

Delayed Administration of Thrombolysis for Cardiac Arrest due to Pulmonary Embolism: Case Report and Review of the Literature

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Abstract:

The use of thrombolytic therapy during CPR in massive pulmonary embolism (PE) has been recognized. Ongoing areas of debate include optimal techniques, aiding in the diagnosis of fatal PE, and the use of thrombolysis or embolectomy in cardiac arrest due to PE. We report a case of Pulseless Electrical Activity (PEA) due to massive PE, successfully treated with a single bolus of streptokinase, and discuss the relevant literature.

MeSH Words: Pulmonary Embolus, Pulseless Electrical Activity, Cardiac Arrest, Streptokinase

Case History

A 50 year old female was brought to the Emergency Department (ED) following sudden onset of epigastric pain, 1 hour prior to presentation to the ED, which remained constant with radiation to the back. There was a 5 day history of dyspnea on exertion. The patient was not a smoker, had no history of recent travel, hypercoagulable state, recent surgery, or coronary artery disease. Occupational history was non-contributory, and the patient did not spend an inordinate amount of time sitting. The patient was on no medications. There was no history of

fever, change in mental status, leg pains, hemoptysis, cough, or chest pain.

On arrival, the patient was pale, diaphoretic, tachypneic, anxious and confused. Her pulse was 130 beats/minute. She was in respiratory distress and unable to complete full sentences. There was prominent jugular venous distention. The lungs were clear to auscultation bilaterally, with good air entry. The radial pulse was thready and auscultation of the heart yielded no murmurs or gallop. The abdomen was soft.

The patient became bradycardic, bradypneic and unconscious within 3-4 minutes after the initial examination. Cardio-pulmonary resuscitation (CPR) was started. Endotracheal intubation was performed and mechanical ventilation was instituted. Venous blood gas values were pH-6.9, PCO₂-68 mmHg, PO₂-13 mmHg, bicarbonate 13mEq/L, base excess -20 mEq/L. An electrocardiogram showed asystole, with ventricular escape beats. Epinephrine and atropine were given intravenously. Hemoglobin was 12.2 gr/deciliter, Na 139 mEq/L, K 3.2 mEq/L, Ca 1.1 mEq/L, anion gap 22.6, glucose 236. A portable chest x-ray was normal. After 10 minutes of CPR, the patient resumed normal sinus rhythm, blood pressure was 40/70 and pulse was 110, end-tidal CO₂ was 15 mmHg.

Bedside transthoracic echocardiography was performed by a technician from the echocardiography unit and interpreted in real-time by a senior cardiologist with experience in echocardiography. This revealed a massively dilated right ventricle, pulmonary artery hypertension according to tricuspid regurgitation estimation, severely decrease right ventricular systolic function, and a dilated right atrium. The left ventricle showed normal wall thickness and wall motion, with an ejection fraction estimated at 55%; the inter-ventricular septum bulged into the left ventricle secondary to a dilated right ventricle with right ventricle volume overload; there was a normal aortic root, normal valve function, no pericardial effusion was noted. Abdominal ultrasound examination was normal.

Within 5 minutes of resumption of sinus rhythm, symptomatic bradycardia recurred which evolved into asystole. CPR was resumed, and epinephrine and atropine given again. After some 10 further minutes of CPR, the patient resumed sinus rhythm electrical activity but had no pulse or blood pressure (i.e. Pulseless Electrical Activity – PEA). End-tidal CO₂ was 8 mm Hg. CPR was continued with administration of 2 units of packed red blood cells, 4 liters of normal saline, and 500 ml of hetastarch solution, in order to increase oxygen carrying capacity and cardiac output. The patient was also placed on an intravenous dopamine drip at 22 mcg/kg/min.

In light of a normal ECG, recurrent PEA, the echocardiographic findings, and low end-tidal CO₂, a diagnosis of massive PE was made. CPR

was continued throughout this time period. Senior cardiovascular surgery staff were consulted to perform emergent embolectomy, but were reluctant to intervene, as they believed the patient to be beyond salvage. A decision was made to administer thrombolysis, in lieu of or as a bridge until surgical embolectomy. Streptokinase 1.5 million units was administered through the right internal jugular venous line, along with 5,000 units of heparin, as a manual bolus over 3 minutes. The patient was transferred to the pre-anesthesia care unit in order to facilitate both monitoring and intensive therapy and a possible rapid transfer to the operating room in case surgery was decided on later. The cardiac monitor demonstrated a sinus arrest with infrequent ventricular escape beats and then sinus rhythm but no palpable pulses. Chest compressions were continued en-route.

While in the pre-anesthesia unit, 15 minutes post streptokinase administration, strong carotid pulses were palpated. A femoral arterial catheter was placed and a blood pressure of 125/85 mmHg were measured; the ECG showed normal sinus rhythm. End-tidal CO₂ was 30 mmHg.

