CASE REPORT

New onset S wave in pulmonary embolism: revisited (something old and something new)

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SUMMARY

We report a case of a young man who had a new onset S wave in lead 1 in his ECG with typical symptoms of acute onset of dyspnoea 2 months after an episode of deep vein thrombosis, S wave disappeared 6 days after thrombolysis. We report this case as the clinical course was very typical plus we have reviewed the literature regarding diagnosis and risk stratification of pulmonary embolism for the student, or the casualty medical officer.

BACKGROUND

We had previously reported how an S wave in lead 1 disappeared after successful thrombolysis or after pulmonary embolectomy in an acute pulmonary embolism.¹ In two of our cases the patients presented with acute dyspnoea, low blood pressure and tachycardia. Their ECGs showed a new onset S wave in ECG lead 1, a Q in lead 3 and a new onset right bundle branch block in V1. As others²⁻³ have reported, we consider that S1 Q3 T3 occurs only when a major pulmonary artery is obstructed and could be a surrogate marker for this obstruction, especially in patients in shock. We want to present one more patient who had a new onset S wave that disappeared with thrombolysis. We will also discuss the updated guidelines since we published our last article.

CASE PRESENTATION

The patient, aged 31 years, had deep vein thrombosis (DVT) 2 months before this admission and was then admitted in December 2011 for sudden onset breathlessness from the previous day afternoon to our intensive cardiac care unit. He presented with a heart rate of 110/min, his respiratory rate was 24/min and his blood pressure was 120/80 mm Hg. On cardiovascular examination his jugular venous pressure was mildly elevated (6 cm from the sternal angle) and his apex beat was in the fifth left intercostal space in the mid-clavicular line. His S1 was normal S2 was widely split P2 was normal. He had a third heart sound (RV). His lungs were clear. His ECG showed: sinus rhythm (figure 1) HR 100/min, PR 140 ms. QRS duration 80 ms and mean frontal QRS axis +90. Lead 1 showed an S wave.

A provisional diagnosis of pulmonary embolism was made and he was sent for spiral CT (figures 2 and 3). Since he was very dyspnoeic and the CT scan showed pulmonary embolism involving the right descending pulmonary artery and the left main pulmonary artery extending into the branches

he was thrombolysed with streptokinase. We followed the report of the radiologist. His RV was also dilated when compared with the LV (figure 4) and his pulmonary arteries appeared dilated (figure 5). His X-ray chest posteroanterior view was unremarkable (figure 6). Finally on the sixth day his ECG reverted to normal and the new onset S wave disappeared from lead 1 (figure 7).

INVESTIGATIONS

Haemoglobin 14.5 g%. His total count was 9850/mm³, erythrocyte sedimentation rate 30. Differential leukocyte count was P62.L37. His blood sugar was 79 mg%, blood urea 18 mg%. His D-dimer was 1.4 mEq (normal value for the lab was 0.5 mEq). His serum creatinine was 1.1 mg%. Total cholesterol 228 mg/dl, low-density lipoprotein (LDL) 152, high-density lipoprotein 28 mg/dL and TG 244, very-LDL 48. PT international normalised ratio (INR) at discharge was 2.18. His creatinine phosphokinase was 111 iu/L, myocardial creatinine phosphokinase was 3/52 iu/L.

His PESI score was 61. And modified PESI score was 1.⁴ Approximately our patient seemed to have a CT obstruction index of 62% (left pulmonary artery score—20 right 5, 25/40=62.5%) and a miller index of 11/16=68.75%.

DIFFERENTIAL DIAGNOSIS

- ▶ Acute pulmonary embolism
- ▶ Chronic obstructive pulmonary disease exacerbation
- ▶ Pneumonia.

TREATMENT

The patient was treated with intravenous streptokinase at 1 lakh units/h infusion for 24 h. Then warfarin was started and till his INR reached 2–3 he was put in intravenous heparin.

His echocardiogram showed resolving RV function. He was also treated with aspirin and clopidogrel as this is our policy. His repeat ECG after 5 days showed the S wave in lead 1 had disappeared (figure 7).

OUTCOME AND FOLLOW-UP

The patient improved and comes for follow-up regularly.

