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Massive Pulmonary Embolism in a Young Female: Multidisciplinary Approach and Therapeutic Interventions

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Type of Publication: Case Report

Conflicts of Interest: Nil

Abstract

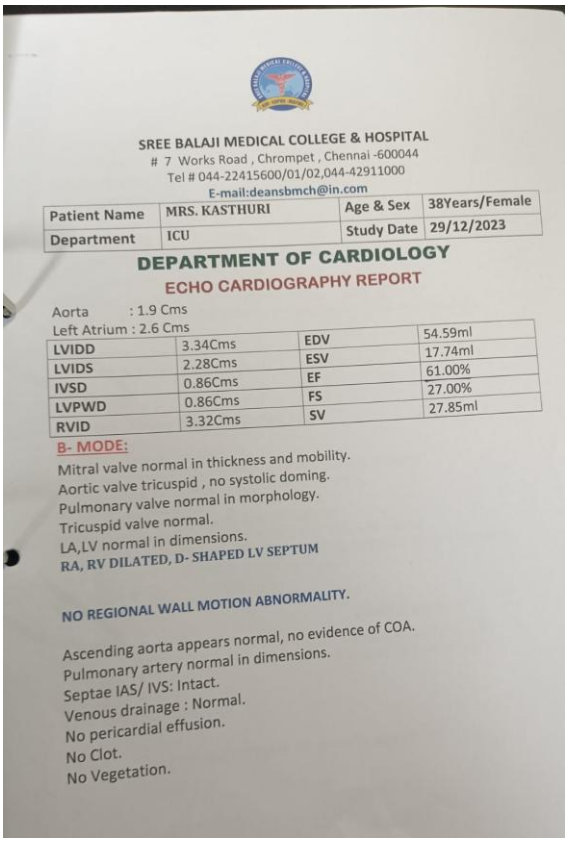
Venous thromboembolism (VTE), comprising pulmonary embolism (PE) and deep vein thrombosis (DVT), poses a significant clinical challenge due to its diverse and often nonspecific manifestations. We provide a comprehensive description of the clinical, electrocardiographic, and radiological findings observed in the patient, elucidating the individualized therapeutic approaches based on their hemodynamic stability.

Recognizing the spectrum of clinical presentations in pulmonary embolism is crucial, given its variability and potential life-threatening consequences. Timely identification and differentiation between hemodynamically stable and unstable cases are pivotal, as immediate initiation of appropriate treatment significantly mitigates the morbidity and mortality risks associated with obstructive shock.

Keywords: Pulmonary Hypertension, Hemodynamically

Case Report

A 38-year-old obese female, a non smoker with no known co-morbidities, presented with complaints of sudden-onset chest pain with breathlessness for four hours, associated with palpitations. The patient was tachypneic with blood pressure of 70/50 mmHg with normal oxygen saturation. Her ECG showed sinus tachycardia with the "S1Q3T3" pattern with right axis deviation and incomplete right bundle branch block (RBBB). D Dimer was elevated. The 2D echo revealed a dilated right atrium and right ventricle and moderate pulmonary hypertension RVSP- 55mmHg.



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Patient Name	MRS. KASTHURI	Age & Sex	38Years/Female
Department	ICU	Study Date	29/12/2023

DEPARTMENT OF CARDIOLOGY
ECHO CARDIOGRAPHY REPORT

Aorta : 1.9 Cms
Left Atrium : 2.6 Cms

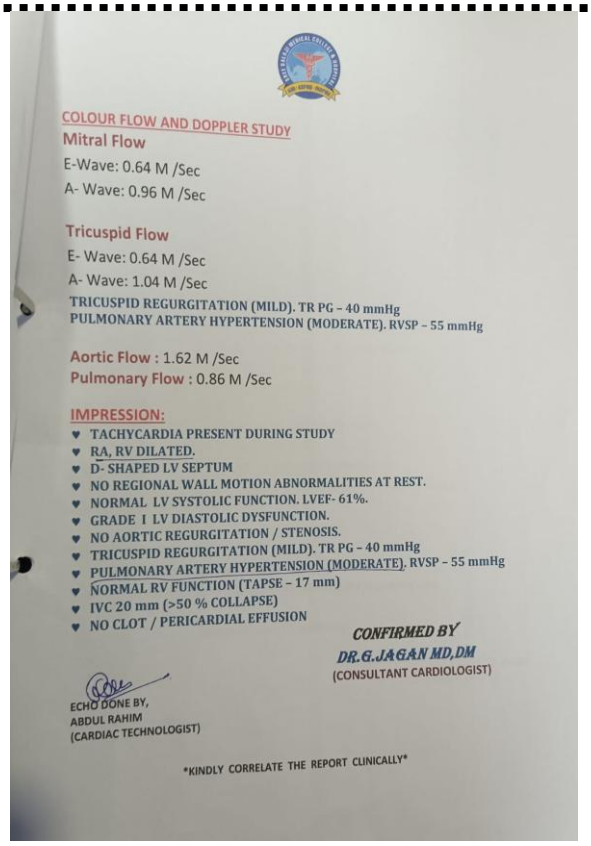
LVIDD	3.34Cms	EDV	54.59ml
LVIDS	2.28Cms	ESV	17.74ml
IVSD	0.86Cms	EF	61.00%
LVPWD	0.86Cms	FS	27.00%
RVID	3.32Cms	SV	27.85ml

