rhythm disorders no other drugs known to prolong the Q-T interval and induce torsades de pointes—for example, diuretics or antiarrhythmics—had been given. Antibiotic treatment was changed to ciprofloxacin. No further arrhythmia occurred and the patient was discharged three weeks later.

Comment

Ventricular arrhythmia associated with prolongation of the Q-T_c interval is a well known side effect of several drugs. In our patient the close temporal relation of prolongation of the Q-T_c interval and infusion of erythromycin with the maximum interval occurring at the end of the infusion and a gradual return to normal over the next few hours strongly suggested a direct dependence of the Q-T_c interval on the plasma concentrations of erythromycin. That we recorded this phenomenon during two separate drug infusions lends support to this suggestion. A possible association of erythromycin with ventricular arrhythmias has been reported.^{1,2} Prolongation of the Q-T interval was the common feature in all cases. Nevertheless, given the widespread use of erythromycin, the low rate of drug associated arrhythmias recognised is remarkable.

In our patient ventricular extrasystoles and torsades de pointes occurred only at the time when peak plasma concentrations would be expected and when the longest Q-T_c interval was seen. How erythromycin alters cardiac repolarisation is not clear, but a recent electrophysiological study has shown that erythromycin exerts a concentration dependent at quinidine-like effect.³ Aside from the mode of administration leading to different peak plasma concentrations other factors must be considered. In patients with pre-existing

prolongation of the Q-T interval—for example, in idiopathic long Q-T syndrome⁴—erythromycin may worsen the repolarisation abnormality and induce arrhythmias. As in other published cases² our patient had a normal Q-T interval before being given the drug. Other factors such as alterations of erythromycin pharmacokinetics resulting in different individual susceptibility may be important. Both structural damage of liver tissue and cotreatment with other drugs competing for the same hepatic cytochrome (P450III_A4) may impair hepatic drug clearance and lead to critically raised plasma concentrations of erythromycin.⁵ The hepatic dysfunction present in our patient may have been a main factor contributing to the erythromycin toxicity.

To minimise the danger of toxic plasma concentrations rapid injections of erythromycin should be avoided as recommended by the manufacturers. In patients at risk close Q-T_c and rhythm monitoring may be advisable. In a patient with impaired hepatic drug metabolism or pre-existing cardiac disease a smaller dose or an alternative antibiotic should be considered.

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Prolonged recovery of renal function after prostatectomy for prostatic outflow obstruction

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High pressure chronic retention from prostatic outflow obstruction presents with few urological symptoms, but the presence of late onset enuresis and a painless, palpable bladder, with hypertension or peripheral oedema, should suggest the diagnosis.¹ Progressive renal failure is a feature of this condition, and prostatectomy is essential to relieve obstruction of the lower urinary tract. Prospective tests of renal function done up to one year after prostatectomy show that appreciable improvement may occur.² Further recovery may, however, occur some years later and these observations form the basis of my report.

Patients, methods, and results

Three men, aged 48, 51, and 63, with typical features of high pressure chronic retention associated with prostatic outflow obstruction, were studied prospectively for between three and nine years after prostatectomy.¹ They formed part of a larger series of six patients, one of whom had died of an unrelated cause and two had not yet been followed up for three years.² All six patients showed a consistent improvement in renal function over a period of 12 months, with their mean glomerular filtration rate rising from 27 ml/min/1.73 m² to 56 ml/min/1.73 m² and their mean effective renal plasma flow rising from 88 ml/

min/1.73 m² to 221 ml/min/1.73 m² (p<0.02 for both values). Their mean filtration fraction was 0.30 preoperatively and 0.25 one year after prostatectomy.

All three patients were hypertensive at presentation, but two became permanently normotensive within days of prostatectomy. The third patient experienced resurgence of severe hypertension two months after prostatectomy requiring control with multiple anti-hypertensive drugs.

The figure shows the effective renal plasma flow and glomerular filtration rate for each patient before and after prostatectomy.³

Intravenous urography, renal tract ultrasonography, and gammacamera renography were performed before prostatectomy and at arbitrary intervals after for several years. Initial urography showed gross bilateral hydronephrosis and hydroureter in all three patients, which changed to normal appearances some years after prostatectomy. These findings were confirmed by ultrasonography.

