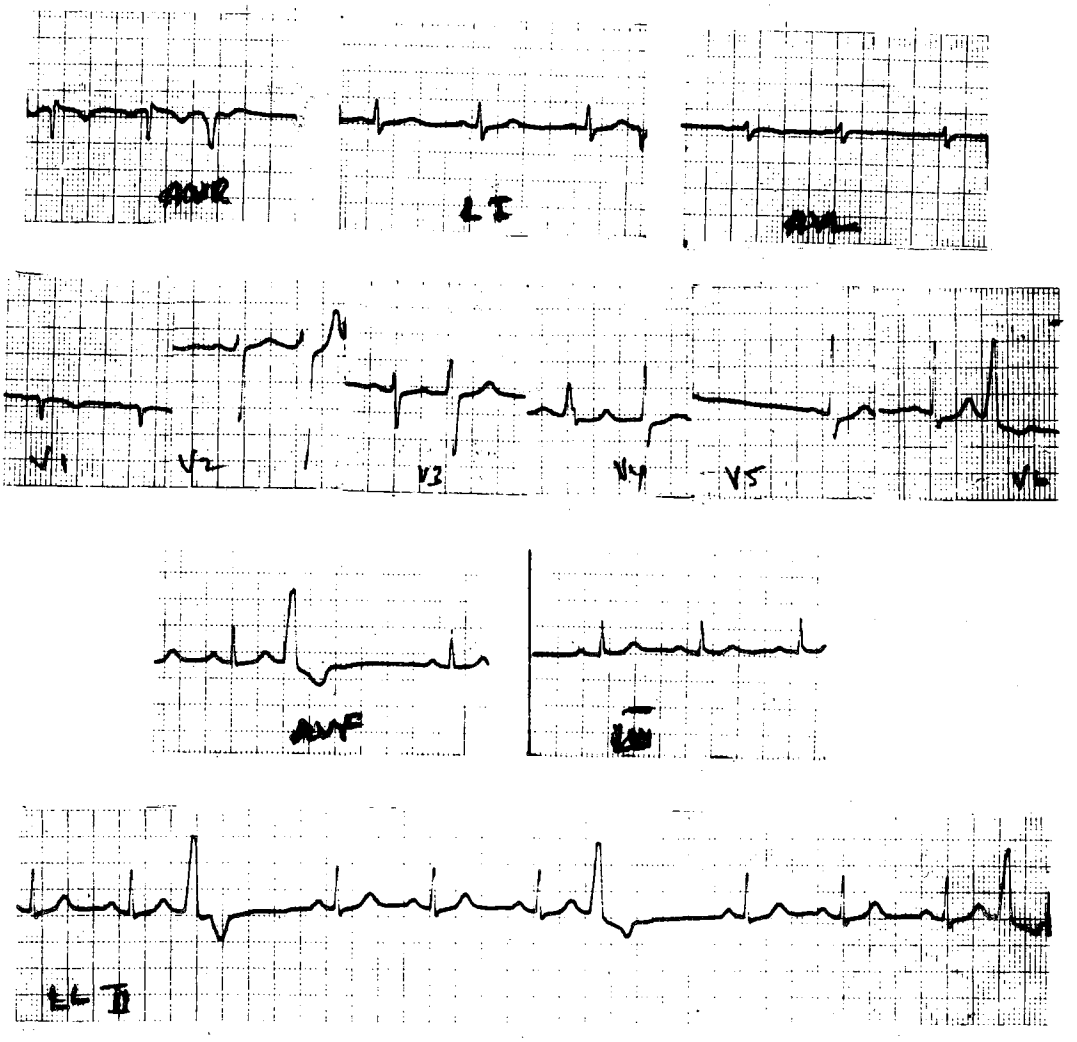

Figure 3: Electrocardiographic Tracing Case Three

Case Four

A 40 year old Indian woman presented with three days history of high fever, chills and rigors. She complained of severe central chest pain, pressing in nature, lasting more than ten minutes, associated with sweating and shortness of breath. She also had headaches and cough with moderate amount of whitish sputum.

