R-on-T with massive ST-segment displacement unmasking dispersion of repolarisation associated with torsade de pointes

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Figure 1. ECG showing a clearly prolonged QT of 660 ms (scale 10 mm/mV and 25 mm/s).

A 70-year-old woman presented to the cardiac emergency department with recurrent near-collapse. Two weeks before admission she had undergone mitral valve surgery for severe regurgitation.

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Physical examination revealed no significant abnormalities. The electrocardiogram showed sinus rhythm with low voltage P waves and incomplete right bundle branch block, and a severely prolonged QT interval of 660 ms at a heart rate of 56 beats/min, with negative T waves in the anterolateral leads (figure 1). Her medication on presentation consisted of digoxin 0.125 mg once daily and sotalol 80 mg three times a day. The serum potassium level was 4.18 mmol/l, magnesium 1.02 mmol/l and creatinine 76 µmol/l. A second ECG showed an R-on-T phenomenon where the premature R wave seemingly fuses with the ongoing T wave (figure 2). This seemingly fused complex shows massive ST depression and ST elevation as a sign of a large electrical gradient between the immediately repolarised or repolarising region in the

Figure 2. R-on-T phenomenon with fusion of the R wave with ST segment (arrow) accompanied by impressive ST elevation and depression (scale 10 mm/mV and 25 mm/s).

heart and the region instantaneously depolarised by the triggered beat. The mean vector of the T wave in the frontal plane immediately before the triggered ventricular complex is approximately $+150^\circ$, pointing towards the septum and the right ventricle, which means that these areas have already repolarised whereas the anterolateral area still needs to repolarise. The already repolarised area will produce the triggered beat,

in this case the triggered beat arises either in the septum or the right ventricle. Considering the duration of the triggered beat of only 80 ms, its frontal plane axis of 0° and its incomplete left bundle branch like configuration, it is quite compatible with an origin in the septum rather than the right ventricular free wall. The subsequent mean ST-segment vector of the triggered beat should point from the area depolarised

Figure 3. Development of torsade de pointes (scale 10 mm/mV and 25 mm/s).

by the triggered beat (septum/right ventricle) to the lateral wall of the left ventricle which in the (short) meantime has repolarised, yielding an expected mean frontal ST vector of around -20° . The T-wave vector before the triggered beat, the axis of the triggered beat and the vector of the subsequent massive ST displacement indicate a rapid sequence of changing vectors potentially setting the stage for torsade de pointes. To produce such massive ST displacement one half of the heart must interact with the other half, which fits with the notion of torsade de pointes. During her stay the patient showed recurrent episodes of torsade de pointes (figure 3) which resolved after magnesium suppletion and isoprotenerol infusion, and discontinuation of the digoxin and sotalol. Our case illustrates the extreme dispersion of repolarisation which may occur with class III antiarrhythmic drugs and which is associated with significant arrhythmias. The electrocardiographic phenomenon of massive ST-

segment displacement presented here illustrates that R-on-T ventricular beats may unmask the extreme dispersion of repolarisation which may occur with class III antiarrhythmic drugs. ■

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