B- MODE:
Mitral valve normal in thickness and mobility.
Aortic valve tricuspid , no systolic doming.
Pulmonary valve normal in morphology.
Tricuspid valve normal.
LA,LV normal in dimensions.
RA, RV DILATED, D- SHAPED LV SEPTUM

NO REGIONAL WALL MOTION ABNORMALITY.

Ascending aorta appears normal, no evidence of COA.
Pulmonary artery normal in dimensions.
Septae IAS/ IVS: Intact.
Venous drainage : Normal.
No pericardial effusion.
No Clot.
No Vegetation.

Image 1



COLOUR FLOW AND DOPPLER STUDY

Mitral Flow

E-Wave: 0.64 M /Sec

A- Wave: 0.96 M /Sec

Tricuspid Flow

E- Wave: 0.64 M /Sec

A- Wave: 1.04 M /Sec

TRICUSPID REGURGITATION (MILD). TR PG – 40 mmHg

PULMONARY ARTERY HYPERTENSION (MODERATE). RVSP – 55 mmHg

Aortic Flow : 1.62 M /Sec

Pulmonary Flow : 0.86 M /Sec

IMPRESSION:

- TACHYCARDIA PRESENT DURING STUDY
- RA, RV DILATED.
- D- SHAPED LV SEPTUM
- NO REGIONAL WALL MOTION ABNORMALITIES AT REST.
- NORMAL LV SYSTOLIC FUNCTION. LVEF- 61%.
- GRADE I LV DIASTOLIC DYSFUNCTION.
- NO AORTIC REGURGITATION / STENOSIS.
- TRICUSPID REGURGITATION (MILD). TR PG – 40 mmHg
- PULMONARY ARTERY HYPERTENSION (MODERATE). RVSP – 55 mmHg
- NORMAL RV FUNCTION (TAPSE – 17 mm)
- IVC 20 mm (>50 % COLLAPSE)
- NO CLOT / PERICARDIAL EFFUSION

CONFIRMED BY

DR.G.JAGAN MD,DM

(CONSULTANT CARDIOLOGIST)

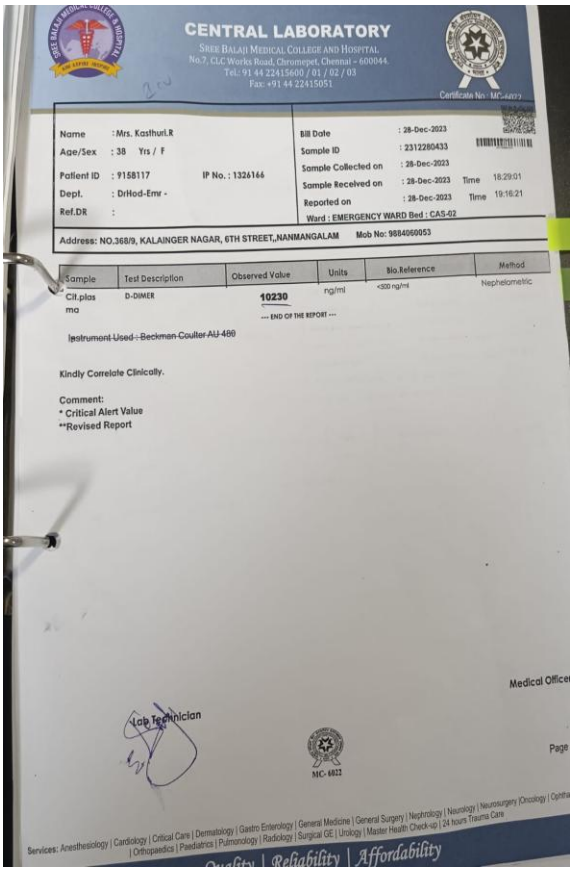
ECHO DONE BY,

ABDUL RAHIM

(CARDIAC TECHNOLOGIST)

