CASE REPORT

Use of transthoracic echocardiography in the evaluation of chest pain: illustrative cases and review of recent guidelines

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SUMMARY

Chest pain (CP) is a very common presentation with a wide range of differential diagnoses, including life-threatening conditions, which need to be considered, diagnosed and treated urgently. Cardiac CP accounts for less than one-third of causes. Echocardiography is a valuable non-invasive tool that can help in diagnosing and treating patients presenting with CP. National Institute for Health and Care Excellence (NICE), European Society of Cardiology (ESC) and American Heart Association (AHA) guidelines state that echocardiography can help in the diagnosis of acute coronary syndromes and also in ruling out other serious conditions. We present three cases where transthoracic echocardiography (TTE) was essential in assessing patients, making the correct diagnosis and offering the right treatment. This article emphasises the importance of using TTE in assessing patients with CP and hopes to promote its consideration in day-to-day practice.

BACKGROUND

Chest pain (CP) or discomfort is a common symptom and accounts for up to 5% of all admissions to the emergency department (ED), and up to 25% of emergency hospital admissions.¹ There are many causes of acute CP, not all of which are necessarily cardiovascular in origin. Examples of cardiovascular causes of CP include acute coronary syndrome (ACS), myocarditis, pericarditis, hypertrophic cardiomyopathy, aortic stenosis and aortic dissection. Non-cardiovascular causes of CP include pulmonary embolism (PE), pneumothorax, pleurisy, gastro-oesophageal reflux disease, oesophageal spasm, oesophageal perforation and musculoskeletal pain.

When first assessing a patient presenting with CP or discomfort, it is important to consider the wide variety of conditions. If the pain is suspected to be cardiac in origin, there are numerous life-threatening conditions that need to be excluded in the first instance. Acute myocardial infarction (MI) carries a poor prognosis if not detected and treated early, and therefore requires prompt and accurate assessment and management. Newer National Institute for Health and Care Excellence (NICE) guidance on CP of recent onset states that transthoracic echocardiography (TTE) can be used to establish the diagnosis of MI by looking for any regional wall motion abnormalities (RWMA) or loss of viable myocardium.² Furthermore, there is a growing emphasis on the use of this non-invasive and easily accessible diagnostic tool in excluding other life-

threatening conditions. TTE is important in assessing patients with shock/unexplained hypotension. These include conditions such as aortic dissection, PE and myocarditis, which often present with a similar clinical picture and have changes in the ECG together with rises in serum troponin I or T levels. TTE may also contribute in preventing unnecessary treatment of non-life-threatening inflammatory cardiomyopathies and musculoskeletal CP.³

We present three illustrative cases where the use of TTE was essential in reaching the correct diagnosis. We discuss the increasing emphasis on the use of TTE in the ED as well as acute admissions, and review the latest NICE and European Society of Cardiology (ESC) guidance along with American Heart Association (AHA) guidelines.

CASE PRESENTATION

Case 1

A 55-year-old man presented acutely after waking from sleep at 23:00 with sudden onset central chest tightness and breathlessness. The pain radiated to the left arm, jaw and back with associated clamminess and was partially relieved by glycerol trinitrate spray sublingually. He was pain free on admission at midnight. He suffered from hypertension. He never smoked and drank a minimal amount of alcohol.

The patient was slightly hypotensive with a blood pressure (BP) of 95/55 and a pulse rate of 65 bpm. Cardiac examination revealed normal heart sounds with no obvious added sounds. There was no radial–radial or radiofemoral delay and no discrepancy in the BP between arms on initial assessment. He had no clinical evidence of fluid retention or pulmonary oedema.

Case 2

A 48-year-old woman was admitted acutely with a 5-day history of non-specific chest and abdominal pain radiating through to the back. She also reported general lethargy, breathlessness and an intermittent fever together with occasional palpitations. She was previously fit and well and on no regular medications. She had no cardiac risk factors and denied any preceding coryzal symptoms. On admission she was hypotensive with a BP of 91/66 and no variation between arms and no radiofemoral delay. There were no added sounds on auscultation of the heart and the chest was clear. There was a 2 cm non-tender hepatomegaly on abdominal examination.

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Case 3

An 82-year-old woman with a history of type 1 diabetes mellitus and previously resected thyroid cancer was admitted after feeling generally unwell for 2 days with breathlessness and dizzy spells. She reported chest tightness associated with the breathlessness, which she described as heaviness across the chest, but denied any specific CP. On examination she was pale and clammy with oxygen saturations at 80% on 15 L oxygen and she was hypotensive with a BP of 82/55. There were no clinical signs of deep vein thrombosis.

