© VERTIENTES Revista Especializada en Ciencias de la Salud, 15(2):72-77, 2012.

ACCUTE PULMONARY EMBOLISM CLINICAL CASE

*Davis Cooper-Ruiz, Diana Romero-Zertuche,
Eugenio Pérez-Jiménez, Ofelia Pedraza-Aguilera
Moisés Jiménez-Santos

RESUMEN

El caso clínico se trata de un paciente en la 7a década de la vida que al realizar ejercicio presenta disnea aguda y síncope por 10 minutos, inicialmente fue diagnosticado como síndrome coronario agudo y enviado a nuestro hospital; en urgencias le efectúan un ecocardiograma, electrocardiograma, laboratorios y lo ingresan a la terapia intensiva con diagnóstico de tromboembolia pulmonar aguda, se estabiliza y se trata con Heparina, después de efectuarle una tomografía computada se decide darle Tenecteplase, fue dado de alta 8 días después en buenas condiciones funcionales.

Palabras Claves: Tromboembolia pulmonar, diagnóstico por tomografía computada, hipertensión pulmonar, hipertensión pulmonar por tromboembolia crónica, tromboembolia pulmonar aguda sintomatica, tratamiento de la tromboembolia pulmonar aguda.

Caso clínico de embolismo pulmonar agudo

ABSTRACT

This is a case of a patient in his seventies that during exercise presents acute dyspnea and blackout for 10 minutes, he visits the physician who diagnosis a probable Acute Coronary Syndrome and sends him to our hospital. At the Emergency Room (ER), after an echo cardiogram, laboratory exams and an electrocardiogram the patient was diagnosed with an acute pulmonary embolism and was sent to the Intensive Care Unit (ICU). He was stable and medicated with Heparin, after a CT Pulmonary Angiography (CTPA) he was treated with Tenecteplase and eight days later he was discharged in good health working conditions.

Key Words: Pulmonary thromboembolism, computed tomography diagnosis, pulmonary hypertension, chronic thromboembolic pulmonary hypertension, acute symptomatic pulmonary thromboembolism, treatment acute pulmonary thromboembolism.

ARTÍCULO RECIBIDO EL 11 DE SEPTIEMBRE DEL 2012 Y ACEPTADO EL 06 DE NOVIEMBRE DEL 2012.

INTRODUCTION

Pulmonary thromboembolism (PTE) is a common cause of morbidity and mortality and remains the leading cause of maternal death in developed countries[1,2]. The incidence of lower extremity deep venous thrombosis during pregnancy is estimated to be 0.1% as diagnosed by ultrasound, whereas the incidence of pregnancy related pulmonary embolism has been found to be 0.01% to 0.03%[1-3]. Although venous disease has generally received less attention than arterial disease, acute and chronic conditions can be associated with significant morbidity and, potentially, mortality.

Approximately 1 per 1000 persons is affected by venous thrombosis annually. The most common condition is *acute venous thromboembolism* involving the deep veins of the legs, with or without pulmonary embolism[1,3,4].

Pulmonary Thromboembolism extrapolation from limited data reported by Dalen and Alpert in 1975 indicates that there are 630,000 cases of PTE annually in the United States. According to a recent investigation, the prevalence of unsuspected PTE is

Institución: Department of Radiology, UMAE Cardiología, C.M.N. Siglo XXI, IMSS.
E-mail: *davis.cooper.ruiz@gmail.com

1.5% on routine helical CT scans and is the highest among patients with cancer[1,5].

Although various imaging modalities can be used, Multidetector Computed Tomography (MCT) is the imaging method of choice. Due to recent advances in multidetector row scanning, CT has become more important than scintigraphy in the diagnosis of (PTE)[1-3].

CLINICAL CASE

The patient MRJ is a 71 year old man, married, unemployed, catholic, resident in the DF metropolitan area; as background he smoked for 10 years, 10 cigarettes a day, he has not smoked in the last 20 years.