KINDLY CORRELATE THE REPORT CLINICALLY

Image 2



CENTRAL LABORATORY

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Fax: +91 44 22415051

Certificate No : MC-6023

Name	: Mrs. Kasthuri.R	Bill Date	: 28-Dec-2023
Age/Sex	: 38 Yrs / F	Sample ID	: 2312280433
Patient ID	: 9158117 IP No. : 1326164	Sample Collected on	: 28-Dec-2023
Dept.	: DrHod-Emr -	Sample Received on	: 28-Dec-2023 Time 18:29:01
Ref.DR	:	Reported on	: 28-Dec-2023 Time 19:16:21
		Ward : EMERGENCY WARD Bed : CAS-02	

Address: NO.368/9, KALAINGER NAGAR, 6TH STREET,,NANMANGALAM Mob No: 9884060053

Sample	Test Description	Observed Value	Units	Bio.Reference	Method
Cit.plas ma	D-DIMER	10230	ng/ml	<500 ng/ml	Nephelometric

--- END OF THE REPORT ---

Instrument Used : Beckman Coulter AU 480

Kindly Correlate Clinically.

Comment:

* Critical Alert Value

**Revised Report

Medical Officer

Lab Technician

MC- 6023

Page

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Quality | Reliability | Affordability

Image 3

Computed tomography pulmonary angiography (CTPA) revealed a near complete hypodense filling defects noted in right and left pulmonary arteries and an impression of acute pulmonary thromboembolism-CLOT BURDEN SCORE (30/40) according to QANADLI INDEX.

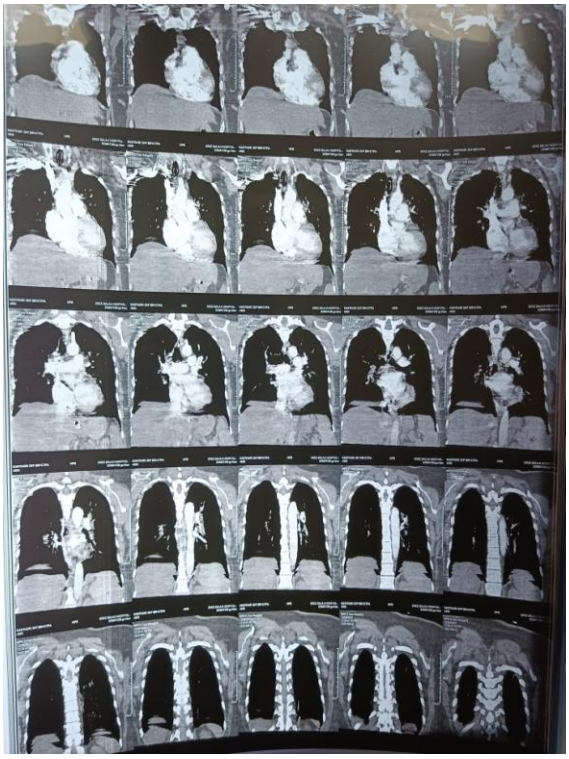
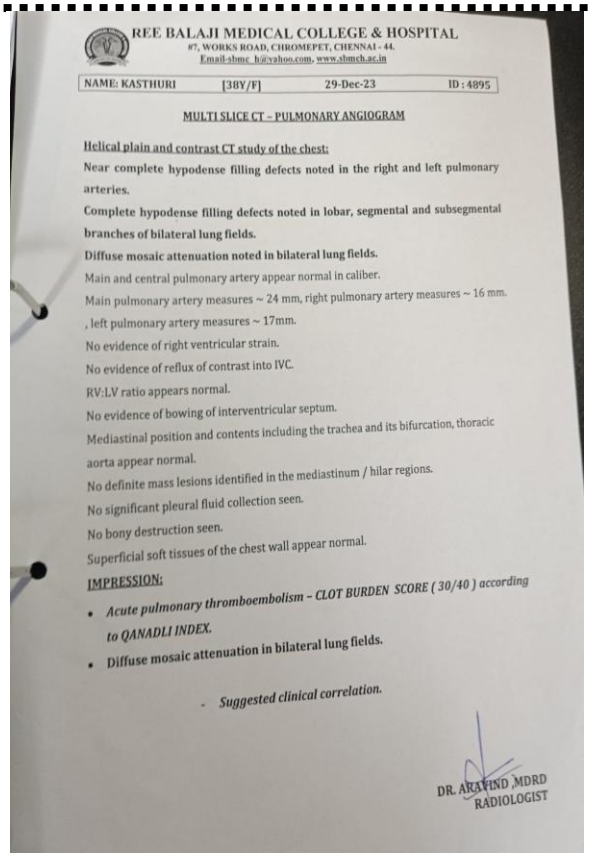