INVESTIGATIONS

Case 1

ECG (figure 1) showed biphasic T wave in V2 lead with T wave inversion in the V3–V6 leads as well as II, III and aVF leads but no Q waves or ST-segment deviation. As his CP sounded cardiac and ECG was thought to be ischaemic, he was initially treated for suspected ACS with dual antiplatelet therapy (aspirin and clopidogrel) and low-molecular-weight heparin and referred for a cardiology opinion the following morning. Repeat bloods were taken at 6 h, which showed that troponin I was 0 but the creatinine had risen acutely from 109 to 174 $\mu\text{mol/L}$.

There was no local catheterisation laboratory in our District General Hospital. Therefore, in view of abnormal ECG and negative troponin, the cardiology team was asked to review after admission. Bedside TTE was performed to look for any RWMA, but surprisingly showed dilated aortic root with possible dissection flap and moderate aortic regurgitation and no pericardial effusion (figure 2A, B). It was also noted that the urine dipstick had 3+ blood. BP was repeated and showed a 24 mm Hg difference between arms with some new radial-radial delay, which suggested extension of dissection more distally to involve subclavian arteries. Therefore, an urgent CT aortogram was arranged as a direct result of these findings. This showed a type A dissection from the aortic root to the iliac arteries, affecting the left renal artery and the aortic valve (figure 3A, B) but sparing the right renal artery.

Case 2

ECG showed small complexes with no ST-segment changes and chest X-ray (CXR) showed a prominent aortic arch. Bloods

were normal other than a random troponin I of 5.81 ng/mL (normal <0.04 ng/mL), deranged liver function (alanine transaminase of 124 IU/L and alkaline phosphatase of 128 IU/L) and mildly raised inflammatory markers (C reactive protein of 22.5 mg/L and white cell count of $8.8 \times 10^9/\text{L}$). An urgent CT of the chest and abdomen was performed to rule out aortic dissection and to look for any intra-abdominal pathology. It showed no obvious abnormality in the thorax but revealed an enlarged pancreas and a left renal infarct secondary to occlusion of the renal artery. Urgent TTE was not possible overnight due to lack of expertise.

Case 3

Bloods revealed a raised troponin at 1.37 ng/mL. CXR showed left mid zone atelectasis. ECG showed T wave inversion in leads II, III, aVF and V3–V5. Bedside TTE (figure 4A–C) showed good left ventricle (LV) function with no RWMA, but interestingly the right ventricle (RV) was severely dilated with impaired function. In short-axis view (figure 4B) it was clear that RV was pressure-overloaded and the septum was bowed towards LV giving it a D-shape. The right atrium (RA) was also dilated with a visible intra-atrial thrombus and a diagnosis of massive PE was made.

DIFFERENTIAL DIAGNOSIS

- ▶ Case 1: ACS, aortic dissection.
- ▶ Case 2: Sepsis, intra-abdominal pathology, pancreatitis, myocarditis, ACS with cardiogenic shock.
- ▶ Case 3: ACS, heart failure, cardiogenic shock, PE.

TREATMENT

- ▶ Case 1: This patient was subsequently transferred to a tertiary centre for urgent aortic root replacement and repair of dissection. Coronary angiogram was not performed prior to surgery as the patient's condition was deteriorating. After diagnosing aortic dissection, the patient revealed that he had a positive family history of aortic dissection, which was not mentioned beforehand.
- ▶ Case 2: The patient remained hypotensive despite adequate fluid resuscitation and was transferred to the high

Figure 1 ECG showing widespread anterolateral T wave inversion suggestive of disease in left anterior descending coronary artery.

Figure 2 (A) Transthoracic echocardiography/parasternal long-axis view showing dilated Ao root with IF and false L. (B) Transthoracic echocardiography/apical three-chamber view showing dilated Ao with IF. LA, left atrium; LV, left ventricle; RV, right ventricle; IF, intimal flap; Ao, aorta; L, lumen.

dependency unit for inotropic support. Her inflammatory markers continued to rise and she was started on broad spectrum antibiotics (meropenem). Pancreatitis was considered, although serum amylase was within normal limits. In view of hypotension and raised cardiac enzymes, the patient had TTE, which showed severe global LV systolic dysfunction and dilation of the LV cavity with an ejection fraction of <20% and no LV thrombus with no RWMA and high LV filling pressures. The RV was also slightly impaired with raised RV systolic pressure and there was no significant valvular disease (figure 5A, B). A clinical diagnosis of myocarditis with severe biventricular systolic impairment and cardiogenic

shock was made, and the patient was transferred to a tertiary heart failure centre for further management.

- Case 3: The patient was thrombolysed and made immediate good clinical response.

