

T Wave Alternans and Acute Rheumatic Myocarditis: A Case Report

K S Tan,**MB, MRCP (I), MRCP (UK)*, Y S Lau,***FAMS, MBBS, M Med (Int Med)*, W S Teo,****FAMS, M Med (Int Med), MRCP (UK)*

Abstract

T wave alternans is an uncommonly recorded cardiac rhythm. We report here an unusual case of a 13-year-old girl with acute rheumatic carditis and acute nephritis, who developed T wave alternans associated with a prolonged QT interval.

These electrocardiographic changes were evident only after the initial acute stage of the disease process and should be borne in mind for patients with acute rheumatic carditis as they may be associated with more malignant arrhythmias.

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Key words: Acute rheumatic carditis, Prolonged QT interval, T wave alternans

Introduction

T wave alternans, an electrical cardiac alternans in which there is a beat-to-beat variation in the amplitude, shape or the polarity of the T wave during sinus rhythm without any changes in the QRS complex, is an uncommonly recorded rhythm.¹ We report here an unusual case of T wave alternans developing in a patient with acute rheumatic fever and myocarditis.

Case Report

A 13-year-old Malay girl presented with a week's history of persistent upper abdominal pain of increasing severity. Furthermore, she had noticed some early morning facial and periorbital oedema prior to admission. She was previously well and had no symptoms of an upper respiratory tract infection in the preceding few weeks. On examination, she was in distress due to her abdominal pain. Her respiratory rate was 36 per min and she had a low grade fever. Her heart rate and blood pressure were 144 bpm and 110/70 mmHg, respectively. Her jugular venous pressure was elevated 8 cm above the sternal angle. There was a third heart sound gallop rhythm and bibasal crepitations in both lung bases were noted. She had a tender, non-pulsatile enlarged liver.

Her electrocardiograph (ECG) on the day of admission showed sinus tachycardia of 145 bpm with right axis deviation, non-specific T inversion and a corrected QT interval (QTc) of 360 ms. Her Chest X-ray (CXR)

showed evidence of acute pulmonary oedema. An echocardiographic examination the next day showed biventricular enlargement with global hypokinesia and a left ventricular ejection fraction of 25%. Her full blood count (FBC) revealed a haemoglobin concentration of 10 g/dl with a normocytic and normochromic pattern, a white cell count of 17,900, a platelet count of 503,000 and an erythrocyte sedimentation rate (ESR) of 28 ml/h. Her serum C-reactive protein was 7.7 mg/l, serum urea concentration 9.5 mmol/l, serum sodium concentration 139 mmol/l, serum potassium 4.8 mmol/l and serum creatinine 96 µmol/l. Her liver function test showed a serum total protein concentration of 63 g/l, serum albumin of 28 g/l, alanine transaminase of 85 IU/L and aspartate transaminase of 18 IU/L. Her serum creatinine kinase concentration was 44 IU/L. Urinary microscopy showed a urinary red blood cell (RBC) count of 300 to 350, a urinary white blood cell (WBC) of 300 to 350 and a 1+ concentration of urinary albumin. No casts were detected. Urinary total protein was 3.0 g/l. A throat swab did not grow any organisms. Her anti-streptolysin O titre was elevated at 400 IU/ml.

Serum anti-nuclear antibody, double-stranded DNA, l-ANCA, p-ANCA, anti SM, RO (SSA) and anti LA (SSB) titres were all negative. C3 and C4 complement titres were 0.17 g/l (0.4 to 1.46) and 0.11 g/l (0.12 to 0.72), respectively.

The diagnoses were that of acute heart failure with

* Consultant
Department of Medicine
Changi General Hospital

** Consultant
Mount Alvernia Hospital

*** Consultant
National Heart Centre

Address for Reprints: Dr Tan Kok Soon, Department of Medicine, Changi General Hospital, 2 Simei Street 3, Singapore 529889.