On September 14th, 2012 during exercise he presents acute onset of dyspnea, blackout for 10 minutes, he rested and recovered after a while, resumed the exercise and presented dyspnea with little effort. He went to a private cardiologist who explored him and performed an electrocardiogram and diagnosed a probable *Acute Coronary Event Syndrome*, he was medicated with an antiplatelet agent, beta blockers and sent to our hospital (Figure 1).

The patient comes to the Emergency Room (ER) on the 18th, September 2012 at 23:00 hrs suffering dyspnea, he had 110/70 blood pressure, pulse per minute 93, respiratory frequency 18 and temperature 36.5° C. Physical exploration shows splitting of S2, decrease of the distal arterial pulse in the right leg and changes in the color of the skin. During his stay at the ER we made EKG (Figure 2) and laboratories (Table 1).

Laboratory tests

Poliglobuly and leukocytosis was observed with normal coagulation tests; also high glucose levels with moderately altered renal function tests were present. Very high levels of D-dimer and B-type natriuretic peptide (BNP) were identified, reflecting high risk for pulmonary embolism with evidence of heart failure, respectively. Normal serum troponin levels were present.

Echo cardiogram taken on September 18th, 2012

Right auricular (RA) 53 mm x 57 mm, with image of thrombosis of approximate 22 mm, 20 mm and 41 mm ejection fraction (EF) 11, systolic pressure pulmonary artery (SPPA) 85 mm Hg. Severe insufficiency of the tricuspid valve.

Haemoglobin	19.8 g/dl
Hematocrit	59 %
Platelets	234,000
Leukocyte	13,5000
TP	18 s
INR	1.43
TTPA	36 s
Glucose	226 mg/dl
Urea	87 mg/dl
Creatinin	2 mg/dl
Dimer D	**3880** ng/ml
B-type natriuretic peptide (BNP)	1050 pg/ml
Troponin I	Less than 0.05 ng/ml

Table 1. Laboratory test taken on September 18th, 2012.

