

An elderly patient mentally confused

Florian Claude, Alain Delabays

Department of Internal Medicine, Ensemble Hospitalier de la Côte, Morges, Switzerland

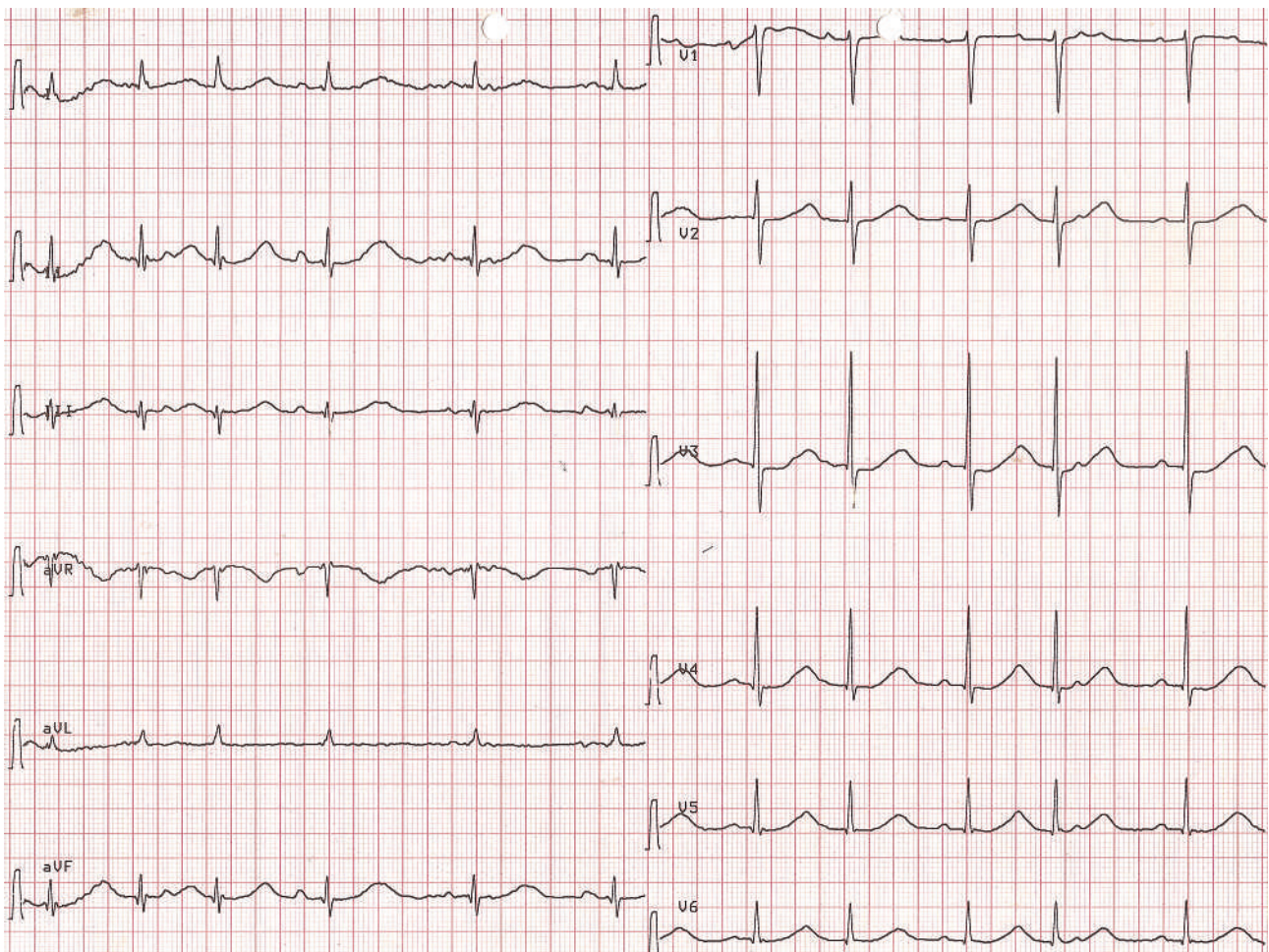
Case presentation

A 68-year-old man with a medical history of supraventricular arrhythmia, cerebral ischaemic stroke, hypertension and cured ORL cancer was admitted to our hos-

pital because of asthenia, weight loss and confusion. The 12-lead ECG at admission in the emergency department is presented below (fig. 1).

