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Editorial

The Post-MADIT II Era: ICD for all Post-infarct Patients with Moderate to Severe Left Ventricular Dysfunction?

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Device therapy has become the preferred treatment for patients who have survived sudden cardiac death. Throughout the last decade, large-scale randomized clinical trials have provided consistent evidence on the clinical efficacy of device therapy. ICD is proven to be superior to anti-arrhythmic drugs in patients with structural heart diseases who suffer from haemodynamically significant ventricular arrhythmias. The role of ICD in primary prevention of sudden death in selected patients with coronary artery disease and left ventricular dysfunction is also widely established after the Multicenter Automatic Defibrillator Implant Trial (MADIT)[1] and the Multicenter Unsustained Tachycardia Trial (MUSTT).[2]

In MADIT, patients with previous myocardial infarction, depressed left ventricular function (ejection fraction <35%) and non-sustained ventricular tachycardia underwent electrophysiology study. Among them, 196 patients with inducible sustained ventricular tachyarrhythmias but not suppressed by procainamide were randomized to receive an ICD or conventional therapy. The ICD group had a 54% reduction in mortality at 2 years, and the benefit was greatest in patients with the lowest left ventricular ejection fraction. The MUSTT trial was designed to compare electrophysiology-guided therapy and no active treatment in high-risk patients with asymptomatic non-sustained ventricular tachycardia. The 2,202 patients with previous myocardial infarction and ejection fraction of 40% or less underwent electrophysiology study before randomization. The non-inducible patients (65%) were followed-up in the registry. The 704 inducible patients (35%) were randomized to receive conventional therapy with no antiarrhythmics or electrophysiologically guided treatment. Patients who remained inducible despite class IA antiarrhythmics, propafenone, or sotalol were randomized to ICD or further drug testing until all patients received either an ICD or an effective drug. At 5 years of follow-up, mortality was 9% in patients who received ICD, 34% in patients treated with an effective drug guided by electrophysiology study, and 32% in patients randomized to no antiarrhythmics. The MUSTT trial confirmed that post-infarct patients with depressed left ventricular function and non-sustained ventricular tachycardia were at high risk for arrhythmic death, and ICD was superior to both electrophysiologically guided therapy and no active treatment.

Both the MADIT and MUSTT trials studied the effect of device therapy in selected high-risk post-infarct populations. Apart from left ventricular dysfunction, non-sustained ventricular tachycardia and inducibility at electrophysiology study were key eligible criteria. To go one step further, in MADIT II, patients with prior myocardial infarction and an ejection fraction of 30% or less were studied.[3] The result of this landmark ICD primary prevention trial was announced in the Annual Scientific Session of the American College of Cardiology this year. Among 1,232 patients with prior myocardial infarction (more than 1 month) and left ventricular ejection fraction of 30% or less were randomized to receive an ICD or conventional medical

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therapy in a 3:2 ratio. No non-sustained ventricular arrhythmias or electrophysiology study was required for enrollment. During an average follow-up of 20 months, the mortality in the ICD group was 14.2% and that of the control group was 19.8%. This study demonstrated that prophylactic ICD offered significant survival benefit in patients with advanced left ventricular dysfunction after myocardial infarction. Subgroup analysis showed that the benefit was greater in patients with age less than 60 years or QRS duration more than 0.15 sec.

There is robust evidence to support that prophylactic device therapy offers survival benefit to patients at high risk of sudden death. However, this therapy is not entirely free of complications. The cost involved is also substantial. In the US, it is estimated that there are approximately 400,000 new MADIT II-alike patients annually. In Hong Kong, if 10% post-infarct patients become eligible for ICD because of the MADIT II data, there will be approximately 300 more implants yearly (Hong Kong Acute Myocardial Infarction Registry data, 1995). The annual cost is nearly 50 million dollars, an amount that is greater than the total sales value of all the ACE inhibitors and AII antagonists used in Hong Kong last year. This does not take into account the cost of replacement of these devices that have an average longevity of 5 years. When the result of the ongoing cost-effectiveness analysis is available, we may have a better picture of how practical it is to apply the MADIT II evidence in our clinical practice. In the long run, it is anticipated that market force and competition may eventually drive down the cost of ICD. In the mean time, prophylactic ICD implantation should be seriously considered in patients with prior myocardial infarction and advanced left ventricular dysfunction, especially in those with a younger age or a wide QRS complex on surface electrocardiogram.

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Predictor of High-Risk Patients after Acute Myocardial Infarction by Serial Echocardiography

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ZHANG ET AL.: ***Predictor of High-Risk Patients after Acute Myocardial Infarction by Serial Echocardiography.*** *In order to distinguish between the low and high-risk patients for ventricular remodeling after acute myocardial infarction (AMI) and to determine whether an early predictor of progressive ventricular dilatation and chronic dysfunction could be identified, 70 consecutive patients undertook serial echocardiography on the entry, 3 days, 2 weeks, 3 and 6 months after admission. The results showed that variables influencing the pattern of remodeling are the location of AMI, initial endocardial surface area index and abnormal wall motion area and reperfusion of infarcted-related artery. Increased endocardial surface area index within 48 hours of onset of AMI can be considered as an echocardiographic manifestation of infarct expansion, and was proposed an early predictor for high risk patients after AMI. (J HK Coll Cardiol 2002;10:100-104)*

Echocardiography, Myocardial infarction, Ventricular remodeling

摘要

探討急性心肌梗塞後心室重構的高低危患者和決定梗塞區膨展是高危患者的早期預測指標。7 0 例連續患者均在入院後 24 小時內、72 小時、2 周、3 個月和 6 個月分別進行超聲心動圖檢查。結果顯示：梗塞部位、入院時心臟大小和梗塞區面積及梗塞相關血管再通是決定心室重構類型的因素。入院後 48 小時內心臟進行性增大是梗塞區膨展的臨床特徵，也是高危患者的早期預測指標。

關鍵詞：超聲心動圖　心肌梗塞　心室重構

Introduction

Two dimensional echocardiography has become established as an ideal noninvasive method to identify and quantitate regional left ventricular morphology that accompanies acute myocardial infarction (AMI).[1,2] A number of studies have emphasized the acute structure and functional changes were dynamic and varied with the size and location of the infarction. In addition, dilatation of the left ventricle might play an important and active role in the development of chronic heart failure, and left ventricular volume was the most powerful predictor of survival in patients with coronary artery disease.[3] Deterioration of cardiac performance correlated with the degree of dilatation in patients with AMI. However, the time course and interaction of regional function of noninfarcted and infarcted myocardium and global left ventricular dysfunction had

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not been evaluated in detail. The purpose of this study was to use computerized echocardiography to prospectively determine the natural history of left ventricular size, area of abnormal wall motion (AWM), i.e. area of functional infarct size, and ventricular function, to distinguish between the low and high risk patients for ventricular remodeling from entry until 6 months after AMI, and to determine whether an early predictor of progressive ventricular dilatation and chronic dysfunction could be identified.

Materials and Methods

Study Group

Patients with a first Q wave AMI were prospectively screened for inclusion into the study. Q wave AMI was defined by the presence of chest pain lasting at least 30 minutes, new ST segment elevation in ECG, appearance of new Q waves on serial electrocardiograms, and a significant rise in creatine kinase (CK) and cardiac isoenzyme fraction (CK-MB). Patients with previous myocardial infarction, primary valvular heart disease, cardiomyopathy, the onset of symptoms more than 48 hours before entry were excluded from the study. In addition, patients with echocardiogram of inadequate quality for quantitative analysis were also excluded. Seventy of the 73 patients completed the study, including 3 patients not being followed up (follow-up rate 95.4%), Eight patients (7 anterior and 1 inferior; mean age 64±6.2 years) died during follow-up (11%). Six months follow-up were completed in 62 patients, 42 of that had anterior infarction, 20 had inferior infarction (male 50 and female 12, mean age 59±11.6 years). Patients were in hospital within 12 hours after disease onset and treated with thrombolytic therapy. Sixteen (11 anterior and 5 inferior, mean age 59.8±10.1 years) of 62 patients had patency of infarct-related coronary artery, which was assessed by uniform clinical criteria (receiving thrombolytic therapy) or by coronary angiography (receiving coronary angioplasty or/and coronary stents). Thirty normal volunteers served as normal controls. This group consisted of 22 men and 8 women (mean age 60.9±15.3 years). Twenty-four of the 70 patients had cardiac events such as cardiogenic death, persistent ventricular tachycardia, ventricular fibrillation, symptomatic heart failure and post-infarction angina pectoris.

