

Pseudocardiomyopathy secondary to chronic incessant supraventricular tachycardia

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SUMMARY A teenager with left ventricular dysfunction complicating chronic incessant supraventricular tachycardia is described. He was unusual in his delayed presentation to hospital, despite having come to medical attention several years previously, and he showed an excellent response to flecainide.

Chronic atrial tachycardia has previously been regarded as a benign process without any adverse myocardial sequelae. Subsequent investigators have established a relationship between incessant tachycardia and significant left ventricular dysfunction that presents clinically as a cardiomyopathy and we report such a case.^{1,2}

Case report

A 6 year old boy was found by his family doctor to have a resting heart rate between 150–170 beats/minute. The general practitioner performed a 'rhythm strip' electrocardiogram at the time (January 1979) which confirmed the child's tachycardia (fig 1a), but as the child was asymptomatic no further action was taken.

Seven years later he was again found to have a tachycardia and was referred to the paediatric

outpatient department. On questioning in the outpatient clinic it was evident that he was unaware of his fast heart rate and had been participating in normal physical activities. His only symptom was of feeling faint if he stood or knelt for any length of time.

On examination he had a resting pulse rate of 148 beats/minute and a poor pulse volume. There was clinical evidence of left ventricular enlargement, but no murmurs or gallop rhythm.

Electrocardiography performed at this time (May 1986) showed ectopic atrial tachycardia with evidence of left ventricular hypertrophy (fig 1b). Chest radiography showed appreciable left ventricular hypertrophy (fig 2a) and 24 hour electrocardiograms on two occasions showed the presence of an almost incessant atrial tachycardia with the occasional short break into sinus rhythm (mainly during sleep). An echocardiogram confirmed the presence of a dilated left ventricle, with significantly impaired contractility. His full blood count, erythrocyte sedimentation rate, and thyroid function tests were normal.

Treatment was initiated with the antiarrhythmic drug flecainide on a dosage of 50 mg twice a day; this was subsequently increased to 100 mg twice a day. This drug was well tolerated and provided consistently acceptable serum drug concentrations.

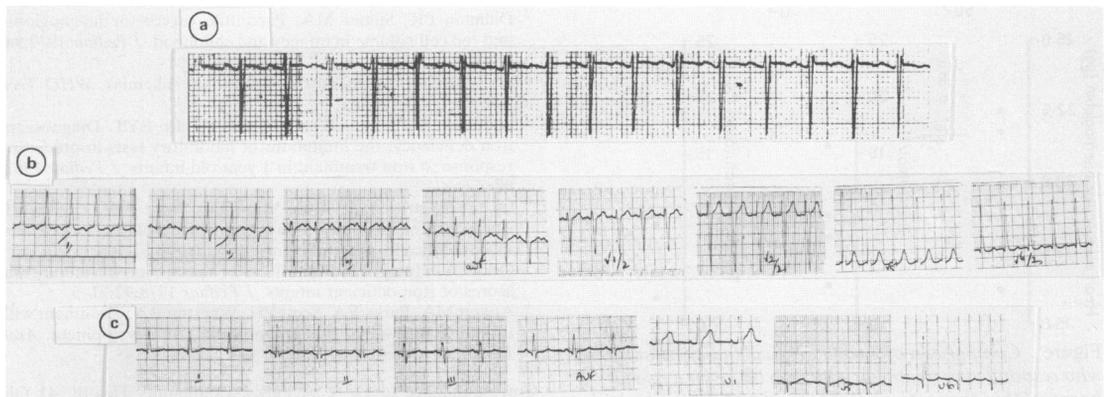


Fig 1 *Electrocardiography performed on the patient (a) in 1979, age 6; (b) in 1986, age 13; and (c) in 1987, age 14.*