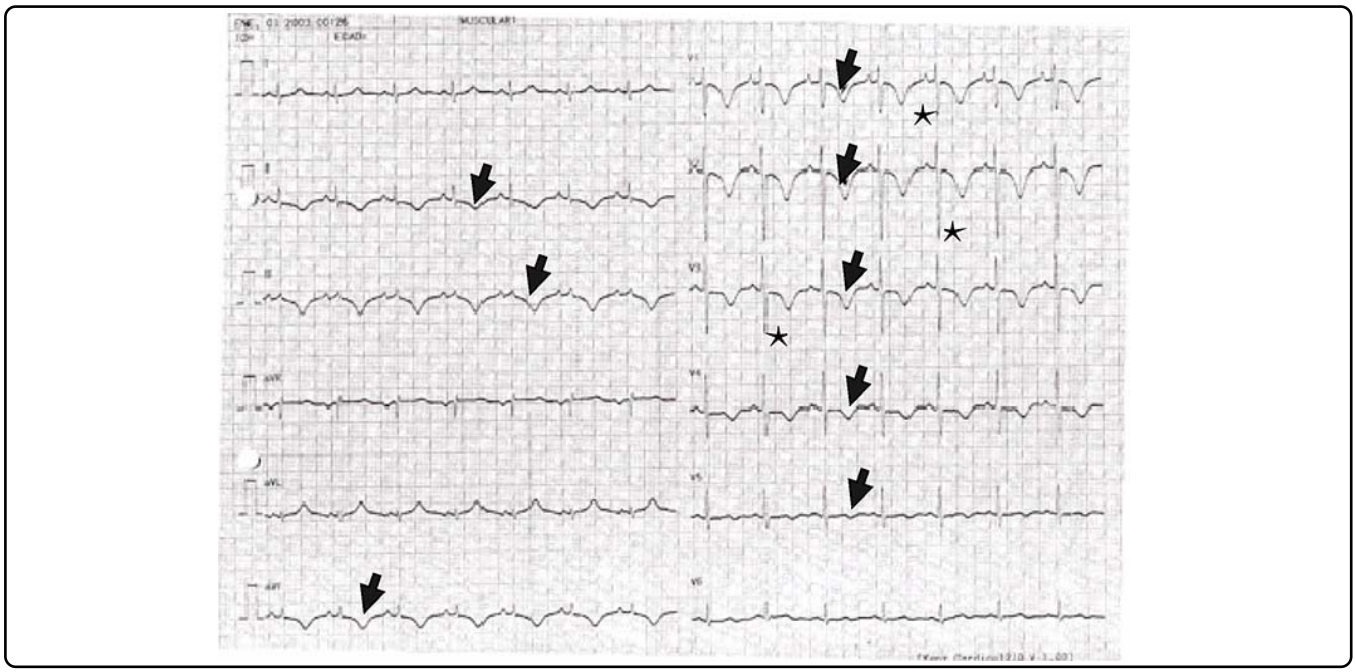

Figure 1. Electrocardiogram performed on September 14th, 2012; shows sinus rhythm, with a heart rate of 85 bpm, the QRS axis is 90º, exists deep inverted and symmetrical T waves in anterior and inferior leads (arrow) with an S1T3 pattern; also deep S waves were observed in V1-V3 leads (⋆). In this case, represents severe right ventricular overload.

Mobility : McConell positive, no movement of the inferior and apical lateral wall.
Right ventricle: 44 mm.
Left ventricle: septum 9 mm, posterior wall 9 mm, diastolic diameter 36 mm, systolic diameter 23 mm.

Evolution:

- He was admitted to the hospital in the Intensive Care Unit (ICU) with a diagnosis of acute pulmonary embolism with a Ginebra score of 11 with no evidence of cardiogenic shock so the treatment was heparin by infusion at 18 U/Kg.
- On September 19th at 10:20 hrs he got a CT scanner for the pulmonary circulation.

CT topogram shows the heart lightly enlarged, there are no data of oligohemic zones in the lung, there is a hipodense round zone on the heart, in the lateral view we see a level that corresponds to the stomach. We applied 70 ml of non ionic contrast media, 370 mg/ml at 5 cm/sec plus a chaser of 40 ml of saline solution. The sequence was started with smart score and the ROI pointer at the main pulmonary artery (image 1 and 2).

CT scans (image 3 and 4) show hipodense image inside the right and left pulmonary arteries, this was seen as thrombi and is known as "horse saddle", in the coronal view is easy to see the air and fecal content of the hernia.

