Acute Myocardial Infarction In Systemic Lupus Erythematosus

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The heart is commonly involved in systemic lupus erythematosus, the usual manifestations of this are LibmanSacks endocarditis, pericarditis, myocarditis, and sometimes vasculitis effecting small coronary vessels. Acute myocardial infarction occurring without other predisposing factors is rare in systemic lupus erythematosus. We report such a case in this paper.

CASE REPORT

The patient was a 37 years old nurse who developed severe idiopathic thrombocytopenic purpura in April 1958. This could not be controlled with corticosteroids but she improved following splenectomy and after this required no other treatment. The blood was examined for L.E. cells and was negative on six occasions.

During the next five years her hair became scanty. She developed acute nephritis in November 1969. L.E. cells were detected then for the first time and a diagnosis of systemic lupus erythematosus was made. She was treated with prednisolone and improved. The blood pressure returned to normal though she still had occasional slight proteinuria. She was maintained on prednisolone 5 mg on alternate days. In October she was admitted to hospital for fever, rigors and generalised bodyache for 5 days. She had a left-sided chest pain, palpitation and shortness of breath the day prior to her admission and was said to have fainted once at home on the day of admission.

She was observed to have a generalised fit as she was wheeled to the ward. She looked pale and had a temperature of 100°F. There was no ankle oedema. The pulse was regular 44/min and the blood pressure was 80/50 mmHg. The apex beat was not palpable. There was varying intensity of the first heart sound. No murmurs or pericardial friction rub was heard. The lungs were clear on auscultation. The rest of the physical examination did not reveal any further abnormality. The electrocardiogram was grossly abnormal. It showed complete heart block (Fig. 1) with atrial rate 125/min and ventricular rate 47/min. Peripheral blood picture showed leucocytosis and L.E. cells were found on three successive days. Urine did not contain protein. Serum lipids were normal: Cholesterol 164 mg%, triglycerides 72 mg%. Serum uric acid 3.7 mg%, serum proteins 7.7 g% albumin 3.7 g% globulin 3.8%. S.G.O.T. 690 Reitman-Frankel units/ml, E.S.R. 57 mm in 1 hour and blood urea 45 mg%. On the next day the S.G.O.T. was 520 units/ml and S.G.P.T. more than 400 units/ml.

She was given intravenous atropine, isoprenaline by infusion and hydrocortisone 200 mg four hourly. The patient reverted to sinus rhythm 14 hours later. The electrocardiogram (Fig. 2) confirmed this, and showed extreme left axis deviation, dominant R and ST elevation in the right praecordial leads, and ST depression and T wave inversion in the left praecordial leads. The tracing indicated severe myocardial ischaemia and possible infarction.

DISCUSSION

Myocardial infarction is not uncommon in other collagen diseases, for example polyarteritis nodosa (Holzniger et al 1962) and rheumatoid arthritis (Swezey 1967 and Sokoloff 1964), but it is rare in systemic lupus erythematosus.

Several authors who reviewed the cardiac manifestations of systemic lupus erythematosus have found no instance of occlusion of a major coronary artery (Shearn 1959, Brigden et al 1960, Hejtmancik et al 1964). On the other hand, there have been a few reports of myocardial infarction (Larson 1961, Anon 1962, Dubois 1966, Keat and Shore 1968, Bonfiglio 1972, E. Sande 1972).

In our case, the patient gave a history suggestive of myocardial infarction on the day before her admission to hospital and of two Adam-Stokes attacks, one at home and the other on admission. The diagnosis of infarction is supported by the electrocardiographic observations and the raised SGOT level. Complete heart block was present initially, but sinus rhythm returned within a short time. The widespread electrocardiographic changes suggest possible involvement of more than one major coronary artery.