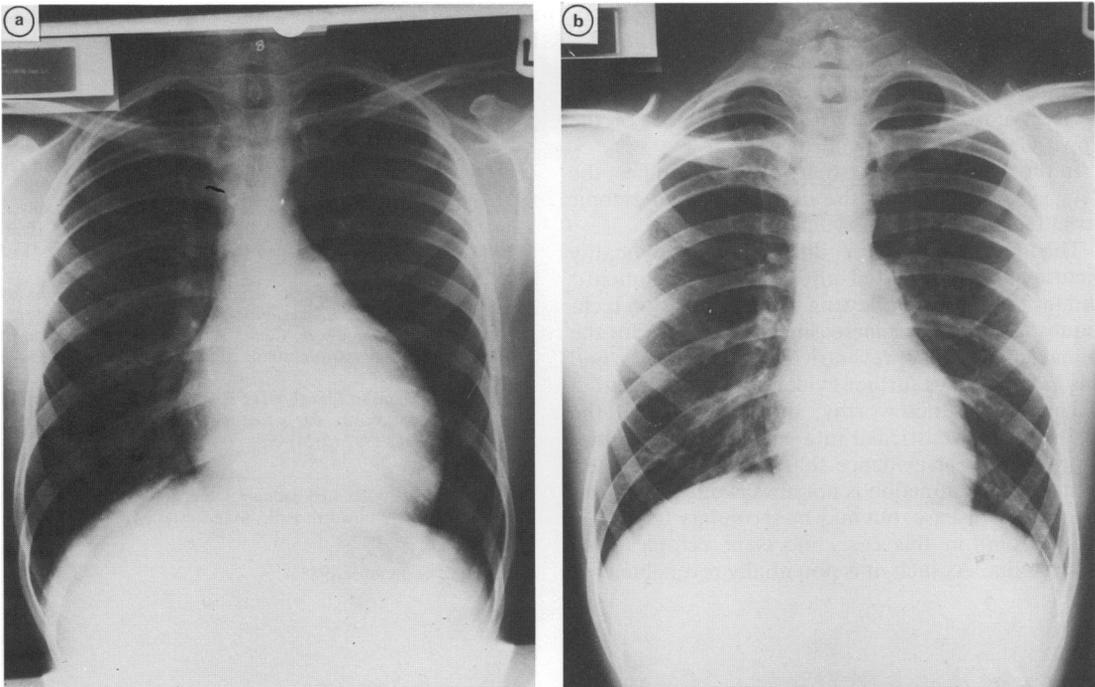


Fig 2 Chest radiographs taken on the patient (a) in May 1986, age 13 (before treatment) and (b) after 18 months treatment with flecainide.

Six months after commencing treatment he developed a fluctuating rash associated with exercise, anxiety, and heat. His full blood count and liver function tests were normal. A dermatological diagnosis of 'cholinergic urticaria' was made and treatment with an antihistamine was effective. The relationship between the rash and flecainide treatment was uncertain.

In the 18 months since drug treatment was commenced a considerable improvement in cardiac function has been noted: his electrocardiogram showed great improvement (fig 1c), a chest radiograph showed a return to normal (fig 2b), and a recent 24 hour electrocardiogram showed no evidence of tachycardia.

Discussion

The long history, normal exercise tolerance, and failure to show any underlying myocardial disease supports the hypothesis that this young man's poor left ventricular function was the result of his chronically uncontrolled tachycardia.

Electrophysiological studies have suggested that supraventricular tachycardias are the result of either

an ectopic focus in the atria or A-V junction triggering abnormal impulses (automaticity) or due to the re-entry of conducted impulses, which may occur at the sinoatrial node; intra-atrial level; the atrioventricular node, or through anomalous atrioventricular connections.

Of these mechanisms, chronic persistent supraventricular tachycardias appear to result from either: (1) an automatic ectopic focus in the atrium (characterised on electrocardiography by 'P waves', of similar size and shape during the tachycardia, but morphologically different from the 'P waves' of sinus rhythm, together with a P-R interval that is variable but is always within normal limits and a narrow 'QRS' complex). (2) An automatic junctional ectopic focus (characterised on electrocardiography by 'P waves' frequently negative in leads II, III and aVF, and often following the 'QRS' complex). (3) A-V node re-entry, either of the 'slow-fast' form ('P waves', if seen, are inverted in leads II, III and aVF, the P-R interval is prolonged and the R-P interval is shortened) or of the 'fast-slow' form (again typified by a superior P wave axis but with a shortened P-R interval and a prolonged R-P interval). (4) A concealed lateral anomalous A-V

pathway (with an apparently normal electrocardiogram when the patient is in sinus rhythm, differentiating this accessory pathway from the manifest anomalous pathway of the Wolff-Parkinson-White syndrome or the A-V node bypass of the Lown-Ganong-Levine syndrome).

In the absence of electrophysiological studies, the data available support the diagnosis of an ectopic atrial tachycardia in this young man.

The regression of a dilated cardiomyopathy picture is well reported after surgical treatment,² and more recently, following catheter ablation techniques.³ Flecainide is increasingly being used for the management of supraventricular tachycardias and this case provides further evidence of its efficacy.^{4 5}

This case is noteworthy, firstly because of the relatively low ventricular rate, and secondly because it provides clear evidence that the presence of left ventricular dysfunction is not always due to primary myocardial disease, but may be secondary to chronic tachycardia—in this case, incessant ectopic atrial tachycardia. As such, it is potentially reversible with

specific antiarrhythmic drugs such as flecainide—hence the term ‘pseudocardiomyopathy’.

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Accepted 21 September 1988

Chronic neonatal Coxsackie myocarditis

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SUMMARY A baby girl, with a birth weight of 2540 g, developed myocarditis with gross, permanent myocardial calcification. The clinical course was progressively downhill, and she died at 14 months of age. Neutralisation antibody to Coxsackie virus group B1 rose from 1:80 after delivery to 1:1280 at age 5 months. The protracted course of this infant's disease represents a new clinical form of fatal neonatal Coxsackie virus group B infection.

The clinical spectrum of neonatal infection with Coxsackie virus group B ranges from a mild, non-specific febrile illness to its severe, and often fatal, manifestation as myocarditis.^{1 2}

We report a newborn girl with proved Coxsackie virus group B1 disease who had an unusually protracted course with chronic cardiac dysfunction, permanent myocardial calcification, and severe failure to thrive.

Case report

A girl, weighing 2540 g, was delivered at 36 weeks' gestation by caesarean section because of maternal fever, accompanied by dry cough and diarrhoea, and hypertension. The mother did not have a rash. Transient respiratory distress in the child was followed by the appearance of fever on the 4th day of life. A systolic heart murmur and a third heart sound became audible; subsequently there were numerous runs of supraventricular tachycardia and development of congestive cardiac failure. The cerebrospinal fluid contained leucocytes at a concentration of $200 \times 10^6/l$, the protein concentration was 1.4 g/l, and glucose 3.1 mmol/l. Serum glucose concentration was 4.1 mmol/l. Cultures of blood, urine, and cerebrospinal fluid were negative for bacterial growth. Despite ventilatory support and treatment with digoxin, diuretics, corticosteroids, and verapamil, the clinical condition did not improve and the arrhythmia persisted for many weeks.