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CASE REPORT

A case of Massive Pulmonary Embolism with Cardiac Arrest Treated with life-saving Emergency Thrombolysis despite Recent Major Spinal Surgery: Dealing Acute Right Ventricular Failure through Passive Leg Raising Test

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ABSTRACT

Background: High-risk (massive) pulmonary embolism is defined by the presence of cardiogenic shock, persistent arterial hypotension, or both.[1] Acute Pulmonary Embolism (PE) is a life threatening, challenging clinical situation, specially if the PE is a massive one. The incidence of PE among the general population is approximately 1/1000 per year.[2] It accounts for 2% to 15% of sudden death.[3,4] When pulmonary artery obstruction is total, external cardiac massage (ECM) is not enough to resuscitate the patient, rather desperate, aggressive measures viz. systemic thrombolysis, emergency thoracotomy, femoro-femoral cardio-pulmonary by-pass, embolectomy may be the only options remaining.[5]

Here, we report a case of massive pulmonary embolism with cardiac arrest. This patient had a major spinal surgery, only 12 days back, when he had massive bleeding.

Key words: Acute RVF, Massive PE, PLR.

Case Report

A 68 years old gentleman weighing 108 kg, presented to Accident and Emergency (A & E) Department & Medical ICU of King Khaled University Hospital, Riyadh, Saudi Arabia with sudden onset of shortness of breath, retrosternal pain and sweating. The patient had a history of major spinal surgery (laminectmy and rod fixation) only 12 days back, when he had massive per-operative bleeding. The patient is also known to have diabetes mellitus, hypertension and had bilateral knee replacement 10 years back. Following the spinal surgery, the patient had significantly restricted movement and was not on any deep-vein-thrombosis (DVT) prophylaxis. On arrival in the Accident and Emergency Department, the patient was tachypnoec, tachycardic, dyspnoeac, sweating and his systolic blood pressure was 100 mmHg which is low for a hypertensive patient. ECG ruled out acute myocardial ischemia. An urgent spiral CT angiogram of chest was done, which showed massive Pulmonary embolism in both the main right and left pulmonary artery branches (Figure 1, 2). Because of the recent spinal surgery, it was a dilemma whether to thrombolyse. Heparin 5000 units iv bolus was administered. While urgent discussion was going on with hematologist, the patient had cardiac arrest. Immediately he was intubated and resuscitated.

After getting 'go-ahead' approval from neuro-surgical team, rtPA(recombinant tissue Plasminogen Activator, Alteplase) 100 mg iv bolus was administered during CPR (cardio-pulmonary resusci-tation) as an immediate rescue measure. Dopamine and Noradrenaline iv infusion started as the patient was in shock after CPR. The patient had another cardiac arrest soon and again resuscitated. Urgent Trans-thoracic Echocardiogram (TTE) was done, which showed hugely dilated, thinned out right ventricle (RV) with severely compromised systolic function but the left ventricular (LV) function was maintained. The patient was shifted to Medical ICU (MICU).

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Contoured Continuous Cardiac Output (PCCO) monitoring through Flo-trac Vigileo system (Edwards Life Sciences, USA) time to time, when the systolic blood pressure (SBP) and Cardiac Index (CI) were dropping, we were performing the Passive Leg Raising (PLR) test to predict the requirement for fluid challenge. We were cautious not to over-stretch the right ventricle (RV) which was already severely stretched. After 2 hours, while turning the patient, nurses noted significant amount of blood under the back. After turning the patient, it was noted that blood was flowing out from the lower part of the recent lumbar incision site. While the bleeding site was compressed, it started to bleed from the upper part of the lumbar incision site. Neurosurgeons were called in and they applied multiple deep bite stitches which failed to arrest the bleeding and finally, they inserted a drain blindly through the incision site. The drain failed to serve the purpose and bleeding continued. Over the next 12 hours, 5 units of packed RBCs were transfused. Simultaneously, we transfused FFP (fresh frozen plasma),cryoprecipitate, ACA (Amino-caproic Acid) and after consultation with hematologist, Factor VIIa (recombinant) was administered.

