Concurrent massive hemoptysis and acute pulmonary embolism: A therapeutic dilemma

Sharad Joshi, Ankit Bhatia, Nitesh Tayal, Ritu Verma, Dheeraj Nair

Department of Pulmonary Medicine, Max Super Speciality Hospital, Vaishali, Ghaziabad, India
Interventional Radiology, Max Super Speciality Hospital, Vaishali, Ghaziabad, India
Department of Emergency Medicine, Max Super Speciality Hospital, Vaishali, Ghaziabad, India

Keywords: Hemoptysis, Pulmonary embolism, Bronchial artery embolism

Abstract

Pulmonary Embolism and Massive hemoptysis are two very potentially fatal emergencies in Respiratory medicine practice. These two conditions are kind of antagonizing conditions requiring completely different and pharmacologically opposite nature of treatment.

We hereby present the case of a 37-year old young male presented to our Hospital with massive hemoptysis, who on evaluation also had a concurrent large pulmonary embolism. The bleed was managed with bronchial artery embolization followed by anticoagulation therapy from a day later for embolism. This case report gives an insight on to how to manage a practical therapeutic challenge which is the concurrence of a massive hemoptysis and life threatening pulmonary embolism.

1. Introduction

The development of pulmonary Thromboembolism in a patient presenting with massive hemoptysis is a highly testing clinical situation in view of competing priorities between the need for anticoagulation to prevent further thrombosis and the concern of pulmonary haemorrhage turning to being potentially disastrous.

Patients presenting with Submassive Pulmonary Embolism can progress to Pulmonary Infarction in 10%–20% patients which can result in hemoptysis in 5%–7% of patients [1]. The resultant ischemic parenchymal necrosis in pulmonary embolism is thought of behind the haemoptysis in these patients [2].

Anticoagulation is needed in the case of hemoptysis since it increases the chances of bleeding.

We present a report of a 37-year old male who presented to the emergency with a massive hemoptysis who on further evaluation was found to have a simultaneous pulmonary embolism. This case will demonstrate the strategy employed to tackle both the complications.

2. Case history

A 37-year-old smoker male, presented to the emergency complaining of blood in sputum throughout the night. He reported having expectorated 4 to 5 times frank blood, each episode approximately 50–100 ml in quantity. He denied any other symptoms, breathlessness, chest pain or fever. On his past records he reported having taken anti-tubercular treatment 5 years back for pulmonary tuberculosis.

His vital parameters were Heart rate- 120/min, Blood pressure- 120/80 mm Hg, Respiratory rate- 32/min, Oxygen saturation-98% on room air. Systemic examination was essentially normal except for coarse crepitations over right infrascapular region. Chest radiograph PA was done that was essentially normal (Fig. 1).

He was admitted and administered intravenous antibiotics (amoxicilline-clavulanic acid), inhaled oxygen, cough suppressants and intravenous tranexamic acid.

Echocardiography was done which showed no regional motion defects, ejection fraction of 55%, Right ventricular systolic ejection pressure of 70 mm Hg, Respiratory rate- 32/min, Oxygen saturation-98% on room air. Systemic examination was essentially normal except for coarse crepitations over right infrascapular region. Chest radiograph PA was done that was essentially normal (Fig. 1).

He was admitted and administered intravenous antibiotics (amoxicilline-clavulanic acid), inhaled oxygen, cough suppressants and intravenous tranexamic acid.

Echocardiography was done which showed no regional motion defects, ejection fraction of 55%, Right ventricular systolic ejection pressure of 70 mm Hg and ECG showed S1Q3T3 complex (Fig. 2). Due to continued hemoptysis CT aortogram was planned for arterial mapping. CT revealed minimal right upper lobe bronchiectatic changes with some fibrosis. Right sided bronchial arteries were grossly hypertrophied. Diffuse ground glass opacities were noted in right middle and lower lobe likely due to aspirated blood. In addition there was a saddle thrombus in the main pulmonary trunk prolapsing into origin of left pulmonary artery and there was occlusive thrombus in right pulmonary artery.

* Corresponding author. Max Superspeciality Hospital, Vaishali, Ghaziabad, Uttar Pradesh, 201012, India.
E-mail address: ankitbhatia85@gmail.com (A. Bhatia).

https://doi.org/10.1016/j.rmcr.2020.101337
Received 9 October 2020; Received in revised form 1 December 2020; Accepted 26 December 2020
Available online 3 January 2021
2213-0071/© 2020 Published by Elsevier Ltd. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).
Due to ongoing massive hemoptysis and hypertrophied bronchial arteries, patient was planned for Bronchial Artery Embolization (BAE) followed by anticoagulation for pulmonary thromboembolism (PTE). BAE was done with 300–500 micron PVA (polyvinyl alcohol) particles (Fig. 6). No extrabronchial systemic arteries were found and complete obliteration was assured.

Work up for PTE was initiated, bilateral leg vein doppler was negative for deep vein thrombosis. Thrombophilia profile- Protein C, Protein S, Factor V, Antithrombin III, ANA were negative. D-Dimer was also found to be negative.

The patient soon after the BAE, developed blood in vomit in the Intensive care unit. The decision to start unfractionated heparin just after BAE was withheld. An Urgent Gastroenterology consult was taken and an Upper Gastrointestinal Endoscopy was done. Upper Gastrointestinal Endoscopy showed ingested blood and no evidence of ac other mucosal erosions.

