

11-1-2011

Saddle pulmonary embolism: is it as bad as it looks? A community hospital experience.

Alejandro Sardi
Thomas Jefferson University

Jill Gluskin
Thomas Jefferson University

Adam Guttentag
Thomas Jefferson University

Morris N Kotler
Thomas Jefferson University

Leonard E Braitman
Thomas Jefferson University

See next page for additional authors

[Let us know how access to this document benefits you](#)

Follow this and additional works at: <http://jdc.jefferson.edu/pulmcritcarefp>

 Part of the [Medicine and Health Sciences Commons](#)

Recommended Citation

Sardi, Alejandro; Gluskin, Jill; Guttentag, Adam; Kotler, Morris N; Braitman, Leonard E; and Lippmann, Michael, "Saddle pulmonary embolism: is it as bad as it looks? A community hospital experience." (2011). *Faculty papers Division of Pulmonary and Critical Care Medicine*. Paper 3.
<http://jdc.jefferson.edu/pulmcritcarefp/3>

This Article is brought to you for free and open access by the Jefferson Digital Commons. The Jefferson Digital Commons is a service of Thomas Jefferson University's [Center for Teaching and Learning \(CTL\)](#). The Commons is a showcase for Jefferson books and journals, peer-reviewed scholarly publications, unique historical collections from the University archives, and teaching tools. The Jefferson Digital Commons allows researchers and interested readers anywhere in the world to learn about and keep up to date with Jefferson scholarship. This article has been accepted for inclusion in Faculty papers Division of Pulmonary and Critical Care Medicine by an authorized administrator of the Jefferson Digital Commons. For more information, please contact: JeffersonDigitalCommons@jefferson.edu.

Authors

Alejandro Sardi, Jill Gluskin, Adam Guttentag, Morris N Kotler, Leonard E Braitman, and Michael Lippmann

As submitted to:

Critical Care Medicine

And later accepted as:

SADDLE PULMONARY EMBOLISM (SPE): Is it as bad as it
looks?
A Community Hospital Experience

November 2011 - Volume 39 - Issue 11 - pp 2413-2418

doi: 10.1097/CCM.0b013e31822571b2

Alejandro Sardi, MD¹; Jill Gluskin, MD²; Adam Guttentag, MD²; Morris N Kotler, MD³;
Leonard E. Braitman, Ph.D⁴; Michael Lippmann, MD¹

¹Division of Pulmonary and Critical Care Medicine. ²Division of Radiology. ³Division of
Cardiology. ⁴Office for Research and Technology Development.

Albert Einstein Medical Center / Thomas Jefferson University School of Medicine.
Philadelphia, Pennsylvania. USA

Saddle Pulmonary Embolism (SPE) : Is it as bad as it looks? A Community Hospital Experience

Alejandro Sardi, MD¹; Jill Gluskin, MD²; Adam Guttentag, MD²; Morris N Kotler, MD³; Leonard E. Braitman, Ph.D⁴; Michael Lippmann, MD¹

¹Division of Pulmonary and Critical Care Medicine. ²Division of Radiology. ³Division of Cardiology. ⁴Office for Research and Technology Development. Albert Einstein Medical Center. Thomas Jefferson University School of Medicine. Philadelphia, Pennsylvania. USA

Background: SPE represents a large clot and a risk for sudden hemodynamic collapse. However, the clinical presentation and outcomes vary widely. Based on the findings of right heart dysfunction on echocardiograms, CT angiography (CTA), or cardiac enzyme elevation, some argue for the use of thrombolytics or catheter thrombectomy even for hemodynamically stable patients,.

Objective: Investigate the outcomes and management of patients with SPE including radiographic appearance (estimate of clot burden) and echocardiographic features.

Methods: Retrospective evaluation of all patients with CTA positive for pulmonary embolism (PE) from June 1, 2004, to February 28, 2009. Two radiologists selected those with SPE and evaluated clot burden score. The clinical information, echocardiography, treatments and outcomes of these patients were extracted via chart review.

Results: SPE was found in 37 of 680 patients (5.4%, 95% CI, 4% to 7%) with documented PE on CTA. For patients with SPE, median age was 60 years and 41% were males. Major co-morbidities were neurologic 24%, recent surgery 24%, and malignancy 22%. Transient hypotension occurred in 14% and persistent shock in 8%. One patient required mechanical ventilation. Echocardiography was performed in 27 patients (73%).

RV enlargement and dysfunction were found in 78% and elevated PASP in 67%. CTA demonstrated a high median pulmonary artery clot burden score of 31 points. Median RV/LV diameter was 1.39. IVC filters were placed in 46%. Unfractionated heparin was administered in 33 (87%) and thrombolytics in 4 (11%). Median hospital LOS was 9 days. Two of 37 SPE patients (5.4%) died in the hospital (95%CI, 0.7% to 18%).