Data Acquisition of Echocardiography

Two dimensional echocardiography was performed prospectively with a 2.5 mHZ transducer using a phased array system (HP1000 or HP2500) by one sonographer at entry, 3 days, two weeks, 3 and 6 months after first AMI. Echocardiographic images of the left ventricle were obtained from five standard imaging planes-parasternal short axis views at mitral valve, papillary muscles and apical level as well as apical four chamber and two chamber views.

Measurement of Endocardial Surface Area Index of Left Ventricle and AWM

Suitable images from the five recorded planes (three short axis views, two and four chamber views) were used to derive the cardiac dimensions including long axis and endocardial length of left ventricle, and the endocardial length of abnormal wall motion segment. The endocardial surface area of left ventricle and AWM area were calculated with a computerized data process system reported and validated previously.[4,5] Because body surface area has been identified as a primary determinate of normal left ventricular dimensions, the endocardial surface area of left ventricle was corrected for the body surface area. This corrected endocardial surface area, i.e., ESAi, and AWM area were used in analyses for between-patients comparisons.

Standards of Ventricular Size

Thirty normal volunteers without coronary artery disease were used as controls. This group, mean age 60.9±15.3 years, consisted of 22 man and 8 women. The mapping technique was then applied to generate a normal range for endocardial surface area. The range of normal ESAi was 63.7±5.1 cm^2/m^2. An ESAi of more than 2 standard deviation above the control group were defined as ventricular enlargement.

Standards of Infarct Expansion

Infarct expansion was defined as left ventricular enlargement, wide-mouthed, thinned-walled myocardial segments that display dyskinetic expansion during systole.

Statistic Analysis

Data were expressed as mean±SD. Statistical comparisons between groups were made by analysis of

variance (ANOVA). Comparisons of frequency of events were performed by x^2 analysis.

Results

Serial Echocardiographic Quantitative Analysis

After AMI, the changes of left ventricle and the infarct area had different trends during 6 months. The effects on left ventricular ejection fraction (EF) value (measured by Simpson's equation) were also different. Based on the data shown in Table 1, patients were divided into four subgroups: (1) 20 had a normal ESAi on admission, no changes during 3 months after AMI, a decrease in left ventricular size at 6 months ($P<0.05$). Decrease in infarct area was noted from entry to 6 months after AMI ($P<0.01$), without EF reduced; (2) 27 had progressive left ventricular dilatation from entry to 3 months after AMI ($P<0.05$), decreased in left ventricular size and infarct area from 3 to 6 months ($P<0.01$), and reduction in EF value ($P<0.01$); (3) 15 had limited dilatation with subsequent reduction. Whether left ventricular dilatation or not at entry, no change in ESAi at two weeks, but decrease in the left ventricular size noted from 2 weeks to 6 months after AMI, and progressive decrease in infarct area from entry to 6 months, without significant reduction in EF value; (4) 7 had an enlarged ESAi at entry, accelerated left ventricular dilatation within 72 hours after AMI. All the patients had infarct area extended greatly with typical manifestation of infarct expansion, and large reduction in EF value. Those 8 patients died in hospital except one were all in this pattern.

Influencing Factors of Ventricular Remodeling after AMI

Based on the size of infarct area, ESAi within 48 hours after AMI and early reperfusion, various remodeling process could be analyzed as follows: In the patients with no dilatation of left ventricle, AWM area was the smallest. Most of them had inferior AMI. Left ventricular size was unchanged except in two patients (ESAi=74 cm^2/m^2). Six of the 20 patients had early reperfusion.

In the group with progressive dilatation of left ventricle, most patients had anterior AMI, 12 of whom

Table 1. Natural history of ESAi, area of AWM, and EF value

Time	Group 1 n=20	Group 2 n=27	Group 3 n=15	Group 4 n=7
ESAi				
24 hours	66.5±5.5	73.8±8.2	72.5±11.2	79.4±9.2
72 hours	67.7±6.3	76.6±9.2	74.3±11.0	91.1±7.7*
2 weeks	69.5±8.6	77.3±8.4	71.1±8.7	
3 months	65.5±9.0	79.6±9.4*	65.2±7.9*	
6 months	62.1±7.5*	69.9±7.9##	61.8±6.7**	
AWM area				
24 hours	24.7±3.2	55.3±12.6	55.1±9.0	66.4±11.3
72 hours	24.6±3.1	56.0±13.7	54.1±9.6	79.7±10.3*
2 weeks	22.6±4.2	56.8±14.4	46.0±7.0**	
3 months	20.4±4.9**	61.0±15.5	42.9±7.0	
6 months	17.0±4.8#	43.2±9.9 **##	38.5±7.1**	
EF				
24 hours	0.47±0.05	0.45±0.07	0.47±0.05	0.42±0.01
72 hours	/	/	/	0.34±0.08*
2 weeks	0.48±0.06	0.44±0.08	0.47±0.08	
3 months	0.47±0.07	0.44±0.05	0.48±0.06	
6 months	0.52±0.06	0.39±0.07**##	0.51±0.06	

Group 1: LV no changes; group 2: progressive dilatation; group 3: progressive decreased; group 4: accelerated ventricular dilatation

*compared with at entry $P<0.05$, **compared with at entry $P<0.01$; #compared with at 3 months $P<0.05$, ##compared with at 3 months $P<0.01$

had initial left ventricular dilatation at entry, with AWM area larger than those without initial dilatation (P<0.001).

In patients with progressive decrease in size of left ventricles in the presence of anterior AMI, 7 patients had initial dilatation, with AWM area larger than those without initial dilatation (P<0.001). All patients had early reperfusion.

All seven patients with accelerated left ventricular dilatation within 72 hours after AMI had initial dilatation and anterior AMI. None had early reperfusion, but they had the most significant left ventricular dilatation and the largest AWM area. The initial left ventricular size and AWM area between progressive dilatation group and progressive decrease group had no significant difference.

Influence on Cardiac Function and Prognosis by Ventricular Remodeling

Patients with accelerated ventricular dilatation in 72 hours after AMI had the lowest initial EF value at entry (P<0.05), which continued to further decrease within 72 hours of onset of AMI. Seven of those patients were dead in hospital. Six months after AMI, EF value in progressive dilatation group was the lowest and differed significantly from the other two groups (P<0.05). EF value was much lower in patients with initial dilatation than without initial dilatation (P<0.01).

Twenty-four of the 70 patients had cardiac events, including 7 patients with accelerated ventricular dilatation within 72 hours after AMI, 14 patients with progressive ventricular dilatation , 2 patients with no ventricular dilatation, and 1 patient with progressive ventricular size reduction. There were 19 patients with anterior AMI with initial dilatation but without early repefusion at entry (12 from ventricular progressive dilatation group, 7 from accelerated ventricular dilatation group), 15 of which had cardiac events (7 died in hospital, 3 had persistent ventricular tachycardia, 6 had symptomatic heart failure, 1 had post-infarction angina pectoris).