Systemic lupus erythematosus is most probably the cause of myocardial infarction in this case as there was no recognized coronary risk factors. It is well known that age and hypertension are predisposing factors for myocardial infarction as in Ben-Asher's patient (1951) who had a blood pressure of 220/110 mmHg and sustained a heart attack at 62 years of age. The heart of his patient was enlarged and there was sclerosis of the vessels of the optic fundi. Our patient was only 37 years old. She was in her menstrual period of her life and was not on oral contraceptive. Her blood pressure was normal and there was no evidence of cardiomegaly.

In view of the absence of predisposing factors, S.L.E. must be considered as the direct cause of myocardial infarction in this case presumably due to vasculitis of the coronary artery in accordance with the experience of the pathological findings of others (Bonfiglio et al 1972, Hejtmancik et al 1964, Dubois 1966, Keat and Shore 1958).

The patient was thus treated with large doses of hydrocortisone 200 mg four hourly to reduce the inflammatory changes due to vasculitis. Isoprenaline infusion was given for her complete heart block to prevent further Adam-Stokes attack and correct her hypotension.

She made a rapid recovery and reverted to sinus rhythm within 14 hours of admission. Her renal function improved and the blood urea fell from 45 mg% to 33 mg% the next day as the urine output increased.

Systemic lupus erythematosus is a common disease with involvement of multiple system. Myocardial infarction has rarely been recorded till the last decade or so. The increasing number of cases being documented (Bonfiglio et al 1972. Sandoe 1972) could be due to setting up of new coronary units, aggressive approach to the management of cardiac arrhythmia, early admission of case with infarction, prolonged life span of cases with S.L.E. and better medical treatment.

SUMMARY

A 37 years old lady with systemic lupus erythematosus who developed acute myocardial infarction is described. There were no predisposing coronary risk factors apart from systemic lupus erythematosus. She developed complete heart block with Adam-Stokes attacks and was treated successfully with hydrocortisone and isoprenalise.

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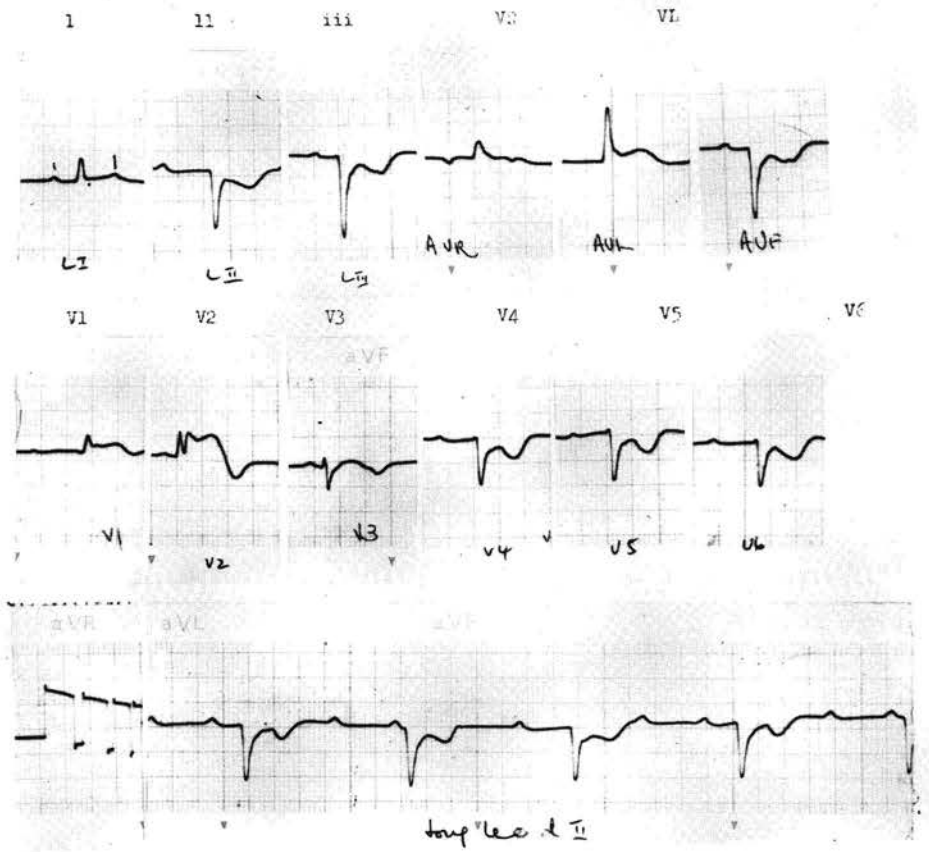