hepatic congestion from acute rheumatic myocarditis and concomitant acute nephrotic/nephritis syndrome. She was commenced on intravenous frusemide 40 mg bd, oral digoxin 0.125 mg, IV crystalline penicillin 1 MU qds and oral aspirin 900 mg qds. Her clinical condition improved over the next few days on the above therapy and by day 4 of admission she was afebrile and was no longer tachypnoeic. Her jugular venous pressure (JVP) was no longer elevated and she was only minimally tender over the liver. There were still some occasional basal crepitations in her lung bases. However, her ESR had increased to 63 mm/h and her serum creatinine concentration to 161 mmol/l. Her ECG at this stage was unremarkable with sinus tachycardia of 124 bpm and non-specific T wave changes in the inferio-lateral leads. The QTc interval was 383 ms. By day 7 of admission, her congestive heart failure had resolved completely. Her ESR had dropped to 28 ml/h and her serum creatinine concentration to 71 $\mu\text{mol/l}$. Her white blood cell was 79,000 and her serum calcium and magnesium concentrations were 2.3 mmol/l and 0.71 mmol/l, respectively. However, the duration of the corrected QT interval on her 12-lead ECG had increased to 590 ms and definite T wave alternans was now evident (Fig. 1). The T wave can be seen to have encroached onto and merged with the U wave, resulting in the fusion of the two waves. This was most obvious in leads V2 and V3. Furthermore, there were obvious monophasic T wave alternans with defi-

nite beat-to-beat variation of the amplitude of the T wave in all except leads aVR, aVL and V1.

A day later, her ECG showed sinus rhythm with a rate of 65 bpm and frequent premature ventricular ectopics. The QTc interval had lengthened to 702 ms. This increase in the length of the QT duration was accompanied by a more pronounced beat-to-beat variation in the amplitude of the T waves. Definite biphasic alternation of the T-wave, with a beat-to-beat change in the polarity of the T wave, was noted in lead III (Fig. 2). There were no other detectable causes for the prolonged QT other than the myocarditis associated with the above acute rheumatic fever.

The QTc interval began to shorten thereafter. By day 9, it measured 569 ms and by day 14, 407 ms. There was a corresponding decrease in the prominence of the alternating T wave, with its disappearance altogether observed by day 14. She continued to improve and was discharged 18 days after admission. When reviewed one month later, she was clinically well. Her ESR was 27 ml/h, and urinary microscopy showed 80 to 100 RBCs, 3 to 5 WBCs with a trace of albumin only. Her ECG showed sinus rhythm.

She was advised to avoid strenuous activities and to continue with aspirin and monthly injections of penicillin.

