S wave in Pulmonary Embolism, a new ECG sign to aid thrombolysis

Thomas John, Sajan Ahmad Z, Prabha Nini Gupta, Praveen GK, Krishna Kumar B, Rajasekharan VR, Suresh, Deepak Madhu, Sanjeev, Shivaprasad K, A George Koshy, Sunita Vishwanathan

Abstract:

Acute pulmonary embolism is a devastating disease that often leads to mortality. Previous workers have found that thrombolysis reduces mortality in men but not significantly in women with pulmonary embolism. Many of the previous studies are with tenecteplase and alteplase. Here, we describe intravenous thrombolysis with streptokinase in 7 patients with pulmonary embolism who survived including 2 women. Further, we have one patient who had a new onset of S wave in lead I and this subsequently disappeared after embolectomy. We also comment on the usefulness of shock sign in deciding on thrombolysis.

We propose a new sign for noninvasive assessment of need for thrombolysis in pulmonary embolism, that is, new onset S waves in Lead I in pulmonary embolism, can be used as a new sign). Further, added to the shock sign it can be used in an emergency to save the patient.

Further, there are no clear end points as to when to stop thrombolysis. In all 4 patients we switched to heparin when spontaneous bleeding/oozing started. In all 4 patients subsequent CT scans showed that the patient has mild to moderate resolution of the pulmonary embolism and patients remained stable and have been discharged and have come for follow up. Hence this sign, bleeding, we propose can be used as an end point for thrombolysis in pulmonary embolism.

Lastly, we also describe a patient who had new onset S wave that disappeared after successful pulmonary embolectomy. Probably, the S wave is a marker of main pulmonary artery branch occlusions.

We propose that new onset S waves in Lead I in pulmonary embolism be used as a new sign for noninvasive assessment of need for thrombolysis.

Pulmonary embolism is a major cause of morbidity and mortality in hospitalized patients. The common electrocardiographic findings are – normal in 30%, S1 Q3 T3 with complete RBBB, P pulmonale, and right axis deviation occur in 26% to 32% of patients who had sub-massive or massive acute pul embolism in those who had otherwise normal hearts.

Q waves do occur in acute myocardial infarction but also occur in pulmonary embolism. Both right and left axis shift occur in pulmonary embolism.

Low voltage QRS complexes may also occur.

The usual indications for thrombolysis in acute pulmonary embolism are a shocked patient with all
the predisposing features for deep venous thrombosis developing acute dyspnea. Usually the diagnosis is confirmed by echocardiography, and spiral CT. There is no strict ECG criteria for the need to thrombolys a patient with pulmonary embolism. Newer guidelines have described both echocardiographic and CT criteria combined with Wells index, modified to assess the need for thrombolysis in pul embolism.

Here we present a case of acute pulmonary embolism that developed new onset S waves during the embolism that regressed with thrombolysis.

**Table 1 –Patients with Pulmonary embolism**

<table>
<thead>
<tr>
<th>Sex</th>
<th>Age</th>
<th>Thrombolysed or not</th>
<th>Shock index</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>F</td>
<td>25</td>
<td>no</td>
<td>130/130/70</td>
<td>Died</td>
</tr>
<tr>
<td>F</td>
<td>54</td>
<td>yes but discontinued after 4 hrs due to bleeding from endotracheal tube On dopamine</td>
<td>120/110/70</td>
<td>died</td>
</tr>
<tr>
<td>F</td>
<td>42</td>
<td>Streptokinase given, dobutamine given</td>
<td>120/120/80</td>
<td>survived</td>
</tr>
<tr>
<td>F</td>
<td>43</td>
<td>Streptokinase given</td>
<td>130/110/80</td>
<td>survived</td>
</tr>
<tr>
<td>M</td>
<td>43</td>
<td>Streptokinase given</td>
<td>130/110/80</td>
<td>survived</td>
</tr>
<tr>
<td>F</td>
<td>29</td>
<td>Streptokinase given</td>
<td>130/90/60</td>
<td>had heavy bleeding</td>
</tr>
<tr>
<td>F</td>
<td>63</td>
<td>pulmonary embolectomy done</td>
<td>74/60</td>
<td>survived</td>
</tr>
</tbody>
</table>

**Table 2 –The electrocardiogram and CT report of the patients with pulmonary embolism**

- **F 25**: ECG – HR 150/min T inversion 2,3,avf showed pul embolism in the upper and lower branches of both pul arteries. Axis + 60.
- **F 54**: rate 100/min, QRS axis +30 showed pul embolism. T inversion V1 to V4. Distal RPA occluded and pul infarct seen in CT.
- **F 42**: RBBB, S1Q3T3 showed MPA saddle embolus (30%) obstructing Lumen, RPA, LPA and segmental arteries.
- **F 43**: rate 130/min S1Q3T3, ST depression V1-V6 showed clot seen partially occluding pul artery and wholly occluding let inferior pul. Artery.
- **M 43**: HR 120/min S1Q3 T3 seen See text.
- **F 29**: HR 120/min S1Q3T3 Had APLA, large thrombus in LPA.
- **F 63**: HR 73/min S1Q3T3 seen sent for embolectomy.