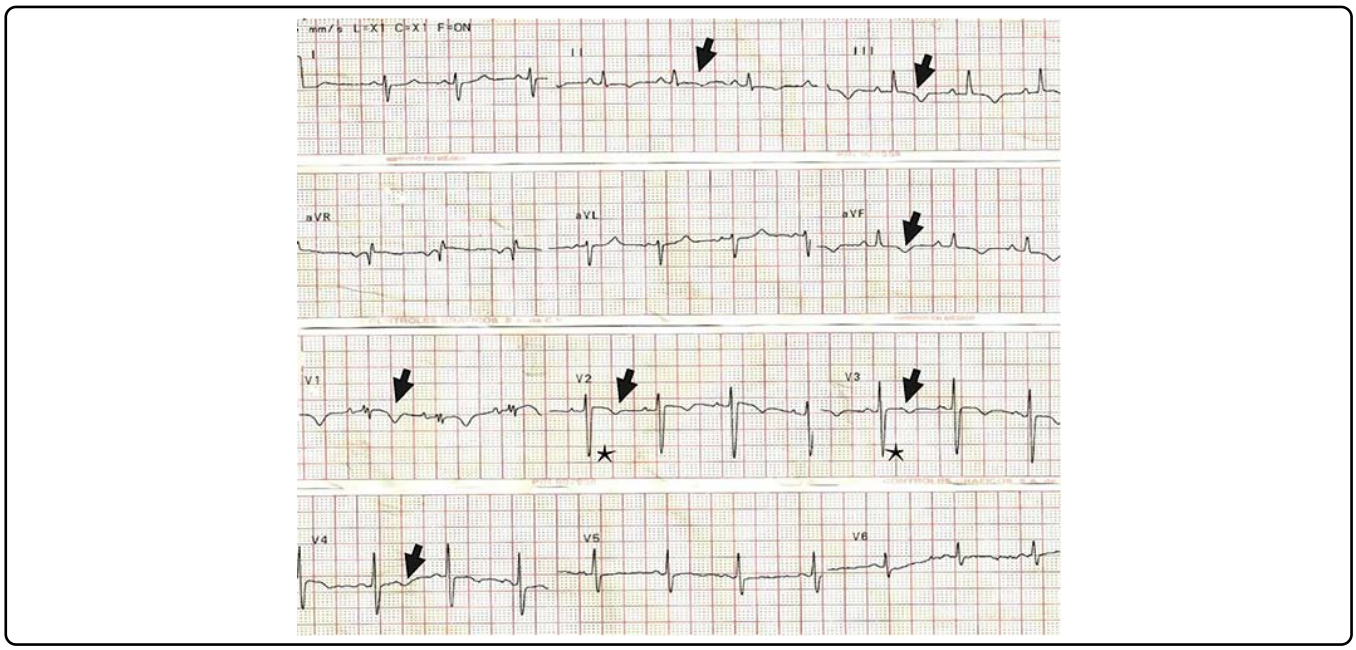

Figure 2. Electrocardiogram performed four days after arrival shows sinus rhythm, with a heart rate of 95 bpm, the QRS axis is -120º (right axis rotation), exists symmetrical inverted and T waves in anterior and inferior leads (arrow) with an S1T3 pattern; also deep S waves were observed in V2-V3 leads (⋆); also incomplete right bundle branch block was identified. In this case, represents right ventricular overload.

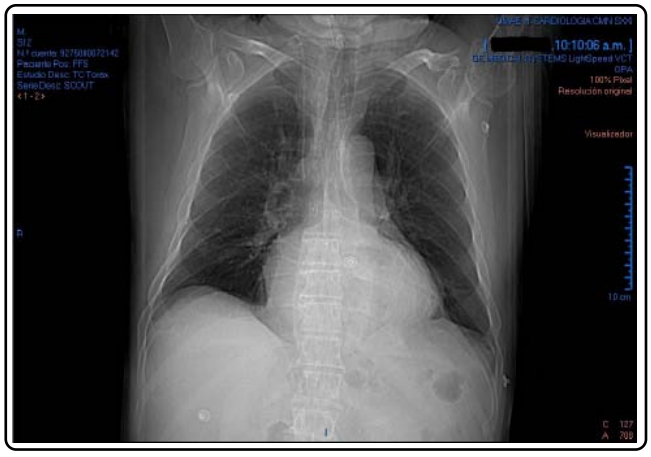

Image 1. CT topogram of the chest shows a slightly grown heart with the right interlobar artery oversize, the heart has the right ventricle enlarged.

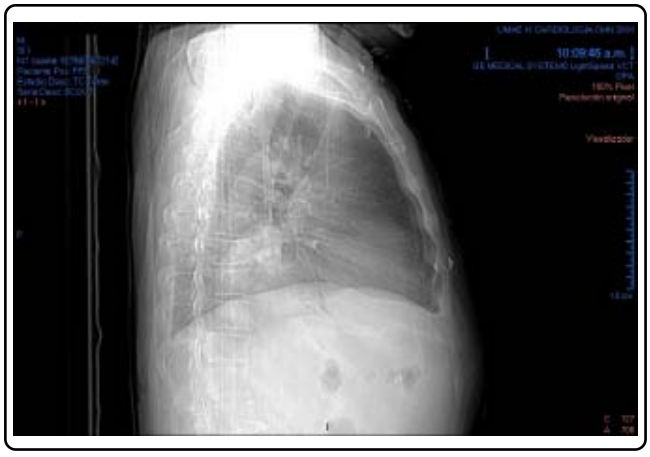

Image 2. CT lateral topogram of the chest shows no other signs.

Also found a thrombi that is filling the lumen of secondary and even tertiary arteries on both lungs (images 5 and 6). At this time he was *diagnosed Acute Pulmonary Thromboembolism*.

