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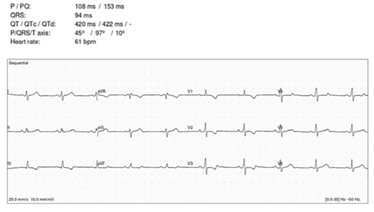
ECG CASE STUDY 1



Case Vignette:
A 29-year-old woman with no cardiac history was found to have an abnormal screening ECG (shown above). She was referred for further cardiology assessment. She was fit and active and played tennis and did Pilates regularly. She had no symptoms of chest discomfort or palpitations although she sometimes felt breathless after a prolonged run. She had no cardiac risk factors or family history of cardiovascular disease and was not on any medication.

Cardiovascular examination revealed a soft mid-diastolic murmur and a split-second heart sound. Lung fields were clear and there were no signs of right heart failure.

What does the ECG show and what investigations would you recommend?

Discussion:
ECG findings - the ECG shows sinus rhythm with the following abnormal features:

- Minor right axis deviation
- Dominant R waves in V1 and V2
- T wave inversion V1-V3
- Crochetage sign in leads II and aVF (notch near the apex of the R wave in inferior leads)

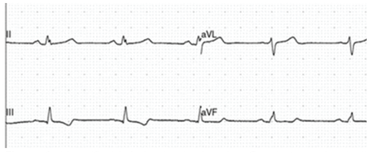


Figure 1. *Magnified inferior leads showing Crochetage sign in II and aVF (notch near apex of R wave)*

Investigations:
She underwent a transthoracic echo which showed a large secundum atrial septal defect (ASD). There was a significant left to right shunt across the ASD with an estimated Qp:Qs ratio of 1.64. The right ventricle (RV) was dilated and there was moderate tricuspid regurgitation (TR). Estimated pulmonary artery systolic pressure was 30mmHg.

A transesophageal echo was performed to obtain more information on the ASD anatomy and margins. The patient was recommended to undergo percutaneous closure of the ASD which was successfully performed.

Discussion:
A dominant R wave in V1 (defined as an R/S ratio ≥ 1) is usually abnormal and may be related to several conditions including right bundle branch block, left ventricular ectopics, right ventricular hypertrophy, acute right ventricular dilation (acute right heart strain), type A WPW- syndrome and a posterior myocardial infarction. It is estimated to be a normal variant in 1% of cases. T wave inversion in leads V1 to V3 can be a normal variant in females and children. The ECG changes in this case may have been due to increased right heart strain and volume overload related to the large ASD with left to right shunt.

The Crochetage sign (also known as the "notch sign" is an ECG pattern with a small notch or "bump" near the apex of the R wave in the inferior leads (II, III, aVF). The term "crochetage" comes from the French word for "hook" as the sinus pattern resembles a hook or crochet-like shape. This sign is most often seen in patients with ASD, although it can also be seen in other conditions such as PFO, pulmonary embolism, right ventricular hypertrophy, and patent ductus arteriosus. This sign may be associated with the severity of the shunt.

Both the American and European guidelines (AHA and ESC guidelines) recommend transcatheter closure of ASDs as the preferred choice, particularly for secundum ASDs that are less than 2 cm in diameter and not associated with other defects. Surgical closure may be recommended for larger ASDs or those not amenable to transcatheter closure. Although patient's ASD diameter was relatively large (2.2cm), the transesophageal echo showed good margins around the ASD for a percutaneous closure device and the patient preferred to close the ASD percutaneously.

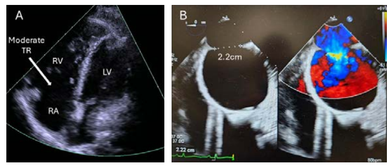


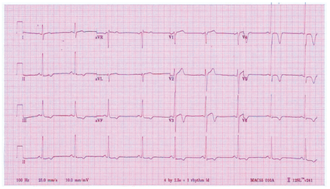
Figure 2*: A) Transthoracic echo image showing dilated right ventricle (RV). There was moderate tricuspid regurgitation (TR; RA= right atrium, LV= left ventricle). B) Transesophageal echo image showing large secundum ASD (2.2cm maximum diameter) with a significant left to right shunt.*

By **Dr. Reginald Liew**
Senior Consultant Cardiologist
The Harley Street Heart & Vascular Centre

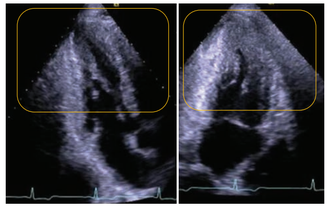
ECG CASE STUDY 2

A 49-year-old IT executive presented to my care through the Emergency Department with a 2-day history of central chest discomfort with radiation to his left shoulder. He looked comfortable during examination; vital signs were stable. Serial cardiac enzymes were not elevated. He had no prior known ischaemic heart disease and no traditional risk factors. An urgent CT coronary angiogram revealed normal, atheroma-free coronary anatomy.

