A NEAR-FATAL CASE OF COXSACKIE B_1 MYOCARDITIS (WITH PERICARDITIS) IN AN ADULT

A. L. AGRANAT, M.D. (DUBL.), F.R.C.P. (EDIN.), D.T.M. & H. (RAND), *Senior Physician, Johannesburg General Hospital*

Fatal cases of Coxsackie B virus myocarditis have been reported in newborn babies, and about 3 cases have been reported in children up to 12 years of age, but, thus far, no such cases have been reported in adults. Connolly[1] of the Virus Reference Laboratory, Queen's University, Belfast, in reporting a case of Coxsackie B_5 myocarditis in a 12-year-old boy, regarded this patient as the oldest one yet reported.

The case to be reported here occurred in a 39-year-old man (a senior laboratory technician) and was nearly fatal.

A review of the literature revealed the following points of interest:

1. *Neonatal deaths* from Coxsackie B myocarditis have been reported by Montgomery *et al.*,[2] Javett *et al.*,[3] and Suckling and Vogelpoel.[4] Kibrick[5] reported a case of Coxsackie A myocarditis.

2. *Coxsackie B myocarditis in children*. The following is quoted from Connolly,[1] who reported this condition in a 12-year-old boy: 'So far as is known the patient whose case is quoted above is the oldest child as yet recorded as having myocarditis during a Coxsackie B infection, and this is only the third recorded case of myocarditis in children infected with viruses of this group. The other cases were in a 5-year-old boy infected with Coxsackie B_2 virus (McLean, Croft, Prince and Heckmann, 1957) and in a $2\frac{1}{2}$-year-old boy during a Coxsackie B_5 infection (Varcasia and Castelli, 1957). Myocarditis has not been reported in adults during Coxsackie group B infections, though pericarditis has been described (Fletcher and Brennan, 1957; Weinstein, 1957.)'.

3. *Laboratory evidence of Coxsackie infection*. This is based on the isolation of the virus in the faeces, type-specific neutralizing antibodies,[6] and pathological findings.[7] Focal necrosis and cellular infiltration of the myocardium are the chief microscopic lesions described in Coxsackie myocarditis. The viral infection is widespread and many other organs may be involved. Histopathological findings have been described to account for the myocarditis (as above), pericarditis, pleurodynia (Bornholm disease), pancreatitis, aseptic meningitis and nephritis.[5,8]

CASE REPORT

R.G.R., a male aged 39 years, a senior laboratory technician at the South African Institute for Medical Research, was referred to me by Dr. L. Schrire on 21 November 1960.

His illness began while invigilating at an examination on 8 November. He felt feverish, sweated, developed a sore throat, and had generalized body pains. He went to bed, and during the next few days developed 'violent chest pains' anteriorly. Pain persisted in both costal regions and radiated to the shoulders on coughing and deep breathing. Symptomatic relief was obtained with analgesics and, although he returned to work, he felt ill. For about a week before I saw him he developed substernal pain radiating to the throat. This became worse in the recumbent position. There was no suggestion of chest pain before this illness and no preceding illnesses of note.

He looked ill, with a temperature of 100·4° F. The pulse rate was 88 and regular. Blood pressure was 105/80 mm.Hg. The heart was normal in size, both clinically and on screening. There was a very loud and extensive pericardial friction rub. The other systems and the urine were normal. An ECG taken on 21 November, about a fortnight after the onset of his illness, showed generalized flat or inverted T waves, no ST deviation, and no other features to suggest a myocardial infarction (Fig. 1). The clinical diagnosis was a pericarditis. Appropriate tests, including a request for isolation of Coxsackie virus in the faeces, were arranged.

He continued his treatment at home and reported progress by phone. On the morning of 29 November, 8 days after I first saw him, his wife phoned to say that he had been very ill during the night, with severe substernal pain and profuse sweating. Arrangements for his urgent admission to a nursing home were made and, while I was examining him, he collapsed. The pulse disappeared, he had a deathly colour, his eyes rolled back and he looked as if he were dying. An immediate intravenous injection of 'aramine' was given, and an intravenous glucose drip with 'levophed' and 'solucortef' was set up. Contrary to expectation (and to my considerable relief) a gradual improvement in his condition took place and the pulse again became palpable.

At this stage there was no pericardial friction rub, but a gallop rhythm was present. Later, a pleural friction rub was heard at the left base. The systolic blood pressure rose to 90 mm.Hg and was maintained there with the above treatment. An ECG at this time showed an inversion of the T waves in standard leads II and III, AVF, and V_3 - V_6 (Fig. 1).

