LETTERS TO THE EDITOR

CANCER : TO TELL OR NOT TO TELL?

Dear Sir,

I read with unbelief and disappointment the results of Tan et al's paper on 'Cancer: To tell or not to tell?'[1]. It is truly disappointing and shocking that in the 1990s, only 43.6% of Singapore doctors will inform their patients of the diagnosis of cancer. Perhaps the results should not be surprising as a similarly designed paper by Oken in 1961[2] revealed the same disastrous results that 90% of American doctors preferred withholding the precious 'privileged' information.

I feel that the wrong population was studied and the wrong questions were asked (or the right question not asked!) in both studies across the globe, hence producing results which must not be accepted as the ideal and correct practice.

I did a brief pilot questionnaire on about forty West Malaysian doctors enquiring whether they would like to be told if they were found to have cancer. The unanimous response was yes. If a doctor who has cancer would like to know, why then shouldn't the patient be similarly regarded?

Subsequently, Dr G C Chong and I, performed a questionnaire survey directed at a sizeable population of patients who knew their diagnosis and who had the diagnosis of cancer established at least 3 months prior to the interview.

Sixty-one patients from a representative section of the population, from all educational and vocational background and equal sex distribution responded. 95.1% of the patients were of the opinion that they want to be told the diagnosis and 77% revealed that it was the doctor who told them and 93.4% felt that the doctor was the best person to break the news. This paper has been presented at a medical congress and is in the process of being submitted to your journal soon.

I strongly believe that it is a myth that patients cannot cope with the truth when they have cancer and hence should not be told and this has been confirmed in the study I conducted. As for chronic illnesses, a significant 83% of patients in Elian's study[3] were in favour of knowing that they had multiple sclerosis. The references below except for Tan and Oken's paper speak forth for informing the truth ie in a professional way.

Doc, please tell your patients, if you too would like to know.

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2 Oken D. What to tell cancer patients. JAMA 1961; 175: 1120-8.

3 Elian M, Dean G. To tell or not to tell the diagnosis of multiple sclerosis. Lancet 1985: July 6: 27-8.

4. Editorial. In Cancer, Honesty is here to stay. Lancet 1980; August 2: 245.

REPLY FROM AUTHORS

Dear Sir,

We are pleased that our paper has aroused discussion among our colleagues. Like the author, we lament the reluctance of Singapore doctors in revealing the diagnosis of cancer to their patients. However we do not support, as the author may have implied, that doctors continue to withhold such information from patients.

We have discussed in our paper the psychosocial reasons why doctors are reluctant to openly discuss the diagnosis of cancer with patients. We have stressed that informed patients and families do better than uninformed ones. We agree with the author that it is a myth that patients are unable to deal with the truth. We sincerely hope that with inclusion of psychosocial aspects of cancer in the medical school curriculum and education of our local doctors, more doctors will tell the truth to patients. We have hope to contribute to the latter with our modest paper.

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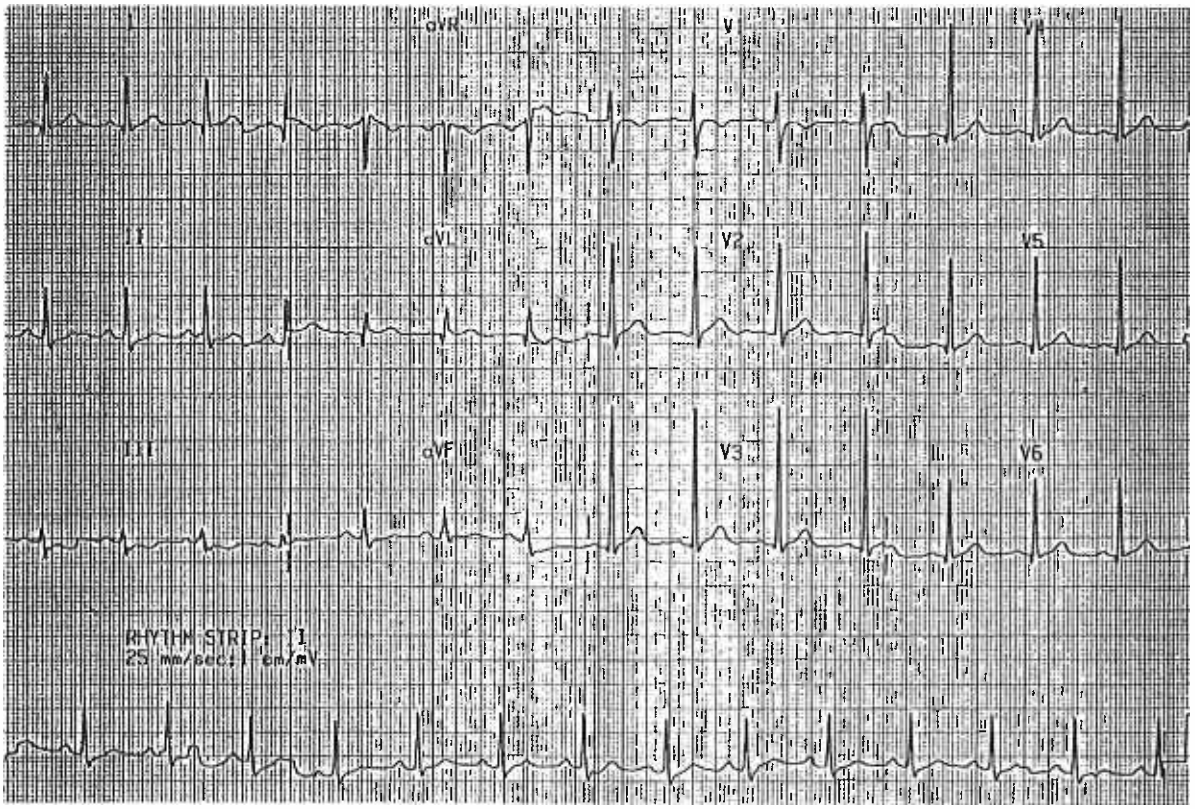
ANSWER TO ELECTROCARDIOGRAPHIC CASE
Diagnosis: Atrial tachycardia.