The patient was placed on IV dobutamine and weaned off dopamine, in order to enhance pulmonary circulation. Given the improving hemodynamic status, a decision was made to postpone surgery. The patient maintained hemodynamic stability on decreasing doses of dobutamine. However, repeat CBC showed Hemoglobin of 9.4 gram/deciliter; platelet count was 75,000/cubic mm. A spiral computerized tomographic angiography (CTA) of the chest was obtained as well as CT of the abdomen. CTA showed filling defects in the left and right sub-segmental pulmonary arteries consistent with a PE, and small bilateral pleural effusions. Abdominal CT showed small free intra-abdominal fluid, suggesting a retroperitoneal hematoma. The patient was transferred to the intensive care unit. Hemoglobin was maintained with 2 units of PRBC's over the subsequent 48 hours. An inferior vena cava (IVC) filter (OPTEASE™ Vena Cava Filter, Cordis Endovascular, Warren NJ) was placed at the bedside 2 days after admission.

Bilateral lower extremity Doppler venous ultrasound showed no thrombus and normal vein compression. CTA on the 10th hospital day was normal, without evidence of remaining PE. On

the same day, ventilation-perfusion pulmonary isotope scan demonstrated normal perfusion and ventilation with no mismatch. Repeat abdominal CT showed retroperitoneal and right psoas mixed dense areas consistent with a retroperitoneal hematoma. The patient's condition continued to improve subsequently, she was extubated on day 3 and discharged in satisfactory status on day 14.

The Chest X-ray on discharge was essentially normal, showing only mild atelectasis in the left lower lobe. Discharge medications included weaning doses of low molecular weight heparin and maintenance therapy of warfarin. A coagulation status work-up, taken on day 10 was negative for antinuclear antibody (ANA), C-ANCA, P-ANCA and anti-factor X.

At 2 months post discharge, the patient reported to have had no breathing difficulties since her discharge, and has returned to work full time as a care giver. Chest X ray at the time was normal. She has since returned to normal daily activity.

Discussion

Diagnosis

At the time of an acute life threatening massive PE, physicians operate in an environment of decision-making under uncertainty. No two cases are exactly alike and often the emergent time frame limits complete diagnostic information gathering. Emergency physicians may not be able to afford the time for definitive tests such as trans-esophageal echocardiography (TEE), CTA, V/Q scans, and/or pulmonary angiography, in association with a clinical risk stratification as recommended by the PIOPED investigators ([1] The PIOPED Investigators 1990). Especially in the case of PE associated with cardiac arrest/PEA, CPR requirements limit more advanced tests. D-dimer assays, even if useful at all for high-probability PE diagnosis, are less useful during prolonged CPR, because CPR activates the fibrinolytic pathway ([2] Bottiger 1995, [3] Gando 1997) and the true negative rate may change.

TEE can provide high sensitivity (92%) and high specificity for the diagnosis of PE (near 100%) in the setting of CPR and PEA ([4] Comess 2001). Trans Thoracic Echocardiography (TTE)

may be less sensitive, but it is more widely and readily available and it has been shown to be a very good addition to the clinical history, physical exam, and basic diagnostic tests such as a CBC, electrolytes, ABG, CXR, pulse oximetry and end-tidal CO₂ ([4] Comess KA 2001). MacCarthy et al reported that TTE can be an excellent adjunct in the Emergency Department and noted: "A Trans Thoracic Echocardiogram is usually abnormal in massive pulmonary embolism, showing right ventricular enlargement, a consequent increase in right ventricular to left ventricular diastolic diameter and paradoxical septal motion, both in systole and diastole. It also allows the simultaneous exclusion of important left heart or aortic abnormality." ([5] MacCarthy 2002). This corroborates that physicians contemplating PE in the setting of PEA should consider emergent TTE even during ongoing CPR. Outcome studies would have to be done to establish the use of TTE as a routine adjunct to ACLS in PEA/EMD, as it can differentiate between fatal PE, tamponade, and a large MI, which are all reversible causes of PEA and have different treatments.

Management of massive PE

Massive PE can be managed through multiple modalities, including surgical embolectomy, transvenous catheter embolectomy or transvenous pulmonary artery catheter lysis, peripheral venous thrombolysis and merely continuing ACLS protocols ([6] Wood 2002, [7] Arkasoy 1991). The aggressiveness and choice of these techniques depend on the prognosis of the patient, other underlying diagnoses such as terminal cancer, the availability of the various modalities, the experience of the surgeons, and the judgment of a group of practitioners. Clinical judgment and an emergent conference at the scene of the code is usually the setting where the choice of modality is made. Attending physicians must make a benefit versus risk decision in conjunction with considering availability of modalities and approximate survival rates for each modality in their center.