Figure 1

12-lead ECG on admission.



Funding / potential competing interests:

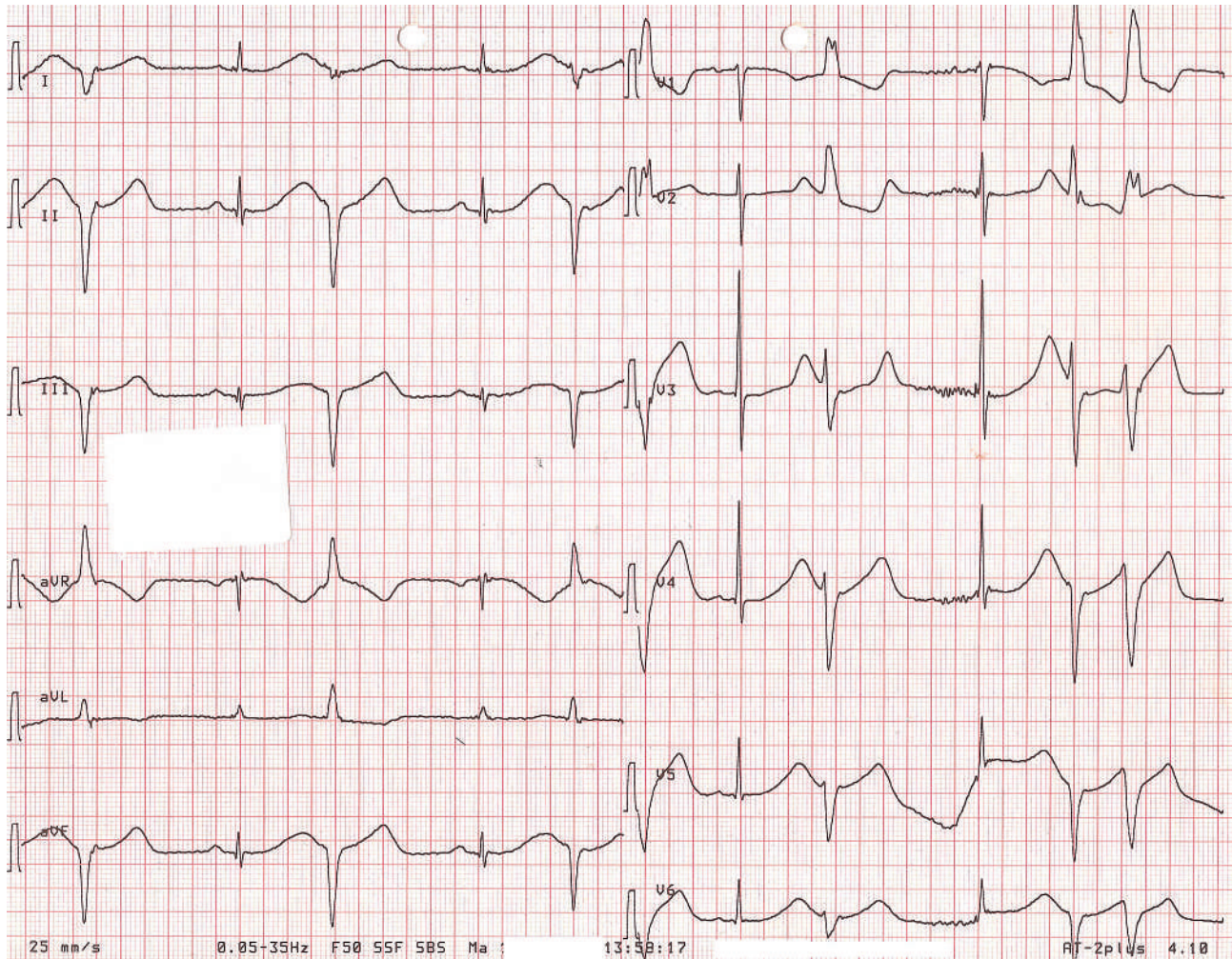
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Correspondence:

Alain Delabays, MD
Department of Internal Medicine
Ensemble Hospitalier de la Côte
2, chemin du Crêt
CH-1110 Morges
Switzerland
alain.delabays[at]ehc.vd.ch

Figure 2

12-lead ECG six hours post admission showing a sinus rhythm with narrow QRS complexes, a prolonged QT interval and R-on-T premature ventricular contractions with a couplet at the end of the recording.



Questions

1. What is your diagnosis?
2. What are the possible causes of this anomaly?

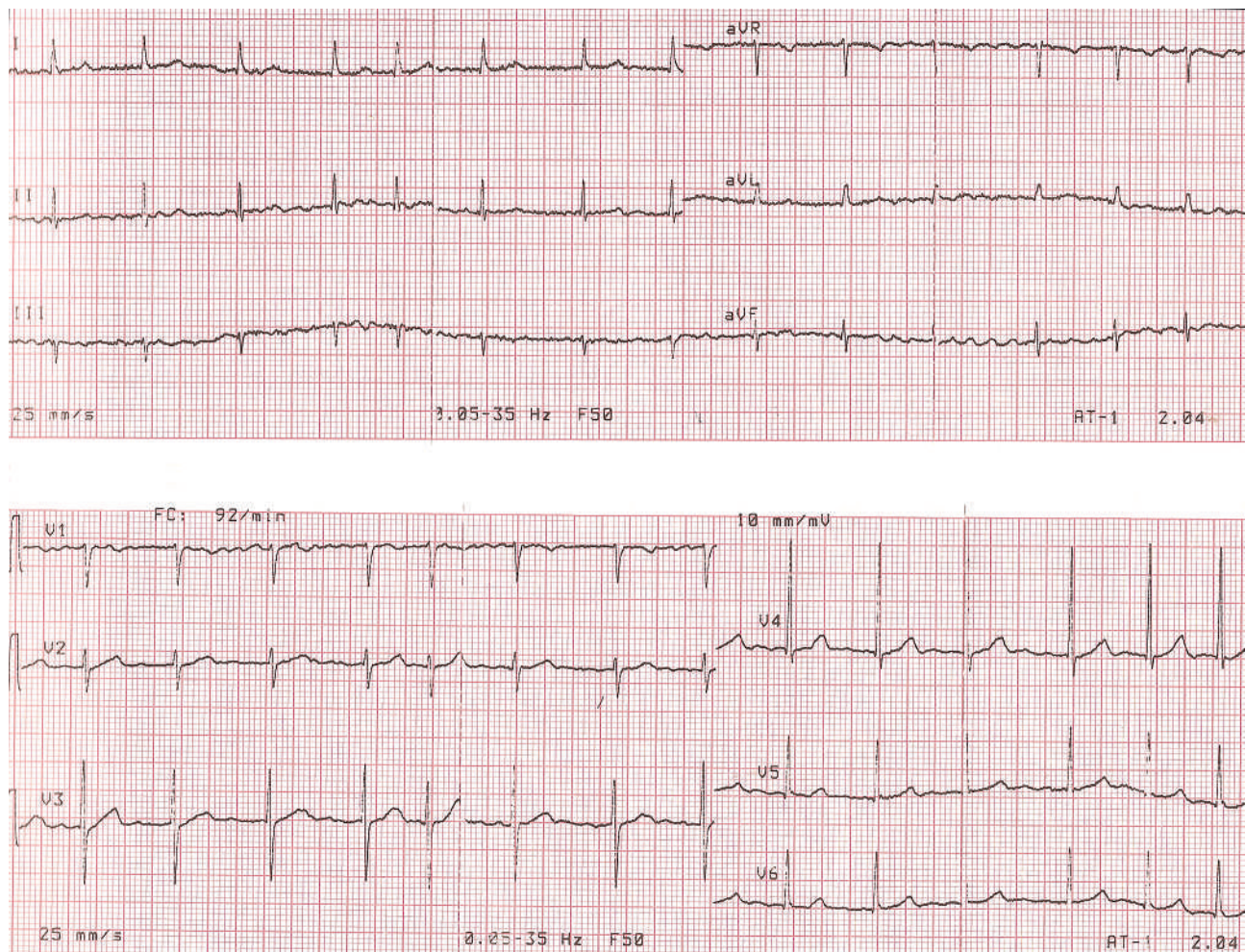
The patient's medication as analysis later showed, included: clopidogrel, sotalol, lecanidipin and torasemid. The laboratory results showed a severe hypokalaemia (2.4 mmol/l) with a normal renal function (creatinine 47 µmol/l). The patient was hospitalised, sotalol was stopped and a parenteral K⁺ substitution was begun. A few hours later, the patient developed bradycardia and hypotension. The 12-lead ECG (fig. 2) showed a sinus rhythm bradycardia with narrow QRS complexes, a dramatically prolonged QT interval (800 ms) and R-on-T premature beats with a couplet at the end of the recording. The plasma K⁺ was 2.7 mmol/l.