Conclusions: Most patients with SPE found on CTA responded to the standard management of PE. Clot burden, RV/LV ratio on CTA and echocardiographic abnormalities were not found to be predictors of adverse outcome in patients with SPE.

BACKGROUND:

Pulmonary embolism (PE) is an important public health problem, accounting for many in-hospital deaths, associated morbidity and immense hospital costs (1). The 3-month all-cause mortality rate for PE varies between 8.5% (2) and 18% (3). The attributable mortality for PE is between 1.68% (2) to 7.9% (3).

Saddle pulmonary embolism (SPE) is defined as the presence of a thromboembolus located at the bifurcation of the main pulmonary artery. Radiological evidence of SPE is found in approximately 2% to 5% (4) (5) of all PE patients. However, the true incidence is difficult to determine since many patients are too unstable to receive a CT angiogram (CTA) and the diagnosis is often established at autopsy. Some have proposed that SPE represents a transient form of acute PE, thus their true frequency may therefore be underestimated (6). Saddle PE represents a large clot burden and is an important risk for right ventricular dysfunction and sudden hemodynamic collapse. Nonetheless the clinical presentation and outcomes for these patients vary widely (7). This is likely due to the variable compensatory physiologic responses and pre-existing cardio-respiratory disease (8). Some investigators argue for more aggressive therapeutic interventions (i.e. thrombolytics or catheter thrombectomy) in those patients with large clot burden even if hemodynamically stable. Such interventions are based on the findings of right heart dysfunction on echocardiograms (9), CTA (10), or cardiac enzymes elevation (11). These interventions may place this group of patients at an increased risk for medication and procedure related complications, lengthen the hospital stay and increase hospitalization costs. The therapeutic value of these interventions in the setting of the normotensive patient with pulmonary embolism is still a matter of ongoing debate.

Few studies have documented the clinical presentation and outcomes of SPE on a CTA. The purpose of this study was to retrospectively review our experience with patients diagnosed with SPE determined by CTA. We investigated the relationship between the radiologic findings and the clinical presentation, co-morbidities, management strategies and clinical outcomes. Our major goals were to determine the in-hospital mortality, the rates of adverse outcomes (i.e. major bleeding, minor bleeding) and in-hospital length of stay and to correlate them with the clot burden. The study was performed at the Albert Einstein Medical Center, a large tertiary teaching institution in Philadelphia.

MATERIALS AND METHODS:

Institutional Review Board approval was obtained with waiver of informed consent, for this retrospective study. Patients with chest CT angiograms coded positive for PE (ICD-9-CM codes 415.11 - 415.19) from June 1st, 2004, to February 28th, 2009 were identified using computer-assisted search of medical records. Patients younger than 18 years of age and those without a chest CT angiogram were excluded.

Two radiologists reviewed all the CT scans and selected only the patients with a radiological finding of saddle pulmonary embolism, defined by a low-attenuation filling defect extending across the bifurcation of the pulmonary artery trunk. Both radiologists evaluated the CT scans for the presence or absence of inferior vena cava contrast reflux,

ventricular septal bowing, measured the ratio between the diameter of the right ventricle to left ventricle (RV/LV), the ratio between the diameter of the main pulmonary artery to the Aorta (PA/Ao), Azygos vein diameter, superior vena cava diameter and scored the embolic burden and pulmonary artery occlusion. Clot burden (CB) and pulmonary artery occlusion index were determined using the Qanadli (12) scoring system. The maximal CB score was 40 (complete proximal obstruction of all distal 20 segmental arteries) corresponding to 100% occlusion. (Figure 1) Both radiologists were blinded to the clinical history. We used 3 multidetector CT scanners: GE Lightspeed 16-slice scanner, GE Lightspeed VCT 64-slice scanner and a Toshiba Aquilion 16-slice scanner.

We extracted the clinical information, including demographics, clinical presentation, echocardiographic and radiographic findings, as well as their treatments and outcomes. All patient identifiers were removed from the database. Echocardiographic data was extracted from the cardiology reports and videotapes and were reviewed by one cardiologist. Only 2D-Echocardiograms done within the first 72 hrs of presentation were included (Phillips 5500 ®, Phillips 7500 ®, GE vivid-I ®, GE vivid-7 ®, and Siemens Sequoia ®). Multiple echocardiographic views are necessary to evaluate right ventricular size and function because the right ventricle is a complex crescent-shaped structure wrapped around the left ventricle and is incompletely viewed in any single two dimensional echocardiographic view (13)

The parasternal long and short axis views, right ventricular inflow, apical four-chamber and subcostal views were evaluated in all patients.

