Lesson of the Week

Prolonged QT interval, atrioventricular block, and torsade de pointes after antiarrhythmic therapy

MARTIN T ROTHMAN

Lignocaine treatment is recommended for most patients with acute myocardial infarction admitted to a coronary care unit.¹ Electrocardiographic (ECG) abnormalities caused by toxic concentrations of lignocaine and other antiarrhythmic agents are not commonly recognised especially because patients receiving treatment may be expected to develop arrhythmias. I report on a patient with supraventricular tachycardia who had ventricular fibrillation, atrioventricular block, and a prolonged QT interval after large doses of antiarrhythmic agents.

Case report

For 18 months a 68-year-old woman had had palpitations several times a week lasting up to 30 minutes at a time. Digoxin 0.25 mg daily and oxprenolol 40 mg three times a day had reduced the number of attacks, but one prolonged attack of tachycardia had been treated with intravenous lignocaine and sinus rhythm returned. Substituting disopyramide by mouth

FIG 1—ECGs showing (a) frequent ventricular ectopics; (b) ventricular fibrillation; (c) second-degree atrioventricular block.

had failed to control symptoms. She was admitted after a six-hour attack of tachycardia. Serum sodium, potassium, urea, and calcium concentrations were normal, and ECG showed supraventricular tachycardia. Because of the success with lignocaine an infusion was started at 2 mg/min, increased to 4 mg/min after 60 minutes, and six hours later sinus rhythm was restored. Tachycardia recurred after six hours. The lignocaine was eventually increased to 8 mg/min and disopyramide 200 mg was given by mouth, but the lignocaine was discontinued three hours later because the patient became light headed and dizzy. A second dose of disopyramide was

Prolongation of the QT interval or torsade de pointes may indicate an electrolyte or metabolic disturbance or high plasma concentrations of antiarrhythmic agents.

given and DC cardioversion (300 W s) re-established sinus rhythm.

Two further doses of disopyramide 200 mg by mouth were given four hours apart, but ventricular ectopics and a 15-beat burst of tachycardia occurred. Two boluses of 100 mg lignocaine were given and the infusion restarted at 4 mg/min. ECG (fig 1a) showed frequent ventricular ectopics. The infusion rate was increased to 6 mg/min. Ventricular fibrillation (fig 1b) was converted to sinus rhythm with a "thump" on the chest. Second-degree atrioventricular block (Wenckebach type) then occurred (fig 1c) and ventricular fibrillation twice more but that responded to DC cardioversion.

Blood was taken for measurement of serum potassium, calcium, and lignocaine concentrations. All treatment was stopped and a bolus of 10 ml of 10% calcium gluconate given. Two minutes later atrioventricular conduction improved, ventricular ectopics disappeared, and the QT interval shortened (fig 2). Five minutes later an infusion of isoprenaline (2 µg/min)

FIG 2—ECG showing improved atrioventricular conduction, disappearance of ventricular ectopics, and shortened QT interval.

was started but ventricular fibrillation developed immediately; DC cardioversion was carried out and treatment stopped. Results from blood taken before the calcium bolus showed serum calcium 2.09 mmol/l (8.36 mg/100 ml) (normal range 2.30-2.60 mmol/l; 9.2-10.4 mg/100 ml), serum potassium 3.7 mmol(mEq)/l (normal range 3.5-5.0 mmol/l), and serum lignocaine 8.2 mg/l (therapeutic range 2-5 mg/l). Calcium and potassium infusions were given until blood concentrations reached 2.69 mmol/l (10.8 mg/100 ml) and 4 mmol(mEq)/l respectively.

Treatment was stopped for 48 hours to allow the toxic effects of the antiarrhythmics to wear off. There was no further ventricular fibrillation but paroxysmal supraventricular tachycardias were controlled with overdrive atrial pacing. The patient was transferred to another hospital to have a permanent ventricular pacemaker system implanted. This and beta-adrenergic blocking agents have kept her free of symptoms for 18 months.