- Taking into consideration the information of the CT, the EKG taken at ER and laboratory findings, treatment with Tenecteplase © 40 mg IV (one doses only) was decided.
- An echo cardiogram three hours later demonstrating no clots in the RA and a systolic pressure of the pulmonary artery (SPPA) of 60 mm Hg
- The B-type natriuretic peptide (BNP) on September 20th was 291 pg/ml.
- At that time he was haemodynamic stable, blood pressure 114/70 mm Hg, heart frequency 61 bpm, respiratory frequency 19 bpm, central venous pressure 13 cm of H_2O, SO_2 94% , thus he was sent to regular beds.
- The next days he was haemodynamic stable, without further diseases and in good clinical condition.
- He went to a study of CT of the pulmonary artery on the September 20th at 1037 hrs.

CT angiography (Images 7 and 8) shows that the thrombi image is smaller in the main pulmonary artery, the hipodense area is located close to the artery wall, eccentrically.

We observe the thrombi further in the secondary and even tertiary arteries on both sides, now we *diagnosed Chronic Pulmonary Thromboembolism* (Images 9 and 10).

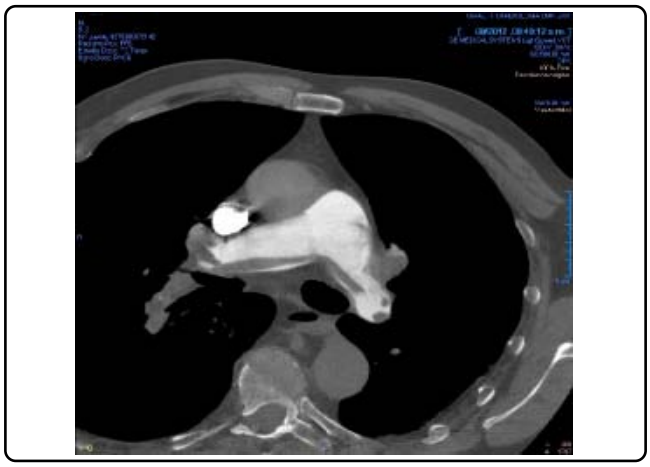

Image 3. CT axial scan shows the main pulmonary artery and the right and left main pulmonary artery division, there is a linear hipodense image that goes into both artery, this is known as the "saddle" sign. This hipodense image is a blood clot. Notice that there are two more round hipodense zones on the left side before the division and a linear image on the right side.

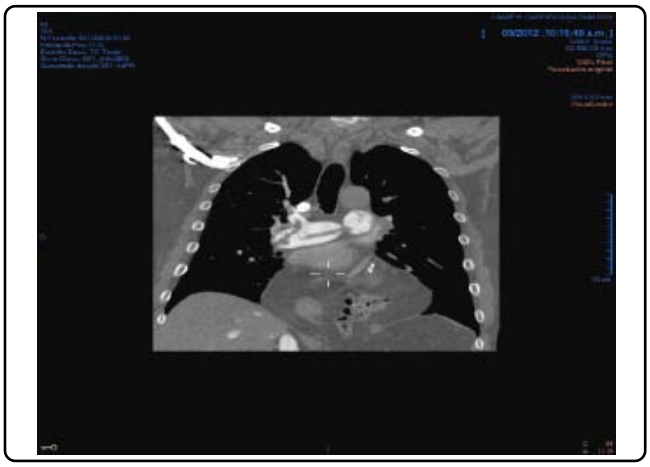

Image 4. CT coronal scan shows the right and left main pulmonary artery division, there is a linear hipodense image that goes into both artery. This hipodense image is a blood clot. Notice that there is blood clot further into the right side. There is a mottled image over the heart that is a diaphragmatic hernia.