Right ventricular size was evaluated in all views but was best determined from the apical four-chambered view. In this view, right ventricular mid cavitory diameter is smaller than that of the left ventricle. Mild right ventricular enlargement was defined as mid right ventricular dimension as equal to that of the left ventricle diameter at that site. Moderate right ventricular enlargement was defined as mid right ventricular dimension slightly larger than the left ventricle but right ventricle shares the apex of the heart. Severe right ventricular enlargement was defined as mid right ventricular dimension clearly exceeding the left ventricular dimension and the right ventricle forming the apex of the heart (14).

Right ventricular systolic function was subjectively evaluated in all views as is customarily in clinical practice. Tricuspid annular plane systolic excursion (an index of right ventricular systolic function) was evaluated in the apical four-chambered view. Tricuspid annular plane systolic excursion greater than 1.6cm was considered normal. Mild right ventricular systolic dysfunction was defined as tricuspid annular plane excursion less than 1.6cm but greater than 1.0cm.

Moderate right ventricular dysfunction was defined as tricuspid annular plane excursions less than 1.0cm but greater than 0.5cm. Severe right ventricular dysfunction was defined as tricuspid annular plane excursion less than 0.5cm (15).

RV systolic pressure was calculated by adding the estimated RA pressure (determined by the inferior vena-cava diameter and its response to inspiratory decline with inspiration) to the tricuspid regurgitation velocity²x 4 (16). The presence of the Mc Connell sign (akinesia of the mid-free wall but normal motion of the apex) which is 77% sensitive and 94% specific for PE was determined. (17).

The highest serum Troponin I (TNI) in the initial 72 hrs was recorded. Electrocardiographic changes documented within the first 24 hrs of presentation were analyzed.

Most authors define shock as a SBP <90mmHg (17) while others have used a threshold < 100mmHg (18). We elected to define it as a sustained or persistent systolic blood pressure \leq 90 mmHg in more than one blood pressure measurement at presentation, and after \geq 500 ml of intravenous crystalloid solutions, requirement of cardio-pulmonary resuscitation (CPR) or the use of vasoactive pressors. Those patients who responded to a fluid bolus were deemed to have transient hypotension. We defined major bleeding as a decrease in hemoglobin levels of 2 g/dL or more, blood product transfusion requirement, intracerebral bleeding, retroperitoneal bleeding, pericardial bleeding, bleeding that required a surgical intervention, bleeding into a major joint, or bleeding into the eye, during the 48 hours following thrombolytic administration. Any other bleeding that that did not fulfill these criteria was considered a minor bleed.(19)

Measured variables were described using the median and interquartile range (IQR). Bivariate median regression was used to compare medians. Percentages were compared using Fisher's exact test. Ninety-five percent confidence interval (95%CI) estimates were presented for percentages. All statistical analyses were performed using Stata 11 (College Station, TX).

RESULTS:

Six hundred and eighty (680) patients were found to have a diagnosis of pulmonary embolism established by a CT angiogram. Saddle pulmonary embolism (SPE) was found in 37 of them (5.4%, 95% CI 4% to 7%).

The demographic characteristics and co-morbidities of the 37 SPE patients are summarized in Table 1. The median age was 60 years (IQR 21). Males comprised 40.5% of the SPE patients and 84% were African-American. Thirty of 37 (81%) presented to the emergency department and only 7 (19%) developed SPE during a hospitalization.

The most frequent co-morbidities were: neurologic deficit (history of a CVA) 9 (24%), surgical intervention within 3 months 9 (24%), and malignancy 8 (22%). There was a low prevalence of prior cardiopulmonary disease 4 (11%) and obesity (BMI>30) 4 (11%). (Table 1)

Dyspnea was the most common symptom (92%), followed by leg pain/edema (43%), chest pain (30%) and syncope (11%). One outpatient (3%) required mechanical ventilation. Persistent shock was seen in 3 patients, (8%, 95% CI 2% to 22%). Transient hypotension was observed in 6 patients (16%, 95%CI, 6% to 32%) but was rapidly corrected with fluid resuscitation. Upon onset of symptoms, the median values of the vital signs were systolic blood pressure of 112 mmHg, diastolic 71mmHg, heart rate of 109 beats per minute, respirations 22 breaths per minute and pulse oximetry saturation of 94% on room air

The median PaO₂ /FiO₂ ratio was 243 mmHg (IQR 168) in the 19 patients in whom these data were available. The median BNP was 251 (IQR 334). Median of the highest Troponin I (TNI) level was 0.08 ng/ml (IQR 0.17). Median D-dimer was 6772.5 (IQR 4039.5). Twenty-eight patients had either CT venography or Doppler ultrasonography (US) of the extremities, identifying 20 patients (71%) with positive deep vein thrombosis (DVT) (Table 2). Electrocardiographic (EKG) abnormalities included: sinus tachycardia (56%), right bundle branch block (RBBB) (22%), S₁Q₃T₃ pattern (25%), new ST changes and non-specific T wave inversions (64%). A normal EKG was observed in 7 patients (19%).