Figure 1. Electrocardiogram of patient on admission

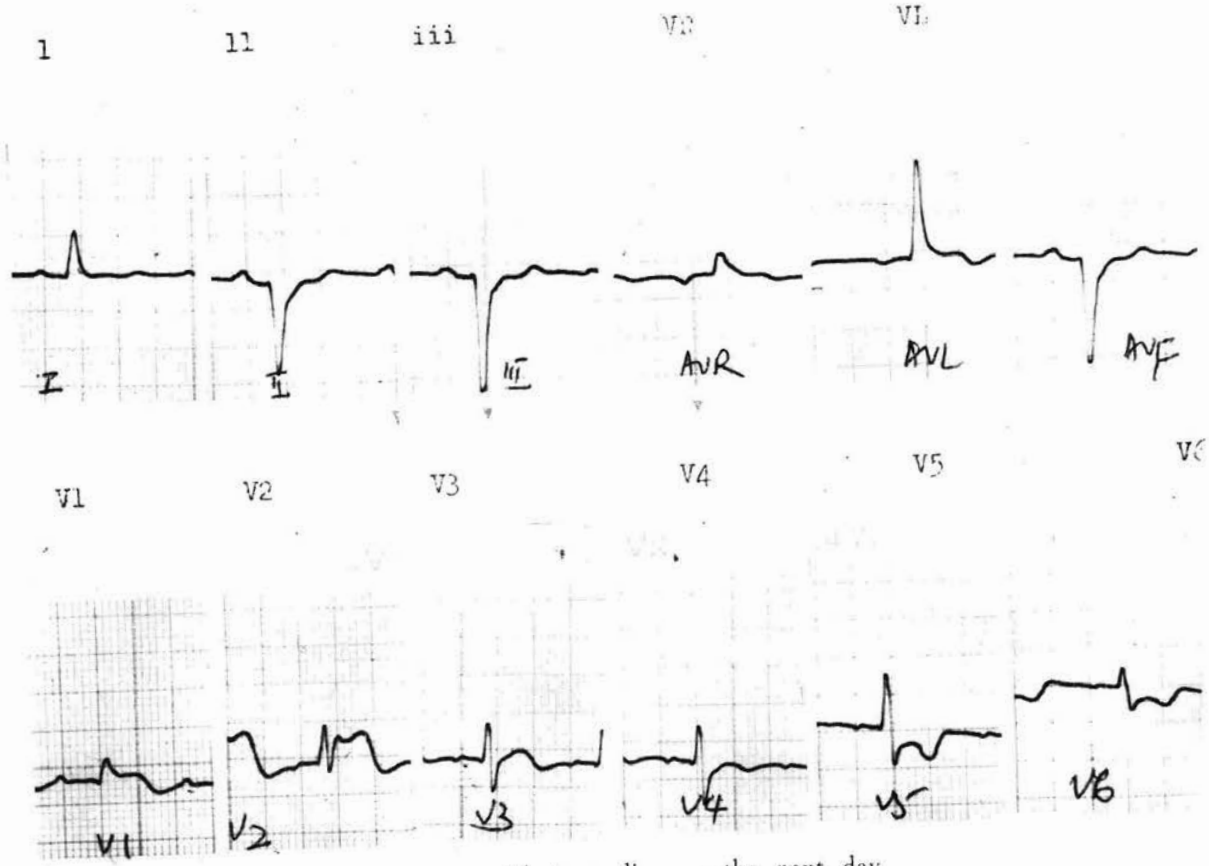

Figure 2. Electrocardiogram the next day