MedMax Publishers

Journal of Clinical Cardiology & Case Reports Review Article

Routine Electrocardiograms (ECGs) Can Be Life Saving to Individuals and Their Family Members - Think Twice Before Concluding as Normal: Important Reminder

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Received: 25 March, 2022; **Published**: 10 April, 2022

Introduction

All routine electrocardiograms (ECGs) are not normal and ignoring subtle abnormalities can be life threatening. Sometimes a routine ECG performed for different reasons (health insurance, sports screening etc.) can pick up abnormal findings. General practitioners (GPs), emergency doctors and non-cardiologist physicians can be the first contact with such patients. Even for individuals who are asymptomatic or describe non-specific symptoms, abnormal findings on ECG should trigger a specialist opinion and possibly further investigations. The aim of this case series is to emphasise the importance of not undermining abnormal findings on routine ECGs as timely investigations and specialist's referral can be life saving not only for the individual, but for the other family members as well.

We present three patients with abnormal ECGs. In the first two patients, the abnormality on the ECG has led to further investigations and diagnosis of an underlying cardiomyopathy. In the third case, patient was an already known case of hypertrophic cardiomyopathy (HCM), but a routine follows up ECG picked up a serious life-threatening arrhythmia, which was timely treated and avoided sudden death. It is very important to recognise the varied pattern of abnormal ECG in these patients, as ignoring or missing the abnormality on ECGs in these patients can be life threatening. Early recognition and specialist referral can save lives.

Case 1: A 52 years old lady was referred to the cardiology clinic due to an abnormal ECG. She had a non-specific history of atypical chest pain. She was previously living in Poland, but has been living in UK for the last 5 years. She has a family history of sudden cardiac death with a brother dying at the age of 28 years from unknown cause of death.

Her 12 lead ECG (Figure 1) was abnormal with evidence of left ventricular hypertrophy (LVH), T wave inversions in precordial leads and evidence of Q-waves as well.

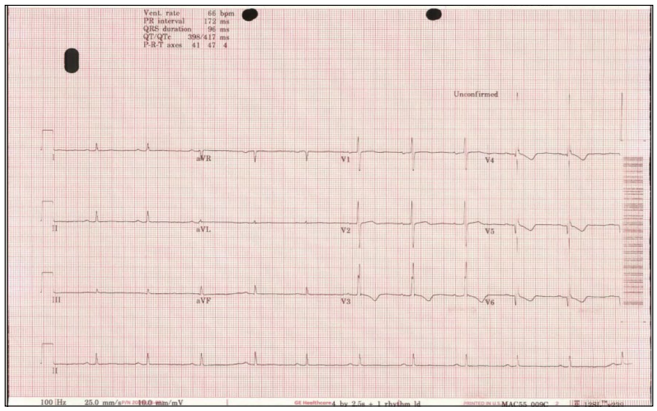

Figure 1: 12 leads ECG suggestive of left ventricular hypertrophy and T wave inversion in leads V3-V6.

Transthoracic echocardiogram (Figure 2) was performed which showed asymmetric septal hypertrophy (25mm) but no evidence of Left Ventricular Outflow Tract Obstruction (LVOTO).

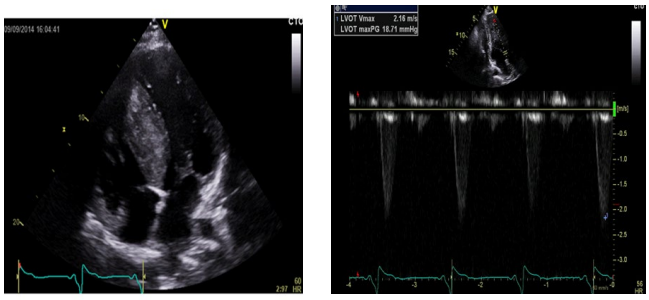

Figure 2: Marked asymmetric hypertrophy of the septal wall but no obvious LVOT obstruction on Doppler echocardiography.

Patient collapsed during the exercise treadmill test, but no cardiac arrhythmias were noted. The blood pressure response to the exercise was not significantly abnormal. She underwent a Cardiac Magnetic Resonance Imaging (cMRI), which confirmed the findings of HCM with a maximum septal thickness of 25 mm. Marked patchy fibrosis was also noted in the hypertrophied segments on late gadolinium enhancing images on the MRI.

She was referred to regional Inherited Cardiovascular Conditions (ICC) clinic where she was started on medical management (beta-blockers) in first place. It was not clear whether the underlying mechanism of her symptoms is due arrhythmias or LVOTO during exercise. In order to clarify the underlying mechanism of her symptoms, an outpatient 72 hours event recorder and stress echocardiogram was arranged with follow up at the ICC clinic. The event recorder not revealed any cardiac dysrhythmias. The stress echocardiogram ruled out any LV outflow tract obstruction as well as exercise induced cardiac dysrhythmias. Currently she is under regular cardiology follow after an implantable loop recorder and no cardiac arrhythmias detected over 2 yrs. She was advised for family screening and discussion was made about the possibility of implantable cardioverter defibrillator (ICD) in future.