Discussion

Significance of Discrimination of Left Ventricular Remodeling by Echocardiography

Our study demonstrated that there were various manifestations of ventricular remodeling, such as no significant changes in ventricular size, progressive dilatation, limited dilatation with subsequent reduction and accelerated ventricular dilatation in 72 hours, which had different effects on left ventricular function and prognosis. The infarct size was critical to ventricular remodeling. Patients with larger anterior infarct area tended to have the higher risk of infarct expansion and progressive dilatation, while those who had smaller infarct areas of inferior infarction had the lower tendency. Measurement of ESAi and AWMA area by echocardiography could serially assess the process of ventricular remodeling accurately, and distinguish between the low and high risk patients for ventricular remodeling for intervention. Variables influencing the pattern of remodeling were the location of AMI, initial ESAi and AWMA size and reperfusion of infarct-related artery.

Assessment of Infarct Expansion by Echocardiography

Infarct expansion may be responsible for left ventricular enlargement in the early stages after AMI, and contribute to the ventricular remodeling in all stages. This occurred in patients with large infarct area. Ventricular remodeling after AMI is an important process affecting ventricular function and survival. Therefore, infarct expansion was the most powerful predictor of high risk patients with ventricular remodeling after AMI. Gaudron et al demonstrated that infarct expansion was present in 50% of patients with anterior infarctions,[6] but only 7 of 44 anterior infarctions had infarct expansion in our study. There were 26 patients of anterior AMI with initial dilatation but without early reperfusion at entry, most of whom had cardiac events. Thus, we believed that increased ESAi within 48 hours of onset of AMI can be considered an echocardiographic manifestation of infarct expansion, was suggested to be used as an early predictor for high risk after AMI.

The relationship between reperfusion and prognosis at the early stage after AMI. Early reperfusion after AMI can reduce infarct area, prevent infarct expansion and subsequent left ventricular dilatation. This study demonstrated that there were 15 anterior AMI with early reperfusion, 7 of whom had infarct expansion at entry, infarct area decreased significantly at 2 weeks after AMI, left ventricular size decreased and EF value

increased markedly at 3 months (0.47±0.06 VS 0.53± 0.04, P<0.05). Thus, echocardiography can serially and accurately assess dynamic changes of infarct expansion and ventricular remodeling after AMI.

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Role of Signal-Averaged ECG in Predicting Results of Flecainide Provocation Test Used in Family Screening for Brugada Syndrome

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MOK ET AL.: ***Role of Signal-Averaged ECG in Predicting Results of Flecainide Provocation Test Used in Family Screening for Brugada Syndrome.*** *Brugada syndrome (BS) is an inherited arrhythmogenic disease with an autosomal dominant mode of inheritance. Flecainide provocation test (FPT) has been shown to be highly sensitive and specific in unmasking the Brugada ECG pattern in affected subjects. We sought to test if late potential (LP) in signal-averaged electrocardiogram (SAECG) is helpful in predicting the results of FPT used in family screening for BS. The study included 17 asymptomatic Chinese subjects from 8 families (M:F 10:7, mean age 24.8±11.4 years) who have undergone family screening for BS. All screened subjects had a normal 12-lead ECG at baseline. None had structural heart disease. SAECG using a time domain analysis was recorded prior to the FPT. LP is defined as positive when at least 2 of the 3 criteria are met: (1) filtered QRS duration >114ms; (2) root-mean square voltage of terminal 40ms of QRS ≦20μV; (3) low-averaged signal <40μV of terminal QRS ≧38ms. Seven subjects had a positive LP on SAECG. Among them 3 had a positive FPT. As for the 10 subjects with a negative LP, none had a positive FPT. Thus in predicting the results of FPT among these subjects, LP on SAECG has a sensitivity, specificity, positive predictive value and negative predictive value of 100%, 76.9%, 42.8% and 100% respectively.* ***Conclusion:*** *LP on SAECG has a high sensitivity and negative predictive value in predicting results of FPT used in family screening for BS. (J HK Coll Cardiol 2002;10:105-108)*

Brugada syndrome, flecainide, signal-averaged electrocardiography, ST-segment elevation

摘要

Brugada 綜合征（BS）是一個遺傳性心律失常性疾病，伴有一個常染色體顯性遺傳模式。氟卡尼激發試驗（FPT）在顯露受累個體 Brugada ECG 波形方面有著很高的敏感性和特異性。我們試圖檢驗單信號平均 ECG（SAECG）中的晚電位（LP）是否有助於預測應用于家族篩查 BS 的 FPT 試驗結果。該研究包括 17 例已進行家族 BS 篩查的來自於 8 個中國家庭的無症狀個體（M:F 10:7，平均年齡 24.8±11.4 歲）。所有篩查的個體均有正常的基礎 12 導聯心電圖。均無器質性心臟病。SAECG 應用時域分析並在 FPT 前進行記錄。LP 陽性被定義為至少滿足下列 3 個條件中的 2 個（1）濾波的 QRS 間期大於 114 毫秒（2）濾波疊加後 QRS 終末 40 ms 振幅的均方根 ≤20 μV（3）QRS 終末部振幅低於 40 μV的時間≧38 毫秒。7 位元個體 SAECG 顯示 LP 陽性。其中 3 個 FPT 陽性。另 10 位 LP 陰性，FPT 均陰性。在這些個體中預測 FPT 試驗結果，SAECG 中 LP 的敏感性、特異性、陽性預測值和陰性預測值分別為 100%、76.9%、42.8%和 100%。結論：SAECG 中的 LP 在預測用於家族篩查 BS 的 FPT 結果方面有著較高的敏感性和陰性預測值。

關鍵詞：Brugada 綜合征　氟卡尼　單信號平均 ECG　ST 段抬高

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Introduction

Brugada syndrome is a genetically determined primary electrical disease characterized by a marked ST-segment elevation in leads V_1-V_3 on electrocardiogram (ECG) during sinus rhythm and a propensity for life-threatening ventricular tachy-arrhythmias.[1] Flecainide provocation test (FPT) has been shown to be highly sensitive and specific in unmasking the Brugada ECG pattern in subjects with a concealed form of the disease[2] and therefore frequently used in family screening for the syndrome. Late potential on signal-averaged electrocardiogram (SAECG) is frequently recorded in patients with Brugada syndrome[3] but only rarely found among healthy subjects. We therefore sought to study if SAECG is helpful in predicting the results of FPT used in family screening for Brugada syndrome in our local Chinese population.

Methods

In Princess Margaret Hospital (PMH), FPT has been used to identify Brugada syndrome among family members of symptomatic patients with the syndrome if their baseline ECG is normal or non-diagnostic. SAECG using a time domain analysis is recorded and echocardiogram performed. This is followed by a FPT if no structural heart disease is found on echocardiogram. LP is defined as positive when at least 2 of the 3 criteria are met: (1) filtered QRS duration >114 ms; (2) root-mean square voltage of terminal 40 ms of QRS $\leqq$20 μV; (3) low-averaged signal <40 μV of terminal QRS $\geqq$38 ms. After an informed consent is obtained, FPT is performed in Cardiac Care Unit under continuous cardiac monitoring. Flecainide at a dosage of 2 mg/kg (maximum 150 mg) is given intravenously over 10 minutes. Leads V_1 and V_2 are recorded in the 2nd and 3rd intercostal spaces in addition to the conventional 4th intercostal space which has been shown to enhance the sensitivity of FPT.[4] A positive FPT is defined as occurrence of terminal R wave and >1 mm ST segment elevation in leads V_1-V_3 following flecainide infusion. Subjects with a positive FPT will be monitored for 6 hours or until the ST segment in leads V_1-V_3 returns to baseline. Sensitivity, specificity, positive and negative predictive values of SAECG in predicting the results of FPT are calculated.