The renographic findings were not diagnostic of obstructive nephropathy before prostatectomy, but they did show bilateral impairment of renal function with poor uptake of isotope. Nevertheless, when renal function had recovered a normal renogram pattern was seen.

Comment

Prognosis in high pressure chronic retention depends on the degree of renal damage at the time of presentation. No test can gauge whether irreversible pathological changes have occurred. Even when renal function is substantially impaired initially, the amount of recovery possible may be established only by surgical relief of the obstruction or by catheterisation

of the bladder, and then by sequential renal function tests. The preoperative reduction in glomerular filtration rate and effective renal plasma flow found in our

Glomerular filtration rates, effective renal plasma flows, and filtration fractions before and after prostatectomy for three patients studied

patients suggested powerful renal vasoconstriction, possibly due to neurogenic or humoral influences. The stimulus to this might be a raised renal pelvic pressure, although this is, however, appreciably lower than the bladder pressure measured concurrently.⁴

Surgical relief of chronic retention was accompanied by a prompt and sustained rise in glomerular filtration rate and effective renal plasma flow that persisted for several years. Possibly this prolonged recovery of renal function might have been due to amelioration of vasoconstrictive changes, with a gradual process of regeneration of the residual nephron population due to functional adaptation. This latent improvement of renal function was not inhibited by recrudescence of hypotension in one patient, and this suggests that hypertension is not an adverse factor for renal function in these circumstances. Renal damage is probably related to the duration and degree of the preceding high pressure chronic retention, but this might be difficult to prove as the high pressure chronic retention syndrome may be clinically silent, or may present with secondary effects such as hypertension or cardiac failure. Unfortunately, the duration of secondary hypertension, which is probably a late manifestation, gives no indication of how long high pressure chronic retention has been present. Whether the renal damage is reversible depends on the timing of surgical intervention. It is therefore imperative to avoid long delays in referral to hospital and to circumvent lengthy waiting lists to expedite urological assessment and early prostatectomy.⁵

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Anaphylaxis induced by peanuts

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We report four cases of severe anaphylactic reaction to peanuts.

Case reports

Case 1—A 23 year old man with asthma who had had allergic reactions to nuts from the age of 3 became unwell while eating a Chinese meal that included satay (ground peanut) sauce. While returning to his hotel to get adrenaline that had been prescribed for self administration he became increasingly short of breath. About 30-40 minutes after starting the meal he collapsed and vomited and had a cardiorespiratory arrest. Cardiopulmonary resuscitation was started, and an ambulance crew intubated him and recorded ventricular fibrillation. On arrival at this hospital he was asystolic and resuscitation was unsuccessful. Post-mortem examination showed features of acute asthma and some aspiration of the contents of the stomach.

Case 2—A 20 year old man with a history of allergic reactions to nuts collapsed after inadvertently eating

part of a meal containing peanuts. He had deliberately induced vomiting but had become increasingly short of breath and cyanosed. He subsequently had a cardiorespiratory arrest. Despite rapid transfer to this hospital and intensive resuscitation he died. Post-mortem examination showed features consistent with death due to acute asthma.

Case 3—A 21 year old man with a history of asthma and allergy to nuts presented to the casualty department of this hospital severely cyanosed and with loud wheezing. He was given oxygen and nebulised salbutamol but within minutes had a generalised convulsion followed by a cardiorespiratory arrest (with asystole). He was intubated, and cardiac massage was started. He was given intravenous adrenaline, which resulted in the return of sinus rhythm, and intravenous aminophylline and hydrocortisone. Very high airway inflation pressures were noted. He recovered rapidly and was extubated within four hours. The next day his condition was normal. A more detailed history was obtained: he had developed an identical reaction two years previously after inadvertently eating walnuts; had had allergic reactions to nuts from the age of 6; and felt panicky and breathless within two to 30 minutes of eating small quantities of several nuts. He deliberately avoided these nuts, and if he accidentally ate even minute quantities he would induce vomiting. On the day that he presented to us he had bought packets of dried food dressing, thinking that they did not contain