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A patient with extensive and severe pulmonary thromboembolism with lung infarct with right Atrial thrombosis – A case report

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Abstract

A patient reported acute complains of severe anemia with features of Congestive cardiac failure with suspicion of obstructive sleep apnea with features of hepatocellular dysfunction. Later turned out to be extensive pulmonary thromboembolism causing pulmonary hypertension leading to Cor pulmonale with hepatocellular congestion.

Keywords: VTE, CVA, MI, PE

Introduction

Venous thromboembolism (VTE) and pulmonary embolism (PE) are responsible for a significant number of cardiovascular deaths, ranking third after myocardial infarction (MI) and cerebrovascular accidents (CVA)[1]. However, due to the difficulty in accurately diagnosing PEs, many cases may go undetected, making it challenging to calculate the true incidence. Nevertheless, PE is a preventable cause of in-hospital mortality and should be taken seriously.

Most clinically significant PEs originate as VTEs in the lower extremities or pelvic veins. Less frequently, upper extremity thromboembolic events lead to PE. Various conditions lead to the generation of VTE. Virchow's triad of hypercoagulability, venous stasis, and vessel wall injury provides a model for understanding many of the risk factors. These factors are usually either inherited or acquired, as shown in Tables Tables1 and 2.

Table 1

Inherited Risk Factors for VTE [2,3]

Weak	Medium	Strong
Hyperhomocy-steinemia	Mutation in the factor V Leiden (FVL)	Deficiencies of coagulation inhibitors including antithrombin (AT), protein C (PC), and its cofactor protein S (PS)
	Mutation in the 3′-untranslated part of the prothrombin (Factor II) gene (prothrombin 20210A, rs 1799963)	Insufficiency of anticoagulant pathways such as tissue factor pathway inhibitor (TFPI), thrombomodulin and endothelial protein C receptor (EPCR)
	Blood group (non-O blood group) C- to T-variation at position of 10034 in the fibrinogen gamma chain (rs 2066865)	Elevated level of factor VIII

Table 2

Acquired Risk Factors for VTE [2–4]

WEAK (odds ratio <2)	MEDIUM (odds ratio 2–9)	STRONG (odds ratio >10)
Bed rest (>3 days)	Arthroscopic knee surgery	Fracture (hip or leg)
Extended immobility (air travel >8 hours)	Central venous lines	Hip or knee replacement
Increasing age (³40 years	Chemotherapy	Major general surgery
Laparoscopic surgery	Congestive heart or respiratory failure	Major trauma
Obesity	Hormone replacement therapy or oral contraceptive therapy	Spinal cord injury
Pregnancy/antepartum	Malignancy	
Varicose veins	Pregnancy/postpartum	
	Previous VTE	

Prompt recognition of a pulmonary embolism (PE) is crucial due to its high mortality and morbidity rates, which can be prevented with early treatment. Failing to diagnose a PE is a serious management error, as untreated patients have a 30% mortality rate, while those who receive effective therapy have only an 8% mortality rate. Therefore, early diagnosis and treatment are essential for improving patient outcomes. [5 PE, or pulmonary embolism, can be a serious condition that may not show any symptoms or can lead to sudden death. The signs and symptoms of PE are not specific and can be present in other conditions such as acute MI, congestive heart failure, or pneumonia. In the PIOPED II trial, patients with PE showed a range of signs and symptoms. The most common signs were tachypnea (54%) and tachycardia (24%). The most common symptoms were dyspnea, which usually occurred within seconds, either at rest or with exertion (73%), pleuritic pain (44%), calf or thigh pain (44%), calf or thigh swelling (41%), and cough (34%). [6].

Pulmonary Embolism can be presented with variable signs and symptoms. There are scoring systems to assist in the determination of likelihood of PE and thromboembolic events. Wells criteria and Geneva score are often used as scoring systems as shown below.

Table 3

Modified Wells Criteria

Symptoms and signs of deep-vein thrombosis	3.0
Heart rate >100 beats per minute	1.5
Recent immobilization or surgery (<4 weeks)	1.5
Previous PE or deep venous thrombosis (DVT)	1.5
Hemoptysis	1.0
Active cancer	1.0
PE more likely than alternate diagnosis	3.0
Total score:	
</= 4 PE unlikely	
>4 PE likely	

Table 4

Revised Geneva Score

Previous DVT or PE	3
Heart Rate	
75–94 beats/min	3
>/=95 beats/min	5
Surgery or fracture within 1 month	2
Hemoptysis	2
Active Cancer	2
Unilateral lower limb pain	3
Pain on lower limb deep venous palpation and unilateral edema	4
Age >65 years	1
Total score:	
</= 5 PE unlikely	
>5 PE likely	

The PE Rule-Out Criteria (PERC) can help rule out pulmonary embolism (PE) in low-risk patients in the emergency department. The PERC criteria are listed below.