Results

From February 1999 to December 2001, a total of 17 asymptomatic Chinese subjects (M:F 10:7) with a mean age of 24.8±11.4 years have undergone family screening for Brugada syndrome in PMH. They came from 8 families. Each family had one proband member suffering from Brugada syndrome (Ventricular fibrillation in 5, recurrent syncope in 2 and asymptomatic in 1). All 17 subjects had a normal 12-lead ECG at baseline. None had structural heart disease found on echocardiogram. Of these 17 subjects, 7 had a positive LP on SAECG. Among these 7 subjects 3 had a positive FPT (Figure 1A) while 4 had a negative FPT. Among the 10 subjects with a negative LP, none had a positive FPT (Figure 1B). Thus in predicting the results of FPT among these subjects, LP on SAECG has a sensitivity, specificity, positive predictive value and negative predictive value of 100%, 76.9%, 42.8% & 100% respectively. Two subjects complained of dizziness during flecainide infusion which rapidly subsided upon completion of the test. Transient unifocal ventricular premature beats were recorded in 1 subject. No bradyarrhythmia or tachyarrhythmia was found in any subject during FPT.

Discussion

Brugada syndrome is potentially life-threatening disease. Symptomatic patients may have up to a 62% chance of arrhythmia recurrence over a 4-year follow-up.[5] As this syndrome is an inherited disease with an autosomal dominant mode of inheritance, it is therefore essential to screen their family members who may also be affected and therefore predisposed to life-threatening ventricular arrhythmias. Genetic data has linked this syndrome to mutations in cardiac sodium channel gene SCN5A in chromosome 3.[6,7] However SCN5A mutation can be found in only 15-25% of patients with this syndrome.[8] Genetic screening for SCN5A mutation is therefore an insensitive test in family screening to identify Brugada syndrome unless SCN5A mutation is

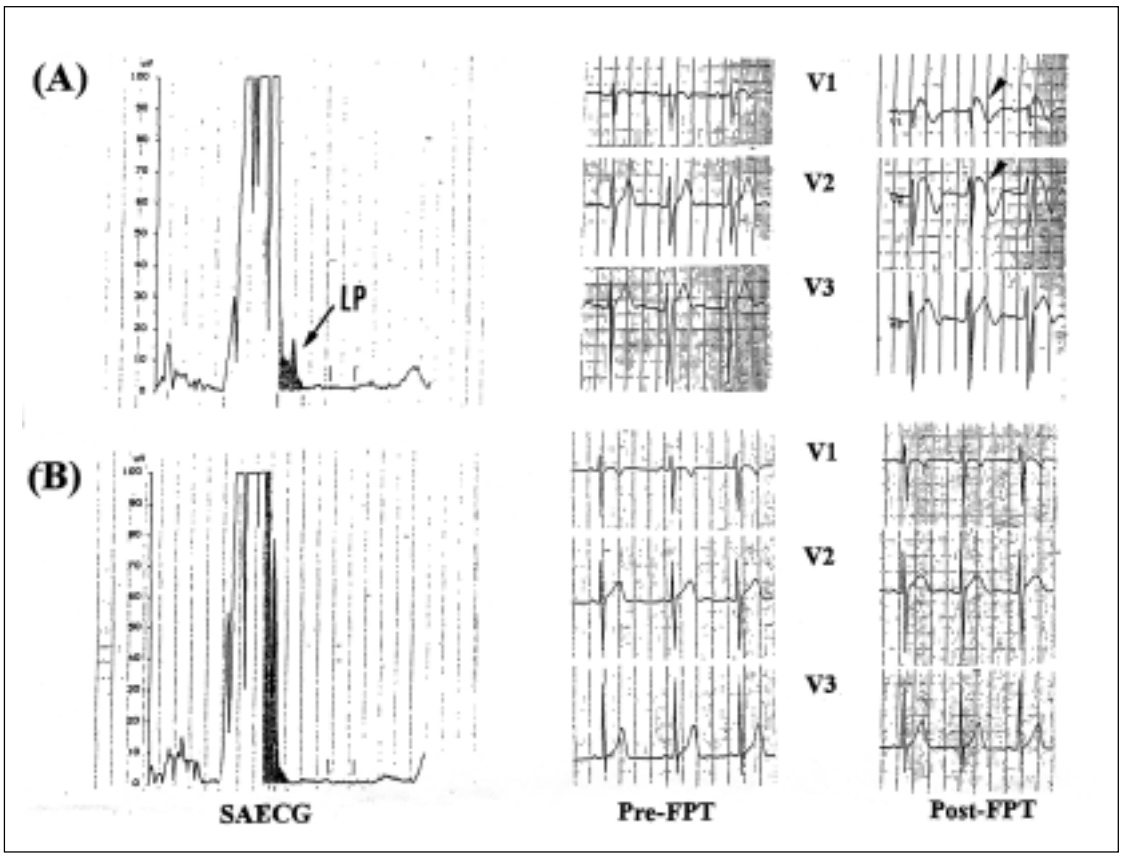

Figure 1. *Late potential (LP) on signal-averaged ECG (SAECG) predicting results of flecainide provocation test (FPT) in family screening for Brugada syndrome (see text). (A) Positive FPT evidenced by coved type ST-segment elevation (arrowheads) in leads V_1-V_2 following flecainide infusion in a subject with a positive LP on SAECG (B) Negative FPT in a subject with a negative LP on SAECG.*

already documented in probands. Moreover, genetic test for channelopathies is expensive and not easily accessible. Using 12-lead ECG to diagnose Brugada syndrome among family members is easy if the typical Brugada ECG pattern is found on their ECG during sinus rhythm. However, dynamic changes or transient normalization of the ST-segment may obscure the correct diagnosis. Flecainide, a potent sodium channel blocker, has been shown to be highly sensitive and specific in unmasking the syndrome in affected subjects who have normal baseline ECG.[2] It is therefore frequently used locally[9] and world-wide for the purpose of family screening in Brugada syndrome.

LP on SAECG reflects conduction delay in the ventricles. Although at present the role of conduction disturbance in the right ventricle in arrhythmogenesis of the syndrome is a matter of some controversy, it is well known that LP on SAECG is frequently found in patients suffering from Brugada syndrome.[3] LP can be found among patients with Brugada syndrome irrespective of whether the Brugada ECG pattern is manifested or concealed.[10] Ikeda et al[11] studied LP among patients with Brugada syndrome and found that it can even be used as a noninvasive risk stratifier in these patients. To date, there is as yet no study to examine whether LP on SAECG is useful in family screening for the syndrome. In this study, we were able to demonstrate in this small cohort of subjects that LP in SAECG has a 100% sensitivity and negative predictive value in predicting the results of FPT. The

clinical implication of these results is that in conducting family screening for Brugada syndrome, we can start by recording both 12-lead ECG and SAECG. If the 12-lead ECG does not show any Brugada ECG pattern, FPT should be followed to look for any concealed form of the Brugada syndrome. But for those asymptomatic subjects who are reluctant to undergo FPT which requires venipuncture and intravenous drug infusion under close cardiac monitoring, a negative LP on their SAECG should reliably predict a negative FPT and FPT might be spared. However, if a positive LP is found, they should be strongly encouraged to undergo FPT.

This study has the following limitations. Firstly, the number of tested subjects in this study is small. Whether the results in this preliminary study apply to family screening in all patients with Brugada syndrome remains to be clarified in future study which should include a larger number of subjects. Secondly, the definitions of positive LP in SAECG and positive FPT vary among different studies. The results of this study are applicable only in situations where the current definitions used in this study are adopted.

Conclusions

LP on SAECG has a high sensitivity and negative predictive value in predicting the results of FPT used in family screening for Brugada syndrome. SAECG using a time domain analysis should be included as an additional non-invasive diagnostic tool in family screening for Brugada syndrome. Asymptomatic subjects with a negative LP could be reassured absence of the disease and FPT might be spared. However, subjects with a positive LP should undergo FPT to look for any concealed form of the syndrome.