King's College Hospital, London SE5 9RS

MARTIN T ROTHMAN, MB, MRCP, senior registrar in cardiology (present address: Cardiology Division, Stanford University Hospital, Stanford, California 94305)

Discussion

Over 29 hours 8.16 g intravenous lignocaine and 0.9 g disopyramide by mouth were administered. Toxicity in this case caused conduction disturbance, prolonged QT interval, and ventricular fibrillation. The ventricular fibrillation (fig 1b) is unusual, as it appears to turn over on itself and is known as "twisting points," or torsade de pointes, and is associated with a prolonged QT interval² from whatever cause. Isoprenaline has been suggested for the management of torsade de pointes and prolonged QT interval,² but ventricular fibrillation recurred with a very small dose. One 10 ml bolus of 10% calcium gluconate corrected the disturbance of rhythm and conduction. This has not been reported. The ECG abnormalities were corrected and ventricular irritability disappeared by giving intravenous calcium and potassium and stopping antiarrhythmic treatment.

Antiarrhythmic agents are commonly used. Recognising the

ECG changes that are associated with toxic concentrations of these agents is essential to avoid life-threatening arrhythmias. Stopping treatment immediately, correcting electrolyte abnormalities, and overdrive atrial pacing were used successfully in this patient.

I thank Dr D E Jewitt, director, cardiac department, King's College Hospital, for permission to report this case.

References

- ¹ Harrison DC. Should lidocaine be administered routinely to all patients after acute myocardial infarction? *Circulation* 1978;**58**:581-4.
- ² Krikler DM, Curry PVL. Torsade de pointes, an atypical ventricular tachycardia. *Br Heart J* 1976;**38**:117-20.

(Accepted 26 October 1979)

Medical Education

Medical education in London

SIR JOHN ELLIS

In October 1941 the Government outlined plans for a post-war hospital policy. It was to provide a co-ordinated, regionalised, largely State-funded hospital service with special arrangements for educational grants to teaching hospitals. In March 1942 the Government set up an interdepartmental committee under Sir William Goodenough to inquire into the organisation of medical schools and their facilities for clinical teaching and research and to recommend how they should relate to the future hospital service.

When the Goodenough Committee reported in 1944 it came out clearly and categorically with the recommendation that medical students should be educated as well as trained. It recommended a preregistration year to be followed by further postgraduate training. It considered whether it would be better: (a) for teaching hospitals to continue to run the medical schools; (b) for the teaching hospitals to be run by the university; or (c) for equally independent schools and hospitals to work together. It opted for the last and recommended that each of the 12 London schools become (as in fact the London School of Medicine had always been and St Bartholomew's had succeeded in becoming by Royal Charter in 1921) an independent corporate body, with strong university representation on its council of governors, able to deal on equal terms with the teaching hospital, and thus marry the needs of care with those of education.

The Goodenough Report was read by few, which was not altogether surprising because although it only cost three shillings and sixpence and was worth every penny, most people in May 1944 were more concerned with the Allied invasion of Normandy. None the less, those recommendations of Goodenough

which required parliamentary legislation were implemented along with the National Health Service. In 1948 the London teaching hospitals became State hospitals, but each under a board of governors responsible direct to the Minister of Health—and the London medical schools became independent corporate bodies, each with its own council of governors empowered to run its finances and own its premises. Also, overnight, the National Health Service solved one of the two requirements for postgraduate training—how to pay the trainee—by providing large numbers of junior hospital posts in which young doctors could practise under supervision while earning what was euphemistically called a salary. The other requirements—training programmes and facilities—did not occur to anyone at the time—except perhaps the trainees. But they were there, not always recognised as such, but getting what training they could—thousands of them—and so without any announcement and really without anyone realising what was happening, Britain embarked on a two-stage preparation for medicine. The third, intermediate stage of preregistration training was implemented in 1953.

In 1957 the General Medical Council abandoned its system of controlling standards by prescribing a minimum timetabled curriculum and issued new recommendations urging the schools to instruct less and educate more, and giving them freedom (which it said should be equally well regarded as a duty) to experiment with the course.

Changes in the provinces and London

Frustrating though it is for young and old alike, while medical education is quickly altered by changes in medical practice, deliberate changes to improve it are inevitably a slow business which proceeds at different speeds in different places. The provincial schools in 1948, comprising both university and hospital, were well situated for the joint advance of both, and

*Based on the Schorstein Memorial Lecture delivered at the London Hospital Medical College on 18 October 1979.