Transthoracic echocardiography (TTE) was performed in 27 patients (73%). RV enlargement was found in 21 patients (78%) and it was categorized as mild in 6 (22%), moderate in 8 (30%), and severe in 7 (26%) (Table 2). RV dysfunction was seen in 21 patients (78%). It was mild in 7 (26%), moderate in 8 (30%) and severe in 6 (22%). Elevated PASP was seen in 18 (67%) patients. Inter-ventricular (IV) septum flattening and/or left IV septum deviation was found in 7 patients (26%). Inferior vena cava (IVC) failure to collapse on inspiration was observed in 6 patients (22%). Six patients had normal right ventricular findings on TTE. A Mc Connell sign was found in 4 (15%) patients. A thrombus in “migration” was found in 3 patients (11%).

CTA demonstrated a very high median pulmonary artery clot burden score of 31 points (IQR 6) of a maximum of 40 points (mean occlusion of 79%). The median pulmonary artery/aorta diameter ratio was 1.0 (IQR 0.21). The median RV/LV diameter ratio was 1.39 (IQR 0.91) (normal value <0.7). The median SVC diameter was 23mm (IQR 5). The median azygos vein diameter was 11mm (IQR 2). IVS bowing was observed in 26 patients (70%) and contrast reflux in the IVC in 28 patients (76%) (Table 2).

Fifteen of 37 (41%) patients were treated in the intensive care unit, 17 (45%) in an intermediate care unit and telemetry and 5 (14%) on the general medical floor (Table 3). Seventeen patients (46%) received IVC filters; the indications included presence of residual clot burden in the lower extremities (DVT) in 5 patients, contraindications to anticoagulation in 8 patients and a physician’s preference in 4 patients. Of the 15 patients admitted to an ICU with SPE, 11(73.3%) received IVC filters compared to 6 of 22 (27.3%) of patients admitted elsewhere in the hospital (p=0.008) by Fisher’s exact test. In other words, those admitted to an ICU were 2.7 times as likely to receive an IVC filter. The median LOS for patients who received IVC filters (11 days) was longer than for those not receiving IVC filters (5.5 days) (p=0.006). Although the median length of stay for patients monitored in the ICU was 11 (IQR 5) and was 7 (IQR 8) for patients monitored elsewhere in the hospital, that difference did not reach statistical significance (p=0.19).

Most patients (87%) received unfractionated heparin (UFH). Only 4 patients (11%) received intravenous thrombolytics (Alteplase, r-TPA) (Table 3). Two of these patients received r-TPA due to concomitant shock. One survived hospitalization (LOS 11 days) without bleeding or other complications. The other patient’s course was complicated by

PEA arrest, ventilator dependant respiratory failure, gastrointestinal bleed (GIB) and sepsis, and died with anoxic brain injury after 13 days in the hospital. One patient who was normotensive received r-TPA for a right ventricular thrombus seen on TTE. This patient had severe RV dilatation and dysfunction and with a CTA showing a CB of 37 (93% obstruction) with a RV/LV diameter ratio 1.89. His course was complicated by a large arm hematoma, requiring blood transfusion and 11 days of hospitalization. A second normotensive patient received r-TPA after presenting with history of syncope, and severe abnormalities on CTA (CB of 33 or 83% obstruction, RV/LV diameter ratio 2.65) and TTE (moderate RV dilatation and dysfunction). His course was complicated by a minor bleed and he was discharged after 11 days.

In those patients that did and did not receive thrombolytics, the median clot burden scores were 34 and 31, respectively. The median RV/LV ratio of those patients that did and did not receive thrombolytics was 1.86 and 1.34, respectively (p=0.13).

One patient with atrial dysrhythmia and recent right atrial radiofrequency ablation, who developed shock, had a large tubular right atrial thrombus extending to the right ventricle. He did not receive thrombolytics but was sent to a referral center for surgical thrombectomy.

In patients who received r-TPA, a major bleed was observed in 2 patients (50%) and a minor bleed in one patient (25%). One of 32 patients (3%) who received unfractionated heparin (UFH) infusion developed a major bleed (GIB).