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Safety and Feasibility of Diagnostic Coronary Angiogram as Day Procedure

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FU ET AL.: ***Safety and Feasibility of Diagnostic Coronary Angiogram as Day Procedure.*** *With the miniaturization of diagnostic coronary catheters and meticulous post-procedural nursing care, puncture site complications are much reduced and patients can be mobilized earlier. Diagnostic coronary angiogram as a day procedure has been practiced worldwide. It can shorten hospital length of stay while improving patient satisfaction. During a pilot period of 6 months at Queen Elizabeth Hospital, Hong Kong, we showed that in selected patients, similar procedure could be performed safely and more cost-effectively. (J HK Coll Cardiol 2002;10:109-113)*

Coronary, cost-savings, day procedure, feasibility, safety

摘要

隨著診斷性冠狀動脈導管的微型化，穿刺部位的併發症已大大減少，病人早期進行活動。診斷性冠狀動脈造影作為日間程序已在世界範圍內得以實施。這可以縮短住院時間，同時提高病人的滿意程度。香港伊利沙伯醫院6 個月的小規模研究表明類似的程序能夠安全的施行，並且有著更好的成本效益。

關鍵詞：冠狀動脈　節省資源　日間程序　可行性　安全性

Background

The concept of cardiac catheterization as outpatient is not new. Its safety has been confirmed in certain selected groups of patient.[1] The major advantages of cost savings and time conservation were attractive. The introduction of high-quality, smaller catheters has played an important role in the development of outpatient laboratory procedures nowadays.

To shorten hospital length of stay and to improve quality of care and patient satisfaction, increasing number of cardiac procedures is being done on day-patient basis at Queen Elizabeth Hospital, Hong Kong. This study aims to assess the safety and feasibility of performing diagnostic coronary angiogram as day procedure.

Methods

A pilot study was carried out at the Cardiac Day Center of Queen Elizabeth Hospital, Hong Kong from July to December 2001.

It was known that vascular complications such as haemorrhage, haematoma and AV fistula formation, pseudoaneurysm and arterial thrombosis might occur following cardiac catheterization. The primary responsibility of the medical and nursing staff is to ensure proper patient selection and to prevent postoperative complications that might be dangerous and associated with patient discomfort. Therefore, special guidelines for selection of day-cath patients, nursing management for optimal care after cardiac

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catheterization were designed (Table 1).[2]

During this pilot study, patients undergoing diagnostic coronary angiogram, with a view to ad hoc angioplasty, done by 1-2 assigned operators were assessed for suitability as day procedure. They will be interviewed by CCU on-duty doctor on the day of preparation. The nurse of the day center would explain all the details of the day procedure and its after care to the patients and their relatives. All suitable day patients fulfilling the selection criteria and with none of the exclusion criteria would be arranged as first or second cases on day of procedure. The operator of the procedure would decide after the angiogram whether the patients were suitable for day procedure or not.

The patients were recruited if they had normal or minor lesions, no active chest pain or symptoms of CHF, easily palpable femoral arteries with no evidence of peripheral vascular disease, adequate blood pressure control for hypertensive patients, adequate diabetic control for DM patients, independent activity of daily

Table 1. Nursing management

Immediately post-operative nursing management after cardiac catheterization

Back from cardiac catheterization lab at _____, BP _____, P ____.
Size of catheter used 4/5/6 Fr.
Total heparin used _____ units.
Circulation and pulse of the affected L/R, Limb/arm is normal/impaired/absent.
Wound oozing: Y/N
Haematoma: Y/N
Chest pain: Y/N
Dyspnoea: Y/N
The head of the bed is elevated to 30 degree / not elevated during bed rest and sand bag is applied: Y/N
Patient resumes diet at _____ and fluid intake is encouraged if no contraindication.
If complication is detected, Dr. _____ is informed at _____.

Rank and Name: _____

Ambulatory period

The patient is allowed to sit out and slowly mobilize after 4 hours of the procedure at _____, BP _____, and P _____.
Wound oozing: Y/N
Any complications: Y/N
Dr. _____ is informed at _____.
Advise on wound management and possible complications by _____.

Rank and Name: _____

Discharge

Patient is discharged at _____(at least 6 hours after procedure) after checking the wound by Dr. _____.
Information sheet on " wound management and complication" is given to him/her.
The patient is discharged alone/accompanied by _____.

Rank and Name: _____

living, accompanied by family members or relatives after discharge, and home with lift to floor. Those patients were excluded if they had ad hoc angioplasty procedure done, significant coronary artery lesions requiring further treatment, active bleeding or known bleeding tendency, marked renal impairment with creatinine >200 umol/L, obvious dehydration, were on warfarin, lived alone with poor social support, or lived far away from hospital.

Sheaths were removed right after the procedure by manual compression until haemostasis achieved. Then a sandbag would be applied over the groin wound for 2-4 hours. The patients started to ambulate 4 hours later[3] and were discharged in 6 hours if wound haemostasis was adequately achieved. Written instructions detailing the wound care and backup medical support were given prior to discharge. They were advised to attend a follow-up session the next morning for wound inspection. Any complications were then recorded.

Results

Out of a total of 125 elective procedures done by the assigned operator during this period, 84 were excluded either because they had ad hoc angioplasty procedure done or did not meet the inclusion criteria. Of the 41 eligible patients, two requested to stay overnight because of personal reasons and one was admitted to ward when wound haemostasis could not be achieved after 8 hours. As a result, a total of 38 patients with normal angiogram or minor CAD were recruited. Twenty-one of them were males and 17 females. Their age ranged from 28 to 71 years (52.3, mean). All of them had stable angina with positive stress test. Four French sheaths were used in 32 patients, 5F in 4 and 6F in 2. No heparin was given during the procedure in 37 patients while 1000 units of heparin were given in one. No major complications were observed and no patients presented to the hospital again the night after the procedure. Clinical follow up was done the next day in all but two of the patients. Mild bruises were observed in 6 patients but with no haematoma, pseudo-aneurysm or AV fistula formation and no major bleeding requiring blood transfusion was seen. All patients were symptom free on subsequent follow up (Table 2). All patients expressed satisfaction with early discharge without staying overnight in the hospital. There was no readmission related to the procedure.

Discussions

This pilot study shows that day-patient diagnostic coronary angiogram can be safely performed if we can identify certain patient subgroups with low risk characteristics. It is especially true with the availability of the new high-quality 4F catheters with bigger inner lumen. These give similar opacification and torquability compared to the 5F or 6F catheters (reported in an abstract form in the coming Tenth Annual Scientific Congress of the Hong Kong College of Cardiology) and it is easier to handle the puncture site wound.

In a number of cardiac centers overseas and locally, heparin is not routinely given during diagnostic catheterization procedures. In our cohort of patients, only one was given intra-arterial heparin injection deemed necessary by the operator. It was not given in 37 of the 38 patients, with no increase risk of thromboembolism. This also helps to achieve a better wound haemostasis after the procedure.

It is worth mentioning that meticulous nursing care with clear nursing management protocol is essential to ensure the safety of the day procedure and adequate haemostasis of the wound. Clear written instruction on how to handle subsequent wound complications and backup medical support greatly improves patient confidence and satisfaction.

In the study, all patients were requested to come back for wound inspection the next morning to document the possible wound complications. They were more willing to come back the next morning to staying in hospital overnight. Two patients did not attend the wound inspection session because of typhoon signal number 8 and heavy rain the subsequent day. Both of them were enquired through telephone. Provided with such a low risk of wound complications, we believe the next-morning-wound-inspection session is not mandatory in future full-scale service as long as the patients are given proper instructions of wound care and channels to report any suspicions.