We feel this S wave sign would be a useful marker of successful thrombolysis for pulmonary embolism.

**Patients and methods:**

Of 2467 admissions for January 2009 to November 2010 in the intensive coronary care unit in Medical College Trivandrum, we had 7 admissions for pul embolism (0.28%). Of these, 3 died (40%). We were unaware about the Shock index previously, hence we have not included earlier years of patients. One previous patient is included to show the point about bleeding from a previous year.

**Patient no. 1**: Our patient is Mr. S aged 43 years.

He had H/o both bones fracture of the left leg with external fixation done 4 weeks earlier. He developed sudden onset of breathlessness on the morning of admission. No other obvious cause of breathlessness was found. His electrocardiogram showed – 23-4-10. Showed – Sinus rhythm, PR interval .16 sec, QRS duration. 12 sec, and deep S waves in lead I and RBBB in V1 and slurred S waves in lead V5 and V6. He had small non pathological Q waves in 2,3 avf (see fig 1).

His heart rate was 130 at admission and his blood pressure was 120 /80. His shock index was positive. He was tachypnea. An emergency echo revealed dilated RA and RV with mild TR and tachycardia. There was no thrombus seen in the MPA or RPA or LPA on echocardiogram. As per institution policy he was sent for CT chest.

This showed pulmonary thromboembolism in the distal lobar segmental branches of right and left pulmonary arteries. It also showed dilated right and left pul arteries.

In view of his back ground, he was provisionally diagnosed to have acute pul embolism and he was thrombolysed with streptokinase, intravenously at the rate of 1 lakh units per hour. The next day early morning the patient developed mild bleeding from the skin wound of his fracture and the thrombolysis was stopped. He was also given tablet of...
enteric coated aspirin 150 mg daily, clopidogrel 75 mg daily and was also started on intravenous heparin infusion after the streptokinase at 1000 i/u per hour.

He was followed up with daily ECG and echocardiograms. His RA and RV dilatation came down. His heart rate fell to 103 /min. and his repeat CT scan showed some residual thrombus but most of the pulmonary emboli had reduced.

Most interestingly on day-3(26/4/10), his electrocardiogram reverted to his pre pulmonary embolism ECG with reversion of the deep S wave in lead one and disappearance of right bundle branch block and that the S wave in I had disappeared (See fig 2). A repeat CT scan showed improvement in the pul embolism

He was then started on warfarin 5 mg and was kept on this till his INR became 2.5. And, he was then discharged. He has come twice for follow-up. The last on being on 19/6/2010. The patient is stable and doing well.

Other noninvasive indicators for thrombolysis in pul embolism are:

Shock index: Heart rate/systolic blood pressure if more than 1 shows increased mortality 2. We also used this point in deciding on thrombolysis.

Hence, we also postulate that bleeding is a good end point to stop thrombolysis with streptokinase.

**Patient No 2:** Was a 43 years old lady who developed sudden onset of dyspnea 1 week after varicose veins surgery. She came from the surgery department with a Doppler of the leg veins. This showed evidence of DVT in the right venous system extending up to the middle of the IVC. She was immediately sent for CT scan which showed thrombus in the proximal pul arteries.

When she reached our ICCU, she was tachypneic, her pulse rate was 120/min, and her blood pressure was 100/60. Since there was documentation of her emboli, we thrombolysed her with I/v streptokinase. The next day early morning she also developed mild bleeding from a venous ulcer on the ankle joint, left. We stopped the thrombolysis. Started her on heparin and warfarin till her INR was 2. She was then sent home. She had 2 follow up visits. Her repeat Doppler of the leg veins was normal. She was gradually ambulated and sent home.

She came for review after 1 month and was doing well.

In this case also, bleeding was the end point and later investigations showed that she had improved. Hence, here also we postulate that bleeding is a good end point to stop thrombolysis with streptokinase.

We include one more patient from a previous year.