After about 12 hours, the pace of bleeding was controlled. But the fear was that he might have bled inside the spinal canal, which may affect the lower limb function. As the patient had GCS (Glasgow Coma Score) 3/15, and was hemodynamically very unstable with pupils 8.0 mm - not reacting to light, and already had cardiac arrest 3 times, it was very risky to transport the patient to the CT scan room for spinal CT scan to rule out intra-spinal bleeding. So the plan for spinal CT scan was postponed till the patient stabilised enough for transport. As DVT prophylaxis, the patient was put on sequential compression device after ruling out DVT. Regular CBC (complete blood count) was done and packed RBC was transfused regularly to keep Hb around 10 gm/dl. Meanwhile, kidney function also rapidly went down. The patient was noted to have anuria on arrival in MICU and with the improvement of hemodynamics, started to pass urine but remained oliguric. CVVH (Continuous Veno Venous Hemofiltration) was started after 40 hours, due to worsening of renal status as revealed by increased serum creatinine and worsened creatinine clearance.

On arrival in MICU, hemoglobin was 7.5 gm/dl, lactic acid level 9.1mmol/l, ABG(arterial blood gas): pH 6.8, PCO_2 53 mmHg , PO_2 115 mmHg, HCO_3 9.9 mmol/L with FiO_2 1.0 mmol/L, INR 3.51, aPTT >120 second, D-dimer > 20 µg/ml. Initial Cardiac index was 1.8 l/min/m^2. After 36 hours, creatinine clearance (24 hours urine) was 19 ml/min, serum creatinine 290 µmol/l and lactic acid level dropped to 0.8 mmol/l. Decision was made to start CVVH (Continuous Veno-Venous Hemafiltration) at this stage to avoid further the metabolic derangement.

If the patients SBP and CI dropped, we did Passive Leg Raising (PLR) test[7,8] and if the SBP and CI improved, controlled fluid challenges were given. Thus by day 4, the patient developed huge cumulative positive fluid balance of 23 litres and there was obvious evidence of generalized edema. Surprisingly, there was no evidence of Pulmonary congestion and oxygenation was never compromised. From the 1st day, Dobutamine iv infusion was also added and with the guidance of SBP and CI monitor, nor-adrenaline and Dopamine were gradually weaned off. With the improvement of hemodynamics, we gradually started removing fluid by ultrafiltration while continuing CVVH, targeting daily negative fluid balance of 1 to 2 liters, if the hemodynamic profile maintained.

As the hemodynamic became relatively stable with inotrope support, the patient was shifted to invasive Angio-lab and IVC (Inferior Vena Cava) filter was inserted. During the procedure there was wide fluctuation of blood pressure requiring frequent resuscitation and interruption of the procedure. The urine output gradually improved and CVVH was discontinued. Keeping dobutamine on-board, maintaining the targeted hemodynamics, smaller doses of Frusemide from 5 to 10 mg iv boluses were administered repeatedly, after CVVH was discontinued, to reduce the generalized edema from the accumulated fluid, without compromising hemodynamics. As the neck became grossly edematous, tracheostomy was done on the 13th day, to avoid extubation failure and related possible catastrophe.

After 48 hours, pupils started reacting to light sluggishly, and there was mild flickering of the head to pain stimuli. So, Fentanyl iv infusion was started to keep the patient pain-free and to avoid sympathetic over activity. On day 5, the patient started to flicker head and both upper limbs on command. On day 6, it was obvious that the lower limbs were not moving at all and it was decided to do the CT Scan of the dorso-lumbar spine, despite that hemodynamics still needed support. CT Scan showed significant perithecal blood, and hematoma over the lumbar spine. Neurosurgeons were following the patient regularly and after reviewing the CT scan, decided not to go for evacuating the perithecal blood as the spinal damage has already been done, patients neuro-recovery was still insignificant and hemodynamics still needed support. Repeat Echocardiogram (TTE) showed the same RV status-thinned out RV wall, poor contractility with gross dilatation. Another surgery may not achieve anything favorable rather may complicate the neuro-recovery. So the decision for spinal clot removal surgery was delayed.