With such a limited number of patients recruited in the study, a total of 66 days of hospitalization were saved (assuming each patient stayed in hospital for 1.5

Table 2. Characteristics, catheter size and incidence of post-cath. complication of 38 patients

Case no.	Sex	Age	Reason for coro	Time to ambulate (hr)	Time to discharge (hr)	Size of catheter (Fr)	Unit of heparin	Complications	Remarks
1	M	45	+ve TMT	4	6	4	0	-	-
2	M	42	+ve TMT	4	6	4	0	-	Typhoon No. 8
3	F	58	+ve TMT	4	6	4	0	-	Heavy rain
4	M	67	+ve TMT	4	6	4	0	-	-
5	M	52	+ve TMT	4	6	4	0	-	-
6	F	57	+ve TMT	4	6	4	0	-	-
7	F	49	+ve TMT	4	6	4	0	-	-
8	F	68	+ve TMT	4	6	5	1000	-	-
9	F	50	+ve TMT	4	6	4	0	-	-
10	F	64	+ve TMT	4	6	4	0	Mild bruise	-
11	M	32	+ve TMT	4	6	4	0	-	-
12	F	28	+ve TMT	4	6	4	0	Mild bruise	-
13	F	51	+ve TMT	4	6	4	0	-	-
14	M	65	Chest pain	4	6	4	0	-	-
15	M	51	+ve TMT	4	6	4	0	-	-
16	M	57	+ve TMT	4	6	4	0	-	-
17	F	68	+ve TMT	4	6	4	0	-	-
18	M	54	+ve TMT	4	6	5	0	Mild bruise	-
19	M	71	+ve TMT	4	6	5	0	-	-
20	M	43	+ve TMT	4	6	4	0	-	-
21	M	61	+ve TMT	4	6	5	0	-	-
22	F	50	+ve TMT	4	6	4	0	-	-
23	F	48	+ve TMT	4	6	4	0	-	-
24	F	65	+ve TMT	4	6	4	0	-	-
25	M	51	+ve TMT	4	6	4	0	-	-
26	M	53	+ve TMT	4	6	4	0	-	-
27	M	53	+ve TMT	4	6	4	0	-	-
28	M	64	+ve TMT	4	6	4	0	-	-
29	M	38	+ve TMT	4	6	4	0	-	-
30	F	57	+ve TMT	4	6	4	0	-	-
31	F	60	+ve TMT	4	6	6	0	Mild bruise	-
32	F	62	+ve TMT	4	6	4	0	-	-
33	F	52	+ve TMT	4	6	4	0	Mild bruise	-
34	M	58	+ve TMT	4	6	6	0	-	-
35	F	53	+ve TMT	4	6	4	0	-	-
36	M	62	+ve TMT	4	5	4	0	Bruise	-
37	M	53	+ve TMT	4	6	4	0	-	-
38	M	32	+ve TMT	4	6	4	0	-	-

days) while patient satisfaction was enhanced with early discharge. This translates into a significant amount of hospital cost savings.

With the demonstrated safety, feasibility, cost-effectiveness and better patient satisfaction, we are proposing full-scale implementation of day catheterization as a routine for diagnostic coronary angiogram and cardiac catheterization in properly selected group of patients.

Conclusion

Diagnostic coronary angiogram as day-procedure is safe, feasible and cost-effective in selected patients. The risks are minimal. Early ambulation is safe after uncomplicated cardiac studies with 4-6F femoral arterial catheters in elective catheterization.

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Percutaneous Revascularisation in Diabetic Patients with Coronary Artery Disease

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CHEN and LAU: ***Percutaneous Revascularisation in Diabetic Patients with Coronary Artery Disease.*** *Diabetic patients with coronary artery disease are associated with an unfavourable long-term outcome. Surgical revascularisation on top of medical therapy has been shown to confer survival benefit in patients with mutivessel and left main disease. With the emergence of the percutaneous approach of revascularisation, several clinical trials have been conducted to compare these two modalities of treatment. Consistent results were demonstrated in the diabetic subgroup that coronary artery bypass grafting is superior to balloon angioplasty in preventing late adverse cardiac event. With the advancement of percutaneous technique using coronary stenting as the predominant mode of catheter-based revascularisation, the latest comparison trial showed that stenting is a comparable alternative but is associated with a higher incidence of repeat revascularisation than bypass surgery. However, the debate is not yet settled because of the promising results of platelet glycoprotein IIb/IIIa antagonists and drug-eluting stents in improving the safety and efficacy of percutaneous intervention. It remains to be proven by ongoing clinical trials the best revascularisation strategy for diabetics with multivessel coronary disease. (J HK Coll Cardiol 2002;10:114-119)*

Coronary artery disease, diabetes mellitus, revascularisation

摘要

患有冠狀動脈疾病的糖尿病病人，其長期預後不理想。對於該類共並發有多發性血管疾患和以此為主要症狀的病人，外科血管再通的治療將給他們的生存帶來益處。隨著經皮血管再通技術的興起，一些臨床研究對比了二種治療方法。過去的研究結果一致表明在糖尿病組對於預防晚期心臟不良事件中，運用冠狀動脈搭橋術要優於氣囊血管形成術。隨著經皮穿刺技術的提高，冠脈支架已成為基於導管技術的血管再通的優越手段。最近的臨床對比研究表明，支架技術是可供選擇的治療手段，但它比冠狀動脈搭橋術則需要更多的反復血管再通。然而對此的爭論並未了結，原因在於血小板糖蛋白 IIb/IIIa 拮抗劑誘人結果和藥物洗脫支架在經皮介入治療的安全性和有效性的提高。期待進一步的臨床實驗以證實患有冠狀動脈疾病的糖尿病病人血管再通的最佳方案。

關鍵詞：冠狀動脈疾病　糖尿病　血管再通

Following randomised clinical trials (RCTs) in the 70's that showed the benefits of coronary artery bypass graft (CABG) over medical treatment alone in certain subsets of patients with multivessel coronary artery disease (CAD) and left main stem disease, CABG has been regarded as the "standard" treatment for these patients. With improvements in expertise and technology, percutaneous transluminal coronary angioplasty (PTCA) evolves from the treatment of simple, focal, proximally located lesions in a single vessel to those complex anatomic subsets previously tackled only by cardiac surgeons.

In the late 1980's, six RCTs were undertaken to compare PTCA and CABG as the revascularisation

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procedure in patients with multivessel CAD. The main results were consistent among the trials. Mortality was not significantly different between the two modalities of treatment (Figure 1). Fewer repeat revascularisations and recurrent angina were achieved by CABG. A trend of reduction of myocardial infarction (MI) was seen in patients undergoing PTCA.[1-6] However, there are two exceptions to the overall trial results. The Bypass Angioplasty Revascularization Investigation (BARI) trial showed that treated diabetics had a distinct survival advantage with CABG. The benefit was seen 6 months after randomisation and the survival curve continued to diverge up to 7 years of follow-up. Survival was 76.4% for the CABG group and 55.7% for the PTCA group at 7 years (p=0.0011).[7] The Emory Angioplasty Versus Surgery Trial (EAST) also showed a trend of better survival among diabetics treated with CABG. Eight-year survival was 75.5% in the CABG group and 60.1% in the PTCA group.[8]

Poor Outcome of Diabetics After Revascularisation

Diabetes mellitus is associated with the early initiation and accelerated progression of coronary atherosclerosis. Diabetics constitute a significant proportion of patients requiring revascularisation for the treatment of advanced coronary artery disease. Patients with diabetes are at increased risk of adverse outcome after PTCA. In the early era of balloon