On examination she was febrile 39°C with a pulse rate of 110/min. regular and blood pressure 110/70 mmHg. Her heart and respiratory systems were normal clinically. ECG showed gross T wave inversion at lateral chest leads V3–V6 (Figure 4).

Cardiac enzymes were CPK172 iu/L, LDH 1120 iu/L, AST 49 iu/L, ALT 25 iu/L. Full blood picture TWBC 8,000 x 10^9/L with lymphocytes 28%, atypical lymphocytes 9%. The platelets count was normal. Serology tests for viruses including influenza A, B, respiratory syncytial virus and mycoplasma were non conclusive. Her ECG and cardiac enzymes returned to normal on discharge.

The clinical features, ECG and laboratory results are summarized in Table 1.

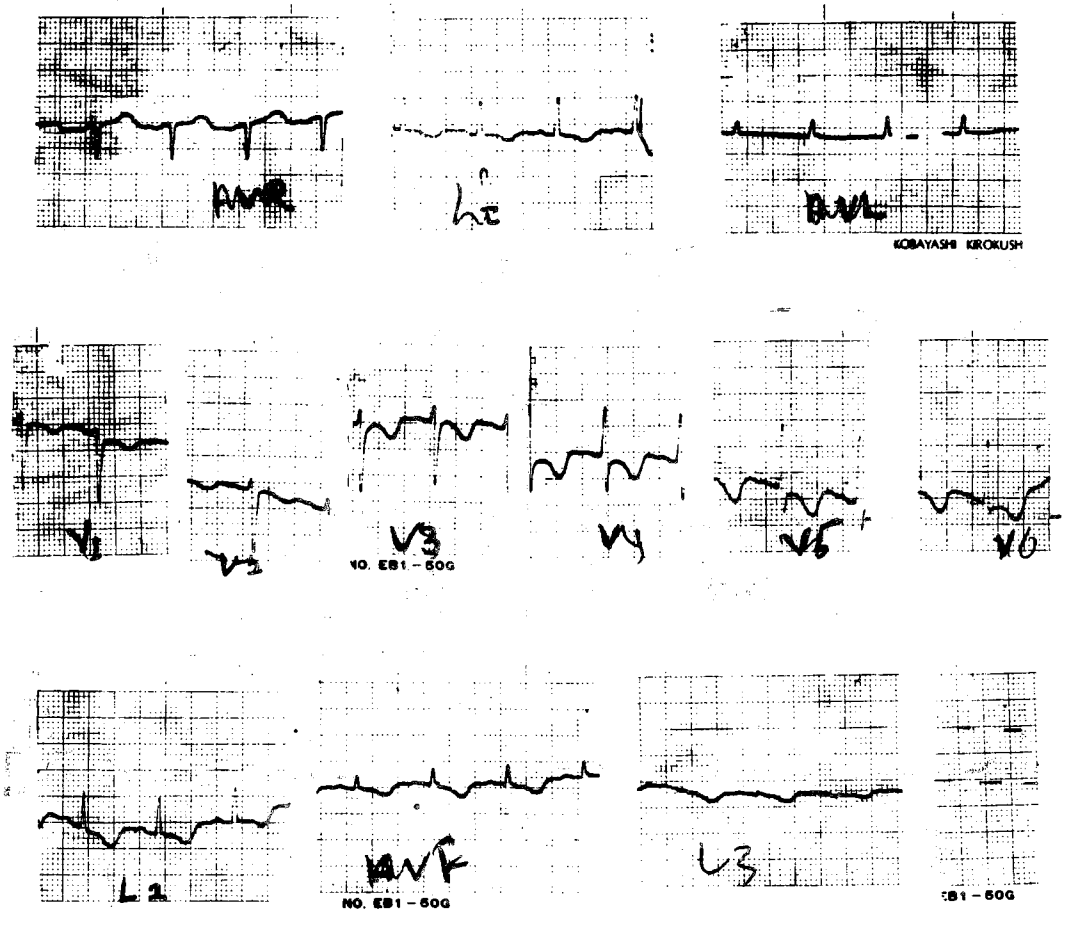

Figure 4: Electrocardiographic Tracing Case Four

Discussion

Myocarditis, an inflammation of the heart has been described with almost every known viral, bacterial, rickettsial and parasitic diseases. Although a cause effect, the relationship between viral infection remains inferrential. Two distinct clinical syndromes can be identified. During the early viral phase, the cardiac manifestations emerge while the symptoms of acute viral infection are still present. During the chronic phase, symptoms of viral infection may be remote or non-existence and the identification of active myocarditis is dependent on endomyocardial biopsy. Both clinical and experimental data suggest that viral myocarditis is biphasic. The initial phase is infective with myocytolysis, lymphocytic infiltration and hormonal immune response. The second phase is associated with a persistent antigen-antibody reaction between virus and myocardium.[2,3] In this report, all 4 cases presented during the acute phase although case 3 was probably in the recovery phase.

A survey of viral myocarditis and idiopathic myocarditis from 65 institutions involving 218 cases in Japan, showed diagnoses were made based on clinical and laboratory alone in 45%, endomyocardial biopsy in 24% and autopsy in 9%.[4] Cardiac symptoms and signs were common in addition to "common cold" symptoms. Electrocardiographic abnormalities, leukocytosis, accelerated erythrocyte sedimentation rate and increased cardiac enzymes were also very common in the acute phase of the disease. Serological tests for viruses performed in 80% of the case were positive in only 21%. There was no apparent correlation between serologic results and endomyocardial biopsy findings.[4] In these four cases, serological tests for viruses were performed but all of them were non-conclusive.