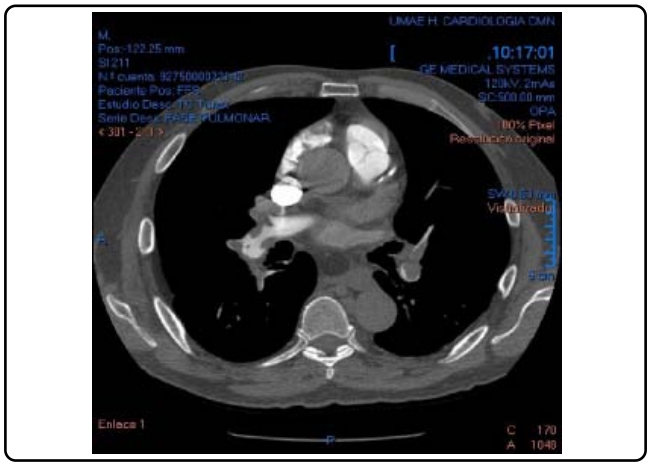

Image 5. CT axial scan shows the right and left main pulmonary arteries, there is a almost 90% of obstruction on the left side and 80% on the right.

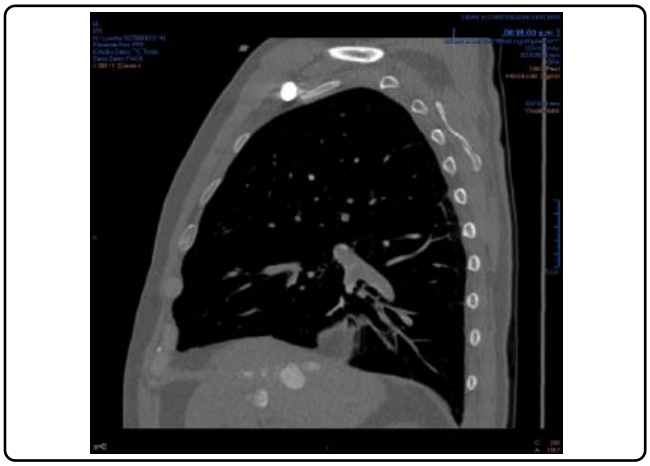

Image 6. CT sagittal plane scan on the right side of the chest shows the right pulmonary artery, there is almost 95% of obstruction.

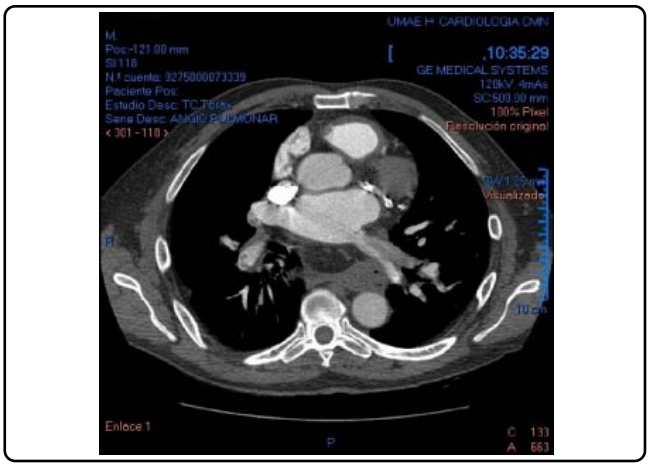

Image 7. CT axial scan taken after treatment shows the right and left main pulmonary artery division, there is a linear hipodense image that goes into the left artery. This hipodense image is residual blood clot. There are two more hipodense zones on the left side and on the right side. Notice the image of the hernia between the aorta and the pulmonary artery.

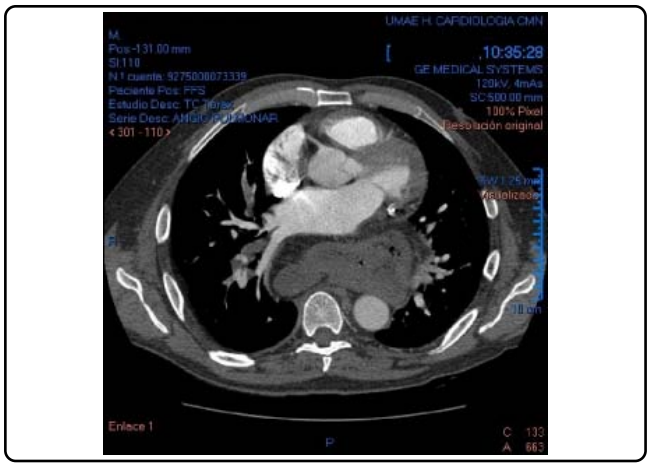

Image 8. CT axial scan taken after treatment shows the right main pulmonary artery, there is a hipodense image that goes into the secondary artery. Notice the image of the hernia between the aorta and the pulmonary artery.

