Letters to the Editor

Embolic stroke following cardioversion of atrial fibrillation to sinus rhythm with oral amiodarone therapy

Sir,

Risk of stroke associated with atrial fibrillation is well recognized.^{1–3} Patients not anticoagulated who undergo elective cardioversion are at risk of developing embolic complications.⁴ Amiodarone is frequently used for treatment of atrial fibrillation and it is known to cause chemical cardioversion. We report a case of disabling stroke after amiodarone-induced cardioversion of atrial fibrillation.

A 66 year old woman with a history of paroxysmal atrial fibrillation was treated with amiodarone as she had symptomatic atrial fibrillation of 5 weeks duration. She was not on prophylactic antiarrhythmic drugs and her previous attacks were infrequent and short lasting. Although she was hypertensive she had not suffered from ischaemic heart disease or transient ischaemic attacks. She was on propranolol 160 mg daily for hypertension.

Clinical examination revealed atrial fibrillation at 120/min and her blood pressure was 120/80 mmHg. There was no evidence of cardiomegaly, valvular heart disease or carotid bruits. Her chest X-ray was normal and the electrocardiogram confirmed atrial fibrillation. Eleven days after commencing amiodarone the patient felt her heart rhythm change as she no longer experienced palpitations or difficulty in breathing. Approximately 4 hours later she developed sudden onset numbness and weakness of the right arm and leg. She had evidence of hemiparesis and extensor plantar response on the right side and her pulse was regular at a rate of 66 per min. The electrocardiogram confirmed sinus rhythm. Routine blood tests including thyroid function tests were normal. M-mode echocardiography revealed normal sized left atrium, left ventricle and a normal ejection fraction. Computed tomographic brain scan showed evidence of an area of cerebral infarction in the left parietal lobe. Anticoagulation was commenced 3 weeks later and amiodarone therapy was continued. With intensive rehabilitation she made a gradual but satisfactory recovery.

It is likely that this patient suffered from a thromboembolic stroke following chemical cardioversion due to amiodarone. Although she received treatment for hypertension the sudden onset neurological deficit with no progression coincided well with the change in her heart rhythm. As she had suffered from atrial fibrillation in the past she was well aware of the physical symptoms relating to changes in her heart rhythm. This episode of atrial fibrillation had lasted longer than usual and the reversion to sinus rhythm was influenced by the recent addition of amiodarone. The importance of anticoagulation prior to electrical cardioversion in established atrial fibrillation is well documented.⁴ Paroxysmal atrial fibrillation is reported to be associated with lower risk of stroke,⁵ particularly in patients with normal sized left atrium.⁶ There is a paucity of information as regards the incidence of embolic complications after chemical cardioversion with amiodarone. However, as exemplified by this case

report, the possibility of cerebral embolization secondary to chemical cardioversion should be borne in mind and anticoagulation should be considered prior to treatment of atrial fibrillation with amiodarone.

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Very long term diarrhoea due to *Campylobacter jejuni*

Sir,

Campylobacter jejuni is a common cause of acute bacterial diarrhoea in children and adults. *Campylobacter* enteritis is usually a self limiting disease and only a few cases of mild chronic diarrhoea have been published. We describe a very unusual case of long term diarrhoea due to this pathogen.

A 20 year old white man came to the clinic in June 1985 because of chronic diarrhoea which started in infancy. He had never been investigated or treated before. For more than 17 years he had had several bowel movements a day and sometimes at night. The stools were liquid but never bloody. The clinical examination was absolutely normal. Laboratory tests including blood iron, calcium, β -carotene, magnesium and immunoglobulins were in the normal range. Bacterial culture of the stools revealed the presence of *Campylobacter jejuni*. There were no parasites. Proctosigmoidoscopy, double contrast barium enema and barium meal were normal. The treatment was erythromycin 500 mg orally four times a day for 10 days. Diarrhoea disappeared during the treatment and the culture of the stools became negative. The patient was followed during three years and never relapsed.

We believe that our case demonstrates that 17-years

long chronic diarrhoea may be due to *Campylobacter jejuni*: we were unable to determine any other aetiology and the patient responded to selective treatment for *Campylobacter* and never relapsed. *Campylobacter* enteritis is usually a self-limiting illness. Symptoms resolve in less than 10 days, even without antibiotic therapy. Sometimes one needs two or three weeks to be cured. We have found only seven well documented cases in which the duration of diarrhoea exceeded 3 weeks from 2 months to 20 months.¹⁻⁵ In our patient the duration was 17 years. It is the longest chronic diarrhoea due to *Campylobacter jejuni* that we know about.

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Mixed enteric infection

Sir,

Between 1977 and 1980, 25,791 cases of *Helicobacter* (previously *Campylobacter*) *jejuni* were reported to the Communicable Diseases Surveillance Centre.¹ Of these 1.8% were multiple infections, and 3% were acquired

overseas. We present a case of mixed enteric infection acquired abroad.

A 35 year old nurse had *H. jejuni* and *Shigella sonnei* isolated from her stool 10 days after returning from Egypt with diarrhoea. She initially settled having taken ciprofloxacin 750 mg twice daily for one week, but presented to us 3 weeks later with rigors and night sweats. *Salmonella paratyphi* A phage type 1 was isolated from blood culture. This phage type of this organism is usually associated with the Indian subcontinent, and is only found in the United Kingdom as an imported infection. Her symptoms settled completely after a further 10-day course of ciprofloxacin. She remains well.

It is interesting that in spite of the initial course of ciprofloxacin, and the in-date TAB immunisation, the patient went on to develop enteric fever. Presumably, the antibiotic was unable to penetrate to adequate levels to the site where the organism was located, perhaps in the reticulo-endothelial system; or its micro-environment was anaerobic, when the antibiotic would be non-active.

S. typhi and *Helicobacter* double infection has been reported,² as has multiple organism infection in children in Ecuador.³ This woman presented with multiple organism infection including *Helicobacter* acquired overseas.

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