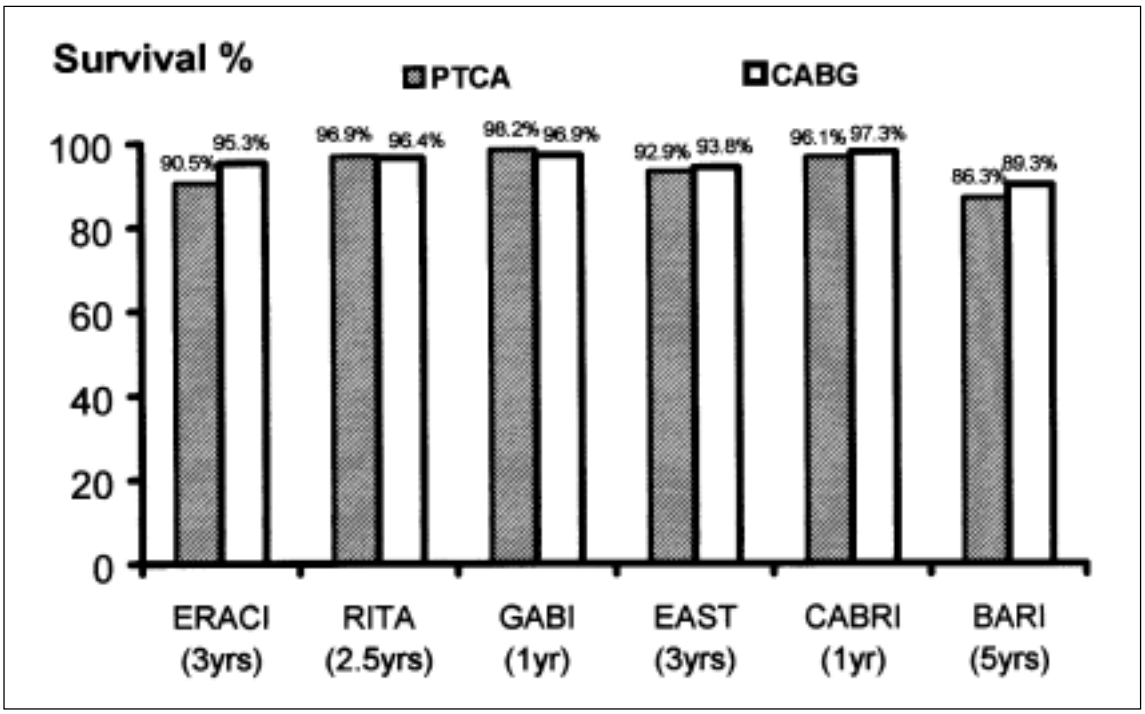

Figure 1. *Survival of diabetic patients with multivessel coronary artery disease after percutaneous transluminal coronary angioplasty or coronary artery bypass graft surgery in randomized controlled trials. BARI = Bypass Angiolasty Revascularization Investigation; CABG = Coronary Artery Bypass Graft; CABRI = Coronary Angioplasty versus Bypass Revascularisation Investigation; EAST = Emory Angioplasty versus Surgery Trial; ERACI = Argentine Randomized Trial of Percutaneous Transluminal Coronary Angioplasty versus Coronary Artery Bypass Surgery in Multivessel Disease; GABI = German Angioplasty Bypass Surgery Investigation; RITA = Randomised Intervention Treatment of Angina; PTCA = Percutaneous Transluminal Coronary Angioplasty.*

angioplasty, diabetic patients had more in-hospital mortality or myocardial infarction (MI) compared with patients without diabetes. The long-term outcome is also plagued by increased risk of restenosis, contributing to greater rates of mortality, MI, and repeat revascularisation.[9,10]

The presence of diabetes is also associated with a higher event rate after CABG.[11] Diabetic patients have more rapid disease progression in saphenous vein grafts and in native vessels whether grafted or not.[12] Diabetes is also an independent risk factor of late mortality after CABG.[13]

Metabolic and haematologic derangements contribute to the increased incidence of adverse cardiac events after revascularisation in diabetics.[14] Platelets are more often activated with increased aggregability, enhanced secretion of vasoconstrictive agents, and greater mitogenic activity. Fibrinogen, factor VII and plasminogen activator inhibior-1 levels are increased while antithrombin III activity is reduced. Diabetes is associated with endothelial dysfunction and excessive smooth muscle cell proliferation and extracellular matrix formation.[15]

The reasons for the inferiority of PTCA compared with CABG for coronary revascularisation in diabetics are usually attributed to a high restenosis rate, incomplete revascularisation, and progression of atherosclerosis. Improvements in the techniques of both percutaneous and surgical techniques have made it necessary to re-examine this issue from time to time. This is especially true for percutaneous intervention as changes in practice are occurring rapidly over the last few years.

Stenting: Improvement over Balloon Angioplasty

Balloon angioplasty is largely replaced by coronary stent implantation as the predominant modality of percutaneous revascularisation because of the advantages of reduction in emergency CABG and restenosis.[16,17] CABG has also undergone changes with the routine use of arterial conduits that has better long-term patency.[18] The Arterial Revascularization Therapy Study (ARTS) was undertaken to compare contemporary percutaneous and surgical revascularisation for the treatment of patients with multivessel CAD. In the diabetic subgroup, the 1-year incidence of the individual components or the composite of death, MI, or stroke was similar between the two groups. The main difference was the increased need for repeat revascularisation in the stent group (14.3% vs 3.1%; p <0.001).[19] Although the reintervention rate has been reduced by ~50% compared with balloon angioplasty results in prior comparison trials, coronary stenting in diabetics is still associated with a high rate of restenosis and the resulting need for repeat revascularisation, confirming the results of retrospective series which reported poorer outcomes of diabetics after stent implantation compared to nondiabetics.[20,21] Moreover, the mortality was 6.4% for the stent group and 3.1% for the surgery group, although not reaching statistical significance. From the BARI and EAST experience, a longer follow-up may unmask the mortality difference between the two groups.

Platelet Glycoprotein IIb/IIIa Antagonist: Reduction in Mortality and Repeat Revascularisation

Another important advance in percutaneous revascularisation emerges from the use of platelet glycoprotein (GP) IIb/IIIa antagonists. In particular, abciximab has been shown to reduce ischaemic cardiac events following balloon angioplasty, artherectomy, and stent implantation.[22-24] In the Evaluation of Platelet IIb/IIIa Inhibitor for Stenting Trial (EPISTENT) diabetic substudy, the 6-month target vessel revascularisation (TVR) rate was significantly reduced from 16.6% in the stent-placebo group to 8.1% in the abciximab-stent group.[25] Angiographic analysis showed a significant decrease in the late loss index. The data are suggesting an anti-restenotic effect of abciximab which may be related to its non-specific inhibitory effects on integrin receptors including IIb/IIIa, $\alpha_v\beta_3$ and Mac-1, which have been implicated in the thrombotic and proliferative response leading to neointima formation after arterial injury.[26-29] In addition to TVR reduction, evidence of mortality reduction was observed in the EPISTENT trial when abciximab was used as adjunct during coronary stenting.[30] Meta-analysis of Evaluation of 7E3 for the Prevention of Ischemic

Complications (EPIC), Evaluation in PTCA to Improve Long-term Outcome with abciximab GP IIb/IIIa blockade (EPILOG), and EPISTENT demonstrated a survival benefit in diabetic patients receiving abciximab during elective or urgent percutaneous intervention. A 44% mortality reduction at 1 year from 4.5% to 2.5% was found.[31] The combination of abciximab and stenting should be considered in diabetic patients undergoing percutaneous revascularisation because of the significant reduction in mortality, MI and re-intervention and should be adopted for future comparison trials.