At the stage of the critical collapse, the clinical condition was like that of a severe myocardial infarction. Laboratory investigations were carried out in an attempt to differentiate between a diagnosis of myocardial infarction and a viral myocarditis in an adult — a case without precedent.

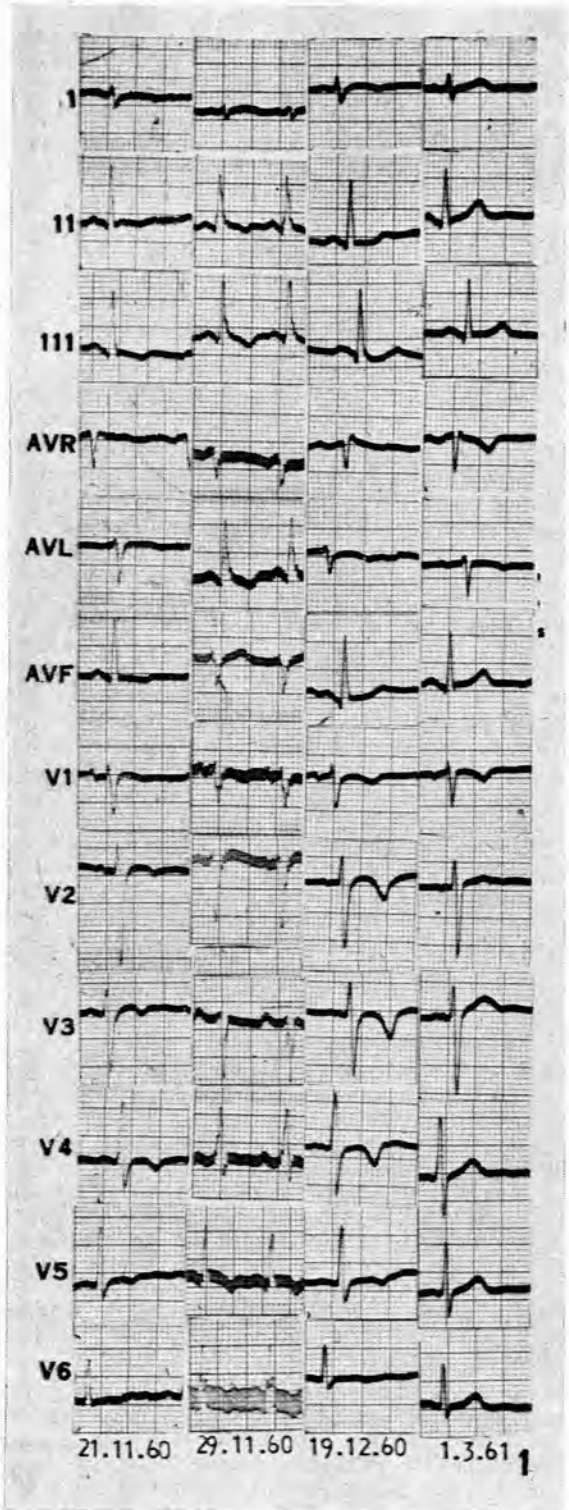

Fig. 1. Serial electrocardiograms.

LABORATORY INVESTIGATIONS (SELECTED)

Blood Counts

Date	Haemoglobin (G. per 100 ml.)	Leucocytes (per c.mm.)	Neutrophils %	Monocytes %	Lymphocytes %	Eosinophils %	Basophils %	Prothrombin index %	ESR (mm. in 1st hour)
22 November ..	14·3	9,600	47	15	31	5	2	71	23
29 November ..	15·2	22,600	81	10	9	—	—	66	—
5 December ..	12·3	8,800	52	11·5	27·5	8·5	0·5	71	45

Transaminase Tests

Date	SGOT (units)	SGPT (units)	Lactic dehydrogenase (units)
22 Nov.	30 (average normal 10) (range 2 - 35)	38 (average normal 10) (range 2 - 35)	200 (average normal 140) (range 100 - 210)
29 Nov.	155 per 100 ml. (range 35 - 100)	230 per 100 ml. (range 12 - 95)	300 at 10 a.m. 350 at 1 p.m. 540 at 3 p.m.
1 Dec.	—	—	470

Liver-function Tests

30 December: Battery tests were normal except for a cephalin cholesterol-flocculation test, +++; 66% cholinesterase; and a prompt direct van der Bergh reaction. Bilirubin direct was 2·3 mg. per 100 ml., and total bilirubin was 4·0 mg. per 100 ml. (Depressed liver function could have accounted for the reduced prothrombin index.)