He declared he was aware his ECG was abnormal on previous health screens but could not elaborate further. His ECG is shown below. The ECG shows sinus rhythm, a normal cardiac exis, inverted T waves (so-called "giant negative T waves") in the precordial leads, V3 to V6. There is Increased QRS voltage, meeting the criteria for left ventricular hypertrophy. ST segment depression is also present within the inferior (II, III, avF) with T wave inversion.



An urgent echocardiogram was arranged which revealed the diagnosis to be apical hypertrophic cardiomyopathy (ApHCM) (Fig 1). There was no evidence of LV outflow tract or mid-cavity obstruction. Fig 1.



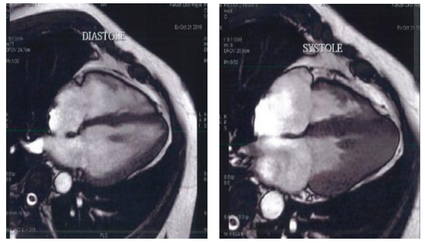
In apical hypertrophic cardiomyopathy (ApHCM), other distinctive ECG findings can include:

1. QRS Complex Changes: The presence of deep, narrow Q waves may be observed in the lateral leads (I, aVL, V5, V6)
2. Arrhythmias: Premature ventricular contractions (PVCs) or other arrhythmias

His chest pain is likely due to myocardial ischaemia. Several of the pathophysiologic features of ApHCM predispose to the development of microvascular angina, which may be induced by an increase in myocardial oxygen demand or a reduction in myocardial blood flow and oxygen supply. Factors that increase myocardial oxygen demand include myocyte hypertrophy and increased muscle mass, myocyte disarray and LVOT obstruction and increased wall stress due to elevated diastolic pressures. Factors that reduce myocardial blood flow in HCM, particularly with exertion, include impaired vasodilator reserve, myocardial bridging with systolic and early diastolic compression of intramural vessels, small vessel disease and microvascular dysfunction, myocardial fibrosis, and increased capillary separation and inadequate capillary density.

The patient was commenced on a beta blocker with good therapeutic effect.

The patient underwent a cardiac MRI (Fig.2) for further prognostic and cardiac event risk assessment. This clearly illustrates thickened myocardium, more pronounced at the apex. Cavity obliteration is also seen in end-systole.

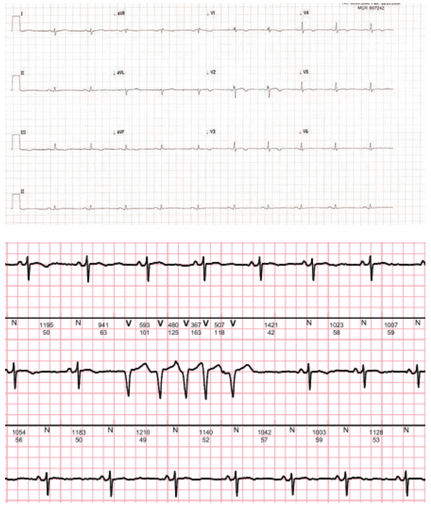


Apical hypertrophic cardiomyopathy (ApHCM) is a subtype of hypertrophic cardiomyopathy characterized by hypertrophy (thickening) of the myocardium (heart muscle) predominantly at the apex of the left ventricle. This localized thickening can result in a distinctive "spade-like" shape of the left ventricle on imaging studies. Diagnosis of ApHCM typically involves a comprehensive evaluation, including clinical assessment, imaging studies, genetic testing, and consideration of ECG findings in the context of the overall clinical picture.

Like other forms of hypertrophic cardiomyopathy, ApHCM is associated with an increased risk of complications such as heart failure, arrhythmias (including atrial fibrillation and ventricular arrhythmias), and sudden cardiac death. Regular monitoring and risk stratification are important for managing patients with ApHCM and preventing adverse outcomes. The management of apical hypertrophic cardiomyopathy focuses on controlling symptoms, preventing complications, and reducing the risk of sudden cardiac death.