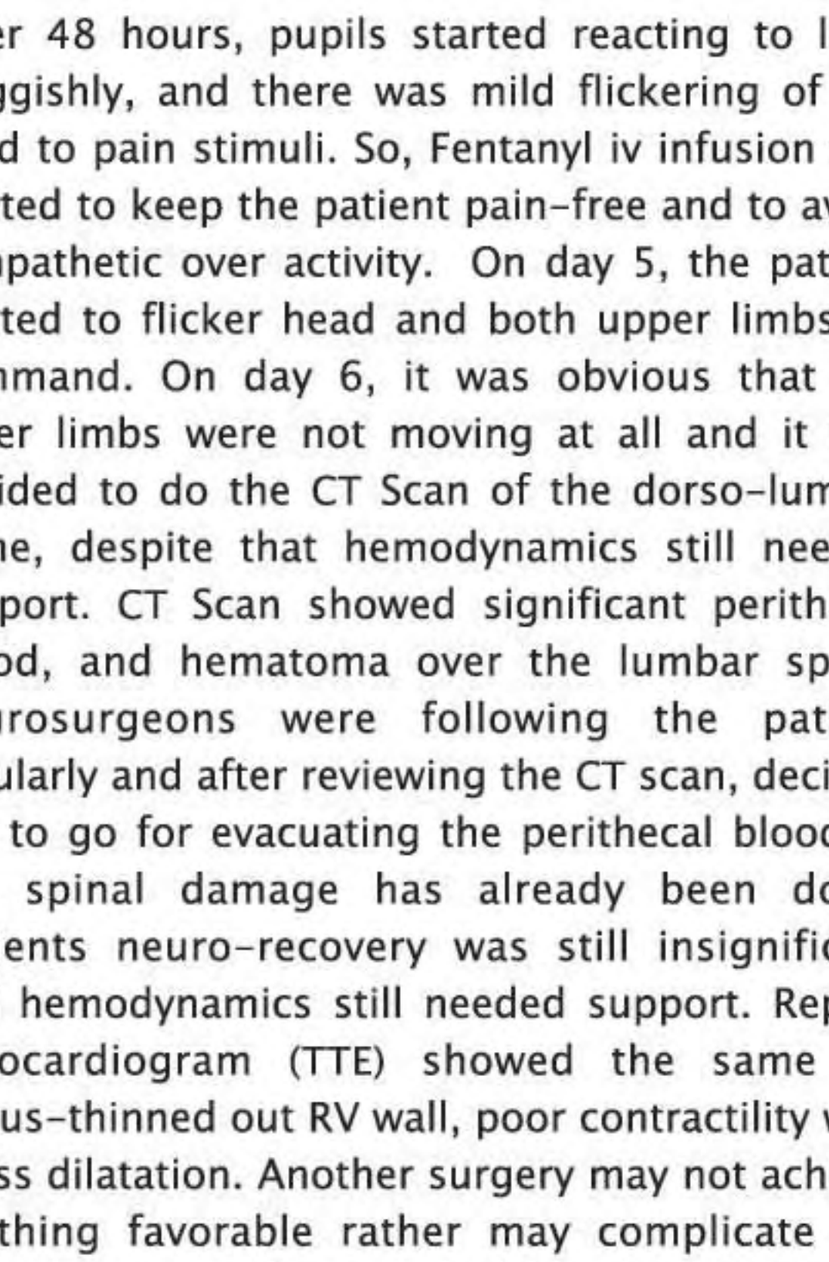

Figure 1: CT angiogram of Chest showing embolus (black arrows) in both right and left main Pulmonary artery branches, on arrival in Accident & Emergency department.

After tracheostomy, sedation was discontinued, the patient's level of consciousness gradually improved, became fully conscious, well-oriented, communicable and moving both upper limbs