**Case 3:** Was a 55 year old lady who had had a hysterectomy 4 days earlier, vaginal hysterectomy. She was slightly obese. On ambulation she suddenly developed syncope. Her ECG revealed only T inversion in the V1 to V4. Since she was obese, the
possibility of coincident non Q MI was thought of(NSTEMI) and metoprolol 25 mg twice daily orally was started. But in view of her post op background she was sent for spiral CT which showed a large thrombus in the pul artery bifurcation. She was thrombolysed with intravenous streptokinase 1 lakh units per hour without the bolus. After 4 hours she developed bleeding vaginally. The gynecologist was called. She found bleeding from the vault of the surgery and packed the vagina tightly. The streptokinase was stopped. One pint of blood transfusion was given and aspirin was continued. Bleeding from the vault of the vagina continued for 48 hours totally. After this the CT was repeated on the 4th day of arriving in cardiology. This showed the thrombus in the PA had substantially come down. Hence, slowly low dose warfarin was started. She was gradually ambulated and discharged. She has come for follow up and is doing well.

Again, bleeding was the end point to stop thrombolysis and the patient was stable after this on warfarin.

Patient no. 4: On 20 11 2010 we had 1 more case of pulmonary embolism. Since the electrocardiographic changes are very interesting we include this case also.

Disappearance of S wave following Surgical Pulmonary Embolectomy

We report an ECG marker of successful pulmonary reperfusion following surgical pulmonary embolectomy in a patient with acute massive pulmonary embolism.

Our patient was a 63 year old lady with Gastrointestinal Stromal Tumor (GIST), for which she underwent distal gastrostomy and anastomosis. On the 2nd postoperative day, she developed mild dyspnea. On the 3rd postoperative day, the dyspnea worsened and she developed hypotension (systolic blood pressure of 60 mm Hg). ECG showed sinus tachycardia, a new onset S wave in Lead 1, Q wave and T inversion in Lead 3 (S1Q3T3). Quantitative Troponin T estimation was positive. A clinical diagnosis of acute pulmonary embolism (massive) was made. An emergency Contrast Enhanced High Resolution CT Chest was done, which revealed extensive thrombi in the right and left pulmonary arteries and their segmental and sub segmental branches(Fig 3).

Since the patient had a major gastrointestinal surgery 2 days ago and because the surgical drains were showing oozing of blood, it was decided not to thrombolys the patient with Streptokinase. A bolus dose of 5000 units of IV Unfractionated Heparin was started, followed by 1000 units/hour infusion.

Thoracic surgery team was alerted and an emergency surgical pulmonary embolectomy was done under cardiopulmonary bypass (Fig 4). Following the surgery, patient became hemodynamically stable and her dyspnea was relieved. The postoperative ECG showed disappearance of the S wave in Lead 1. Heparin infusion was restarted and the patient was initiated on Warfarin. The patient has been ambulated and is now stable.

Discussion:

Pulmonary embolism is a fatal disorder known to recur in spite of successful treatment. Urgent decisions regarding the use of thrombolytic agents are often lifesaving. However very often whether or not to thrombolysy is a difficult decision. So we felt the presence and disappearance of an S wave added to the so called shock index, which we used really aided our decisions and we wanted to share this experience.

Due to cost constraints we usually do not use tenectaplace. 50% of all patients with pulmonary embolism present as clinically stable without hypotension or circulatory failure 2, though they have RV dysfunction.
These patients are at high risk of death even during the first few days after admission. Patients with a possible Shock Index should be thrombolysed.

Shock index= HR/SBP if more than 1, the patient has increased mortality.

Our patient had an HR of 130 and a systolic BP of 120

Shock index was more than 1 130/120=1.08

Using this, we thrombolysed the patient.

In addition, the presence of a new onset S wave in an otherwise normal ECG in lead 1 can be used like ST elevation in myocardial infarction to help thrombolysis in acute pulmonary embolism.

When successfully performed thrombolysis will reduce and reverse right sided heart failure by physical dissolution of an anatomically obstructing pulmonary arterial thrombus, thereby reducing right ventricular overload.

Thrombolysis also prevents the need for escalation of therapy like need for pressers, mechanical ventilation, and cardiopulmonary resuscitation. Most of the current recommendations are for alteplase, as an infusion over 2 hours.

Pulmonary embolism has been classified as Massive, sub-massive and small to moderate. Our patient 1 probably had a sub-massive pulmonary embolism.

Prognosis in Pulmonary embolism:

In the International Cooperative Pulmonary Embolism Registry, among 2454 patients with pulmonary embolism, intracerebral hemorrhagic complications occurred in 3% of patients treated with thrombolysis. The major risk factor for bleeding with thrombolysis was found to be increasing age.