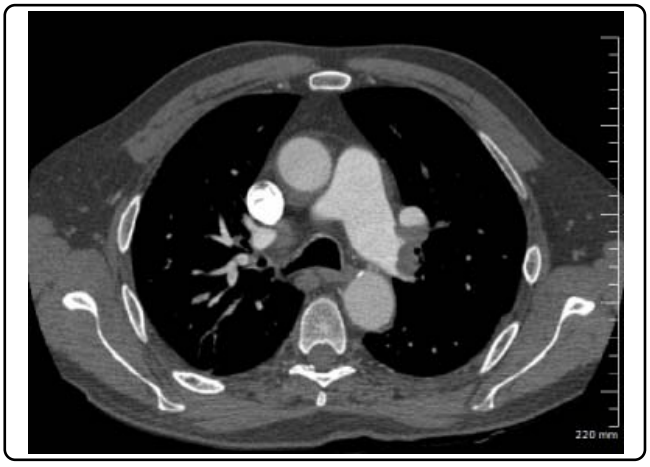

Image 9. CT axial scan taken after treatment shows the left pulmonary artery, there is a hipodense image that causes 90% of obstruction.

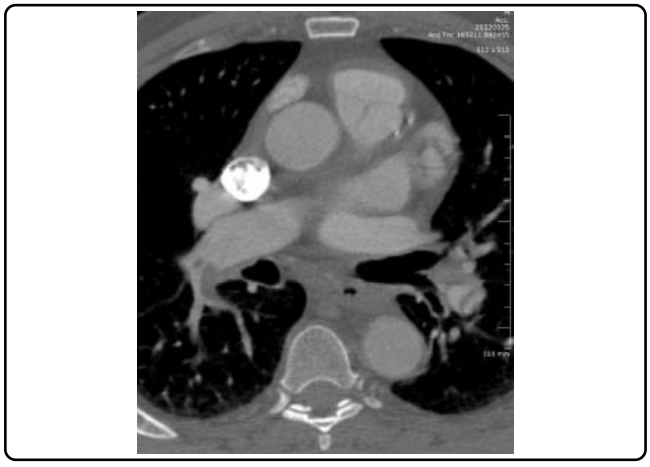

Image 10. CT axial scan taken after treatment shows the right and left pulmonary artery, there is a hipodense image that obstruct 50% on the right side and 80% on the left.

- A second echo cardiogram was taken on September 25th at 18:40 hrs and the results are shown in Table 2.
- In the inter consult with angiology on September 27th they consider treatment with oral anticoagulants *International normalized ratio (INR)* between 2 and 3, venous care, support tights of medium pressure.
- He was sent home on September 28th with oral warfarina and an INR of 2.2 and in good health working conditions.

Commentary

Pulmonary Thromboembolism is a frequent cause of admission in our hospital, patients require of some clinical signs which are qualified in Ginebra score[1], CT Angiography[2-9], echo cardiogram[1,5,10,11] and laboratories[1,4,6]. Much has been done in the PIOPED[4,6,12] II and III to give guides to study and treat these patients, these guides are mandatory to know if you attend on this kind of patients. Another part of PIOPED II study shows that CT Venography and Compression Sonography are diagnostically equivalent and should be used[12]. Look for the PIOPED IV although we have some reserves as PIOPED II and III are working fine.

Left heart	Left auricle 42mm	Diastolic diameter 45mm	Diameter systolic 26mm	Septum 11mm	Posterior wall 10mm
Right heart	Right Auricle 42mm	Right ventricle 30mm	Pulmonary systolic pressure 65mm	Systolic displacement of the tricuspid ring 24mm	Tricuspid S wave 11 cm/s
Diastolic function	E wave 69	A wave 89	Relationship E/A 0.8	Mitral deceleration 201	Isovolumetric relaxation 116
LVEF 66%	FAC 36%	Final systolic volume 31ml			

Table 2. Echo cardiogram taken on the September 25th, 2012.

