Unusual presentation of more common disease/injury

Pulmonary embolism mimicking left-ventricular hypertrophy on ECG

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Summary

Patients with pulmonary embolism (PE) can show changes on the ECG. Here, we report the case of a 48-year-old man who initially presented with calf discomfort and swelling. He was discharged with no anticoagulation after a negative complex duplex venous ultrasonography for deep vein thrombosis (DVT). He presented 4 days later with shortness of breath and pleuritic chest pain. Multiple pulmonary emboli were found on CT pulmonary angiogram. His ECGs showed left-ventricular strain which is unusual for PE. He was anticoagulated and discharged. Despite presenting 2 days later with PE-related complications, he eventually recovered well.

BACKGROUND

Pulmonary embolism (PE) can be associated with changes on the ECG which are well documented in the literature. Left ventricular strain pattern, which we describe here however, is unusual. Although deep vein thrombosis (DVT) was missed in our patient, despite a complete duplex venous ultrasonography (including the calf veins), we stress that utilisation of this imaging modality is generally sufficient. This case also reminds us that PE can lead to complications including pleural effusion and hepatic congestion.

CASE PRESENTATION

A 48-year-old normally fit man presented to our emergency assessment unit with a 3-day history of right calf discomfort and swelling. Apart from a left-leg DVT sustained 7 years ago following an ankle fracture, he had no other identifiable risk factors for venous thromboembolism. There was no history of thrombophilia, hypertrophic obstructive cardiomyopathy, sudden cardiac death or use of a pacemaker in his family. He was not on any regular medications and had no known allergies. He worked as a postman, was a non-smoker and only drank alcohol occasionally. Physical examination revealed right calf swelling and tenderness. His vital signs were unremarkable apart from a blood pressure reading of 160/92. He was not known to have hypertension. The D-dimer was raised at 923 ng/ml.

A routine ECG (figure 1) showed inverted T waves in V4 and V5, with the sum of S1/S2+RV5-6 at 35 mm suggestive of left ventricular hypertrophy (LVH). Complete duplex ultrasonography of the right leg veins (including the calf veins) showed no evidence of DVT. The patient was discharged and advised to return if he developed progressive leg swelling or pain or symptoms of PE.

We met the patient for the first time, when he presented 4 days later with breathlessness and pleuritic chest pain. The right calf was unchanged. He was afebrile and had a respiratory rate of 18/min, heart rate of 100/min,

blood pressure of 146/89 and was saturating at 97% on air. C reactive protein (CRP) was 23 mg/l (normal <10 mg/l) and alanine transaminase (ALT) was 59 IU/l (normal <40 IU/l). Troponin was negative. Chest radiograph was normal. T wave inversion in leads V3–V6 had worsened and the sum of SV1 and RV5/V6 had increased to 45 mm (figure 2). Additionally, the ECG showed no signs of right ventricular strain to suggest acute pulmonary hypertension. CT pulmonary angiogram (CTPA) was performed next. It showed filling defects consistent with multiple emboli in the right main pulmonary artery, right upper-lobe and middle-lobe pulmonary arteries and in the segmental arteries of the right lower lobe. There was no pleural effusion or pericarditis.

As the ECG changes for an incidental LVH in a relatively young patient were concerning, an echocardiogram was performed on the same day. It was normal, with no evidence of LVH. A repeat duplex ultrasonography of the right leg veins again showed no evidence of DVT. He was appropriately anticoagulated and discharged home the following day.

The patient presented again 2 days later with an increasing dyspnoea. He was markedly tachypnoeic at 32/min, saturating at 100% on 10 litres of oxygen and tachycardic at 102/min. His blood pressure was 166/105 and he was afebrile. Examination suggested bibasal pleural effusions. His blood results had worsened (CRP 55 mg/l and ALT 117 IU/l). However, the hyperacute T wave changes on ECG had improved. Chest radiograph and repeat CTPA confirmed bilateral pleural effusions with no other new changes. Subsequent tests were done to exclude underlying diseases including antinuclear antibody (ANA), rheumatoid factor (RF), carcinoembryonic antigen (CEA), carbohydrate-associated antigen (CA) 19-9 and prostate-specific antigen (PSA), all of which were unremarkable. Ultrasound of liver and biliary systems was also normal. A clinical decision was made for lifelong warfarin. A thrombophilia screen was therefore felt unnecessary at that time. However, with hindsight, a timely thrombophilia screen

Figure 1 Routine ECG at the first presentation.

would have been appropriate for genetic counselling of the patient's family. After 5 days of hospital stay, our patient was clinically much better and was discharged. Follow-up blood tests and chest radiograph about 2 months later in out-patients showed normal CRP and ALT, respectively, and resolved pleural effusions. His recovery was uneventful.