Surgical embolectomy: The availability and surgical experience varies by medical center and surgical embolectomy requires the use of extracorporeal bypass. Centers with a high volume of diverse cardiac cases may have an advantage in surgical embolectomy experience.

In the setting of strict contra-indications to thrombolysis, surgical embolectomy is the treatment of choice. The survival rate of surgical pulmonary embolectomy in massive PE not in the setting of cardiac arrest is as high as 89% ([9] Aklog 2001, [10] Aklog 2002). In the setting of cardiac arrest and post-CPR situations however, survival rates are as low as 7% ([11] Matis 1999). Consultation with a cardiovascular surgeon during the code clarifies the experience and judgment of surgeons who feel confident in surgical embolectomy techniques, and especially in PEA, as a last resort. In the above case, cardiovascular surgery was consulted. It was felt that surgical embolectomy was needed to be offered as an option after unsuccessful thrombolysis.

Transvenous catheter embolectomy is a newer technique with less widespread acceptance. The development of catheters is still in evolution and reported survival rates are 70% ([12] Suarez 2004). This option should be considered for patients in whom surgical embolectomy or thrombolysis are strictly contra-indicated ([13] Meyer 2001).

Thrombolysis via pulmonary artery catheter (PAC) is a well described and successful treatment for massive PE in stable patients ([14] Uflacker). However, insertion of a PAC during CPR would be very difficult and there are no reports of this technique during CPR.

Thrombolysis is suggested as a preferred treatment for PE with hemodynamic compromise ([6] Wood 2002). The choice of specific thrombolytic agents has not been established, so the clinician needs to make the choice between streptokinase, urokinase or recombinant tissue Plasminogen Activator (rtPA), based on availability. Some published regimens include: streptokinase (250,000–750,000 units by intravenous bolus or 1,500,000 units rapidly infused), urokinase (500,000–1 million units bolus), or TPA (100 mg in various schemes). In addition to thrombolytic agents, generally 5,000 U of Heparin is given by intravenous bolus, followed with 1,000 u/hr continuous infusion, or the body weight equivalent ([15], Bettiger 2001).

Research and literature reviews over the last 10 years have indicated that thrombolytics can be safely given in the setting of cardiac arrest not related to PE ([16] Ruiz-Bailen 2001). However,

strict contra-indications remain and the package insert should be reviewed before each administration. Updates can be found on the PDR.net website. Because thrombolytic therapy increases the risk of bleeding, thrombolytics are contraindicated in the following situations: a) active internal bleeding; b) recent (within 2 months) cerebrovascular accident; c) recent intracranial or intraspinal surgery; d) intracranial neoplasm; and e) severe uncontrolled hypertension. ([17] PDR.net 2004). Relative contra-indications such as recent major surgery, recent trauma, and major GI bleeding have to be weighed in a benefit risk ratio on an individual basis. In the setting of in-hospital massive PE and PEA, many patients are post-surgery with relative contra-indications to thrombolysis. A small case series (n=7) by Hopf et al. reported successful thrombolytic treatment in the post-operative state using rTPA; 6/7 patients survived neurologically intact, 6/7 required CPR ([18] Hopf 1991). This would suggest that thrombolysis can be administered during PE with cardiac arrest.

In considering any therapy, mortality rates need to be assessed in order to weigh benefit versus risk. Literature reviews were done on thrombolysis during PE and CPR, thrombolysis in PE and PEA, and thrombolysis during PEA. The published literature provides mixed results. Case reports and series show promise in giving thrombolytics during PE and PEA, but suffer from selection bias. Bettiger et al reviewed thrombolysis treatments of PE with CPR in 2001, with an aggregated survival rate of 36/63 patients or 57% ([15] Bettiger 2001). It is important to note that the survival rate in smaller series is generally higher, possibly secondary to the selection bias of authors publishing successful cases. In the smaller series within the meta analysis (n<3), only surviving patients were reported. In larger series (n>=5), the survival rate was about 60%, such as streptokinase bolus and infusion, survival 11/20 ([19] Kohle 1983), rTPA infusion, survival 5/9 ([20] Westhoff-Bleck 1991), and urokinase or streptokinase bolus, survival 3/5 ([21] Scholz 1990).

