

Management of Thromboembolism (Pulmonary Embolism) in Coronary Cardiac Unit

Faiza Naeem* and Saleha Sadeeqa

Institute of Pharmacy, Lahore College for Women University, Pakistan

***Corresponding author:** Faiza Naeem, Institute of Pharmacy, Lahore College for Women University, Lahore, Pakistan, Email: faizanaeem12@hotmail.com

Case Report

Volume 3 Issue 3

Received Date: July 01, 2019

Published Date: July 16, 2019

DOI: 10.23880/vij-16000215

Abstract

Pulmonary embolism and deep vein thrombosis is considered thromboembolism which is the occlusion of blood vessels by thrombus that has fragmented away from its site of formation. Pulmonary embolism can be fatal which occurs when the Deep vein thrombosis breaks free from wall of a vein and blocks some or all of the blood supply to the lungs. Clinical presentations are pleuritic chest pain, rapid breathing, increased heart rate of patient and shortness of breath. It involves hypercoagulability, circulatory stasis and endothelial damage causing stagnation of blood flow which can be diagnosed by CT angiography. Computed tomography, or CT angiography is most often used but sometimes ventilation-perfusion lung scan is also used. Recommended treatment involves anticoagulant therapy, thrombolytics and surgery. In this case report, referred patient of 42 years old male is suffering from pulmonary embolism having chest pain along with diabetes and he was treated by thrombolytic therapy as well as by life style modifications which finally stabilizes his chest pain and condition of pulmonary embolism.

Keywords: Pulmonary Embolism; Hypercoagulability; Venous Stasis; Occlusion

Abbreviations: VTE: Venous Thromboembolism; PE: Pulmonary Embolism; DVT: Deep Vein Thrombosis.

Introduction

Venous thromboembolism (VTE) including pulmonary embolism (PE) & Deep vein thrombosis (DVT) is the major source of morbidity as well as higher mortality rate [1]. Primary pulmonary thrombi commonly occur in upper lobe of human lungs than the lower lobes, although thromboemboli either small or large can be more numerous in lower lobes of lung if they happen [2].

Multiple studies showed reported rates for pulmonary embolism recurrence ranging from 2 % to 50 % [3].

Epidemiologic prospective data evaluated a frequency of nearly 4% pulmonary thromboembolism. In clinical manifestation, unexplained dyspnea persistent nature, greater perfusion defects are also seen. Parenchymal signs including scars, mosaic pattern for perfusion, bronchial anomalies and focal opacities are also evaluated [4]. VTE is 3rd prominent disease associated with cardiovascular system in United States. Thrombosis frequency increases along with age and 2 folds higher in people older than 65 yrs [5].

Risk factor for venous thrombosis development as well as propagation includes increased coagulability, venous stasis and endothelial injury. Smoking will increase the plasma fibrinogen level and it will activate the pathway for intrinsic coagulation by anoxia or endothelial damage. Diabetes, physical inactivity and obesity all are connected with greater coagulation and lower fibrinolytic potential which can cause venous stasis. Lipids, predominantly elevated lipoprotein or triglycerides (TGs), increases coagulation and interact with fibrinolytic cascades [6]. Venous stasis has shown to improve the formation and propagation of intravascular deep vein thrombosis (DVT) [7].

Spiral CT angiography evidenced as effective in the recognition of PE in pulmonary arteries up to segmental level [8]. Treatment with heparin and streptokinase or heparin alone (both thrombolytic therapies) can decrease the death rate of acute stage of massive pulmonary embolism [9]. Anticoagulation therapies are capable of improving the patient outcome. Thrombolytic therapy in acute stage and use of inferior vena cava filters both are effective in treatment of PE [10]. Patients diagnosed with chronic stage of PE are more prone to

surgical therapeutic interventions [11].

Case Presentations

A 42-year male having 66kg weight presents with complains of Chest pain and nausea. He was suffering from Chest pain from 3 hours along with rapid breathing. He smokes 7-8 cigarettes per day. He frequently uses fried food in his diet.

Past Medical History

Patient was suffering from Headache and diabetes type 2 since 1 year.

Past Medication History

He was using Panadol 500mg for headache and vildose (vidagliptin) 50mg once daily for treatment of diabetes since 1years.

Lab Findings

Troponin level is raised and pulmonary embolism is confirmed by CT pulmonary angiography. Other parameters of patient profile are tabulated in Table 1.

Tests	Normal Range	Unit	Values	Comments
WBCs	4-11	$\times 10^3/\mu\text{L}$	12.7	Above Normal
Total RBC Count	4.0-5.5	$\times 10^6/\mu\text{L}$	4.5	Normal
Hemoglobin	13-17 (Male)	g/dL	13	Normal
Plateles	150-400	$\times 10^3/\mu\text{L}$	230	Normal
HCT (PCV)	40-75	%	40	Normal
MCV	20-45	fl	89	Normal
MCH	1-20	Pg	29	Normal
MCHC	65-110	%	33	Normal

Table 1: Complete blood count (CBC) of patient.

Vital signs

Blood pressure: 120/80mmHg

Pulse: 100 beats per min

Temperature: 98°F

Brands	Generics	Dose	Frequency	Indications
Sustac	Glyceryl Trinitrate	2.6mg	BD	Chest pain
Merol	Metoprolol	25mg	BD	Stabilizes rapid breathing
Nadroparin	LMW Heparin	5000 IU	8Hourly	Thromolysis
Noclot	Clopidogrel	75 mg	O.D	For preventing strokes, heart attacks
Dispirin	Aspirin	300mg	OD	Blood thinning

Table 2: Medication therapy.