The median length of stay in the hospital was 9 days (IQR= 7). Only two of 37 patients with SPE (5.4%, 95% CI 1% to 18%) died in the hospital. One patient died after 13 days in the hospital with multiorgan failure, major bleeding and sepsis. A second patient died in the emergency room with a bradycardic cardiopulmonary arrest due to an acute SPE. That patient had terminal lung cancer, was bedridden due to spinal and brain metastasis and TPA was contraindicated. None of our patients received an autopsy.

The 2 patients who died had clot burdens of 30 and 31, respectively. Although almost half of the surviving patients had a higher clot burden than the two who died, the small number of deaths made it impossible to determine which characteristics were associated with mortality.

DISCUSSION

The cumulative incidence of SPE (5.4%) was similar to prior studies. by Pruszczyk, et al. (5.2%) (4) and Ryu, et al. (2.6%) (5). However, this incidence of SPE may be an underestimate, since not all patients with SPE survive the initial event and many more delay medical care. In our study, only 2 of 37 patients (5.4%) died in the hospital.

This investigation contains one of the largest and best-documented group of patients with saddle pulmonary embolisms described in the literature. It incorporates a radiographic score of clot burden, echocardiographic features and outcomes. Although ominous in

appearance, a SPE may be a more common feature of many embolic events than previously thought when CTA's are performed soon after symptoms begin.

Clinical and hemodynamic characteristics appear to be the most important predictors of adverse outcomes (2). Systolic hypotension (<90mmHg) increased the risk of death substantially (Odds Ratio=18.6, 95% CI, 11.2 to 31.1) (2). In the decision to utilize thrombolytic therapy, there is no consensus on any of the following: an absolute number for the SBP, mean pressure, duration and response to fluid resuscitation in the definition of "systolic hypotension".

Six (16%) of the 37 patients had persistent shock in the emergency room that corrected prior to the diagnosis and treatment of SPE. These patients did not receive thrombolytics. Persistent shock was seen in only 3 patients (8%, 95% CI 2% to 22%), two of whom received thrombolytics. Similar to our study, Ryu et al. (5) found an incidence of hypotension of 14% (2 patients), but only one patient received thrombolytics.

Despite the high frequency of an abnormal finding on the EKG (81%) and on the echocardiogram (74%), the in-hospital mortality of SPE was low. RV enlargement and dysfunction were found very frequently (78%), as well as an elevated mean PASP (67%). Ryu et al. (5) also found a high incidence of echocardiographic abnormalities in patients with SPE but had no in-hospital deaths. Pruszczyk et al. (4) found no difference in the initial echocardiographic characteristics between patients with saddle and non-saddle PE. These findings are not surprising as Miller (21) demonstrated discordance between the degree of perfusion abnormalities on V/Q scans and RV enlargement or dysfunction. Similarly in hemodynamically stable patients with PE, Wolde demonstrated a very poor positive predictive value (4% to 5%) of echocardiographic RV dysfunction predicting PE related mortality (22). We feel that this also applies to clot burden score on CTA, hemodynamics and cardiac function.

One concern is that RV dysfunction and dilatation are relatively subjective determinations. Also, some studies that have concluded that the echocardiogram was useful in such patients may have had a selection bias (i.e. echocardiograms ordered mainly in sicker patients).

Only two in-hospital deaths occurred from SPE (5.4%) in our study, despite the presence of a high pulmonary artery clot burden (median 31 and 80% obstruction) and other ominous CTA signs such as RV/LV diameter ratio (mean 1.47), IVS bowing (70%), IVC reflux (76%). Neither of these two patients had underlying cardiopulmonary disease. Araoz et al, found that IVS bowing in CTA was associated with a low sensitivity and high interobserver variability for predicting mortality in 30 days, and that neither RV/LV diameter ratio nor embolic burden was associated with death due to PE (23). This differs from two small CT studies (24,25) where clot burden was associated with death due to PE over a three month period. The differing conclusions of these studies may have been a consequence of differing demographics or underlying cardiopulmonary co-morbidities of those study groups.

An interesting observation was the more frequent use of IVC filters in those patients admitted to the ICU. This could perhaps be explained by the number of patients who had residual clot burden in the lower extremity (found in nine of the 14 patients who received IVC filters). A contraindication to anticoagulation or physician preference also may have influenced the decision to insert a filter. The threshold to insert an IVC filter has been reduced since the introduction of retrievable filters. This may partially explain the increase IVC use in patients with high clot burden (26).

Pruszczyk, et al (4) and Ryu, et al (5) reported infrequent use of thrombolytics (29% and 7% respectively). In our study, 4 patients (11%) received thrombolytics. The rate of major bleeding due to thrombolytics in our study was high compared to other studies (27). Two of the four patients who received thrombolytics sustained major bleeding and one had a minor bleed, prolonging their hospital stay. One major bleed occurred with UFH requiring its discontinuation and hence IVC filter placement. Even though there was a trend towards administration of thrombolytics with a higher CB score and RV/LV ratio, this did not reach statistical difference. A larger study is needed to investigate these associations.