Commentary

The QT interval is the time between the beginning of ventricular myocardial depolarisation and the end of the repolarisation. It is measured between the beginning of the QRS complex and the end of the T wave. A U wave can sometimes hinder the measurement. In this case, the lowest point between the two waves may be considered as the end of the T wave. If the U wave merges with the T wave, the end of the U wave should be considered as the approximate end of the interval QT. In other situations, it is possible to use the aVL derivation where the U wave is invisible because of the perpendicular axis. The QT interval depends on sex, age and heart rate. It may be adjusted to the heart rate with correction formulas. The Bazett's formula is mostly used. ($QT_B = QT/\sqrt{R-R}$). However, it is not accurate, and over-corrects at high heart rates and under-corrects at low heart rates. The Fridericia formula ($QT_F = QT/\sqrt[3]{R-R}$) is less dependent on the heart rate.

Figure 3

12-lead ECG seven days post admission showing an atrial fibrillation with normalised QT interval (QTc 400 ms).



The long QT syndrome is defined by a QTc ≥ 450 ms in men and ≥ 460 ms in women.

Clinicians should look for each cause of acquired long QT syndrome when it is present on ECG. In our case, Sotalol and hypokalaemia were responsible for bradycardia, long QT interval and premature ventricular contractions putting our patient at risk for torsade de pointes. Many common medications can lengthen the QT interval in otherwise healthy people and cause a form of acquired long QT syndrome known as drug-induced long QT syndrome. Therefore, it is important to get a complete medication history and to stop the responsible medications. Information about medications which prolong QT interval can be found at www.qtdrugs.org. Electrolytic disturbances such as hypokalaemia, hypomagnesaemia and hypocalcaemia can also induce or aggravate a long QT syndrome and have to be looked for and treated. Finally, there are paramete-

ters that promote long QT syndrome and torsade de pointes like advanced age, female sex, myocardial infarction, congestive heart failure and impaired hepatic drug metabolism (hepatic dysfunction or drug interaction).

Concerning our patient, the QT interval normalised (320 ms) after the hypokalaemia was corrected and sotalol was stopped, but seven days later he developed atrial fibrillation (fig. 3) for which anticoagulation therapy was started.

References

- 1 McKibbin JK, Pocock WA, Barlow JB, et al. Sotalol, hypokalemia, syncope and torsade de pointes. *Br Heart J*. 1984;51:157.
- 2 Makkar RR, Fromm BS, Steinmann RT, et al. Female gender as a risk factor for torsades de pointes associated with cardiovascular drugs. *JAMA*. 1993;270:2590.
- 3 Roden DM. Drug-induced prolongation of QT interval. *N Engl J Med*. 2004;350:1013.