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COXSACKIE B INFECTION IN A GENERAL MEDICAL UNIT

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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.

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Normal subjects. Between 1973 and 1978, one of us (EJB) examined the Coxsackie B antibody status of several 'normal' populations in the West of Scotland (Table II). These subjects had no known history to suggest a recent viral illness. Their ages, where given, ranged from 14 to 59 years and the male/female ratio was approximately equal.

Methods. All sera were tested for neutralising antibodies to the 6 group B coxsackie viruses by the modified micro-metabolic inhibition test (3). As in our previous studies (4) titres of ≥512 and of 256 were regarded as indicative and suggestive respectively of recent Coxsackie B infection.

Results

The results in the hospital patients are presented in Table I. No four-fold or greater rise in neutralising antibody titres was detected. Antibody titres of ≥256 were found in 49 per cent of all 220 patients studied. Using a more rigorous threshold, titres of ≥512 were found in 33 per cent of all patients and in 32 per cent of the in-patients and 35 per cent of the out-patients who presented with chest pain.

No seasonal variation was observed. Not unexpectedly, antibody to Coxsackie B4 virus was that most commonly encountered (5).

Results of antibody tests in the normal subjects are given in Table III. On average, titres of ≥256 were observed in 10 per cent and titres of ≥512 in only 4 per cent in these populations. These findings are similar to those recorded in a previous six year study of patients with non-cardiac disease (4).

Table I. Results of Coxsackie B neutralisation tests in 220 patients.

	No. patients tested	No. patients with antibody titre ≥512	256	Total ≥256
Chest pain—in-patients	101	32	13	45
Chest pain—out-patients	98	34	20	54
CNS/respiratory	13	2	3	5
Gastro-intestinal	2	0	0	0
Renal	1	0	0	0
Thyroiditis	1	1	0	1
Labyrinthitis	1	1	0	1
Erythema nodosum	1	1	0	1
Diabetes	1	1	0	1
P.U.O.	1	0	1	1
Total	220 (100%)	72 (32.7%)	37 (16.8%)	109 (49.5%)

Table II. Details of 'normal' populations studied.

Group	Year sampled	Number persons	Age range (yr)	Male: female
Rubella negative women	1973	95	17–40	0:1
Prisoners	1973–75	100	20–59	1:3
S.T.D.* clinics	1978	346	14–30	3:2
B.T.S.† donors	1977–78	409	18–40	NK

*Sexually transmitted disease
†Blood Transfusion Service

Table III. Results of Coxsackie B neutralisation tests in 950 'normal' subjects.

Group	Total exam.	Number with antibody titres ≥512	256	Total ≥256
Rubella negative women	95	8	10	18
Prisoners	100	8	12	20
S.T.D. clinics	346	5	10	15
B.T.S. donors	409	13	28	41
Total	950 (100%)	34 (3.6%)	60 (6.3%)	94 (9.9%)

Discussion

Although raised static Coxsackie B antibody titres are notoriously difficult to interpret, past studies (4,6) suggest that the higher the titre observed, the greater the probability of recent infection.

In the present study titres of 256 were detected approximately five times as often in the hospital population as in the normal population; with titres of ≥512 this frequency rose to eight times.

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).

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