In a German multicenter pulmonary embolism registry 3 thrombolysis was associated with a 79% reduction in mortality in men but no significant reduction in women. In this study, we describe 2 female patients who successfully went home after thrombolysis in spite of bleeding.

We postulate the start of bleeding in a patient with pulmonary embolism is a good reason to stop thrombolysis. And usually the repeat CT scans would show almost complete resolution of the pul artery thrombus. And after this heparin and oral anticoagulants are sufficient.

All 3 of our patients had bleeding. This was the end point of thrombolysis.

A word about streptokinase. This causes a systemic lytic state. It is possible that bleeding denotes adequate systemic levels of the drug and fibrinolysis. So it's possible that this is why even after stopping the drug no further deterioration occurred in these 3 patients.

Clinical presentation and need for thrombolysis:

When a typical patient with typical predisposing factors presents with shock and dyspnea, especially if there is no other diagnosis clinically suspected, pulmonary embolism should be suspected. And if the echocardiogram is supportive, the ecg shows new onset S wave in lead 1 and RBBB or S1 Q3 T3 and the CT scan is diagnostic, then thrombolysis should be started.

If spiral CT is not available and the echocardiogram is very suggestive of pul Embolism and the patient is in shock again, thrombolysis should be given.

The common predisposing factors for pul embolism are deep venous thrombosis, surgery for varicose veins(we have seen this on 3 occasions) heart rate more than 100, immobilization within 4 weeks or plane travel or long journey. Cancer is also a reason for pulmonary embolism.

Previously it was believed that only patients with angiographic presence of thrombus should be thrombolysed but now the guidelines allow CT
documentation or echocardiographic findings for decision for thrombolysis. In a recent algorithm even transthoracic or transeosophageal echocardiography could be used to decide on thrombolysis 6.

ECG changes in Pulmonary embolism:
It has been believed by many authors that ECG is a poor indicator of pulmonary embolism 4. It is true that the sensitivity of S1Q3T3 is low. But we believe in the appropriate setting it may be used to start thrombolysis in a life saving situation. These workers have even postulated that ECG in pulmonary embolism should be used only to rule out acute myocardial infarction. McMinn and White 4 were the first to note the S1Q3 T3 pattern in acute pulmonary embolism.

The commonly described ECG changes in pulmonary embolism include 4, normal ECG (9-30%), sinus tachycardia (8-69%) RBBB -6-67%.RBBB has previously been shown to persist for 3 month to 3 years. This is believed to be due to massive pulmonary trunk obstruction and RV acute overload. It is possible that the S wave in lead 1 is a marker of major pulmonary artery block. This is shown by the early resolution of the S wave in the pulmonary embolectomy patient. We hope this sign will help treatment of this group of sick patients.

The QRS axis can change in pulmonary embolism LAD , RAD and Indeterminate axis can occur in pulmonary embolism. Right axis deviation is supposed to be the classical finding but it has been found that Left axis deviation of the QRS axis is more common and believed to be due to associated cardiopulmonary disease.

Transition zone shift-usually an R and S in V1 to V6 becomes equal in lead V3. In acute pulmonary embolism the R/S become equal in V5. This is postulated to be because of hypoxia to the myocardium and slowing of the conduction of the Purkinje system of the right ventricle. It is also because of acute right ventricular dilatation.

Low voltages can also occur in pulmonary embolism ST T changes have been used to prognosticate patients with pulmonary embolism. If T inversion occurs in more than 7 leads there is likely to be right ventricular dilatation on echocardiography. This also predicts the need for cardiopulmonary resuscitation, catecholamine and the need for mechanical supports.

S1Q3T3 has been shown to occur in 10-50%, P pulmonale in 2-31%, P pulmonale and atrial arrhythmias can occur due to acute atrial dilatation. In some series atrial flutter and fibrillation have been reported in 18-35%.

During pulmonary embolism there is secretion of vasoactive substances. The ECG changes may be due to this.

Sreeram 4 has described a predictive index for pulmonary embolism. The presence of 3 of the criteria would predict accurately pulmonary embolism in 76% of cases. The following ECG criteria are included:
1. Incomplete or complete RBBB with ST elevation and positive T wave
2. S in I and avl of larger than 1.5 mm
3. Shift in the transition zone to V5.
4. Q in III avf but not in II.
5. Low voltage QRS less than 5 mm in the limb leads
6. T inversion in III avf and or V1-V4.

If 3 are positive this has a sensitivity of 94%. This can also be used to decide on thrombolysis.

References:
1. Stein PD, Sostman DH, Russel DH, et al; Am J Cardiol 2009:103; 881-886, “Diagnosis of Pulmonary Embolism in the Coronary Care Unit”.