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Case Report

Prolonged asystole following direct-current cardioversion for atrial flutter

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We describe the case of a 64-year old lady with multiple established cardiovascular risk factors including non insulin-dependent diabetes mellitus, hypertension and previous history of stroke, with known to have atrial fibrillation, who presented for emergency admission with acute dyspnoea due to the onset of a fast ventricular response. Satisfactory rate control was achieved with digoxin. The cause of atrial fibrillation in this patient was presumed to be mild-to moderate regurgitation at the mitral valve, as evidenced by echocardiographic examination, in the absence of alternative positive findings.

CASE REPORT

The patient was admitted as an elective day-case two months later for direct-current cardioversion (DCC) following four weeks of adequate anticoagulation with warfarin, having discontinued digoxin three days previously. Other medications were: carbamazepine and thyroxine. The electrocardiograph at this time revealed atrial flutter with variable block, a ventricular rate of 108 per min, QRS axis of 15° and a T-wave axis of - 90°. She was sedated with 500 micrograms of alfentanil and 4mg of midazolam intravenously according to standard hospital practice. Flutter persisted despite 1x50Joule and 2x100Joule DC synchronised shocks. Following a further 200Joule shock the patient became asystolic. Percussion pacing was required to maintain cardiac output. The patient continued to breathe spontaneously and remained conscious, though heavily sedated, the rhythm being assessed every minute for three minutes until sinus rhythm developed at a rate of 56 ventricular beats per min. At this time she was normotensive and was monitored in hospital for 24 hours during which sinus node dysfunction, manifest as Tachycardia-Bradycardia Syndrome, was revealed. Serum electrolytes and thyroid function tests were found to be within normal reference ranges.

A permanent pacemaker was implanted three weeks later after which the patient has been generally well; although she relapsed into atrial fibrillation once.

Discussion

DCC, originally described for treating atrial fibrillation and flutter in 1963 by Lown *et al*, is a common procedure and is considered to be a simple and safe technique for restoring sinus rhythm[1]. A Medline and PubMed search revealed only one similar case of prolonged asystole following DCC. Hansen *et al*[2] described asystole preceded by a few seconds of atrial flutter and followed by severe nodal arrhythmia following DCC for atrial flutter. The authors postulated that while the precipitant may have been the direct current energy, this effect was facilitated by the adverse effects of pharmacological agents used which included sertraline, sotalol, digoxin and thiopental.

Kabutan *et al*[3] described cardiac arrest at induction of general anaesthesia with isoflurane in a patient who, as in the case we have described, had sick sinus syndrome. In one study of post-DCC arrhythmias[4] asystole was noted to be a transient

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characteristic, almost always lasting for less than two seconds. While several post DCC arrhythmias have been recorded in such studies, we were unable to establish any incidence of prolonged asystole[1,4,5].

It is difficult to confidently propose a mechanism for this complication since the salient characteristics of the reported case previously mentioned as well as the case we describe, are shared with all elective DCCs: sedation and DC shock against a backdrop of concomitant medical therapy often accompanied by underlying cardiac pathology. Synchronised DC shocks are known to have arrhythmogenic effects, however, these typically induce ventricular tachyarrhythmias. Perhaps the outcome of asystole rather than a slow nodal or ventricular rhythm may suggest generalised conducting system dysfunction; while ischaemia would seem the most likely basis for this, particularly in view of cardiac risk factors, there was no direct evidence of coronary artery disease. Carbamazepine has been reported to have effects on conducting tissue and indeed alfentanil is known to be a cause of asystole; unfortunately we do not know what the serum digoxin level was at the time of the procedure. It is possible to postulate that in this case the combined action of drugs, DC shocks and a susceptible substrate of sinus node dysfunction may have collectively led to the asystolic event; the contribution of each, however, cannot be confidently concluded.

Since DCC is a procedure which is commonly carried out in district general hospitals in a general medical day-patient setting by relatively junior medical staff, while the outcome we report is uncommon, we feel that its potential gravity justifies its consideration. It is therefore of critical importance that those who carry out this procedure should be aware of the possibility of asystole and be adequately trained and experienced so as to feel capable of managing this as well as other more common complications. The trend towards developing nurse-led elective DCC[6] adds further weight to such a position.

Sinus node dysfunction may be a prerequisite for prolonged post DCC asystole. Clinicians should be alert to this as it may be unmasked by DCC itself.

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