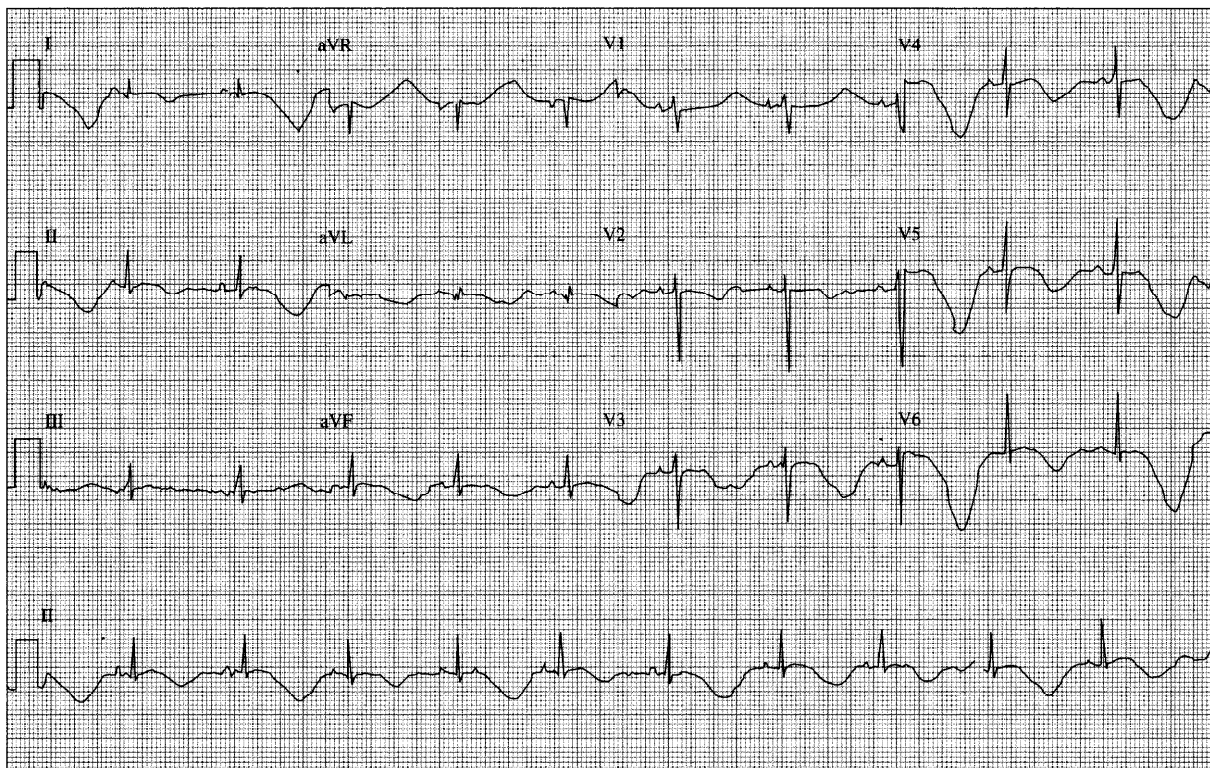


Fig. 1. 12-lead ECG showing sinus rhythm with a rate of 62 bpm, a QTc interval of 590 ms and T or QU wave alternans. The alternans waves were most obvious in leads I, II and in V4 to V6. The T waves could be seen to have encroached on to the U waves, resulting in a fusion of both. This was best seen in lead V3. Premature ventricular beats too were noted.

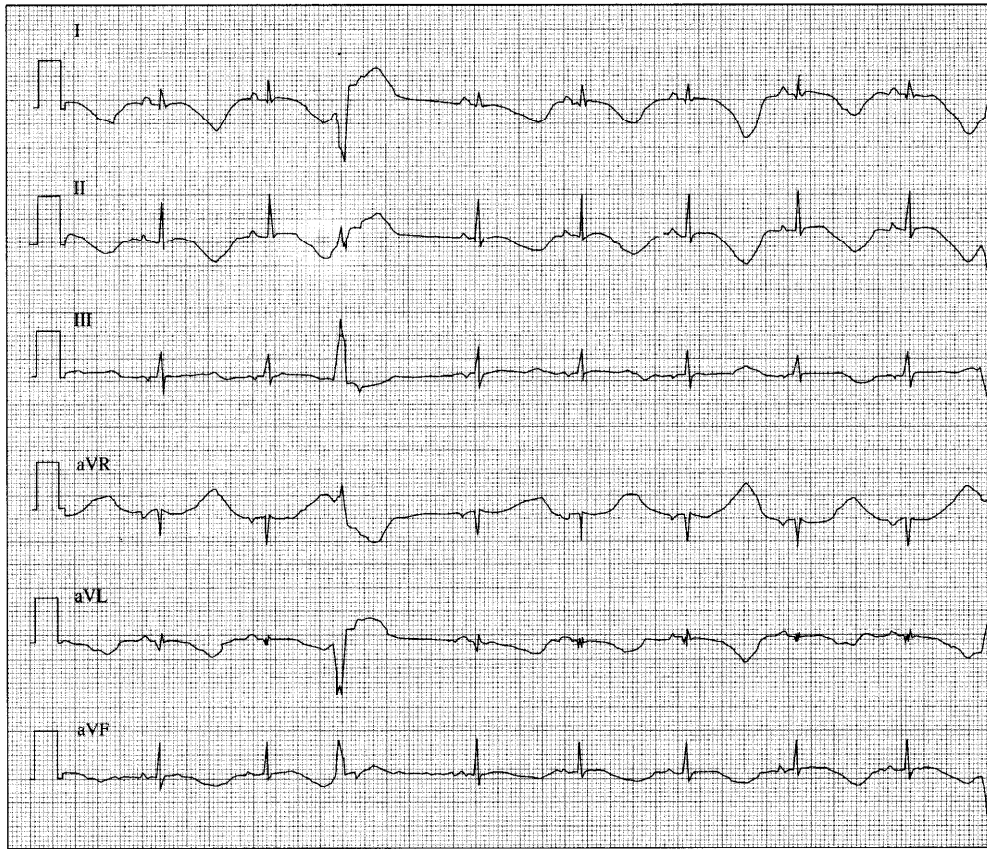


Fig. 2. ECG showing sinus rhythm with a rate of 65 bpm. The QTc interval had lengthened to 702 ms. This increase in the length of the QT duration was accompanied by a more pronounced beat-to-beat variation in the amplitude of the T waves. Definite biphasic alternation of the T wave can be seen in lead III. Frequent premature ventricular ectopics were noted.

Discussion

T wave alternans associated with a long QT interval has been reported in congenital long QT syndrome, hypocalcaemia, hypokalaemia, hypokalaemia with hypocalcaemia and hypomagnesaemia, cardiomyopathy associated with hypomagnesaemia and alcoholism, after ventricular fibrillation and in patients treated with quinidine.²⁻⁶ It has further been associated with changes in heart rate or with premature extrastimuli, though in some cases its occurrence is unexplained.^{7,8} However, to our knowledge, it has never been reported in association with acute rheumatic carditis. The more commonly reported ECG changes in acute rheumatic carditis are those of prolonged PR interval, non-specific ST-T wave changes, less commonly nodal rhythms and transient second and third degree heart blocks which generally do not require any specific interventions.⁹ Interestingly as this case illustrated, the appearance of a prolonged corrected QT interval and T wave alternans did not occur during the early stages of the acute disease process but was apparent only a week after the presentation with acute heart failure and at a time when the patient was clinically improving and the inflammatory process waning as suggested by the falling levels of the ESR value and the white cell count.