DISCUSSION

Early decisions regarding the treatment of pulmonary embolism have helped improve survival in pulmonary embolism. Sam *et al*⁵ have compared the

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Figure 1 ECG of the patient showing an S wave in lead 1.

PESI with modified PESI (sPESI) score, and with the shock index to prognosticate pulmonary embolism.

Both PESI and sPESI scoring delineate low risk pulmonary embolism patients. Shock index prognosticates high risk patients. We feel that adding new onset S wave to the diagnosis will help decisions regarding thrombolysis or embolectomy.

PESI scoring

Briefly the PESI scoring system is

Class 1: score less than 65

Class 2: score 66–85

Class 3: score 86–105

Class 4: score 106–125

Class 5: score more than 125.

Classes 1 and 2 are defined as low risk.

The components of this risk scoring are: age in years, age greater than 80 years, men, cancer, heart failure, chronic lung disease, pulse rate, pulse greater than 110, systolic blood pressure less than 100 mm Hg, respiratory rate more than 30/min, temperature below 36°C, altered mental status, and SaO₂ less than 90% (the scores were age in years, +10, +30, +10, +10, +20, +30, +20, +20, +60, +20, respectively).

The modified PESI score or sPESI score that was simplified by Jimenez,⁴ is much simpler to be used especially in the emergency department and its use has been found to be equal in

prognosticating survival in pulmonary embolism. The simplified PESI score is the following age greater than 80 years –1 point, cancer 1, history of heart failure or chronic lung disease 1, pulse rate more than 100 is 1 or systolic blood pressure less than 100 is 1, and arterial oxygen saturation less than 90% 1 score.

In this simplified scoring a score of 0 was low risk and more than 1 was high risk.

So according to this our patient had a high risk. He was thrombolysed (by our previous experience of combining the following parameters—classical dyspnoea of new onset in a patient with a history of DVT, new onset S wave in pulmonary embolism, a spiral CT showing obstruction to the major branches of the pulmonary artery, dilated RV in the CT scan, dilated RA and RV on echocardiogram, and shock index of 1).

Since he was not hypotensive we feel if we had not observed the new onset S wave, we might have treated him as a low risk patient. In our experience not thrombolysing such patients later leads to death. Sam *et al*⁵ have also compared the PESI and

Figure 2 CT scan of the patient showing a thrombus in the pulmonary artery obstructing both the right and left main pulmonary arteries.

Figure 3 CT scan showing a filling defect in the right pulmonary artery, probably 50% obstruction of the upper lobe artery.

Figure 4 Another view of the CT scan showing the dilated right ventricle, almost the same size as the left ventricle, indicating a significantly large pulmonary embolism.

sPESI scoring to the shock index. The shock index is⁶ defined as the heart rate divided by the systolic blood pressure.⁵ This is an independent predictor of 30-day mortality. If more than 1 it is associated with more shock, death or inotropic needs.⁶ Our patient's shock index was 100/120, less than 1. His shock index was negative.

We think that this report will help the causality medical officer make rapid decisions.

The ACCP guidelines also recommend thrombolysis for those patients who have a risk of developing hypotension (2C).^{7 8} Our patient falls in this category.

The American Heart association has published guidelines on the massive and submassive pulmonary embolism.⁹ This study commented on the use of RV to LV size in the CT angiogram as a marker of massive pulmonary embolism with a poor prognosis. Our patient has a dilated RV on CT angiogram. Further they have also commented that elevated troponin T, elevated brain natriuretic peptide and elevated D dimer are also adverse prognostic factors in pulmonary embolism. They also comment on the shock index.⁹ The long-term benefits of pulmonary thrombolysis are beyond mortality benefit.

In the Emperor registry the mortality of submassive pulmonary embolism treated with heparin was less than 3%.

Figure 5 Contrast CT scan showing the branches of the pulmonary artery appear plump and dilated showing indirectly the presence of a thrombus burden.

Figure 6 Chest X-ray posteroanterior view.

Thrombolysis usually causes a mortality reduction of 30% (relative). So in submassive pulmonary embolism⁹ the absolute mortality reduction with thrombolysis would be around 1%. But thrombolysis has been prospectively shown to reduce CTPEH at 6 months and improves the quality of life, the 6 min walk test and the persistent RV dysfunction.

