CASE REPORT

24(105), September - October, 2020

ISSN 2321–7359 EISSN 2321–7367

Medical Science

Status epilepticus presenting feature of pulmonary embolism: Rarest of rare combination

Charan Bagga[1]✉, **Iftekhar Ansari**[1], **Amrutha Garikapathi**[1], **Irshadvs**[1], **Srinivas Naik**[1], **Sachin Agrawal**[2], **Sunil Kumar**[3]

[1]Post Graduate Resident, Department of Medicine, Jawaharlal Nehru medical college, DattaMeghe Institute of Medical Sciences (Deemed to be University), Wardha, Maharashtra, India
[2]Professor, Department of Medicine, Jawaharlal Nehru medical college, DattaMeghe Institute of Medical Sciences (Deemed to be University), Wardha, Maharashtra, India
[3]Professor and Head, Department of Medicine, Jawaharlal Nehru medical college, DattaMeghe Institute of Medical Sciences (Deemed to be University), Wardha, Maharashtra, India

✉**Corresponding author:**
Post Graduate Resident, Department of Medicine, Jawaharlal Nehru medical college, DattaMeghe Institute of Medical Sciences (Deemed to be University), Wardha, Maharashtra, India; Email: drcharanbagga@gmail.com

Article History
Received: 03 July 2020
Reviewed: 04/July/2020 *to* 16/August/2020
Accepted: 17 August 2020
E-publication: 24 August 2020
P-Publication: September - October 2020

Citation
Charan Bagga, Iftekhar Ansari, Amrutha Garikapathi, Irshadvs, Srinivas Naik, Sachin Agrawal, Sunil Kumar. Status epilepticus presenting feature of pulmonary embolism: Rarest of rare combination. *Medical Science*, 2020, 24(105), 3277-3280

ABSTRACT

Generalized seizures secondary to underlying pulmonary embolisim is rare phenomenon. Pulmonary embolism is a significant cause of mortality due to difficulty in diagnosis especially in absence of any obvious clues, as its presentation ranges from symptomless to

sudden death. Here by presenting a case of 49-year-old male who presented with continuous episodes of generalized tonic clonic seizure for 30 minutes, due to pulmonary embolism associated with Hyperhomocysteinemia.

Keywords: Seizure, Pulmonary embolism, Hyperhomocysteinemia.

1. INTRODUCTION

A thrombus in any of the branch of pulmonary artery can be regarded as pulmonary embolism (PE). Its origin is somewhere in the venous system (Pichereau et al., 2015). Frequently, patients with Pulmonary Embolism had no history of findings of Deep Vein Thrombosis because the clot has already embolized to the lungs. Risk factors of Pulmonary embolism are history of PE in past, Known Hyper Coagulable Disorder, Trauma, Surgery, Pregnancy, Immobilization, obesity, Cigarette use, Cancer, Hormone Replacement Therapy and Oral Contraceptives. However, in 30% of PE patients there are no detectable provoking factors (Kumar et al., 2016; Heit et al., 2000). Diagnosing pulmonary embolism is a very tricky situation as it mimics various clinical conditions such as breathlessness, cough, chest pain, hepatic congestion which has multiple causes. Most common physical findings can be tachycardia, fever, abnormal findings on pulmonary examination and collapse of the peripheral vessels. The tests commonly used for its diagnosis is D-dimer which has low sensitivity and specificity, while high cost of pulmonary angiography makes the things more confusing (Heit et al., 2000; Pineda et al., 2001). Generalized seizure as first presentation of pulmonary embolism is very rare, only documented in few case reports (Kumar et al., 2016). Hereby, we are reporting a case of a 49-year-old male who had presented with generalized tonic clonic seizure that had lasted for 30 minutes (Status Epilepticus), due to pulmonary embolism without any common risk factors, and on investigation was associated with Hyperhomocysteinemia.

2. CASE

A 49 year old male was brought to the casualty by wife with complaints of loss of consciousness and continuous episodes of generalized tonic clonic seizure since 30 minutes. There was no history of fall, trauma, surgery, vomitting, cough, hemoptysis, chronic disease or addiction, Deep Vein Thrombosis, seizure disorder, psychiatric illness, medication. On asking leading questions he revealed that there was sudden onset of chest pain, palpitation, breathlessness followed by continuous episodes of generalized tonic clonic seizure. He was non alcoholic and nonsmoker. He was not on any oral anticoagulants medication. On general examination, patient was afebrile, His blood pressure was 110/70 mmHg, pulse of 120/min and respiratory rate was 24/min with oxygen saturation of 94% on room air. CVS examination was normal with rapid heart sounds and no clinical murmur. On respiratory system examination, chest was bilateral clear with no adventitious sounds. On CNS examination, patient was drowsy and responding to verbal commands with normal power, tone, exaggerated deep tendon reflexes and bilateral plantar flexion.