By **Dr Rohit Khurana**
Senior Consultant Cardiologist
The Harley Street Heart & Vascular Centre

ECG CASE STUDY 3



Clinical Vignette:
A 32-year-old woman with no significant symptoms presented for evaluation after an incidental finding of abnormal T-wave inversions on a routine ECG. She is an active individual who regularly participates in running, with no reported episodes of chest pain, palpitations, or fatigue.

Past Medical History: In 2020, during an unrelated medical evaluation, she was noted to have T-wave inversions on an ECG. Subsequent investigations, including a cardiac ultrasound (U/S), stress echocardiogram, and 24-hour Holter monitoring, were normal.

Social History: She is a non-smoker, does not consume alcohol.

Family History: Her father died suddenly at the age of 54, and her paternal grandmother died at age 48, both suspected from sudden cardiac death (SCD). No autopsies are available.

Clinical Examination: Normal.

Investigations:

- **Resting ECG:** Sinus rhythm with inferior and anterolateral T-wave inversion.
- **Echocardiogram:** Normal structure and function.
- **48-hour Holter Monitoring:** Showed frequent ventricular ectopics (VEs) and a non-sustained ventricular tachycardia (NSVT) run of 5 beats.
- **Cardiac MRI:** Revealed mild biventricular dysfunction, and dilatation with linear fibrosis in the interventricular septum.
- **CT Coronary Angiogram:** Normal coronary arteries.

Discussion:
This case presents several red flags for potential cardiac disease despite the patient's asymptomatic status and active lifestyle. Most notably, her family history is concerning, with her father and grandfather both experiencing sudden cardiac death at a relatively young age. Her resting ECG findings of inferior and anterolateral T-wave inversion, along with the discovery of ventricular ectopics and NSVT on Holter monitoring, raise the suspicion of an underlying cardiomyopathy.
The cardiac MRI findings of mild biventricular dysfunction and septal fibrosis further support a diagnosis of a cardiomyopathy.

Diagnosis:
Mild dilated cardiomyopathy (DCM) is the most likely diagnosis in this case, particularly with the evidence of fibrosis on MRI, abnormal ECG findings, and frequent arrhythmias. While she currently remains asymptomatic, her strong family history of sudden cardiac death, coupled with her ECG abnormalities and imaging findings, suggests she may be at increased risk for future adverse cardiac events, including arrhythmias.

Take home message:
This case highlights the importance of ECG screening and the need to assess the full clinical picture, even in asymptomatic individuals, especially those with a strong family history of cardiac events. Echocardiography can often miss the early signs of cardiomyopathy, and cardiac MRI is the gold standard for assessing heart function with the added benefit of being able to assess for fibrosis in the heart.

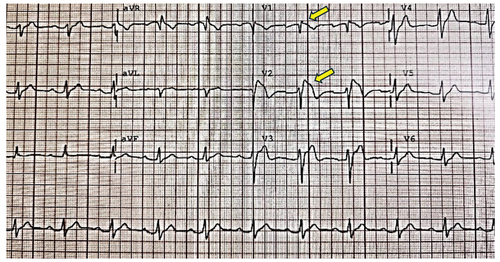
By **Dr Michael MacDonald**
Senior Consultant Cardiologist
The Harley Street Heart & Vascular Centre

ECG CASE STUDY 4

Patient profile
A 37-year-old Chinese male with no past medical history presented with syncope to my care. This was his second episode in 2 months. The first occurred in his workplace where he had a deskbound job in the IT sector. He had mentioned that his colleague had noticed him lose balance from his chair in a meeting with transient loss of consciousness for about 1 minute. He saw a GP that evening and was told it was likely a vasovagal episode.

The 2nd episode in which he saw the emergency department happened while he was walking. He fell, sustained facial abrasions and was being helped by a passerby once he regained his consciousness. He did not complain of palpitations or chest discomfort in the lead up to his fall.

The ECG done in the emergency department showed the following red flag finding.



ECG Analysis
While the ECG showed a normal sinus rhythm, **there was a characteristic coved shaped 1-2 mm ST elevation in V1 and V2 (yellow arrows).** Without classical chest pain or corresponding reciprocal inferior leads ST depression changes, this was not in keeping with a ST elevation myocardial infarction.