BIBLIOGRAPHY

1. Torbicki A., Perrier A., Konstantinides S., Agnelli G., Galiè N., Pruszczyk P., Bengel F., Brady A. J .B., Ferreira D., Janssens U., Klepetko W., Mayer E., Remy-Jardin M., Bassand J. P. Guías de práctica clínica de la Sociedad Europea de Cardiología. Guías de práctica clínica sobre diagnóstico y manejo del tromboembolismo pulmonar agudo. Rev Esp Cardiol. 2008;61(12):1330.

2. Kimura-Hayama E., Canseco-León N., Santiago-Serra R. Angiotomografía computarizada multidetector: una nueva era en la evaluación de tromboembolia pulmonar. Arch Cardiol Mex 2011;81(2):137-150.

3. Wittram C., Maher M. M., Yoo A. J., Kalra M. K., Shepard J. O., McLoud T. C. CT Angiography of Pulmonary Embolism: Diagnostic Criteria and Causes of Misdiagnosis. RadioGraphics 2004; 24:1219 - 1238 Published online 10.1148/rg.245045008.

4. Torbicki A., Perrier A., Konstantinides S., Agnelli G., Galie N., Pruszczyk P., Bengel F., Brady A. J. B., Ferreira D., Janssens U., Klepetko W., Mayer E., Remy-Jardin M., Bassand J. P. Guidelines on the diagnosis and management of acute pulmonary embolism. European Heart Journal (2008) 29, 2276-2315 doi:10.1093/eurheartj/ehn310.

5. Castañer E., Gallardo X., Ballesteros E., Andreu M., Pallardó Y., Mata J. M., Riera L. CT Diagnosis of Chronic Pulmonary Thromboembolism. RadioGraphics 2009; 29:31-53, Published online 10.1148/rg.291085061.

6. Stein P. D., Woodard P. K., Weg J. G., Wakefield T. W., Tapson V. F., Sostman H. D., Thomas A. S, Quinn D. A., Leeper K. V., Hull R. D., Hales C. A., Gottschalk A., Goodman L. R., Fowler S. E., Buckley J. D. Diagnostic Pathways in Acute Pulmonary Embolism: Recommendations of The PIOPED II Investigators. The American Journal of Medicine (2006) 119, 1048-1055.

7. Deonarine P., Wet C., McGhee A. Computed tomographic pulmonary angiography and pulmonary embolism: predictive value of a d-dimer assay. BMC Research Notes 2012, 5:104.

8. Stein P. D., Fowler S. E., Goodman L. R., Gottschalk A., Hales C. A., Hull R. D., Leeper K. V., Popovich J., Quinn D. A., Sos T. A., Sostman H. D., Tapson V. F., Wakefield T. W., Weg J. G., Woodard P. K. Multidetector Computed Tomography for Acute Pulmonary Embolism. N Engl J Med 2006;354:2317-27. Copyright © 2006 Massachusetts Medical Society.

9. Masotti L., Righini M., Vuilleumier N., Antonelli F., Cappelli G. L. R., Ray P. Prognostic stratification of acute pulmonary embolism: Focus on clinical aspects, imaging, and biomarkers. Vascular Health and Risk Management 2009:5 567-575 ,© 2009 Masotti et al, publisher and licensee Dove Medical Press Ltd.

10. Tapson V. F. Advances in the Diagnosis and Treatment of Acute Pulmonary Embolism . F1000 Medicine Reports 2012, 4:9 (doi:10.3410/M4-9) | 02 May 2012.

11. Miniati M., Cenci C., Monti S., Poli D. Clinical Presentation of Acute Pulmonary Embolism: Survey of 800 Cases. PLoS ONE www.plosone.org, February 2012 | Volume 7 | Issue 2 | e30891.

12. Goodman L. R., Stein P. D., Matta F., Sostman H. D., Wakefield T. W., Woodard P. K., Hull R., Yankelevitz D. F., Beemath A. CT Venography and Compression Sonography Are Diagnostically Equivalent: Data from PIOPED II. AJR 2007; 189:1071-1076.