Fig 3 – 12-lead electrocardiogram after radiofrequency catheter ablation of ectopic atrial focus.



DISCUSSION

The electrocardiogram in Fig 1 shows a regular narrow complex tachycardia with a rate of 180 beats per minute. The T wave of each QRS complex is deformed by a P wave which is positive in leads I and aVL, biphasic in lead II, and negative in leads III, aVR and aVF. The R-P interval is slightly less than the P-R interval. The QRS complexes are alternately bigger and smaller (QRS alternans). In Fig 2, the heart rate has slowed down to 90 beats per minute. However, there are now two P waves for every QRS complex. The P waves still have a rate of 180 beats per minute and are identical in morphology to those in Fig 1. Verapamil has blocked the atrioventricular (AV) node allowing only 2:1 conduction from the atria to the ventricles.

The differential diagnoses of a regular narrow complex tachycardia are sinus tachycardia, atrial flutter, atrial tachycardia (either due to increased automaticity or reentry), atrioventricular nodal reentrant tachycardia (AVNRT) utilising dual AV nodal pathways or orthodromic atrioventricular reentrant tachycardia (AVRT) involving an accessory pathway and the AV node. Atrial flutter is characterised by flutter waves commonly with a rate of 300 beats per minute and is best seen in the inferior leads on the electrocardiogram (sawtooth pattern). The AV conduction ratio in untreated cases is usually 2:1 giving a heart rate of 150 bpm. Atrial tachycardia may be due to increased automaticity from an ectopic focus, or reentry circuits within the sinus node (sinoatrial reentry) or any part of the atria (intraatrial reentry). Atrial tachycardia differs from atrial flutter in that it has a rate of less than 250 beats per minute. The P wave morphology depends on the site of origin or exit site of the reentrant circuit in the atria. The P wave is distinctly separated from the QRS complex and is closer to the next QRS complex than to the preceding one, ie R-P interval greater than P-R interval[1]. AVNRT utilises a fast and a slow pathway. When antegrade conduction is down the slow pathway and retrograde conduction is via the fast pathway, the common (slow-fast) form of AV nodal reentry results. The uncommon (fast-slow) form is due to antegrade fast and retrograde slow pathway conduction. AV nodal reentry results in a P wave polarity that is negative in leads II, III and aVF. In the common slow-fast form, the P wave is hidden within the QRS complex or distorts the terminal or initial part of the QRS complex and the R-P interval is less than the P-R interval. The uncommon fast-slow form results in the R-P interval being greater than the P-R interval[1]. Activation of the ventricles in orthodromic AVRT is via the AV node and retrograde atrial activation is via an accessory pathway. The P wave is separated from the QRS complex but the R-P interval is less than the P-R interval. Uncommonly, a slowly conducting accessory pathway may cause the R-P to be greater than the P-R interval[1]. The morphology of the P wave depends on the location of the accessory pathway. It is negative in leads I and aVL in a left sided accessory pathway and negative in leads II, III and aVF in a posteroseptal pathway. The commonest form of paroxysmal supraventricular tachycardia is AVNRT, followed by AVRT and atrial tachycardia[2-4]. Drugs or vagal manoeuvres that block the AV node can terminate AVNRT or AVRT but not atrial tachycardia. Atrial tachycardia can continue despite the presence of AV block but AVRT cannot, although AVNRT can sometimes continue in the presence of 2:1 AV block. QRS alternans which persists after the first five

seconds of the tachycardia is said to be highly specific for orthodromic AVRT[2], and is found particularly at heart rates above 200 per minute. However this is not true in all cases, as in this patient.

This patient had an automatic atrial tachycardia. Automatic atrial tachycardia is rare in adults but relatively more common in children[5]. The characteristic feature of automatic atrial tachycardia is that it starts and stops spontaneously and cannot be predictably induced or terminated by single premature atrial or ventricular depolarisations. If its onset is observed, the first complex usually occurs late in the cardiac cycle and the cycle length progressively shortens for several cycles until its ultimate rate is achieved (warm-up phenomenon). The first and subsequent P waves of the tachycardia are identical[4]. The P wave morphology in this patient points to a right atrial origin, vector being right to left. Incessant automatic atrial tachycardia (atrial tachycardia being present most of the day) can result in tachycardia-induced congestive cardiomyopathy and heart failure. Removal of the ectopic focus, either by surgery[6] or radiofrequency catheter ablation[7], can cure the arrhythmia and reverse the cardiomyopathy[8].

Management of the atrial tachycardia in this patient is aimed at controlling the ventricular rate initially by drugs which slow AV node conduction. This patient was treated with a combination of digoxin, verapamil and propranolol. She delivered vaginally at full term to a healthy baby and, on follow up, was found to be still in incessant atrial tachycardia. Her left ventricular ejection fraction was found to be mildly impaired on echocardiography. She subsequently underwent electrophysiological study which localised the ectopic focus to the anterior mid-right atrium. Radiofrequency catheter ablation of the ectopic focus resulted in restoration to sinus rhythm and the electrocardiogram after ablation is shown in Fig 3 (compare sinus P wave morphology to ectopic focus).

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