COXSACKIE B INFECTION IN A GENERAL MEDICAL UNIT

E. J. Bell, K.G. Irvine, A. J. S. Gardiner and J. C. Rodger
Regional Virus Laboratory, Ruchill Hospital, Glasgow and
Medical Unit, Monklands District General Hospital, Airdrie, Lanarkshire

Summary. From January 1979 until December 1980, neutralizing antibody titres to Coxsackie B1-6 viruses were measured in sera from 220 patients presenting to a general medical unit, the majority of whom had chest pain. No four-fold or greater rises in antibody titre were detected in these hospital patients. However, 49 per cent had titres of ≥256 to one or more Coxsackie B virus types compared with 10 per cent of the 950 persons studied during a 'normal population' survey in the West of Scotland. When a more rigorous threshold was adopted, titres ≥512 were found in 33 per cent and 4 per cent respectively. We conclude that Coxsackie B infections are not uncommon in the practice of a general medical unit and that they may account for more cases of both acute and subacute left chest pain than is usually realised.

Key words: Coxsackie B infection; chest pain; normal population survey.

Coxsackie B viruses are endemic in the United Kingdom. They can cause a variety of illnesses varying from mild respiratory infection to acute myopericarditis (1,2). The actual incidence of infection and the proportion of infections assuming clinical significance is unclear.

We report the results of Coxsackie B antibody studies made over a two year period in patients who presented to a general medical unit with symptoms suggestive of Coxsackie B infection in general and of myopericarditis in particular. We examine the significance of our findings by comparing them with the Coxsackie B antibody status of several normal populations in the West of Scotland.

Patients, subjects and methods

Patients. From January 1979 to December 1980, neutralising antibody titres to Coxsackie B1-6 viruses were measured in sera from 220 patients presenting to the medical unit of Monklands District General Hospital.

The patients (134 male, 86 female) were aged from 14 to 76 (mean 39.5) years. Of these, 110 were in-patients and 110 were out-patients.

Symptoms varied, but the majority of patients presented with chest pain (Table I). Of the in-patient group, 101 were admitted with acute chest pain which was considered to be possibly pericardial while 98 of the out-patients had non-acute atypical left chest pain. Though 11 patients were considered to have coexisting ischaemic heart disease, no patient had evidence of acute myocardial infarction.

Requests for reprints to: Dr Eleanor J. Bell, Regional Virus Laboratory, Ruchill Hospital, Glasgow G20 9NB.
We accept that our normal population study does not represent an ideal control group. However, the normal subjects and the patients studied were broadly similar in terms of age and sex. Although the normal populations were sampled from 1973–78, there were no epidemics of Coxsackie B infection to bias the results; the last recorded UK outbreak was in 1965 when Coxsackie B5 was the predominant virus. We have no reason to believe that the Coxsackie B antibody status of our normal population groups is other than representative of the population in the West of Scotland as a whole.

It seems reasonable to conclude that we have observed a real difference in Coxsackie B antibody status between our hospital and our normal populations. We further conclude that Coxsackie B infections are not uncommonly encountered in the practice of a general medical unit and that they may account for more cases of both acute and subacute left chest pain than is usually realised.

Others have investigated Coxsackie B antibodies in patients with acute myocardial infarction (7-9). This was not our intention; patients with acute myocardial infarction were excluded. While we have evidence of coexisting ischaemic heart disease in a small number of our patients and it cannot be firmly excluded in the remainder, it must be emphasised that only patients with pain which was thought to be atypical and possibly pericardial were included in the study.

We recognise the limitations of interpretation of high static or high single neutralising antibody titres in relation to onset of patient's illness. Our findings highlight the need for the more rapid and reliable specific Coxsackie B IgM detection systems which are currently being developed (10).

REFERENCES

4 Grist NR, Bell EJ. A six-year study of coxsackievirus B infection in heart disease. J Hyg (Camb) 1974; 73:165-72

5 Doerr HW. Coxsackie B virus neutralizing antibodies in myocarditis and pleurodynia. Dr Med Wschr 1973; 98:1396-1400


7 Woods JD, Nimmo MJ, Mackay-Scollay EM. Acute transmural myocardial infarction associated with active coxsackie B infection. Am Heart J 1975; 89:283-87