ECG in pulmonary embolism

We thought we were the first to show regression of ECG changes on thrombolysis or embolectomy after treatment of pulmonary embolism. But unknown to us, Todd has showed an S wave disappearing after embolectomy for pulmonary embolism. We would also like to report the article of Cheng¹⁰ that was published in *Heart* 2005, here the authors show that a large P wave in lead 2 regressed after thrombolysis with alteplase. The ECGs are beautifully shown in this article. They believed this P wave was due to right atrial strain, and that regressed after treatment.

Ferreri *et al* have reviewed T inversion in anterior precordial leads as a sign of massive pulmonary embolism. They correlated 80 cases with this sign with their right heart pressures and the Miller index. They again reported amelioration of these changes with normalisation of the right-sided pressures.¹¹ It was considered a strong marker when it appeared on day 1 and a good prognostic sign when it disappeared on day 6 or earlier.

What was the Miller index?^{12 13}

This was an angiographic scoring system for pulmonary embolism (1971). The T wave changes in the anterior leads were found to correlate with the Miller index. Briefly, after angiography of the right and left pulmonary arteries the extent of thrombus was scored. The right pulmonary artery was considered to have nine major branches and the left seven. An embolus in any of these was scored as 1 and a total score of 16 was obtained.

To assess whether the pulmonary embolism was significant or not, the lungs were divided into an upper, middle and lower zone where each got a score of 3—in no flow at all, if severely reduced flow; 2 points mildly reduced flow; 1 point—0 was normal flow. Thus the Miller score ranged from 0 to 34. The higher the score the worse the involvement.

Miller index still has relevance in this day and age. It has been used to validate a new CT scan scoring system.¹⁴

Figure 7 ECG showing regression of the S wave in lead 1, compared with figure 1. There is no S wave in lead 1.

Obstruction to the major pulmonary arteries on a CT scan has been correlated with the Miller index.¹⁴ With the CT scans evaluated by two independent observers the Miller index correlated with the CT scan index ($r=0.904$, $p<0.0001$). Further the CT scan obstruction index and the Miller index were correlated with the ratio of RV to LV. If the ratio of the RV to LV was more than 0.6 then this is correlated to a higher CT scan obstructive index and more RA and RV dilatation on echocardiography. A ratio of more than 0.6 correlated with an obstruction index of more than 40% of the pulmonary vascular bed.

We believe our patient had an RV–LV ratio of more than 0.6 (almost=1) and a CT obstruction index of 63%. So by these criteria our decision to thrombolysed was correct. He also had a dilated RA and RV on echocardiogram.

How to calculate the CT obstruction index

The arterial tree of each lung was regarded to have 10 segmental arteries. Each artery was scored separately. (Three arteries in

the upper lobe, two in the middle lobe and five to the lower lobes and two to the lingula.) The presence of thrombus in a segmental artery was scored as 1 if partially obstructing, and 2 if totally obstructed. So the total score was 40 for both lungs. So the index was total score/40 $\times$ 100 expressed as percentage.

In our patient we thought the left pulmonary artery was totally obstructed=score 20 and right only 50% score 5. So 20+5=25, 25/40 $\times$ 100=62.5%.

Contributors PNG wrote the article and followed up the patient. SBP retrieved the CT scan and the ECG. SZA found the reversion of the S wave and SMB reviewed the manuscript and treated the patient in the ICU.

Competing interests None.

Patient consent Obtained.

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Learning points

- New onset S wave in lead 1 in patients with a background for it should make one suspect pulmonary embolism.
- Apart from echo some risk stratification can be carried out on clinical features, using the PESI or sPESI scores.
- Shock index is useful in deciding on thrombolysis, if RA and RV are dilated and the patient cannot be taken for CT scan.
- CT scans of the patient with pulmonary embolism can give an idea of the magnitude of the thrombus in the pulmonary artery using the CT obstruction index.
- The RV-to-LV size ratio in CT scan of more than 0.6 gives an idea about the severity of pulmonary embolism, and can be used to help decide on the line of treatment (thrombolysis vs only intravenous heparin).
- The ECG changes of pulmonary embolism can regress if the patient is treated by thrombolysis.

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