PE Rule-out Criteria [7]

If a patient meets all PERC criteria, has low Wells score and physician's gestalt, PE can be ruled out. [7]

The diagnostic workup for a patient suspected of having a pulmonary embolism (PE) will vary depending on whether the patient is hospitalized and whether there is hemodynamic instability. If a patient has a suspected PE, the diagnosis of proximal deep vein thrombosis (DVT) in a symptomatic patient or in an asymptomatic patient who has contraindications to CT angiography is sufficient to confirm the presence of PE. [8]

Case Report

A Female patient,42 year old came to the hospital emergency for the complain of dry cough and generalised weakness since one-month, Pedal edema and dyspnea on

exertion since 2 weeks along with on and off vomiting since one week. She is newly diagnosed diabetes. No other co-morbidity was present. She had past history of snoring since 1 year. She do not have any H/O bleeding or coagulation disorder. Her pregnancy was uneventful, had full term normal delivery with 2 children. No h//o hospitalisation, CAD, HTN.

On general examination BP was 120/84 mm hg, Pulse rate 98/minute, regular in rhythm Spo2 98% on 4 litre oxygen. RBS was 188 mg/dl .

On general examination, severe pallor was present. Respiratory rate was 20/minute. Rest examination was normal.

On chest examination, b/l basal crepitations were present. No other added sound was present.

On Cardiovascular examination P2 was loud at pulmonary area, parasternal heave was present and a pansystolic murmur was present at tricuspid area with inspiration accentuation suggestive of Tricuspid murmur.

Per abdomen examination was soft, non-tender, no organomegaly present. Patient was conscious, oriented and responding to commands.

She was referred to ICU. She was kept propped up, and advised antibiotics, diuretics, antiemetics, regular insulin according to sliding scale 8 hrly and other symptomatic treatments. Blood transfusion was done. She was nebulised with Prevent thrice a day and budecort twice a day.

Tab Tadalafil 20 mg twice a day was advised. Tadafil 20mg Tablet belongs to a group of medicines called PDE5 inhibitors. It works by relaxing blood vessels and can help lower the blood pressure in the arteries of your lungs.

ECG was done showing S1Q3T3 with sinus tachycardia. Right axis deviation with RBBB present in ECG.

1st 2D echo was suggestive of severe Pulmonary arterial hypertension with LVEF 61%. Normal left ventricle systolic function was seen. PASP was 75 mm Hg. Summary of other findings on echo- Right atrium and right ventricle was dilated.

RA diameter 54 × 46 mm, RV basal diameter = 48 mm

TAPSE (Tricuspid annular plane systolic excursion) 12 mm, S' 6.8 cm/s

LV dysfunction grade 1

NO PE/clot/vegetation

Repeat 2Decho done few days after-

Ejection fraction- 62%

Shortening fraction – 33%

Dilated RA/RV

RA Basal diameter- 42mm

RV dysfunction (TAPSE-14)

Thrombus noted in right pulmonary artery at bifurcation(14×13mm)

LA thrombus (14×7mm)

IVC 18mm (>50% collapsible with inspiration)

Normal LV systolic function, LVEF 62%, No RWMA

Moderate TR, Moderate PAH (PASP=67mmHg)

No clot in IVC

No PE/Vegetations

CT Thoracic Angiography

Angiography- Large non-enhancing thrombusis noted in the right pulmonary artery at the level of hilum causing near complete luminal obliteration. There is extension of the thrombus into right inferior descending pulmonary artery with dilatation. There is further extension into subsegmental branches of right, middle and lower lobe arteries. It is culminating into a peripheral wedge-shaped consolidation with peripheral as well as central ground glass opacity giving rise to Halo/atoll sign is noted in the right middle lobe- infarct.

Thrombus noted at the bifurcation of left pulmonary artery with extension into the left upper lobe branches and left descending pulmonary artery. Small areas of consolidation with central Halo is also noted in the adjacent left lower lobe.

Thrombus is also noted in the left atrium and in the visualised part of infra renal IVC causing >50% 0f luminal occlusion.

Thorax

A peripheral wedge shaped consolidation with peripheral as well as central ground glass opacity giving rise to halo/atoll sign is noted in the right middle lobe- infarct.

Small areas of consolidation with central halo are also noted in the adjacent left lower lobe.

There is mild right pleural effusion. Trachea, Bronchi and esophagus, chest wall all normal.

Impression: Diffuse severe Pulmonary thromboembolism with arears of lung infarct.

IVC and Right Atrial thrombi.

Other reports

HBsAg -ve, Anti HCV -ve ,HIV -ve

Thyroid profile normal

S. folic acid 6.32 ng/ml, S.vit B12 441 pg/ml , Hs Trop-I 74 ng/L

S. bilirubin 2.13 mg/dl, SGOT/AST 3145 U/L, SGPT/ALT 2725 U/L, S.Alk phosphatase 106U/L after treatment S. Bilirubin 1.37 mg.dl, SGOT 37 U/L ,SGPT 246 U/L

S.cholesterol(total) 115 mg/dl,HDL 19.10 mg/dl,S.LDL 61 mg/dl,S.VLDL 27.04 mg/dl,S.TG 135 mg/dl

Urine routine normal, NT-PROBNP 3749 pg/ml, S. electrocyte normal, KFT normal,

Hb 8.4, (after BT 11.40 gm/dl), TLC 15.40×10^3/microlitre, platelet count 200×10^3/microlitre

PT/INR 10 /1.5

GBP – Moderate anisotosis, predominantly normocytic normochromic with few microcytic hypochromic and fair no. of polychromatophilic macrocytes. Neutrophilic leucocytosis. Platelets adequate.