The mean LOS found in our SPE study (mean=10, median=9) is slightly higher than the most recently reported LOS for all PE in the USA (8.6 days) (1). This likely reflects the safety concerns that many physicians have when discharging patients with SPE.

Only two (5.4%) patients with saddle pulmonary embolism died in the hospital. Pruszczyk et al (4) also observed low mortality also observed (1 of 17, 5.8%). In Ryu et al. (5) none of the 14 patients died. The low prevalence of cardiopulmonary comorbidities in our patients as well as in these two studies may explain their relatively good outcomes. Patients with underlying cardiopulmonary disease (CPD) would have excess risk of an adverse outcome with the amount clot burden produced by a SPE. (17,28). Wood (8) however, reported no consistent relationship between the degree of cardiovascular and RV impairment and the magnitude of angiographic obstruction in those with underlying CPD..

In one of our patients, death was directly attributed to an SPE event. The second death was related to complications of a prolonged hospitalization, bleeding from thrombolytics and multiple co-morbidities appearing after a massive PE. In contrast, the only death from SPE in Pruszczyk et al. (4) was attributed to a recurrent PE. While Ryu, et al (5) reported that all the patients with SPE who died at three months after discharge died due to progression of an underlying malignancy.

CONCLUSIONS:

In this study we found that clot burden, the degree of pulmonary artery obstruction, RV/LV ratio on CTA and the echocardiographic abnormalities found in patients with

SPE were not statistically correlated with an adverse outcome. Perhaps a larger sample size or a prospective study would be necessary to demonstrate a correlation.

Thrombolytics are rarely needed, as most patients with SPE on CTA are hemodynamically stable with hypotension being frequently transitory and shock infrequent. Sustained hypotension and the need for mechanical ventilation remain the major criteria for the use of thrombolytics in the management of SPE.

Our study is one of the largest series of patients with SPE reported in the literature to date, but suffers from being retrospective and observational. Although the low mortality in SPE is encouraging, the small number of deaths made it impossible to determine which characteristics were associated with mortality. Nonetheless, in patients with SPE found on CT angiography, the majority appear to respond to the standard management with UFH regardless of the ominous radiological appearance.

Table 1. Demographics and Co-morbidities

	Median (IQR)
Age (years)	60 (21)
Gender	Number (Percent)
Female	22 (59.5)
Race	
African American	31 (83.7)
Asian	1 (2.7)
White	5 (13.5)
In/Out patient	
Outpatient	30 (81)
Inpatient	7 (19)
Co-morbidities	
Malignancy	8 (22)
CHF	3 (8)
COPD	1 (3)
Obesity	4 (11)
OAS/OHS	2 (5)
Rheum	6 (16)
Infections	5 (14)
Neurologic	9 (24)
Prior PE/DVT	6 (16)
Thrombophilia	2 (5)
Recent Surgery	9 (24)
Recent Trauma	4 (11)
Smoking	9 (24)
Hormonal contraceptives	4 (11)

Deleted: .

Table 2. Imaging, Echocardiographic and CT findings in SPE Patients.

Imaging		Number (Percent)
Positive DVT * (n=28)		20 (71)
2-D Echocardiogram (n=27)		
RV enlargement	Mild	6 (22)
RV enlargement	Moderate	8 (30)
RV enlargement	Severe	7 (26)
RV dysfunction	Mild	7 (26)
RV dysfunction	Moderate	8 (30)
RV dysfunction	Severe	6 (22)
Elevated PASP (≥ 35 mmHg)		18 (67)
IV septum flattening/deviation		7 (26)
IVC failure to collapse w/insp		6 (22)
McConnell sign		4 (15)
Thrombus in migration		3 (11)
Normal		7 (26)
CT angiogram		Median (IQR)
Clot Burden (Qanadli)		31 (6)
Pulm. Artery/Aorta diameter ratio		1 (0.2)
RV/LV diameter ratio		1.4 (0.9)
Superior Vena Cava (SVC) diameter (mm)		23 (5)
Azygos Vein diameter (mm)		11 (2)
		Number (Percent)
Inter-ventricular septum (IVS) bowing		26 (70)
Inferior Vena Cava (IVC) contrast reflux		28 (76)

* In Doppler US and CT venogram.

Table 3. Management and Outcomes.