Case 2: A 23 years old manual warehouse worker, was referred to cardiology clinic as his insurance was declined due to abnormal ECG. He has had a history of collapse 5 years ago and at that time he was told that his ECG was normal. Clinical examination is unremarkable. His 12 lead ECG in clinic showed T wave inversion in lateral leads (Figure 3).

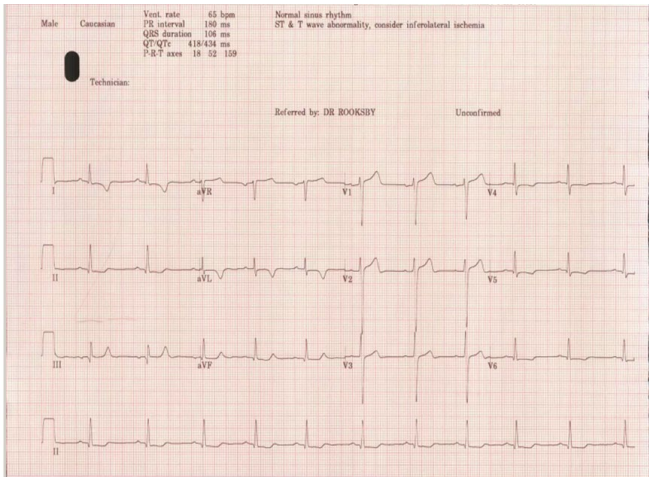

Figure 3: 12 lead ECG showing lateral T waves inversion

A trans-thoracic echocardiogram was arranged which showed increased apical and apical lateral thickness as shown in figures 4 and 5.

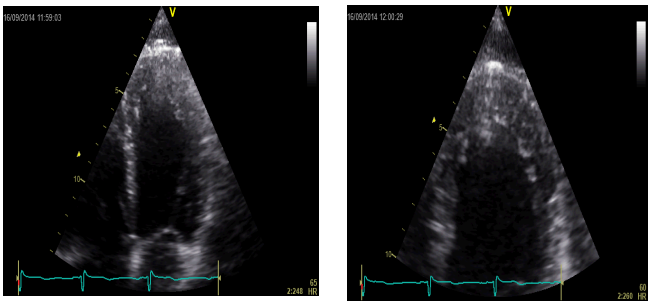

Figure 4: Increased apical wall thickness as demonstrated in a 4 chamber view of transthoracic echocardiography.

Cardiac MRI was arranged which showed prominent trabeculations of left ventricular apex and lateral wall, raising the suspicion of non-compaction type of HCM (Figure 5).

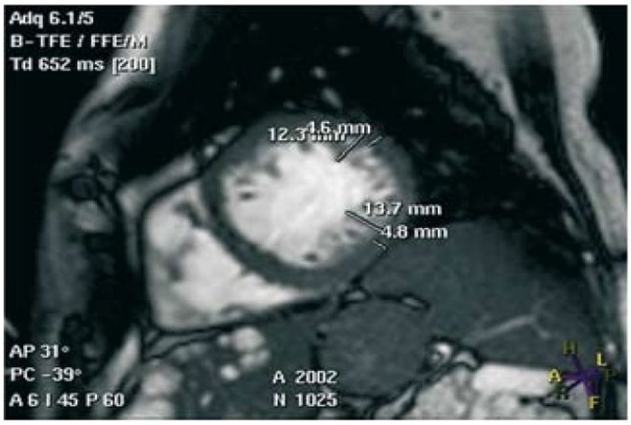

Figure 5: Prominent trabeculations on cardiac MRI. Also notice the almost 3:1 ratio of non-compacted to compacted segments.

Patient was referred to regional ICC clinic where it was thought to be possible early stage of non-compaction type of HCM. He was planned for repeat cardiac MRI and regular follow up at the ICC clinic.

Case 3: A 20 years old male, who has been diagnosed with hypertrophic cardiomyopathy (HCM) since birth, has been under regular follow up. He is asymptomatic and clinical examination is unremarkable. He works as a roofer and doing regular weight lifting and playing football. He has got family history of HCM, with his father and paternal uncle, both diagnosed with HCM while father had an ICD implanted as well. He had a routine ECG which showed features of Wolf-Parkinson's White (WPW) Syndrome with short PR interval and delta waves (Figure 6).

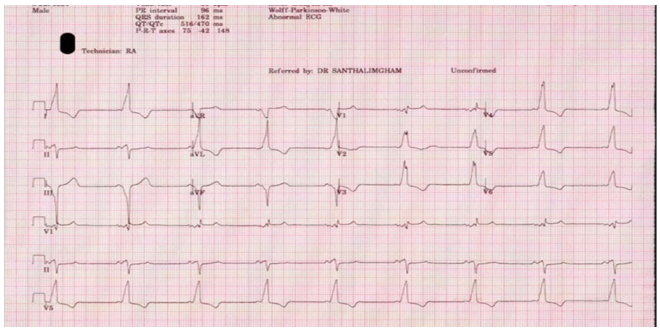

Figure 6: 12 lead ECG showing short PR interval (96 msec), broad QRS and slurring of the upstroke of the QRS complex (delta waves).