Image 4

Image 5



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NAME: KASTHURI [38Y/F] 29-Dec-23 ID : 4895

MULTI SLICE CT – PULMONARY ANGIOGRAM

Helical plain and contrast CT study of the chest:

Near complete hypodense filling defects noted in the right and left pulmonary arteries.

Complete hypodense filling defects noted in lobar, segmental and subsegmental branches of bilateral lung fields.

Diffuse mosaic attenuation noted in bilateral lung fields.

Main and central pulmonary artery appear normal in caliber.

Main pulmonary artery measures ~ 24 mm, right pulmonary artery measures ~ 16 mm. , left pulmonary artery measures ~ 17mm.

No evidence of right ventricular strain.

No evidence of reflux of contrast into IVC.

RV:LV ratio appears normal.

No evidence of bowing of interventricular septum.

Mediastinal position and contents including the trachea and its bifurcation, thoracic aorta appear normal.

No definite mass lesions identified in the mediastinum / hilar regions.

No significant pleural fluid collection seen.

No bony destruction seen.

Superficial soft tissues of the chest wall appear normal.

IMPRESSION:

- *Acute pulmonary thromboembolism – CLOT BURDEN SCORE (30/40) according to QANADLI INDEX.*
- Diffuse mosaic attenuation in bilateral lung fields.

- *Suggested clinical correlation.*

DR. ARAVIND MDRD
RADIOLOGIST

Image 6

This is a case of massive PE and the patient was thrombolysed with streptokinase. Post thrombolysis, anticoagulation was started with heparin and later discharged with novel oral anticoagulant after clinical recovery.

During follow-up, evaluation for thrombophilic states was done which revealed intermediate hyperhomocysteinemia (78 micromol/l) with normal vitamin B12, folate, protein C, and S levels as well as negative antinuclear antibody (ANA) and antiphospholipid antibodies (APLA) profile.

Discussion

Pulmonary embolism (PE) occurs when there is a disruption to the flow of blood in the pulmonary artery or its branches by a thrombus that originated somewhere else. In deep vein thrombosis (DVT), a thrombus develops within the deep veins, most commonly in the lower extremities. PE usually occurs when a part of this thrombus breaks off and enters the pulmonary circulation.

Most pulmonary embolisms originate as lower extremity DVTs. Hence, risk factors for pulmonary embolism (PE) are the same as risk factors for DVT. Virchow's triad of hypercoagulability, venous stasis, and endothelial injury provides an understanding of these risk factors.

Risk factors can be classified as genetic and acquired. Genetic risk factors include thrombophilia such as factor V Leiden mutation, prothrombin gene mutation, protein C deficiency, protein S deficiency, hyperhomocysteinemia, among others. Acquired risk factors include immobilization for prolonged periods (bed rest greater than three days, anyone traveling greater than 4 hours, whether by air, car, bus, or train), recent orthopedic surgery, malignancy, indwelling venous catheter, obesity, pregnancy, cigarette smoking, oral contraceptive pill use, etc

It is extremely crucial to divide PE based on the presence or absence of hemodynamic stability.

Hemodynamically unstable PE (previously called massive or high-risk PE) is PE which results in hypotension (as defined by systolic blood pressure (SBP) less than 90 mmHg or a drop in SBP of 40 mm Hg or more from baseline or hypotension that requires vasopressors or inotropes), the old term "massive" PE does not describe the size of the PE but describes its hemodynamic effect. Patients with hemodynamically unstable PE are more likely to die from obstructive shock (i.e., severe right ventricular failure).

Hemodynamically stable PE is a spectrum ranging from small, mildly symptomatic or asymptomatic PE (low-risk PE or small PE) to PEs, which cause mild hypotension that stabilizes in response to fluid therapy, or those who present with right ventricle dysfunction (submassive or intermediate-risk PE), but is hemodynamically stable.

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