INVESTIGATIONS

First presentation

- Blood tests—D-dimer 923 ng/ml, CRP 3 mg/l, ALT 29 IU/l, urea 5.4 mmol/l, creatinine 84 μ mol/l, full blood count (FBC) normal.
- ECG—inverted T waves in V4 and V5, sum of S1/S2 + RV5-6=35 mm.
- Duplex ultrasonography right leg veins—no evidence of DVT.

Second presentation

- Blood tests—CRP 23 mg/l, ALT 59 IU/l, urea 4.4 mmol/l, creatinine 92 μ mol/l, troponin negative, FBC normal.
- Chest radiograph—normal.
- ECG—T wave inversion V3–V6, sum of SV1 and RV5/V6=45 mm.
- CTPA—right-sided pulmonary emboli. No pleural effusion or pericarditis.
- Echocardiogram—normal (no LVH, right ventricular strain or pericardial effusion; normal contractility).
- Duplex ultrasonography right leg veins—no evidence of DVT.

Third presentation

- Blood tests—CRP 55 mg/l, ALT 117 IU/l, urea 3.5 mmol/l, creatinine 99 μ mol/l, FBC normal.

Figure 2 ECG at the second presentation.

- ▶ Chest radiograph—bilateral pleural effusions.
- ▶ ECG—some improvement over the previous condition.
- ▶ CTPA—no new changes apart from bilateral pleural effusions.
- ▶ Ultrasounds of liver and biliary systems—unremarkable.
- ▶ Special blood tests—autoantibodies negative (ANA 0.3 IU/ml, RF 18 IU/ml) Tumour markers negative (CEA 0.8 ng/ml, CA 19.9 < 1 U/ml, PSA 0.4 ng/ml)

Follow-up in clinic (about 2 months later)

- ▶ Blood tests—CRP 1 mg/l, ALT 27 IU/l.
- ▶ Chest radiograph—resolution of pleural effusions.

DIFFERENTIAL DIAGNOSIS

The first presentation: suspected DVT but disproved by complete duplex venous ultrasonography.

The second presentation: despite no evidence of DVT on previous ultrasonography, our main differential diagnosis was still PE. Pneumonia and pneumothorax were possibilities, but these were disproved by a normal chest radiograph. Given the clearly pleuritic nature of the chest pain and the absence of a viral prodrome, acute coronary syndrome and myocarditis, respectively, were thought to be highly unlikely. This was supported by the negative troponin. PE was confirmed on CTPA. However, given unusual ECG changes, we had to exclude incidental LVH secondary to undiagnosed hypertension or cardiac pathologies such as hypertrophic obstructive cardiomyopathy. The echocardiogram was normal. This provided confirmation that PE was the sole condition responsible for the abnormal ECGs.

Third presentation: main differential diagnosis was PE with complications (pleural effusions, hepatic congestion). Although unlikely, we were concerned to exclude systemic autoimmune diseases such as systemic lupus erythematosus or an underlying malignancy as predisposing factors for the patient's susceptibility to venous thromboembolism. These could independently cause pleural effusions and deranged liver function tests. Tests for autoantibodies and tumour markers were unsurprisingly negative in keeping with the lack of supporting evidence from the patient's history and examination.

TREATMENT

After confirmation of PE, our patient was anticoagulated with a low molecular weight heparin, Tinzaparin at a dose of 175 Units/kg. He was also started on warfarin at the same time. Tinzaparin was stopped when the international normalised ratio (INR) reached a therapeutic level. Treatment was mainly supportive (oxygen, intravenous fluids, analgesia) when he presented the third time.

OUTCOME AND FOLLOW-UP

Eleven days from the first presentation, our patient was discharged home with no further related readmissions. He remains well to date.