Location	Number (Percent)
Medical ICU	9 (24)
Surgical ICU	1 (3)
Cardiac ICU	5 (14)
Intermediate Care	17 (46)
General Medical Floor	5 (14)
Treatment	
Un-fractionated Heparin (UFH)	32 (87)
Low Molecular Weight Heparin (LMWH)	4 (11)
IVC filter	17 (46)
Thrombolytics (Alteplase)	4 (11)
Surgical Thrombectomy	1 (3)
Outcomes	
Minor Bleeding	1 (3)
Major Bleeding	3 (8)
In Hospital Mortality	2 (5)
In Hospital Length of Stay (days)	9 (7)*

* Median, IQR

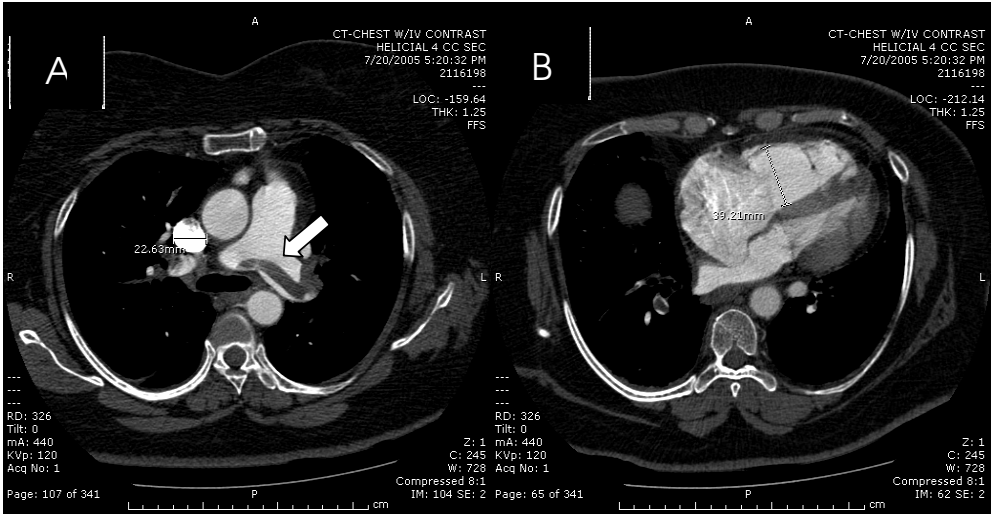


Figure 1. Chest CT angiogram: A) Saddle pulmonary thrombus. Clot Burden score: 28 of 40 points. Occlusion index: 70% (12). B) increased RV/LV diameter ratio: 2.52

References

1. Park B, Messina L, Dargon P, Huang W, Ciocca R, Anderson FA. Recent trends in clinical outcomes and resource utilization for pulmonary embolism in the united states: Findings from the nationwide inpatient sample. *Chest*. 2009 Oct;136(4):983-90.
2. Laporte S, Mismetti P, Decousus H, Uresandi F, Otero R, Lobo JL, et al. Clinical predictors for fatal pulmonary embolism in 15,520 patients with venous thromboembolism: Findings from the registro informatizado de la enfermedad TromboEmbolica venosa (RIETE) registry. *Circulation*. 2008 Apr 1;117(13):1711-6.
3. Goldhaber SZ, Visani L, De Rosa M. Acute pulmonary embolism: Clinical outcomes in the international cooperative pulmonary embolism registry (ICOPER). *Lancet*. 1999 Apr 24;353(9162):1386-9.
4. Pruszczyk P, Pacho R, Ciurzynski M, Kurzyna M, Burakowska B, Tomkowski W, et al. Short term clinical outcome of acute saddle pulmonary embolism. *Heart*. 2003 Mar;89(3):335-6.
5. Ryu JH, Pellikka PA, Froehling DA, Peters SG, Aughenbaugh GL. Saddle pulmonary embolism diagnosed by CT angiography: Frequency, clinical features and outcome. *Respir Med*. 2007 Jul;101(7):1537-42.
6. Enzweiler CN, Wiese TH, Lembcke AE, Taupitz M, Rogalla P, Kivelitz DE, et al. Electron beam tomography of interpulmonary saddle embolism: Extent and vascular distribution. *J Comput Assist Tomogr*. 2002 Jan-Feb;26(1):26-32.
7. Parmley LF, Jr, Senior RM, McKenna DH, Johnston GS. Clinically deceptive massive pulmonary embolism. *Chest*. 1970 Jul;58(1):15-23.
8. Wood KE. Major pulmonary embolism: Review of a pathophysiologic approach to the golden hour of hemodynamically significant pulmonary embolism. *Chest*. 2002 Mar;121(3):877-905.
9. Kucher N, Rossi E, De Rosa M, Goldhaber SZ. Prognostic role of echocardiography among patients with acute pulmonary embolism and a systolic arterial pressure of 90 mm hg or higher. *Arch Intern Med*. 2005 Aug 8-22;165(15):1777-81.
10. Gibson NS, Sohne M, Buller HR. Prognostic value of echocardiography and spiral computed tomography in patients with pulmonary embolism. *Curr Opin Pulm Med*. 2005 Sep;11(5):380-4.
11. Becattini C, Vedovati MC, Agnelli G. Prognostic value of troponins in acute pulmonary embolism: A meta-analysis. *Circulation*. 2007 Jul 24;116(4):427-33.