Reticulocyte count 2.3 RBCs, absolute reticulocyte count 2.1, Reticulocyte index 1.41 (hypoproliferative) (Sysmex XN-1000,Sysmex XT- 2000/medonic M-series/Sysmex XNL 550/Alinti HQ)

S.LDH 4555 U/L, S. iron 12.61 µg/dl, UIBC 457 µg/dl, TIBC 470, S. ferritin 47 ng.ml (BECKMAN COULTER-AU480)

RBS 291 mg/dl

HBA1C 7.6% (broad D-10 Analyser)

PFT suggestive of normal with restriction and small airway involvement.

USG abdomen showing 17 cm, normal echotexture, GB and CBD normal, Portal vein normal, other organs normal except uterus which is bulky (~ 10.0× 6.0× 4.9 cm). A well-defined heterogeneously, hypoechoic lesion measuring 48×43mm with few internal cystic areas is seen in anterior myometrium and is seen pushing endometrial cavity posteriorly- intramural fibroid with cystic degeneration.

Venous color doppler study of B/L lower limbs- normal

Rarity of the case is that patient had no h/o hemoptysis, no evidence of DVT, age<50 years, No estrogen use, No prior DVT or PE, No unilateral leg swelling, No surgery/trauma requiring hospitalization within the preceding four weeks. But investigation depicted extensive pulmonary thromboembolism.

Discussion

Obtaining lower extremity ultrasound for the diagnosis of pulmonary embolism (PE) may not be a reliable method to check for pelvic venous thrombosis. Additionally, there are instances where patients have PEs, but no evidence of extremity thrombosis. According to a study,

the use of ultrasonography alone for suspected PE had a sensitivity of only 29%. [9]. In this case Venous doppler USG was normal.

It is important to note that false positive results can lead to unnecessary and potentially dangerous anticoagulation in patients who do not actually have pulmonary embolisms (PEs). Nowadays, helical CT angiography is the preferred method for diagnosis as it is quick, allows for direct visualization of PE, and can also identify other conditions in patients without PE. However, in patients who cannot receive intravenous contrast dye, a V/Q scan is used. A normal lung scan can effectively rule out the diagnosis of PE, but only 25% of patients with suspected PE have a normal scan. In the PIOPED II trial, which is one of the largest studies evaluating V/Q scanning, it was found that V/Q scans can effectively rule out PE in patients with a very low probability scan and a very low clinical likelihood of PE.EKG findings are often non-specific, as tachycardia is frequent. Evidence of right heart strain on EKG or echocardiography may support the diagnosis as well as provide information about the severity of the PE. One finding on echocardiogram, known as the McConnell's sign, is strongly suggestive of PE. In those patients with suspected PE and right ventricular dysfunction, the finding of normal wall motion of the RV apex but akinesia of the mid-RV free wall has a positive predictive value of 71% for the diagnosis of PE and a negative predictive value of 96%.[10]

Venous thromboembolism (VTE) and pulmonary embolism (PE) are still major causes of illness and death. However, by combining patient symptoms, clinical suspicion, and scoring systems, diagnosis can be made more efficiently, and unnecessary treatment can be avoided. Nowadays, more physicians have access to portable ultrasound devices, which can help in the early detection and treatment of VTE. However, in-hospital patients, especially those who are critically ill, can still pose diagnostic challenges. In such cases, clinical scoring systems and imaging may not provide conclusive results. Nevertheless, the accuracy of helical CT scans has improved our ability to detect PE in many of these patients.

The reversed halo sign (RHS) is characterised by a central ground-glass opacity surrounded by a more or less complete ring of consolidation on high-resolution CT (HRCT) [11]. It has also been described as the “atoll sign” because of its resemblance to a coral atoll [12]. First reported in cryptogenic organising pneumonia, it was initially thought to be specific for this disease [13] but was subsequently described in a variety of pulmonary diseases [14,15]. Despite being no longer considered specific, its presence in association with ancillary CT findings and the clinical history can be useful in narrowing the differential diagnosis. In this article, we present the spectrum of neoplastic and non-neoplastic diseases that may show the RHS and present clues that are helpful in the differential diagnosis. When the RHS is recognised on CT, by integrating the ancillary radiological and clinical data, the radiologist should be able to reach a succinct differential diagnosis to guide clinical decision making and/or determine treatment options.

Treatment

Those without contraindications to systemic anticoagulation, parenteral anticoagulation with subsequent conversion to vitamin K antagonists is the mainstay of therapy. Patients who present to the emergency room with acute PE have decreased mortality if anticoagulation treatment commences in the emergency room, rather than waiting until after admission.[16]

Supportive care of hypoxemia and hemodynamic instability should be instituted. Hemodynamically unstable patients may benefit from fibrinolytic therapy.

Newer options for anticoagulation include oral factor Xa inhibition with agents under investigation, such as rivaroxaban. [17,18]

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