DISCUSSION

Apart from the post-ankle fracture left-leg DVT, our patient had no personal or family history of cardiovascular ill-health including hypertension, hypertrophic obstructive cardiomyopathy, sudden cardiac death or use of a pacemaker. The underlying PE, complicated by the

Table 1 Sensitivity and specificity for selected ECG criteria for LVH⁴

	Sensitivity	Specificity
Sokolow-Lyon voltage SV1 + RV5/RV6 > 35 mm	22	100
Cornell voltage criteria R in aVL + S in V3 (> 28 mm in ♂, > 20 mm in ♀)	42	96
Cornell voltage duration criteria > 2400 mm/ms	51	95
RaVL > 11 mm	11	100
Romhilt-Estes > 4 points	54	85
Romhilt-Estes > 5 points	33	94

aVL, left augmented limb lead; LVH, left ventricular hypertrophy; RaVL, R wave in the left augmented limb lead.

presence of bilateral pleural effusions and hepatic congestion, explains our patient's clinical presentation.

ECG changes in PE are well documented with the commonest change being sinus tachycardia. The ECG is abnormal in over 70% of patients with PE, the earliest descriptions dating back to the mid-1930s.¹

Other ECG findings include

1. Dominant R wave in lead V1
2. Right bundle branch block
3. Right axis deviation
4. Q wave and inverted T wave in III
5. S1Q3T3
6. T wave inversion V1–V4
7. P pulmonale
8. Supraventricular arrhythmias
9. Persistent S wave in V6

The patient's ECG pattern of left ventricular strain secondary to PE was unusual. To the best of our knowledge, this is the first description in the literature. ECG mimicry of intracardiac conditions like myocardial infarction is well established.^{2–3} The ECG criteria for diagnosing LVH have specificity above 85% (table 1).⁴ Both the Sokolow-Lyon voltage criteria and the Romhilt-Estes' scoring system (table 2) were met by the patient, suggesting LVH. However, the echocardiogram, which is highly

Table 2 Romhilt-Estes' scoring system for LVH.⁴ Probable LVH is diagnosed if 4 points are present and definite LVH is present if 5 or more points are present. Our patient scored 6 points (voltage, and ST segment and T-wave changes without digitalis)

Voltage (any of the following):	3
R or S in limb lead 20mm or more	
S in V1, V2 or V3 25mm or more	
R in V4, V5 or V6 25mm or more	
ST segment and T-wave changes (typical strain)	
Without digitalis	3
With digitalis	1
Left axis deviation > -15° or more	2
QRS duration > 0.09s	1
LA involvement	
P-terminal force in V1 > 1mm depth with a duration > 0.04s	3
Intrinsicoid deflection in V5 or V6 > 0.05s	1
LVH	5
Probable LVH	4

sensitive for detecting LVH,⁵ was negative. LVH, therefore, was not the cause of the left ventricular strain pattern. The negative troponin also served to suggest that intracardiac conditions such as myocardial infarction or myocarditis, whether as a consequence or incidental to the PE, were unlikely contributors for the left ventricular strain pattern on the ECG. The mechanism of LVH morphology on these ECGs, secondary to PE, is unclear.

We would like to emphasise that although DVT was missed on duplex venous ultrasonography of the proximal and calf veins, this is an uncommon phenomenon. The sensitivity of venous ultrasonography for the diagnosis of symptomatic proximal DVT and isolated calf vein thrombosis is 97% and greater than 90%, respectively.⁶ It is generally safe to withhold anticoagulation in patients with negative venous ultrasonography that include both the proximal and calf veins, short of performing contrast venography which is invasive, technically difficult and more costly and therefore not practical for routine clinical evaluation of possible DVT.⁶

Learning points

- ▶ Left ventricular strain pattern on the ECG is a very rare manifestation of pulmonary embolism.
- ▶ Deep vein thrombosis (DVT) can be missed by venous ultrasonography. However, in the vast majority of cases, this investigation is sufficient for diagnosing DVT.
- ▶ Pleural effusion and hepatic congestion are potential complications of pulmonary embolism.

Competing interests None.

Patient consent Obtained.

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Please cite this article as follows (you will need to access the article online to obtain the date of publication).

Tan A, Yikona J. Pulmonary embolism mimicking left-ventricular hypertrophy on ECG. *BMJ Case Reports* 2012;10.1136/bcr-2012-007092, Published XXX

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