Laboratory tests were done in which Prothrombin time: - 14.4 (INR: 1.15) and an activated partial thromboplastin time of 30.1 seconds (control: 30 seconds) Complete Blood Count, Kidney function test, Liver function test and serial cardiac biomarkers was within normal range. The ECG showed sinus tachycardia with right axis deviation, and T-wave inversion in V1-V5, incomplete right bundle branch block, without excitation abnormalities. Chest X-ray showed the diaphragm in normal and regular, no evidence of pneumonia, no mediastinal widening, normal sized heart, aorta was also normal. His Brain magnetic resonance imaging and electroencephalography was unremarkable. Two dimensional echocardiography was done which was suggestive of Ejection Fraction of 42%, dilated right atrium and right ventricle, without any regional wall motion abnormality (Figure 1).

As there was no intracranial or cardiac clue was found, D dimer was sent, which was found to be positive. Complete blood count was done in which HB was 13.5 gm%, WBC was 13,200 platelets were 2.8 lakhs, lymphocytes were 12, Liver functional test and kidney function test were normal. Fasting lipid profile was done in which total cholesterol was 200, triglycerides was 207,dHDL was 20,LDL was139,VLDL was 41,Serumhomocysteine (level) was elevated.Contrast enhanced computed tomography of thorax with pulmonary angiography revealed partial to complete pulmonary embolism of right middle and distal pulmonary artery and left distal pulmonary artery with embolus extending into right lower lobar pulmonary artery and its segmental branches and on left side embolus was extending into lingular lobar and lower lobar segments (Figure 2). His Arterial and venous Doppler of the lower limb was normal. Carotid Doppler was also normal. After heparinization patient received infusion of intravenous heparin in the intensive care unit and was overlapped with warfarin with a target of international normalized ratio (INR) of 2 to 3 and anti-epileptics.was started.

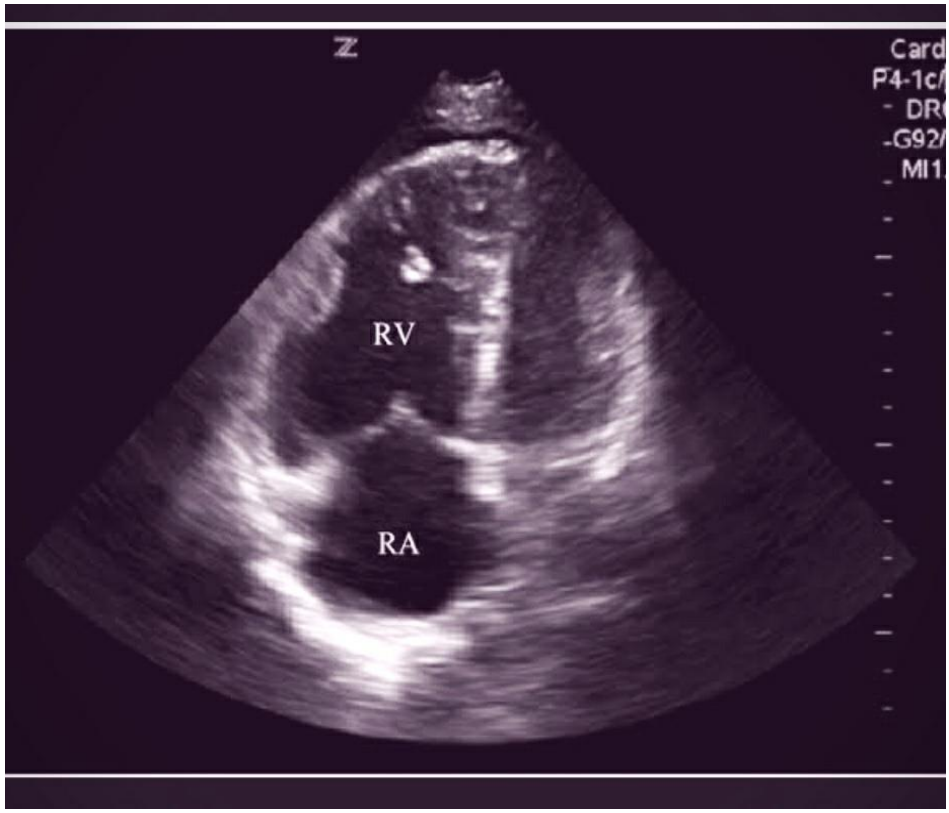

Figure 1 2D Echo suggestive of Dilated right atrium and right ventricle, without any regional wall motion abnormality (April 2020)