To elucidate the etiology of respiratory infection and pharyngitis associated myocarditis, a serological study was performed in Russia on 201 patients who were admitted with clinical diagnosis of myocarditis.[5] Coxsackie virus group B, influenza A and B, parainfluenza, adenovirus (as well as beta haemolytic streptococcus group A) were determined. Preceeding Coxsackie infection was established in 38.3% of patients, Influenza A and B in 27.5%, adenovirus in 3.6% and para influenza in 1.7%. Beta haemolytic streptococcus was the cause of myocarditis in only 4.9% of the patients. In all four cases in this report, no etiological agent was identified because the serological virus titres tested were non-conclusive.

Case reports of viral myocarditis with endomyocardial biopsy are still few in the literature although endomyocardial biopsy is becoming a standard procedure. In the majority of cases, diagnosis were made based on clinical features supported by non-invasive investigations. Immunological tests used to identify the causative organism are only of value when they are positive.

The ECG changes in myocarditis are variable as shown in the four cases. Non-specific T wave changes are common.[1-5] Abnormalities of rhythm are also found.[1-2] These changes all revert to normal. None of the four cases had cardiac failure or hypotension and all four recovered spontaneously.

Acknowledgement

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References

1. O'Connel JB Robinson JA, Gunner RM, Sconlon PJ. Clinical aspect of virus/immune myocarditis. Heart vessels 1985 supp I 102–6.

2. Lowny BS. Viruses and heart diseases: A problems in pathogenesis. Annual Clin. Lab Sci 1986 Sept–Oct 16(s) 358–6.

3. Richardson PJ. Clinical aspect of myocarditis: Heart vessels 1985 supp. I 97–100.

4. Kawamura K, Kitaura Y, Morita H, Deguchi A, Kotaka Ng. Viral and Idiopathic myocarditis in Japan. A Questionnaire Survey. Heart vessels 1985 supp. I 18:22.

5. Morikor, Luisyalova MA. Viral myocarditis (the etiology clinical diagnostic and treatment problems). Ter. Arkh. 1985 57(9) 49–56.