48 hours heart monitor did not reveal any underlying arrhythmias. Treadmill exercise test showed no drop or flattening of blood pressure, but the ECG changes to narrow complex with exercise with associated dizziness. No chest pain was reported.

Patient was referred to regional inherited cardiac conditions (ICC) clinic where, after seeing a specialist, he underwent successful ablation right posteroseptal accessory pathway. He also underwent full genetic study for HCM and was advised about life style and arranged repeat cardiac magnetic resonance (CMR) with follow up at ICC clinic.

Discussion

Hypertrophic cardiomyopathy (HCM) is a cardiac condition characterised by increase left ventricular thickness. In an adult, HCM is defined as LV wall thickness of 15mm or more in one or more LV myocardial segments as measured by any of the imaging modality including echocardiography, cardiac MRI or

cardiac CT scan, that cannot be explained by any other loading conditions [1]. It is a rare genetic disorder with an annual incidence reported of about 0.3-0.5 per 100,000 in population based studies [2,3]. Various studies across the world have shown that, the prevalence of HCM is similar in different racial groups [4-10].

Many cases with HCM can be asymptomatic or with minimal or atypical symptoms. In such cases the diagnosis can be incidental on routine tests or during screening [11]. It can prove fatal and results in sudden early cardiac deaths if the diagnosis is missed, particularly in young otherwise healthy individuals. Early diagnosis and proper workup can not only be life saving for the individual but also can prevent sudden cardiac deaths of their family members, by early screening and recognition of the HCM in other family members. As in our second case, patient sadly had a family history of sudden death of a brother at very younger age, which, could be have been due to underlying HCM. In this review article we wanted to emphasize the importance of early recognition of this disorder by hospital physicians and general practitioners, as these patients can present with wide range of symptoms. Physicians and general practitioners can easily undermine the ECG changes and discharge these group of patients, without any further follow up with cardiologists. In contrary, early recognition and prompt referral to the cardiologists can be lifesaving. Furthermore, we want to draw the attention of the readers to the fact that patients with already diagnosed HCM, can later on develop abnormal ECG due to complications/ arrhythmias related to the HCM (as in case number 3).

References

1. UK CO. 2014 ESC Guidelines on diagnosis and management of hypertrophic cardiomyopathy: web addenda.
2. Lipshultz SE, Sleeper LA, Towbin JA, Lowe AM, Orav EJ, et al. (2003) The incidence of pediatric cardiomyopathy in two regions of the United States. N Engl J Med 348: 1647-1655.
3. NugentAW, Daubeney PE, Chondros P, Carlin JB, Colan SD, Cheung M, et al. (2005) Weintraub RG. Clinical features and outcomes of childhood hypertrophiccardiomyopathy: results from a national population-based study. Circulation 112: 1332-1338.
4. CoddMB, Sugrue DD,Gersh BJ, Melton LJ III (1989) Epidemiology of idiopathic dilated and hypertrophic cardiomyopathy.A population-based study in Olmsted County, Minnesota, 1975–1984. Circulation 80: 564–572.
5. Corrado D, Basso C, Schiavon M, Thiene G (1998) Screening for hypertrophic cardiomyopathy in young athletes. N Engl J Med 339: 364–369.
6. Maron BJ, Mathenge R, Casey SA, Poliac LC, Longe TF (1999) Clinical profile of hypertrophic cardiomyopathy identified de novo in rural communities. J Am Coll Cardiol 33: 1590–1595.
7. Maron BJ, Gardin JM, Flack JM, Gidding SS, Kurosaki TT, et al. (1995) Prevalence of hypertrophic cardiomyopathy in a general population of young adults. Echocardiographic analysis of 4111 subjects in the CARDIA Study. Coronary Artery Risk Development in (Young) Adults. Circulation 92: 785–789.
8. Maron BJ, Spirito P, Roman MJ, Paranicas M, Okin PM, et al. (2004) Prevalence of hypertrophic cardiomyopathy in a population-based sample of American Indians aged 51 to 77 years (the Strong Heart Study). Am J Cardiol 93: 1510–1514.
9. Nistri S, Thiene G, Basso C, Corrado D, Vitolo A, et al. (2003) Screening for hypertrophic cardiomyopathy in a young male military population. Am J Cardiol 91: 1021–1023.
10. Zou Y, Song L, Wang Z, Ma A, Liu T, et al. (2004) Prevalence of idiopathic hypertrophic cardiomyopathy in China: a population-based echocardiographic analysis of 8080 adults. Am J Med 116: 14–18.
11. Rapezzi C, Arbustini E, Caforio AL, Charron P, Gimeno-Blanes J, et al. (2013) Diagnostic work-up in cardiomyopathies: bridging the gap between clinical phenotypes and final diagnosis. A position statement from the ESCWorking Group on Myocardial and Pericardial Diseases. Eur Heart J 34: 1448-1458.

Citation: Mohammad Waleed, Evangelos Tzolos, Manoharan Santhalingam. Routine Electrocardiograms (Ecgs) Can Be Life Saving to Individuals and Their Family Members - Think Twice Before Concluding as Normal: Important Reminder. J Clin Cardio & Case Res, 2022, 2: 1-3.