Herpes zoster pericarditis

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SUMMARY A healthy 22-year-old man developed acute varicella pericarditis, characterised by an enanthem with diagnostic rising titres of varicella zoster antibodies but without the typical exanthem. This, the third reported case of varicella pericarditis, is the first to be documented without a typical varicella eruption.

Pericarditis and myocarditis are well-known complications of viral infections. These include Coxsackie B viruses, Coxsackie A viruses, Echoviruses, notably types 3, 6, and 19, influenza virus, Epstein-Barr virus, and polio virus (Bennett and Forbes, 1967; Bell and Grist, 1970; Grist, 1972; Haynes *et al.*, 1972; Shapiro *et al.*, 1973; Sainani *et al.*, 1975). Pericarditis may follow smallpox vaccination (Bengtsson *et al.*, 1966; Matthews and Griffiths, 1974). Though varicella myocarditis has been reported (Hackel, 1953; Moore *et al.*, 1969; Morales *et al.*, 1971), only 2 cases of varicella pericarditis have been reported (Mandelbaum and Turk, 1959; Helmly *et al.*, 1963). In both cases the pericarditis followed a typical varicella exanthem; this case is unique in that the typical rash was absent and the cardiac manifestations dominated the clinical picture.

Case report

A 22-year-old man, previously healthy, woke with discomfort in the upper chest, which, within a few hours, developed into severe central chest pain with dyspnoea and paraesthesiae in his fingers. On admission he was distressed, pale, and sweating, with a fever of 38.3°C. No other abnormality was noted; the heart sounds were normal and there was no pericardial friction rub.

Routine biochemical and haematological investigation was normal except for a white blood cell count of $14.9 \times 10^9/l$. Chest x-ray film was normal but the electrocardiogram showed prominent elevation of the ST segments in leads II, III, aVF, V5 and 6 (Fig.). An initial diagnosis of inferolateral myocardial infarction was made. On the second day

his temperature rose to 40°C and he developed severe painful ulcers in the left mandibular buccal sulcus associated with enlarged and tender cervical lymph glands. The white blood cell count was $11.9 \times 10^9/l$ (61% polymorphs and 35% lymphocytes). A pericardial friction rub was heard on the third day and his temperature became normal on the fifth day. Serum aspartate transaminase rose to 62 IU/l and the hydroxybutyric-dehydrogenase to 176 IU/l on the second day (normal values in the laboratory being 40 and 150 IU/l, respectively). The ST segment elevation of the electrocardiogram resolved by the third day, with subsequent T wave inversion in the same leads (Fig.).

Clinical recovery was uneventful. Virus serology showed a fivefold rise in herpes zoster complement-fixing antibodies from 4 to 128 and a similar, anamnestic, rise in herpes simplex antibodies (Table).

In view of the patient's youth, it was important to exclude coronary heart disease. As the electrocardiographic findings were compatible with either pericarditis or myocardial infarction, coronary arteriography was performed; this showed a normal coronary vascular tree without coronary artery disease and with normal left ventricular function. The electrocardiogram returned to normal after 5 months (Fig.), and the patient remains in good health 3 years later with no recurrence of pain, other cardiac symptoms, or electrocardiographic abnormalities.

Discussion

The association of varicella infection and myocarditis has been previously reported and has a high mortality probably because of systemic infection (Hackel, 1953; Moore *et al.*, 1969; Morales *et al.*, 1971). Varicella with pericarditis alone is rare. The only two previously reported patients (Mandelbaum and

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Table Titres of viral complement-fixing antibodies

	Days after onset of illness	
	1	16
Influenza A	4	<4
Influenza B	<4	<4
Adenovirus	8	<4
Psittacosis— <i>Lymphogranuloma venereum</i>	<4	<4
<i>R. burneti</i>	<4	<4
Respiratory syncytial virus	8	8
<i>N. pneumoniae</i>	4	4
<i>H. zoster</i>	<4	128
<i>H. simplex</i>	<4	128
Coxsackie B	8	16

Terk, 1959; Helmly *et al.*, 1963) were young men. As both had typical varicella eruptions, the clinical diagnosis of varicella was straightforward. This case had an enanthem and a history of chest pain only. The electrocardiographic changes initially suggested myocardial infarction but the elimination of coronary disease and the raised antibody titres permitted correct diagnosis.

Although this patient lacked the characteristic cutaneous eruption of herpes zoster, the mouth ulcers noted in the early phase of the illness were probably a zoster eruption in the territory of the buccal nerve, a branch of the third division of the trigeminal nerve. The rise in complement-fixing antibodies was diagnostic of herpes zoster infection according to the criteria of Grist (1972), with an anamnestic rise in herpes simplex antibodies.

A review of findings in infants with varicella has suggested that electrocardiographic abnormalities may be more common than is usually suspected (Paci *et al.*, 1960). It is possible that pericardial or myocardial involvement in the absence of clinical symptoms may occur frequently and has been postulated in several viral illnesses (Ahlborg *et al.*, 1966; Lewes *et al.*, 1974). This case suggests that a rising titre of varicella antibodies in the viral serological screening of patients with acute benign

pericarditis may increase the frequency with which varicella infection is diagnosed.

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Fig. Electrocardiographic changes showing ST segment elevation on day 1, maximum T wave inversion on day 17, and the return to normal after 5 months

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