Lipoprotein Partition (22 November)

	Lipoprotein %	Cholesterol %
Alpha$_1$ and alpha$_2$ lipoprotein ..	36	20
Beta lipoprotein	64	80

Total lipid 427 mg. per 100 ml., phospholipid 169 mg. per 100 ml., total cholesterol 130 mg. per 100 ml., free cholesterol 39 mg. per 100 ml., cholesterol esters 91 mg. per 100 ml., percentage esters to total 70%, cholesterol-phospholipid ratio 0·77, total fatty acids 241 mg. per 100 ml., triglycerides 61 mg. per 100 ml., and naked-eye appearance—clear.

Urine

30 November: protein + +, glucose + +, microscopic examination showed the presence of occasional polymorphonuclears per high-power field, and hyaline and granular casts.

6 December: a trace of protein present, glucose absent.

Blood Urea

52 mg. per 100 ml.

Isolation of Coxsackie Virus

Specimen of faeces submitted 25 November. Report from the Poliomyelitis Research Foundation, 17 December: 'A Coxsackie group B_1 virus was isolated from this specimen'. There was an extensive epidemic of group B type 1 infection in Johannesburg at the time and large numbers of patients during this epidemic suffered from signs and symptoms of Bornholm disease from Coxsackie group B type 1 (personal communication from Dr. J. H. S. Gear).

Electrocardiograms

About 3 months after the onset (6 February 1961) the ECG still showed T-wave inversion in the chest leads, although the standard leads had returned to normal.

1 March. The ECG returned to normal (Fig. 1).

DISCUSSION

On the basis of the following facts — a clinical course compatible with an infection, laboratory investigations indicating widespread pathology involving the liver, kidneys and pancreas, serial ECG changes not specific of

a myocardial infarction, a normal lipogram, the clinical progress of the case and the isolation of Coxsackie B_1 virus in the stool — the diagnosis of a Coxsackie myocarditis in an adult is suggested in this case. The nearly fatal issue indicates that the myocarditis may be as severe in the adult as it is in the infant. The incidence of pericarditis in this condition is not infrequent and, when this is suspected, the patient should be treated with strict bed rest and appropriate therapy. There is no specific antiviral therapy, and no evidence that steroids have any place in the treatment, unless cardiovascular collapse occurs. A complete recovery in due course may be anticipated.

An important point made by Connolly is that unexplained ECG changes in an adult may in some cases be caused by a viral myocarditis and not coronary-artery disease.

SUMMARY

A most severe case of Coxsackie myocarditis in an adult is reported. As far as is known, this is the first recorded case in an adult. The infection at one stage was nearly fatal.

My thanks are due to Dr. J. H. S. Gear, Director of the Poliomyelitis Research Foundation, Prof. J. H. Gear, and Dr. L. Schrire, as well as my Registrar, Dr. G. Clennar, and the resident staff at the Johannesburg General Hospital, for their invaluable help. My thanks are due also to Mr. M. Shewitz and the Photographic Unit, Department of Medicine, University of the Witwatersrand, for the reproduction of the electrocardiograms.

REFERENCES

1. Connolly, J. H. (1961): Brit Med., J., **1**, 877.
2. Montgomery, J., Gear, J., Prinsloo, F. R., Kahn, M. and Kirsch, Z. G. (1955): S. Afr. Med. J., **29**, 608.
3. Javett, S. N., Heymann, S., Mundel, B., Pepler, W. J., Lurie, H. T., Gear, J., Measroch, V. and Kirsch, Z. (1956): J. Pediat., **48**, 1.
4. Suckling, P. V. and Vogelpoel, L. (1958): Med. Proc., **4**, 372.
5. Kibrick, S. (1959): Med. Clin. N. Amer., **43**, 1291.
6. McLean, D. M., Croft, C. C., Prince, J. T. and Heckmann, E. E. (1957): Ohio St. Med. J., **53**, 907.
7. Simenhoff, M. L. and Uys, C. J. (1958): Med. Proc., **4**, 389.
8. Dowling, H. F., Lepper, M. H. and Jackson, G. G. (1960): Med. Clin. N. Amer., **44**, 5.
9. Weinstein, S. B. (1957): New Engl. J. Med., **257**, 265.