Comment

The cardiologist's nightmare

Electrical storm is one of cardiologists' worst nightmares. Patients have to be repeatedly shocked out of ventricular fibrillation, and it is difficult to know what to do. Stian Ross et al. have reported on one such instructive case.

Up until the mid-1990s, antiarrhythmic agents were used more widely than they are now. The medicines caused proarrhythmia, and cessation was the most important and most usual treatment for electrical storm. Today, the fundamental objectives are to reduce ischaemia (revascularisation and beta-blockade), reduce wall tension (optimal heart failure treatment) and correct acidosis and electrolytic imbalances (particularly hypokalaemia). The next steps are infusion of magnesium, which reduces the calcium overload of the cells (1), and administration of amiodarone. This procedure was followed with the patient in the present report, but strong sedation was necessary in addition. When the patient suffered relapses on recovering consciousness, there were still some therapeutic options available:

One possibility is rapid ventricular pacing, which is often effective (2). A higher heart rate results in shorter action potentials (less calcium overload) and shorter diastoles, with a correspondingly shorter excitable gap in which an extrasystole can trigger arrhythmia. Pacing is particularly effective when, as in this case, the arrhythmia is triggered by a pause followed by an early coupled ventricular extrasystole. My experience is that one must start with rapid pacing (120–130 beats/minute), and after a while smoothly reduce the pacing rate to just under 100 beats/minute, and usually keep it there for a day or two. The authors considered this possibility, but decided that the patient was too unstable for such intervention. Ventricular pacing results in poorer haemodynamics, and the pacemaker catheter may irritate the ventricle. However, our experience is that rapid atrial pacing, which results in improved heart function, can also function well.

Another possibility is to discontinue potentially proarrhythmic medications. Amiodarone seldom causes proarrhythmia, but it does happen (3). We do not know if this was the case with the patient in question. In such a case, the drug must be discontinued, and possibly another (unregistered) antiarrhythmic agent administered, for example bretylium (4).

A third possibility is catheter ablation of the suspected arrhythmic focus. The authors chose this option, which requires a high-tech arrhythmia laboratory, haemodynamic back-up treatment and an unusually experienced operator. The fact that the procedure was technically successful was a feat in itself – but unfortunately the patient had been suffering from heart failure so long that his life could no longer be saved.

What can we learn from this? The primary hospital must, as in this case, swiftly transfer such patients to the regional hospital. The treatment algorithm should then be followed with a brief observation period at each step, so as not to exhaust the patient. Occasionally, catheter ablation can be used as a last resort. A patient who has survived an electrical storm should have his or her genetic status examined for mutations that result in a long QT interval, even if the initial ECG is normal (5).

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References
1. Tzivoni D, Keren A, Cohen AM et al. Magnesium therapy for torsades de pointes. Am J Cardiol 1984; 53: 528–30.
2. Sowton E, Leatham A, Carson P. The suppression of arrhythmias by artificial pacemaking. Lancet 1964; 2: 1098–100.
3. Hohnloser SH, Klingenheben T, Singh BN. Amiodarone-associated proarrhythmic effects. A review with special reference to torsade de pointes tachycardia. Ann Intern Med 1994; 121: 529–35.
4. Nakstad AR, Eek C, Aarhus D et al. Survival after prolonged resuscitation with 99 defibrillations due to Torsade De Pointes cardiac electrical storm: a case report. Scand J Trauma Resusc Emerg Med 2010; 18: 7.
5. Lehtonen A, Fodstad H, Laitinen-Forsblom P et al. Further evidence of inherited long QT syndrome gene mutations in antiarrhythmic drug-associated torsades de pointes. Heart Rhythm 2007; 4: 603–7.

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