Unfractionated heparin (UFH) infusion was started the next day and was continued for 48 hours. Patient did not have any further episodes of hemoptysis. A repeat Echocardiography was done which showed right ventricular systolic pressure of 45 mm Hg. UFH was switched over to Dabigatran 150mg twice daily, which he tolerated well. The patient was discharged soon after.

Fig. 1. Chest Radiograph AP view: Showing essentially normal lung parenchyma.

(Figs. 3–5).

Fig. 2. ECG: S1Q3T3 complex

Fig. 3. CT aortogram with soft tissue and lung windows : Axial section at the level of main pulmonary artery showing complete thrombus in right pulmonary artery and partial thrombus near left pulmonary artery origin (solid arrows).
Massive hemoptysis is defined as expectoration of large blood volumes within the feeding vessel as to achieve haemostasis deploying a variety of permanent materials (isobutyl-2-cyanoacrylate or glue, micro-particles, metallic coils or absorbable gelatin pledgets) within the feeding vessel to achieve haemostasis. Hemoptysis control ranges from 65 to 92% in various reports. The recurrence rate unfortunately remains frequent ranging from 10 to 55% for follow-up as long as 46 months. Particles (PVA) are preferred over glue as they reach distally till the inflamed pulmonary bed in the regions of arterio-pulmonary shunting. They also have better injection control that reduces the risk of non target embolization. Coil is also less preferred embolizing agent as they tend to block proximally and don’t go distal in the inflamed pulmonary bed thereby having high chances of recurrence.

Unfractionated heparin (UFH) given intravenously with six hourly monitoring of the activated partial thromboplastin time (aPTT) is the anticoagulation of choice when there is a risk of recurrent haemorrhage as it can be quickly stopped in the event of a life threatening bleed. We planned to start UFH 6 hours after the bronchial artery embolization, however patient vomited blood just after the procedure. We wanted to rule out concurrent gastrointestinal bleed before starting anticoagulation.

The patient presented to the hospital not only with hemoptysis but also with acute Pulmonary embolism. Anticoagulation is the management of choice in patients with Pulmonary embolism, it prevents both early mortality death and recurrence. However, anticoagulation is contraindicated in patients with severe hemoptysis as it may cause fatal haemorrhage in the airways. From the management perspective these two conditions are extremely opposite. In the case of hemoptysis, procoagulants enhancing coagulation are give. On the other hand for management of pulmonary embolism, anticoagulants are the mainstay of treatment. Therefore such an antithetical clinical scenario was a real management challenge and
this case may guide future management of such clinical corundrum.

4. Conclusion

This case gives an insight on how to manage a real Therapeutic problem for massive hemoptysis who at the same time was found to suffer from pulmonary embolism. This case report gives an answer on how to manage a real therapeutic challenge which is the concurrence of a massive hemoptysis and life threatening pulmonary embolism.

Declaration of competing interest

No conflict of interest.

References

[1] J. Belohlavek, V. Dytrych, A. Linhart, Pulmonary embolism, part I: epidemiology, risk factors and risk stratification, pathophysiology, clinical presentation, diagnosis and nonthrombotic pulmonary embolism, Exp. Clin. Cardiol. 18 (2013) 129–138. Medline, Google Scholar.

[2] Corey R. Hemoptysis. In: rd, HK Walker WD Hall JW Hurst , (eds). Clinical Methods: the History, Physical, and Laboratory Examinations. 1990. Butterworth-Heinemann, Boston, MA. Google Scholar.

[3] J.S. Earwood, T.D. Thompson, Hemoptysis: evaluation and management, Am. Fam. Physician 91 (2015) 243–249.

[4] K.E. Najarian, C.S. Morris, Arterial embolization in the chest, J. Thorac. Imag. 13 (1998) 93–104.

[5] W.H. Ibrahim, Massive haemoptysis: the definition should be revised, Eur. Respir. J. 32 (4) (2008) 1131–1132.

[6] A.S. Botenga, [Broncho-bronchial anastomosis A selective angiographic study], Ann. Radiol. 13 (1970) 1–16.

[7] J.L. Lordan, A. Gascoigne, P.A. Corris, The pulmonary physician in critical care * Illustrative case 7: assessment and management of massive haemoptysis, Thorax 58 (2003) 814–819.

[8] E.I. Hsiao, C.M. Kirsch, F.T. Kagawa, J.H. Wehner, W.A. Jensen, R.B. Baxter, Utility of fiberoptic bronchoscopy before bronchial artery embolization for massive hemoptysis, AJR Am. J. Roentgenol. 177 (4) (2001) 861–867.

[9] J.L. Lordan, A. Gascoigne, P.A. Corris, The pulmonary physician in critical care * Illustrative case 7: assessment and management of massive haemoptysis, Thorax 58 (2003) 814–819.

[10] C.A. Moen, A. Burrell, J. Dunning, Does tranexamic acid stop haemoptysis? Interact. Cardiovasc. Thorac. Surg. 17 (2013) 991–994.

[11] A. Khalil, B. Fedida, A. Parrot, Severe hemoptysis: from diagnosis to embolization, Diagn. Interv. Imag. 96 (2015) 775–788.

[12] D.R. Sokko, T.P. Smith, Bronchial artery embolization for hemoptysis, Semin. Intervent. Radiol. 28 (1) (2011 Mar) 48–62, https://doi.org/10.1055/s-0031-1273940. PMID: 22379276; PMCID: PMC3140255.

[13] C. Kearon, E.A. AKL, J. Orencio, Antithrombotic therapy for VTE disease: CHEST guideline and expert panel report, Chest 149 (2016) 315–352.