The diagnosis is that of **Brugada syndrome**. This is a sodium channelopathy associated with sudden cardiac arrest which can be as a result of a spontaneous mutation or an autosomal dominant genetic transmission. The most common gene accounting for 20-30% of patients is the SCN5A gene. There is a risk of malignant arrhythmias (ventricular tachycardia or ventricular fibrillation) and in a patient with suspicious symptoms such as syncope, treatment with an implantable cardiac defibrillator (ICD) is advised. After appropriate counselling, the patient opted for the procedure.

In patients with Brugada pattern ECGs (but without symptoms), the risk of cardiac events annually is low (<1%). These patients can be conservatively managed with 3 main precautions to reduce their arrhythmic risk.

- Avoidance of certain drugs (https://www.brugadadrugs.org)
- Controlling fever adequately with antipyretic drugs
- Avoidance of alcohol intake

The ECG shown above is that of a Type 1 Brugada pattern. There are also Type 2 and Type 3 less apparent Brugada patterns that have been described and a flecainide challenge test can be done to aid in confirmatory Type 1 diagnosis making.

Conclusion
For patients presenting with syncope, performing an ECG helps with risk stratification for their presentation. While this patient's symptoms were initially thought to be vasovagal in origin, a timely ECG in a subsequent presentation showed Brugada syndrome which changed the whole outlook of management – from a conservative strategy to sudden cardiac arrest prevention with an ICD.

By **Dr Pinakin V Parekh**
Senior Consultant Cardiologist
The Harley Street Heart & Vascular Centre

QUIZ

Question:

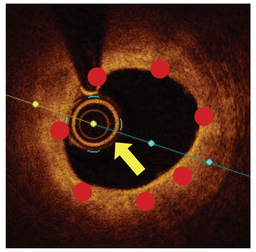


Figure A

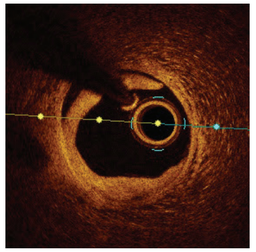


Figure B

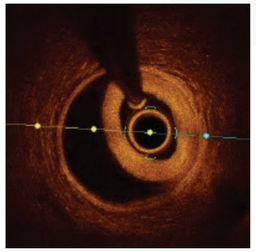


Figure B

Modern day precision coronary angioplasty involves the use of intracoronary imaging to look at the coronary vessels from the inside. Figure A is how the round imaging catheter (yellow arrow) looks like in relationship to the vessel diameter with its smooth continuous lining of intima (red dots).

Figure B and C are abnormalities in the same patient that has the normal architecture of the vessel altered. Can you make a guess to what the differences are?

Answer is available on our website:
http://www.harleystreet.sg/quiz - answers/medbulletin-nov-2024/

By **Dr Pinakin V Parekh**
Senior Consultant Cardiologist
The Harley Street Heart & Vascular Centre

MEDBULLETIN
NOV2024

THE HARLEY STREET
HEART & VASCULAR CENTRE

INTRODUCTION

Dear Colleagues,

Welcome to this year's second edition of the Harley Street Heart & Vascular Centre newsletter. As part of our commitment to supporting you with practical, relevant cardiology insights, we are pleased to share four challenging ECG cases in this issue. These cases highlight key diagnostic elements and complexities in ECG interpretation that we encounter in practice.

Building on these cases, we invite you to our Annual ECG Reading Workshop, scheduled for January 18, 2025. This hands-on symposium will allow you to explore similar ECG scenarios, with interactive lectures and small group discussions tailored to deepen your interpretation skills. It's a unique opportunity to learn directly from our experienced team and engage in collaborative case analysis with peers.

To learn more, please visit www.harleystreet.sg (information on workshop will be posted in Dec 2024) or drop us an email at enquires@harleystreet.sg to indicate your interest.

From The Harley Street Heart and Vascular Centre

From left to right:
Dr Pinakin V Parekh, Dr Sriram Narayanan Dr. Reginald Liew, Dr. Rohit Khurana, Dr. Michael MacDonald

http://www.harleystreet.sg
Email **enquiries@harleystreet.sg**

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Mount Elizabeth Novena Specialist Centre
#05-30, 38 Irrawaddy Road
Singapore 329563

Gleneagles Hospital #02-38/41 (Annexe Block)
6A Napier Road
Singapore 258500

Mount Elizabeth Medical Centre
#11-07, 3 Mount Elizabeth
Singapore 228510