Larger reviews and prospective trials are much less promising. In a series of 30 patients with CPR for PEA due to PE, Kurkiyan et al found that only 2/15 patients with central PE and PEA survived with thrombolysis versus 0/15 patients not receiving thrombolysis ([22] Kurkiyan

2000). Of note, 70% of patients were diagnosed clinically via history, ABG, and ECG and only 30% were diagnosed on the basis of TTE and later confirmed by autopsy. Both patients who survived received CPR immediately, underwent a diagnosis by TTE or CT, and had TPA administered within 15 minutes of arrival. In one patient, thrombolysis served as a bridge to embolectomy. The patient survived neurologically intact to be discharged 21 days later. Ruiz-Bailen et al. comment that the high global mortality in Kurciyan et al's study could be explained in part by the fact that most of the patients suffered prolonged cardiac arrest and died from untreatable shock or severe cerebral damage ([16] Ruiz-Bailen 2001). However, Abu-Laban et al ([23] Abu-Laban 2004) found no difference in the timing of thrombolysis administration in (non PE related) PEA, and rate of return of spontaneous circulation (ROSC). The limitation of the study is, that a previous paper from the same group ([24] Abu-Laban 2002) indicated that survival was only a dismal 1/117 patients in the tPA group. In our case, thrombolysis was delayed approximately 40 minutes.

Previous studies have shown no survival difference in hemodynamically stable patients with PE receiving rtPA, TPA, urokinase, or streptokinase ([7] Arcasoy 1999) A large study by Konstantinides ([8]Konstantinides 2002) prospectively looked at alteplase 100 mg over 2 hours plus heparin vsus heparin and placebo in 256 hemodynamically stable patients with acute submassive pulmonary embolism. The primary end point was in-hospital death or clinical deterioration requiring an escalation of treatment, (catecholamine infusion, secondary thrombolysis, endotracheal intubation, cardiopulmonary resuscitation, or emergency surgical embolectomy or thrombus fragmentation by catheter). The incidence of the primary end point was significantly higher in the heparin-plus-placebo group than in the heparin-plus alteplase group ($P=0.006$). However, mortality was low and similar in both groups (3.4 percent in the heparin-plus-alteplase group and 2.2 percent in the heparin-plus-placebo group, $P= 0.71$). No fatal bleeding or cerebral bleeding occurred in patients receiving heparin plus alteplase.

We are aware of no studies comparing medications and regimens that have been performed in patients undergoing CPR.

In terms of the risk benefit ratio, the risk of hemorrhage is the main concern in patients receiving thrombolysis. In general, hemorrhage is a possibility, but death from hemorrhage is uncommon. In a retrospective cohort study of 132 patients who received TPA thrombolysis during prolonged CPR (not due to PE); 13/132 had hemorrhage (10%), with none dying of hemorrhagic complications ([25] Kurciyan 2003). In addition, a second study focusing on massive PE and CPR revealed a 25% hemorrhage rate vs. 10% without thrombolysis. The survival rate post TPA was much higher and the authors noted: "In thrombolized patients survival after 24 h was higher (19/36 (53%) vs. 7/30 (23%), $P=0.01$). ([26] Janata 2003). Although severe bleeding complications tend to occur more frequently in patients undergoing thrombolysis, the benefit of this treatment might outweigh the risk of bleeding." In the above case, the patient survived thrombolysis and then developed a spontaneous retroperitoneal hemorrhage which was managed with blood transfusions and supportive care. The benefit of treatment with thrombolysis certainly outweighed the risk of hemorrhage.

Conclusions

Massive PE is an important diagnostic consideration in patients presenting with cardiac arrest. Even without available clinical history, PE should be considered in patients who have PEA and no other clear underlying cause of arrest. The use of TTE can quickly confirm the diagnosis. Preferred therapy would be thrombolysis or surgical embolectomy, with thrombolysis being available in almost all centers and embolectomy limited to experienced centers. Clinical judgment and surgical experience can help guide the most effective therapeutic choice. While hemorrhage is a concern, it is seldom fatal. The literature suggests a positive risk/benefit ratio for thrombolytic therapy in the setting of massive PE requiring CPR. Both the use of TTE in the ACLS protocol and the preferred choice of thrombolytic agent in PE requiring CPR need to be further studied in larger trials. The above

case demonstrates that thrombolytic therapy for massive PE needing CPR confirmed by TTE can be successful. Given the multifactorial and emergent nature of the decision regarding the best therapeutic modality for near-fatal PE, it is probably best for each medical center to have a concrete protocol, clearly communicated to everyone potentially involved in the management of such cases.

Short take-home points for massive PE and PEA:

- 1) TTE helps make the diagnosis of PE during the code;
- 2) Attending physicians must make an individual assessment of experience in their center and produce suggested local protocols;
- 3) Thrombolysis is the preferred option, but debate remains;
- 4) Unsuccessful thrombolysis does not necessarily preclude subsequent surgical or catheter embolectomy;
- 5) Catheter embolectomy is experimental, but can be used in settings of surgical and thrombolysis contra-indications;
- 6) Hemorrhage occurs in 10% of patients given thrombolysis, but is seldom fatal;
- 7) Thrombolysis may be successfully administered in PE after prolonged resuscitation attempts.

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