Drug-eluting Stents: Reduction of Restenosis; Complete Revascularisation

Enormous efforts have been invested in the attempt to reduce restenosis soon after the inception of PTCA. Among the numerous devices and drugs that have been tested, stents were shown in the early 1990s to be the first device capable of reducing restenosis compared with balloon angioplasty. The improved but still disappointing rate of in-stent restenosis (ISR) especially in diabetic patients was the impetus for continued effort to overcome this enemy of percutaneous revascularisation. Although proven to be an effective treatment for ISR, vascular brachytherapy was disappointingly unsuccessful in diminishing restenosis when applied to de novo lesions after coronary angioplasty or stenting.[32] Following early promising results of reduction in restenosis in animal models,[33-35] a pilot study with the sirolimus-eluting stent was shown to achieve 0% ISR up to 1-year angiographic follow-up in 29 patients.[36] The dramatic result of ISR reduction was recently joined by the Randomised double-blind study with the Sirolimus-eluting Bx VElocity™ balloon expandable stent in the treatment of patients with de novo native coronary artery lesions (RAVEL) which showed again no ISR at 6 months in patients receiving sirolimus-eluting stents. The diabetic subgroup in RAVEL is no exception to this remarkable anti-restenotic effect of sirolimus. The ISR was 0% in the sirolimus-eluting stent group versus 42% in the bare stent group.[37] Many other medications including paclitaxol and actinomycin-D are being tested in clinical trials of drug-eluting stents. The TAXUS I trial using paclitaxol-eluting NIR™ stents also demonstrated a promising 0% ISR.[38] When the safety and efficacy are confirmed by longer-term follow-up, the new era of percutaneous revascularisation will begin. Small vessels, long lesions, bifurcations, and unprotected left main stenosis can be confidently tackled by drug-eluting stents with little hindrance from the worry of restenosis. A more complete revascularisation comparable to CABG may be achieved by the percutaneous approach. With development in the technology of detecting vulnerable plaques,[39] e.g. optical coherence tomography, thermography, elastography, those haemodynamically insignificant but unstable lesions may be prophylactically stented for the prevention of future coronary events from plaque disruption.

Conclusion

Advances in interventional cardiology will most likely impact upon the shortcomings of catheter-based revascularisation in diabetics. Equipped with GP IIb/IIIa antagonists and drug-eluting stents, interventional cardiologists are ready again for another comparison trial with CABG. The ARTS-2 is underway to compare percutaneous revascularisation incorporating these improvements with CABG in patients with multivessel disease. The best revascularisation strategy for diabetic patients with multivessel CAD remains to be elucidated by ongoing clinical trials.

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ECG Quiz

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A 17-year-old boy attended Accident and Emergency Department because of palpitation. He described as fast and regular heart beats. There was no associated dizziness. He had experienced intermittent fast palpitations for few months, but never as severe as this time. Otherwise he enjoyed good past health and he had no history of syncope. Family history was unremarkable. He was hemodynamically stable and fully conscious. A 12-lead electrocardiogram (ECG) was performed immediately (Figure 1).

The patient was admitted to coronary care unit (CCU). The tachycardia was aborted when the patient was admitted to CCU. A 12-lead ECG was repeated and shown in Figure 2. It showed delta waves and the diagnosis of Wolff-Parkinson-White Syndrome was made. Electrophysiology study confirmed the presence of right free wall accessory pathway and radiofrequency ablation was performed. There was no recurrence of symptoms for six months after the procedure.

Question

What is the ECG diagnosis?

A) monomorphic ventricular tachycardia (VT)
B) supraventricular tachycardia (SVT) with aberrant conduction
C) SVT with anterograde conduction over an accessory atrioventricular pathway

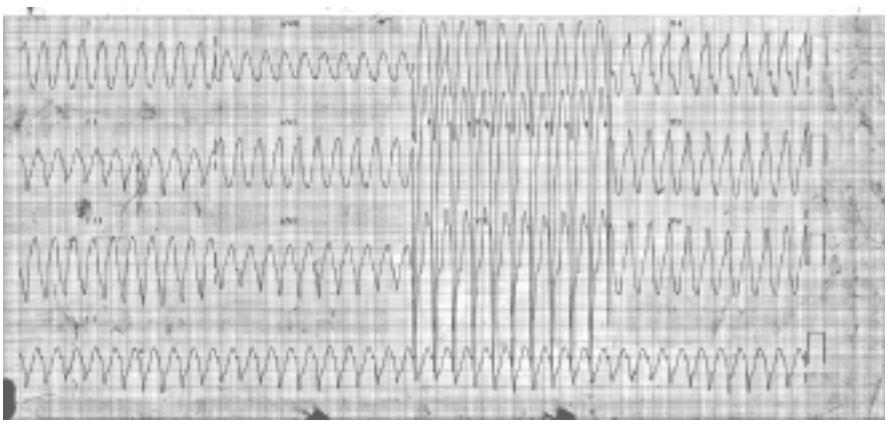

Figure 1.

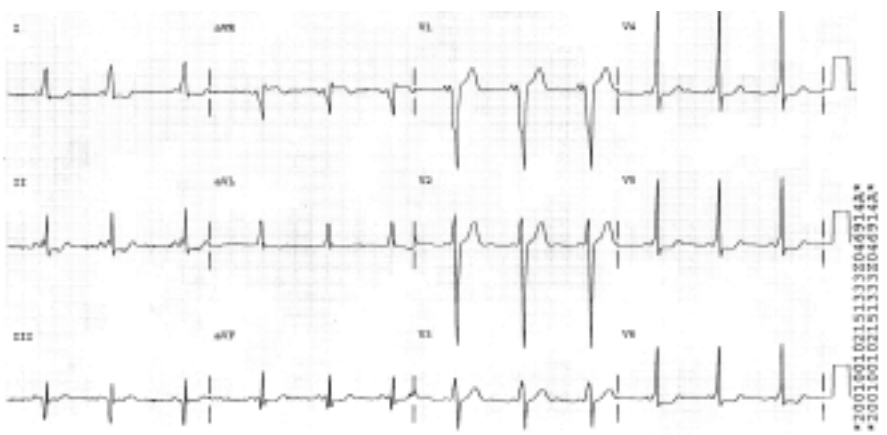

Figure 2.

Answer: C

Discussion

It is always a difficult clinical task to have a correct ECG diagnosis for regular wide complex tachycardia. Differential diagnoses include VT, SVT with aberrant conduction and SVT with anterograde conduction over an accessory atrioventricular pathway (antidromic SVT). Differentiation among these is important because the immediate management is different. However, fast and reliable method is not always available. We adopted the stepwise approach suggested by Antunes et al published in September 1994 in PACE Vol. 17.[1]

The first step aims to differentiate VT or antidromic SVT from SVT with aberrant conduction. There are four questions:

1. Absence of an RS complex in all precordial leads? If the answer is yes, it suggests VT or antidromic SVT. If not, go to question 2.
2. Onset of R to nadir of S interval >100 ms in 1 precordial lead? If the answer is yes, it suggests VT or antidromic SVT. If not, go to question 3.
3. More QRS complexes than P waves? If the answer is yes, it suggests VT or antidromic SVT. If not, go to question 4.
4. Classic morphology criteria for VT present in leads V1 and V6 (i.e. R, qR, QR or RS in V1; R:S ratio <1 in V6 if frontal axis showed left axis deviation; qR or QS in V6 if left bundle branch morphology)? If the answer is yes, it is VT or antidromic SVT. If the answer is "no" again, VT or antidromic SVT is excluded, and the diagnosis of SVT with aberrant conduction is made.

The second step is to differentiate VT from antidromic SVT. There are three questions:

1. Predominantly negative QRS complexes in the precordial leads V4 to V6? If yes, it is VT. If the answer is no, go to question 2.
2. Presence of a QR complex in one or more of the precordial leads V2 to V6? If yes, it is VT. If the answer is no, go to question 3.
3. AV relation different from 1:1? (More QRS complexes than P waves?) If yes, it is certainly VT. If the answer is no, VT is excluded and the diagnosis of antidromic SVT is made.

The admission ECG of the patient showed RS complex in V4 to V6. However, the R to S interval is 140 ms in V6. Based on step one, it suggested VT or antidromic SVT. We proceeded to the second step. The QRS complexes of V4 to V6 were predominantly positive, and there was no QR pattern seen in V2 to V6, and there was no atrioventricular dissociation identified. The answers to all 3 questions in step 2 were "no". This excluded ventricular tachycardia and the diagnosis of antidromic SVT was suggested. Antidromic SVT with anterograde conduction via right free wall accessory pathway was inducible in electrophysiology study with identical morphology as admission ECG.

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