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Electrocardiographic Case: A Middle Aged, Seriously Ill Woman with an Unusual ECG and Wide Complex Tachycardia

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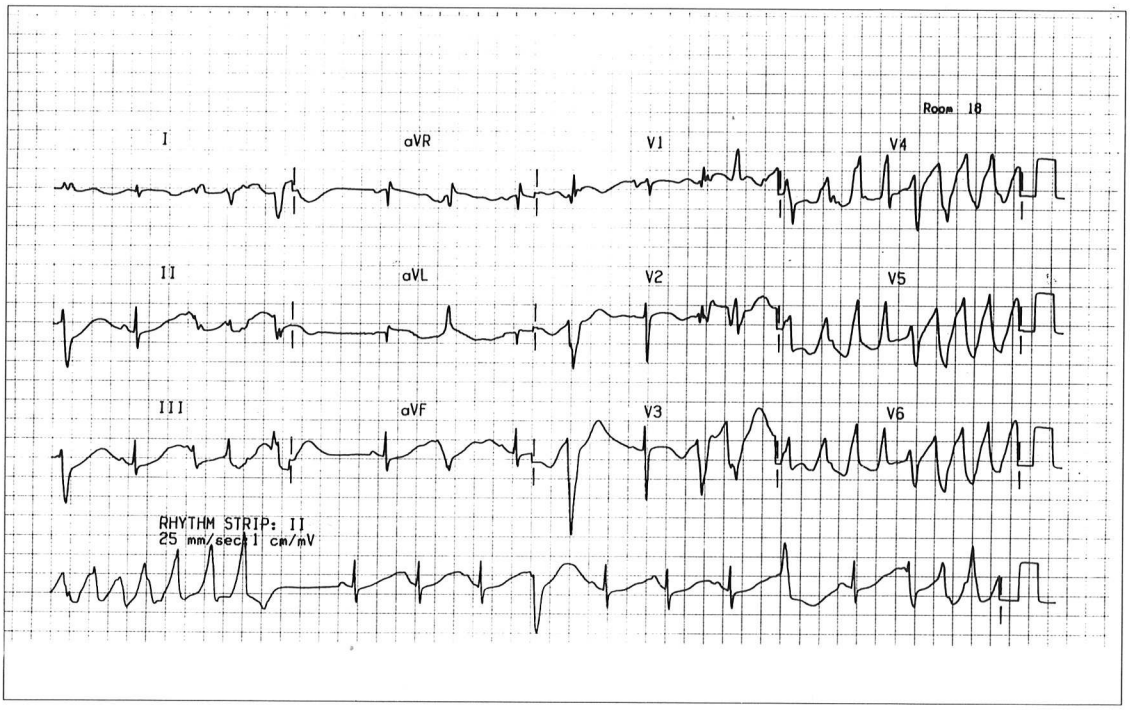

Fig 1

This 48-year-old female patient, who has multiple problems including hypothyroidism, constrictive pericarditis and Type II respiratory failure, was admitted to the intensive care unit and was intubated for acute on chronic respiratory failure. She was treated with antibiotics, diuretics, nitrates and L-thyroxine. A few days after admission, the following ECGs were recorded.