OUTCOME AND FOLLOW-UP

- Case 1: The patient made a good postoperative recovery. His renal function returned to normal postoperatively.
- Case 2: The patient was weaned off inotropes and started on heart failure treatment. A few days later, she had a cardiac MRI, which showed a mildly dilated LV with normal systolic function and no abnormal delayed myocardial hyperenhancement. She did not have a myocardial biopsy at the tertiary centre; and she did not receive any corticosteroids or immunosuppressive therapy as evidence for such treatment is not clear. She was discharged home on standard heart failure treatment and made good recovery.
- Case 3: The patient was discharged with follow-up CT of the chest, abdomen and pelvis, which did not show any malignant cause for PE. She had a repeat TTE later on, which showed resolution of the RA thrombus and improvement in RV size and function.

DISCUSSION

Patients with CP represent a very large proportion of all acute medical hospitalisations worldwide. Diagnosing ACS in patients with suspected cardiac CP can be a challenge, especially in individuals without clear symptoms or ECG features. Non-cardiac life-threatening causes must be considered and ruled out.

Less than one-third of CP presentations are due to cardiac causes. Among non-invasive imaging techniques, echocardiography is the most important modality in the acute setting because it is rapidly and widely available. The role of echocardiography cannot be over-emphasised. Rapid recognition and treatment of true ACS while at the same time ruling out other life-threatening conditions such as PE, aortic dissection and myopericarditis, will minimise mortality and morbidity, facilitate early discharge, thus reducing cost, as well as minimising litigation/claims from misdiagnosis.

The most common cause of CP is ACS. Other cardiac conditions are rare. One of the most important challenges in this field is ACS detection in patients with CP with negative ECG and troponin. Here TTE can be helpful by looking for any LV

Figure 3 CT images showing aortic dissection: (A) sagittal view and (B) coronal view.

Figure 4 Transthoracic echocardiography (A) parasternal long-axis view showing dilated RV; (B) parasternal short-axis view showing dilated RV and septal bowing (S); and (C) apical four-chamber view showing dilated RV with strain and dilated RA with visible mass (T). Ao, aorta; LA, left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle.

RWMA if ACS is suspected. In 2010, Parato *et al*⁴ published a study demonstrating the utility of LV wall motion monitoring by serial echocardiography as part of a diagnostic protocol that can be implemented in chest pain units.

Echocardiography has a high sensitivity but moderate specificity for the diagnosis of acute MI.⁵ Previous studies have shown that although sensitivity is high, specificity is much poorer.^{6 7}

Although TTE is less sensitive compared with trans-oesophageal echocardiogram (TOE) in diagnosing type A aortic dissection, it can help to show if the aortic root is dilated and whether dissection can be visualised. It can also show new aortic regurgitation or pericardial effusion suggestive of aortic dissection. Moreover, TTE can assess the right side of the heart and any strain pattern that might suggest PE as a cause of CP. The pericardium can also be assessed and any pericardial effusion can be detected with its haemodynamic effect.

Echocardiography can be used to confirm or rule out MI diagnosis when it is not clear from the history, ECG or biochemical markers. In addition, it can also help to rule out other

Figure 5 Transthoracic echocardiography (A) parasternal long-axis view showing dilated LV and (B) apical four-chamber view showing dilated LV with poor function. LA, left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle.

causes for acute non-cardiac CP. NICE guidelines as well as several other guidelines highlight the use of echocardiography in the management of patients with CP without diagnostic ECG changes,⁸ or when aortic dissection is suspected.⁹

So why is echocardiography not used more widely in the UK?

We feel that the major obstacle to the routine use of echocardiography in the ED is the need for an echocardiography machine and a competent operator to perform the test and interpret its findings. These limitations can be overcome partially by obtaining help from cardiology teams and cardiac physiologists when available. Courses such as The Focused Echocardiography in Emergency Life support (FEEL) course and Echo in Life Support (ELS) course can also be helpful in making echocardiography more understandable to the emergency clinician, enabling its use more often.

Learning points

- ▶ A cardiologist's opinion should be sought early where diagnosis of chest pain is unclear.
- ▶ Urgent echocardiography can help in reaching early diagnosis and may therefore help save lives by establishing early treatment.
- ▶ Haemodynamically unstable patients with unclear diagnosis should have an urgent transthoracic echocardiography (TTE) to help guide further management.
- ▶ The ability to perform a focused TTE to rule out sinister pathologies in patients who are unstable with unclear diagnosis is a useful skill and we encourage all doctors dealing with these situations to attend such echocardiography courses to help them learn the skill.

Conclusion

We propose that echocardiography should be used more often for the evaluation of patients presenting with CP, where the diagnosis remains unclear. The scope for providing echocardiography to all patients remains impractical but it should be routinely available as a front line investigation in all patients where the diagnosis is not clear due to equivocal history, ECG and/or biochemical assays. We acknowledge the limitations of use of TTE in assessing patients with CP and breathlessness, but courses such as FEEL course and ELS course may widen its use in daily practice. Getting help from cardiology registrars, consultants and cardiac physiologists is another potential solution to this problem.

Competing interests None.

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