12. Qanadli SD, El Hajjam M, Vieillard-Baron A, Joseph T, Mesurolle B, Oliva VL, et al. New CT index to quantify arterial obstruction in pulmonary embolism: Comparison with angiographic index and echocardiography. *AJR Am J Roentgenol.* 2001 Jun;176(6):1415-20.
13. Lang RM, Bierig M, Devereux R, Flachskamp FA, et al Recommendation's for Chamber Qualification: A Report from the American Society of Echocardiography's Guidelines and Standards Committee and the Chamber Qualification Writing Group. *Journal of American Society of Echocardiography* 2005;18:1440-1463.
14. Samad BA, Alam M, Jensen-Urstad K Prognostic Impact of Right Ventricular Involvement as Assessed by Tricuspid Annular Motion in Patients with Acute Myocardial Infarction. *American Journal of Cardiology* 2002; 19:145-54.
15. Rudski LG, Wyman WL, Afilalo J, et al Guidelines for the Echocardiographic Assessment of the Right Heart in Adults: A Report from the American Society of Echocardiography. *Journal of American Society of Echocardiography* 2010; 23:685-713.
16. Currie PJ, Seward JB, Chan KL Continuous Wave Doppler Determination of Right Ventricular Pressure: A Simultaneous Doppler Catheterization Study in 127 Patients. *Journal of American College of Cardiology* 1985; 6:750-6.
17. McConnell MV, Solomon SD, Rayan ME, Come PC, Goldhaber SZ, Lee RT. Regional right ventricular dysfunction detected by echocardiography in acute pulmonary embolism. *Am J Cardiol.* 1996 Aug 15;78(4):469-73
18. Wood K. Major Pulmonary Embolism-Review of a Pathophysiologic Approach to the Golden Hour of Hemodynamically significant Pulmonary Embolism *Chest* 2002;121:877-905.
19. Aujesky, D, Roy PM, LeMANach CP et al Validation of a Model to Predict Adverse Outcomes in Patients with Pulmonary Embolism. *European Heart Journal* 2006;17:476-81.
20. Wells P, Forgie M, Simms M. The outpatient Bleeding Risk. *Archives Int. Med* 2003;163:917-920
21. Miller R, Das S et al Association Between Right Ventricular Function and Perfusion Abnormalities in Hemodynamically Stable Patients with Acute Pulmonary Embolism. *Chest* 1998; 113:665-70
22. ten Wolde M, Sohne M, Quak E, Mac Gillavry MR, Buller HR. Prognostic value of echocardiographically assessed right ventricular dysfunction in patients with pulmonary embolism. *Arch Intern Med.* 2004 Aug 9-23;164(15):1685-9.

23. Araoz PA, Gotway MB, Harrington JR, Harmsen WS, Mandrekar JN. Pulmonary embolism: Prognostic CT Findings¹. *Radiology*. 2007 March;242(3):889-97.
24. van der Meer RW, Pattynama PMT, van Strijen MJL, van den Berg-Huijsmans AA, Hartmann IJC, Putter H, et al. Right ventricular dysfunction and pulmonary obstruction index at helical CT: Prediction of clinical outcome during 3-month follow-up in patients with acute pulmonary Embolism¹. *Radiology*. 2005 June;235(3):798-803.
25. Wu AS, Pezzullo JA, Cronan JJ, Hou DD, Mayo-Smith WW. CT pulmonary angiography: Quantification of pulmonary embolus as a predictor of patient outcome--initial experience. *Radiology*. 2004 Mar;230(3):831-5.
26. Mismetti p, Rivron-Guillot K, Quenet P A prospective Long-term study of 220 pts. with a Retrievable vena cava Filter for Secondary prevention of Venous Thromboembolism *Chest* 2007 131: 223-9)
27. Stein PD, Hull RD, Raskob G. Risks for major bleeding from thrombolytic therapy in patients with acute pulmonary embolism. consideration of noninvasive management. *Ann Intern Med*. 1994 Sep 1;121(5):313-7.
28. Morpurgo M, Schmid C. The Spectrum of Pulmonary Embolism. *Chest* 1995; 107:18S-20S