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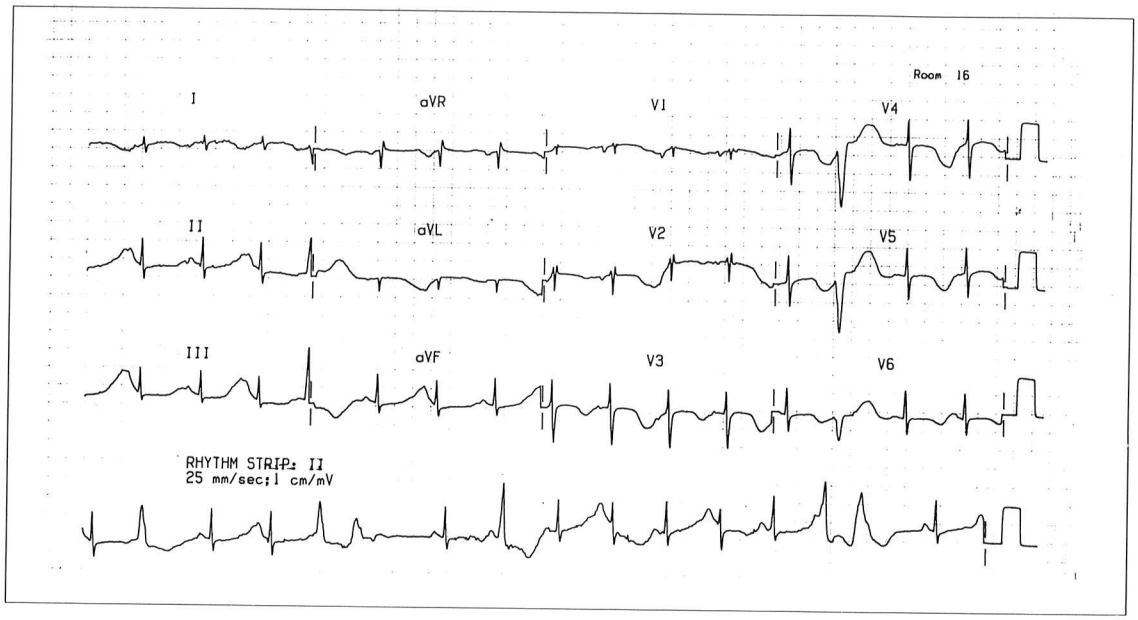

Fig 2

DIAGNOSIS

ECG 1) Torsades de Pointes triggered off by R-on-T premature ventricular complexes.

ECG 2) Markedly prolonged corrected QT interval (QTc) with T wave alternans. Biphasic 'p' waves in lead V1 due to left atrial enlargement.

The first ECG shows frequent isolated ventricular ectopics with a R-on-T type of phenomenon and runs of non-sustain polymorphic ventricular tachycardia (VT) in leads V4 – V6. The QRS complexes are markedly widened and bizarre with a 'rotating' axis. The term 'Torsades de Pointes', which was introduced by Dessertenne[1] in 1966, is defined as polymorphic VT that is associated with an unduly prolonged QTc.

The second ECG shows a markedly prolonged QT interval and T wave alternans. The measurement of the QT segment has been a source of controversy for a long time. The definition of QT is 'from the beginning of the QRS complex to the end of the T wave[2]. This is however fraught with a lot of problems including the variability of the QT segment with the heart rate and the autonomic state, the exact end of the T wave and the presence of the 'U' wave[3]. Presently, the QT interval is usually measured in lead II[4] (which tends to separate the T from the U wave) and the Bazett's formula[5] is used to correct for the heart rate. T wave alternans is rather uncommon and is usually associated with a markedly prolonged QTc ($>$ 0.60 secs)[6]. There is a relationship between the presence of T wave alternans and ventricular tachyarrhythmias, but this is thought to be primarily due to the prolonged QTc per sec[6].

QT interval prolongation can be divided into the acquired and congenital types. The congenital types are the Jervell and Lange-Melsen syndrome[7] (associated with deafness) and the Ward-Romano syndrome[8,9] (unassociated with deafness). The treatment consists of beta-blockade, although occasionally left cervicothoracic sympathetic ganglionectomy or implantable defibrillators are required. The acquired types are secondary to ischaemia, drugs (type IA, IC, III anti-arrhythmic agents, tricyclic antidepressants), electrolyte abnormalities (hypocalcaemia, hypomagnesaemia), cardiomyopathy, rheumatic carditis, subarachnoid haemmorhage and severe bradyarrhythmias[10]. Management of this condition includes correction of the precipitating cause, overdrive pacing, IV magnesium, Type I b antiarrhythmics and occasionally, an implantable defibrillator.

In this patient, the serum magnesium level was found to be very low (0.36 μmol/L). Correction of the magnesium level led to resolution of the QT changes and abolition of the torsades de pointes. The aetiology of the severe hypomagnesaemia was the prolonged use of high doses of loop diuretics.

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