The T wave alternans persisted for about 72 hours and throughout this period, the amplitude of its alternans corresponded closely to the duration of the QTc interval. The alternating T waves became apparent on the surface ECG when the QTc interval reached 590 ms, becoming more pronounced as the QTc lengthened and disappeared as the interval decreased to 409 ms. This close relationship between T wave alternans and the duration of the QTc interval was noted in a series of patients with idiopathic long QT syndrome where T wave alternans were recorded mainly in those patients with a QTc interval of 500 ms or more.¹⁰ Furthermore, patients with biphasic T wave alternans had significantly longer QTc values than those with monophasic beat-to-beat variation of the T wave.¹⁰ Hence, the presence and persistence of the monophasic alternating T waves in our case was likely to be a result of and a reflection of the prolongation and changes in the duration of the QTc interval.

A number of hypotheses have been put forward to explain the fundamental mechanism behind the occurrence of T wave alternans. This included enhanced early after-depolarisation, alteration in the sympathetic nervous system and shortening of the diastolic period.¹⁰

In this case, the development of T wave alternans is

probably a result of the complex interplay between a number of these factors. The critical alternating prolongation and shortening of the diastolic interval and the duration of the following action potential would explain the close relationship between the QT interval and the appearance of T wave alternans as seen in this case in which the T wave alternans is only evident when the QTc interval was longer than 590 ms.¹¹

Another factor which may be implicated in the development of T wave alternans in this case is that of inter-regional heterogeneity of repolarisation.¹⁰ Normal myocardium usually displays a duration of cellular action potential that is not absolutely uniform among the various cellular units that make up the myocardium. We postulate that in our case, the normal inter-regional heterogeneity of the myocardium is markedly enhanced in the recovery phase of the acute inflammatory process as different parts of the myocardium are likely to be at a different stage of recovery. This leads to a prolongation of the action potential (repolarisation) and thus to the predisposition to develop T wave alternans.¹⁰ This would explain why the phenomenon of T wave alternans in this case was only evident during the later stage of the disease process when the inflammatory process was subsiding.

The occurrence of T wave alternans is of not only electrophysiological interest but has considerable clinical importance also as the presence of T wave alternans is associated with a propensity for the development of fatal ventricular tachyarrhythmias, in particular those of torsades de pointes and ventricular fibrillation. This association is probably not causal, as a prolonged QT interval can not only precipitate the development of T wave alternans but in addition indicate the presence of an unstable heterogeneous pattern of ventricular repolarisations that predisposes to the development of torsades de pointes.¹¹ Although it may not be causally implicated as a harbinger of polymorphic ventricular tachycardia, the development of T wave alternans in patients with idiopathic QT syndrome has been shown to identify a subset with a high risk of experiencing a major cardiac event.¹⁰ In fact, even subtle electrical alternans not visible on a surface electrocardiogram, but detectable during electrophysiological study may predispose towards ventricular arrhythmias.¹² Hence, as this case demonstrated, the possibility of prolongation of the QTc intervals and the development of T wave alternans should be borne in mind in patients with acute rheumatic carditis. Furthermore, the development of T wave alternans may only manifest itself in the later stages of the disease process when the patient is clinically and biochemically improving.

This case is also unusual in that both the acute rheumatic fever and post-streptococcal acute nephritis

occurred concurrently. No renal biopsies were carried out in this case because the acute nephritis and nephrotic syndrome recovered quickly. Although her nephritis may be a result of a connective tissue disease, this was unlikely in view of the fact that the autoimmune markers in this case were negative. It has been postulated that only certain strains of group A streptococci are capable of causing acute rheumatic and that others cause acute post-streptococcal glomerulonephritis.¹³ However, this has not been proven and as this case demonstrated, both these non-suppurative sequelae of group A streptococcal infection can occur together.

In summary, this case demonstrated that T wave alternans and a prolonged QT interval can develop in patients with acute rheumatic carditis. These electrocardiographic features should be borne in mind in patients with acute rheumatic carditis as its presence is associated with a propensity for the development of fatal ventricular arrhythmia. Furthermore these repolarisation alternans may appear after the initial acute stage of the cardiac process.

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