Pharmacist Intervention

Drug Related Intervention

- Add metoclopramide 10mg in therapy for the treatment of nausea.
- There is no need to prescribe clopidogrel, although patient is using LMW heparin for thrombolytic effect, so skip clopidogril from medication therapy.

Life Style Modification

- Life style modification especially smoking cessation is recommended for this patient.
- Monitor blood glucose level regularly for good diabetic control
- Reduce fried food intake and use unsaturated fats in diet.
- Take healthy balanced diet having low cholesterol.

Outcomes

Chest pain of patient was relieved after using Glyceryl Trinitrate (Sustac) and recurrence of thrombus formation was reduced by life style modification especially by smoking cessation and reduced fried food intake.

Discussion

Pulmonary embolism remains an imperative medical problem with greater mortality rate [12]. Venous thromboembolism (VTE) is a blood clot in veins which is highly predominant health problem causing disability and raising the death rate. This patient was complaining about the chest pain which was due to blockage by embolus in pulmonary region. He was using fried food more frequently and he smokes 7-8 cigarettes per day. Both of these are contributing etiologic factors for development of pulmonary embolism. Most of the studies showed the effect of smoking on function of platelets resulting in the thrombus formation [13]. Lipids or lipoproteins accelerate the thromboembolism by prostaglandins which are synthesized by fatty acids resulting in activation of platelet and coagulation cascade [14].

Symptomatically, Patient was treated with thrombolytic drugs and further life style modifications. Thrombolytic drugs have shown proper effect in patient for thrombolysis and relieving the chest pain by Glyceryl Trinitrate (sustac). Life style modifications including stoppage of smoking and fried food intake can help this patient from recurrence of thromboembolism. Metoclopramide 10mg was added to his treatment for

nausea by pharmacist. As patient is suffering from co morbidity as diabetes so it is mandatory to evaluate the glucose level. As regular glucose monitoring showed maintained glucose level after use of vildagliptin 50mg. So, the patient is managed properly by doctor's recommendations as well as by pharmacist interventions.

Conclusion

In this case history, the patient was suffering from PE and DM type 2. Now-a-days, PE is leading cardiovascular event so aim was to treat chest pain and embolus formation in pulmonary region. He was treated with thrombolytic therapy. Patient was stable after treatment with thrombolytic agents.

References

1. Tsai AW, Cushman M, Rosamond WD, Heckbert SR, Polak JF, et al. (2002) Cardiovascular risk factors and venous thromboembolism incidence: the longitudinal investigation of thromboembolism etiology. *Archives of internal medicine* 162(10): 1182-1189.
2. Wagenvoort CA (1995) Pathology of pulmonary thromboembolism. *Chest* 107(1): S10-S17.
3. Carson JL, Kelley MA, Duff A, Weg JG, Fulkerson WJ, et al. (1992) The clinical course of pulmonary embolism. *New England Journal of Medicine* 326(19): 1240-1245.
4. Castaner E, Gallardo X, Ballesteros E, Andreu M, Pallardo Y, et al. (2009) CT diagnosis of chronic pulmonary thromboembolism. *Radiographics* 29(1): 31-50.
5. Cushman M, Tsai AW, White RH, Heckbert SR, Rosamond WD, et al. (2004) Deep vein thrombosis and pulmonary embolism in two cohorts: the longitudinal investigation of thromboembolism etiology. *The American journal of medicine* 117(1): 19-25.
6. MacCallum PK, Meade TW (1999) Haemostatic function, arterial disease and the prevention of arterial thrombosis. *Best Practice & Research Clinical Haematology* 12(3): 577-599.
7. Symington IS, Stack BH (1977) Pulmonary thromboembolism after travel. *British journal of diseases of the chest* 71(2): 138-140.

8. van Rossum AB, Pattynama PM, Ton ER, Treurniet FE, Arndt JW, et al. (1996) Pulmonary embolism: validation of spiral CT angiography in 149 patients. *Radiology* 201(2): 467-470.
9. Jerjes-Sanchez C, Ramírez-Rivera A, de Lourdes García M, Arriaga-Nava R, Valencia S, et al. (1995) Streptokinase and heparin versus heparin alone in massive pulmonary embolism: a randomized controlled trial. *Journal of thrombosis and thrombolysis* 2(3): 227-229.
10. Kanter B, Moser KM (1988) The Greenfield vena cava filter. *Chest* 93(1): 170-175.
11. Moser KM, Daily PO, Peterson K, Dembitsky W, Vapnek JM, et al. (1987) Thromboendarterectomy for chronic, major-vessel thromboembolic pulmonary hypertension: immediate and long-term results in 42 patients. *Annals of internal medicine* 107(4): 560-564.
12. Goldhaber SZ, Visani L, De Rosa M (1999) Acute pulmonary embolism: clinical outcomes in the International Cooperative Pulmonary Embolism Registry (ICOPE). *The Lancet* 353(9162): 1386-1389.
13. Hawkins RI (1972) Smoking, platelets and thrombosis. *Nature* 236(5348): 450.
14. Goodnight Jr SH, Harris WS, Connor WE, Illingworth DR (1982) Polyunsaturated fatty acids, hyperlipidemia, and thrombosis. *Arteriosclerosis: An Official Journal of the American Heart Association, Inc* 2(2): 87-113.