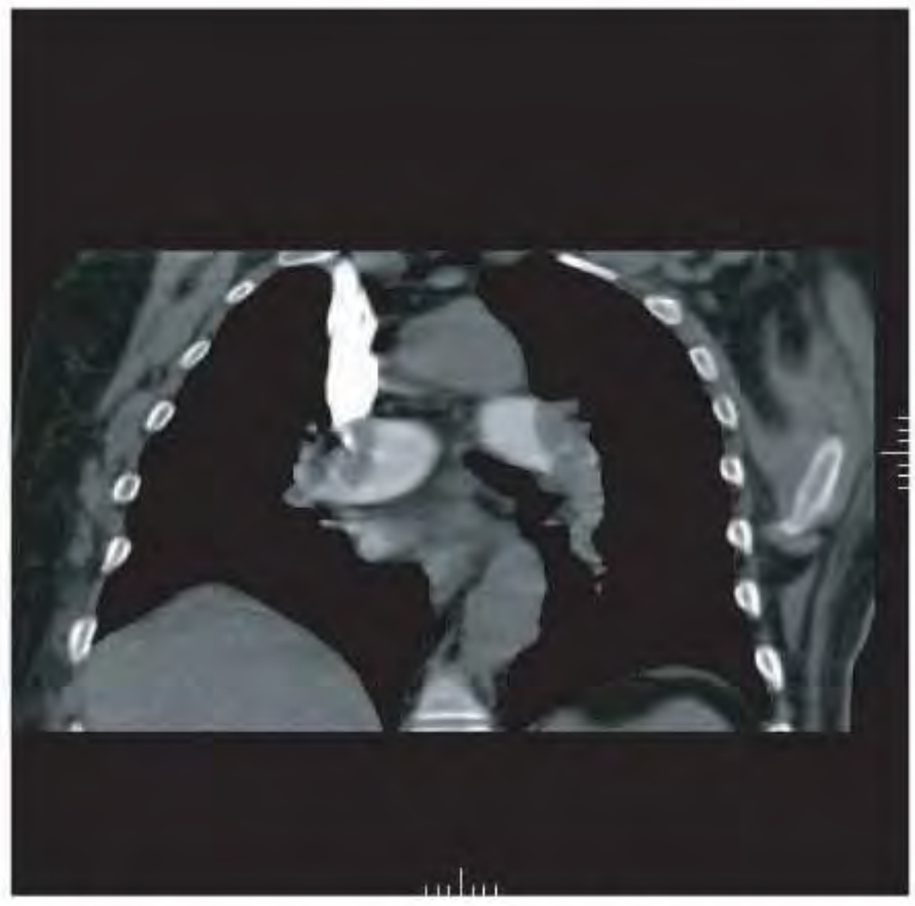

Figure 2: Coronal plane recontruction of CT angiogram of chest, showing embolus (black arrows) in both right and left main pulmonary artery branches.

perfectly but both lower limbs remained paralysed. Dobutamine was weaned-off on day 15. The patient started taking food orally. On day 18, the patient was transferred to floor. Follow up spiral CT angiography of the chest, on day 25, showed no thrombus in pulmonary arteries (Figure 3), but there was a small infarction in the right upper lobe. Follow up Echocardiogram (TTE) after one month showed normal RV size and function and normal PAP (pulmonary artery pressure).

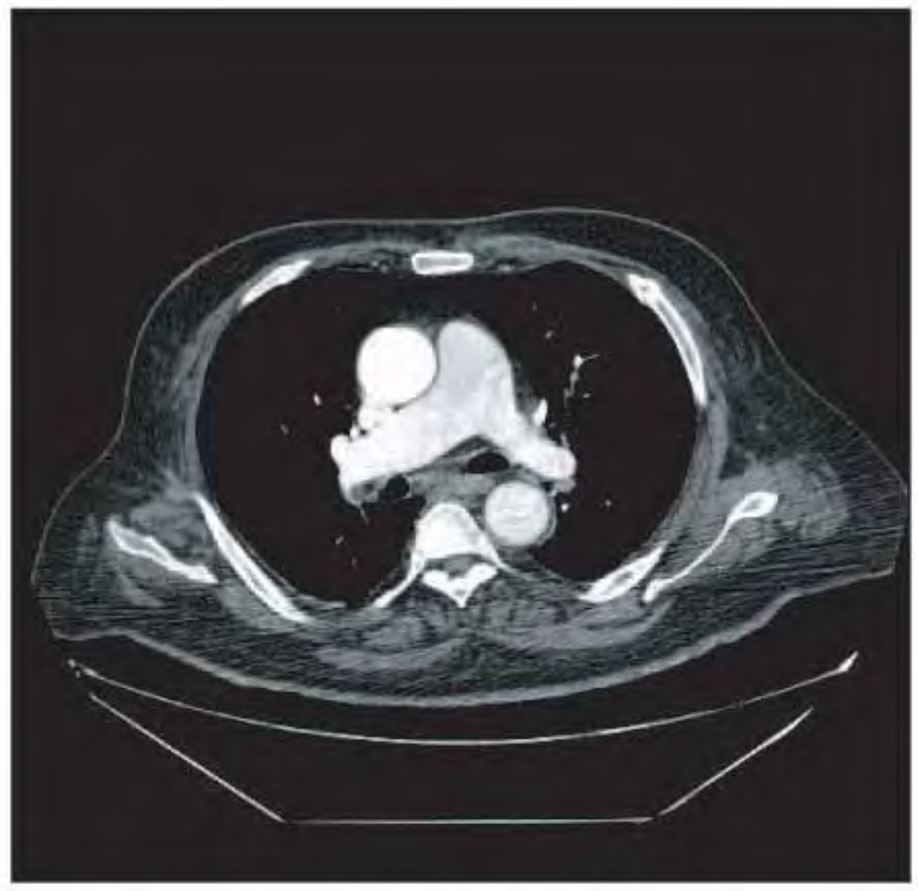

Figure 3: CT angiogram of chest, 25 days after thrombolysis. No embolus in the Pulmonary artery main branches

