9 mm/h. Ureterolysis and ureteric reimplantation was planned, but had to be delayed when the patient had a major myocardial infarction.

COMMENT

Asbestos bodies have also been reported in various retroperitoneal organs, including the kidney and adrenal glands,³ and two previous case studies have reported a link between asbestos exposure and retroperitoneal fibrosis.^{4,5} Uliba *et al.*⁶ found that patients with significant asbestos exposure (>10 fibre-years) were at nine-fold excess risk with a mean latency period from first exposure of 30–33 years.⁶ Our patient had worked as a carpenter and ceramic tiler all his life, and the likely cause of his disease was occupational exposure to asbestos.

Isolated retroperitoneal fibrosis within the true pelvis is very rare, and involvement of single organs presents special diagnostic challenges. Symptoms can range from dyspareunia, when the cervix is affected,² to a change in bowel habit with rectal involvement.¹ The features can resemble those of malignant disease both clinically and radiologically. We have not found a previous case in which retroperitoneal fibrosis gave the appearance of prostate cancer.

Once a malignancy is excluded, the aim of treatment is to restore normal renal function by relief of obstructive uropathy. Primary treatment with steroid therapy and ureteric stenting has been shown to be effective in most patients.⁷ Failure of medical therapy necessitates open ureterolysis.

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Thirty years on quinidine for paroxysmal ventricular tachycardia

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Quinidine is no longer widely used for the termination and prevention of arrhythmias because of concern about cardiac and systemic side-effects. This concern was not shared by the patient reported here, who took the drug continuously for 30 years.

CASE HISTORY

The patient had experienced missed beats since the age of 25, but at age 51 he developed episodes of paroxysmal ventricular tachycardia. The electrocardiogram showed that the arrhythmia originated in the right ventricular outflow tract, QT interval 0.43 ms (Figure 1). Physical examination

revealed a late systolic click at the apex. Chest X-ray and echocardiogram were normal. Procaine amide and practolol had failed to give control and I started him on quinidine sulphate, in the form of Kinidin Durules (Astra), 2 g per day. Blood levels were satisfactory at 2.0–2.3 mg/dL; the QT interval increased to 0.47 ms. He continued to have extrasystoles and short runs of tachycardia but usually these were well tolerated. Addition of propranolol and then mexiletine did not give improvement and the patient felt better on quinidine alone. He continued to live a very active life as a horticulturist. At age 78 amiodarone was substituted but he felt less well on this and asked for the quinidine to be reinstated. The following year he developed biventricular failure, for which he was prescribed celiprolol, losartan and furosemide; he insisted on continuing the quinidine. At age 83 he was experiencing infrequent episodes of ventricular extrasystoles and tachycardia, and he died after an operation for colon cancer.

COMMENT

Quinidine was promoted by Thomas Lewis for the termination and prevention of atrial fibrillation,¹ after Karel Wenckebach had shown in 1914 that the related drug quinine could halt paroxysms of this arrhythmia.² In 1951 Maurice Sokolow reported conversion to normal rhythm with no important side-effects in 80% of 111 personal cases of atrial fibrillation and flutter, using blood level control.

Figure 1 Pre-treatment electrocardiogram

One patient with a high blood concentration got ventricular fibrillation.³ Later he showed that quinidine was successful in preventing recurrences of those arrhythmias.

By 1927 Samuel Levine in Boston was using quinidine with success to terminate episodes of ventricular tachycardia complicating acute myocardial infarction, and in 1950 he reported the treatment of 107 patients with paroxysmal ventricular tachycardia, not all due to ischaemic heart disease.⁴ Later studies by others showed that quinidine could prevent paroxysms of ventricular tachycardia in only about 50% of patients.⁵ However, in 1990 a survey was done of 252 American cardiologists asking which drugs they preferred for malignant ventricular arrhythmias. Quinidine, procainamide and mexiletine were ranked first for initiation of treatment, with a very low use of amiodarone.⁶ Ventricular tachycardia occurring with myocardial infarction may still be treated with a drug; namely lidocaine, in preference to electrical cardioversion.

My patient had no significant heart disease when he was first seen and a study of 123 patients with malignant ventricular arrhythmias showed that 13.8% had normal

findings on investigation.⁷ Coronary heart disease is present in about 70% of cases whilst other causes include the prolonged QT syndromes either congenital or acquired. Although the QT interval was slightly above normal in the present patient, one could not make a case for him having long-QT syndrome. The commonest cause of the acquired type is the use of anti-arrhythmic drugs such as quinidine, disopyramide and flecainide.

There are very few reports as to the length of duration of treatment with quinidine but Paul White mentioned a patient successfully treated for 20 years.⁸ I estimate that my patient ingested about 22 kg of quinidine during his 30 years of treatment.

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