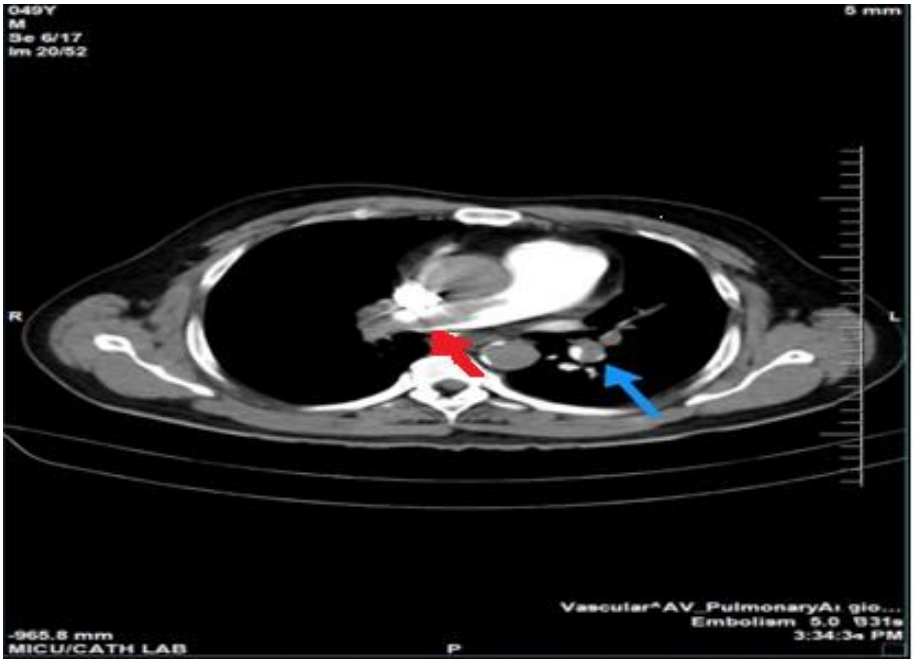

Figure 2 Right sided arrow (Red) showing partial to complete pulmonary embolism of right middle and distal pulmonary artery with extension of the embolus into the right lower lobar pulmonary artery and its segmental branches. Left sided arrow (Blue) showing almost complete pulmonary embolism involving the left distal pulmonary artery with extension of the embolus into the lingular and lower lobar pulmonary artery (April 2020)

3. DISCUSSION

We detailed an unusual case of a male patient with PE, which was eventually demonstrated to be related with Hyperhomocysteinemia on the grounds that it was raised and was the just a single positive finding which can be observed. In addition common signs of PE, for example, palpitation and dyspnea along with the patient gave an atypical symptom of seizure. This

demonstrated potential relationship with these 3 disease manifestations. There has been a sign towards a huge connection among's hyper homocysteinemia and cardiovascular disease and its complications for example, coronary failures and strokes (Goldhaber & Bounameaux, 2012). It is accepted that hyperhomocysteinemia prompts endothelial cell damage, decrease in the flexibility of vessels, and modifies the procedure of hemostasis. Research has demonstrated towards a connection between moderately raised homocysteine levels and the danger of CVD (coronary, heart, cerebrovascular and peripheral artery diseases) (Kapoor, 2010). It has been noticed that PE can be related with seizure (Goldhaber & Bounameaux, 2012; Kapoor, 2010). The pathophysiology hidden PE-related seizure proposes that massive PE causes transient right ventricular failure and diminished cardiac output, prompting transient global cerebral hypoperfusion. In expansion, massive PE with respiratory disappointment results in hypoxemia and acidosis, which is possible contributor of seizures. In our case, PE can be clarified by pulmonary vascular thrombotic event, thrombosis was not found in brain imaging. This perception demonstrated that the seizure in our patient may have been seen due to massive PE, a non-thrombotic presentation of Hyperhomocysteinemia.

4. CONCLUSION

This case highlights about awareness of atypical presentation of PE and seizure due to increased level of homocysteine. Moreover, physicians should consider hyperhomocysteinemia while surveying the possible underlying diseases in patients presenting with PE and seizure, even in a young man.

Informed consent

Informed consent was obtained from participant included in the study. Additional informed consent was obtained from participant for whom identifying information is included in this manuscript.

Conflict of interest

The authors declare that they have no conflict of interest

Funding

This research received no external funding

Data and materials availability

All data associated with this study are present in the paper.

Peer-review

External peer-review was done through double-blind method.

REFERENCES AND NOTES

1. Goldhaber SZ, Bounameaux H. Pulmonary embolism and deep vein thrombosis. Lancet. 2012; 379:1835-46.
2. Heit JA, Silverstein MD, Mohr DN, Petterson TM, O'Fallon WM, Melton LJ. Risk factors for deep vein thrombosis and pulmonary embolism: a population-based case-control study. Arch Intern Med. 2000;160:809–815.
3. Kapoor VK. Venous thromboembolism in India. Natl Med J India. 2010;23:193-5.
4. Kumar Sunil, Shashiraj Lahoti, Prasad Gurjar, Nitin Reddy. Hyperventilation: aura for absence seizure? European Journal of Biomedical AND Pharmaceutical sciences 2016; 3(1): 269-271.
5. Pichereau C, Maury E, Monnier-Cholley L, et al. Post-mortem CT scan with contrast injection and chest compression to diagnose pulmonary embolism. Intensive Care Med. 2015; 41:167–168.
6. Pineda LA, Hathwar VS, Grant BJ. Clinical suspicion of fatal pulmonary embolism. Chest. 2001;120:791–795