Discussion

The issue of thrombolysis during CPR (Cardiopulmonary Resuscitation) for cardiac arrest following massive pulmonary embolism, is not well addressed in literatures as most such cases end up in fatality. Only few case-reports have been published.[9,10,11] Even in advanced Centres where emergency cardiopulmonary bypass (CPB) facilities are available, emergency surgical embolectomy under CPB, had high mortality around 85% if the patients had cardiac arrest.[12] The issue of thrombolysis gets further complicated if there is history of recent major surgery. Our patient had such a history. He had a history of recent major spinal surgery and during surgery he bled profusely. He had three witnessed-cardiac arrests in our hospital immediately after confirmation of massive PE, and every time he was successfully resuscitated. There are many case reports of thrombolysis following post-operative massive pulmonary embolism[13,14,15,16] Some bled[13] while others did not bleed.[14,15,16] There are conflicting reports. Agnelli et al[17] reported that following thrombolysis, pooled risk of bleeding is 13% and this rate exceeded the rate of bleeding in heparinised patient by almost 50%. While dealing with bleeding, we used FFP, cryoprecipitate, amino-caproic acid, recombinant Factor VIIa, packed RBC, to avoid intra-spinal tamponade from bleeding which we could not stop very fast. For reversing shock from acute right ventricular failure, when RV was hugely dilated with very poor contraction, we used inotrope and also, did 'Passive Leg Raising (PLR) test[7,8] to make sure that the BP and CI would improve with cautious fluid resuscitation, without further overloading the RV or compromising LV lumen. To our knowledge (through PubMed search), there is no report of using PLR test to guide fluid administration in the event of shock related to acute right ventricular failure (RVF). We are the first to report such manouver to deal with fluid administration in acute RVF. In the literature, there is always caution against overloading the RV in acute RVF.[18]

During the shock-resuscitation, we monitored the BP invasively. We used Vigileo monitor to monitor cardiac output and other hemodynamic profiles so that we could see the achievements of Inotrope manipulation and fluid resuscitation. Since the patient was anuric initially, and later, with slow improvement of hemodynamics the urine output improved but remained oliguric, we decided to avoid metabolic and circulatory assault on kidney and the heart. Hence, we started CRRT (Continuous Renal Replacement Therapy) support in CVVH modality and it did help in improvement of urine output. There are increasing evidence that early application of CVVH improves mortality in acute kidney injury (AKI).[19,20]

In our case, the patient had three episodes of cardiac arrest following massive pulmonary embolism and every episode was successfully resuscitated and thrombolysed during CPR. There was absolute contraindication for thrombolysis but, in the event of life-threatening catastrophe, thrombolysis was the quickest possible rescue therapy available in our hand and was done. In this event, a quick clearance was obtained from Neurosurgeon, judging between risk and benefit. GCS 3 with dilated fixed pupils after three CPR made the decision-making on the nature of aggressive support fairly confusing. But the patient was treated with all the modern support system available in a standard ICU setting. The inotropic support was guided by continuous cardiac output monitor. Acidemia, oliguria and volume overload was dealt by CVVH modality of CRRT. Tracheostomy was done to avoid extubation failure, prolonged use of narcotics, ventilator associated pneumonia. IVC filter was inserted to avoid recurrent PE which could inflict catastrophic blow on the already severely damaged RV. IVC filter reduces the 90 days incidence of recurrent PE to nil.[22] However, we had several limitations while dealing with this patient. We could not stop the spinal bleeding very fast, which gave a blow to both the lower limbs. We could not explain whether the extensive third spacing of fluid was hastened by mediators,[22] in response to prolonged shock, in addition to acute RVF. Also, we could not assess the C-reactive protein (CRP) level, since at that particular time our laboratory ran out of kit for estimating CRP level. The hemodynamics was so unstable and GCS was terribly low for many days, that the target was mainly to keep the patient alive and to watch for the neurologic recovery which was very much unpredictable. After one year, the patient is para-paretic, using wheel-chair but otherwise, fully conscious using both upper limbs perfectly.

Conclusion

Our case was unique in terms of multiple serious comorbidities and thrombolysis-related serious complications, aggressive invasive procedures - despite serious coagulopathy, invasive monitoring of hemodynamics and planning of resuscitation, and dealing with acute renal failure and massive 3rd spacing of resuscitated-fluid overload, with CVVH modality. Above all, the decision to thrombolyse during CPR, despite absolute contra-indication, was worth in saving the life of the patient. In the absence of facilities for surgical embolectomy, there should not be any hesitation to perform 'rescue' thrombolysis in the event of cardiac arrest related to massive pulmonary embolism even if there is contra-indication for pharmacological thrombolysis.

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