



# Unusual Presentation of A Massive Pulmonary Embolism

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Received 20 January 2010; Accepted 23 May 2010

## Abstract

A pulmonary embolus clogs the artery that provides blood supply to a part of the lung. The embolus not only prevents the exchange of oxygen and carbon dioxide but also decreases blood supply to the lung tissue itself, potentially causing the tissue to die (infarct). A 52-year-old man presented with syncope and anxiety. He had sinus tachycardia and dilated right ventricle with trabeculations. A differential diagnosis of arrhythmogenic right ventricular dysplasia and pulmonary embolism prompted CT angiography, which revealed a bilateral massive pulmonary embolism. The patient was treated successfully with thrombolytic drugs.

*J Teh Univ Heart Ctr 2011;6(1):41-44*

**This paper should be cited as:** Safi M, Tajik Rostami R, Taherkhani M. Unusual presentation of a massive pulmonary embolism. *J Teh Univ Heart Ctr 2011;6(1):41-44.*

**Keywords:** Pulmonary embolism • Tachycardia, ventricular • Arrhythmogenic right ventricular dysplasia • Syncope

## Introduction

The etiology of about one third of all cases of syncope is unknown. Be that as it may, 5% of all cases of syncope are usually of cardiac origins and are thus known as cardiac syncope. Pulmonary embolism is a relatively rare cause of cardiac syncope, which may explain why it is frequently ignored.<sup>1</sup> The variability of presentation sets the patient and clinician up for potentially missing the diagnosis. The challenge is that the “classic” presentation with abrupt onset of pleuritic chest pain, shortness of breath, and hypoxia is rarely the case. Studies of patients who die unexpectedly of pulmonary embolism reveal that they complained of nagging symptoms often for weeks before death related to pulmonary embolism. Forty percent of these patients had been seen by a physician in the weeks prior to their death.<sup>2</sup> When syncope

is the presenting symptom of pulmonary embolism, it should raise the clinical suspicion for a massive clot burden or saddle embolism requiring prompt treatment.

## Case report

A 52-year-old man was referred to our hospital because of two episodes of syncope within a fortnight. He had suffered from pleuritic chest pain and dry coughs for the previous two weeks, and antibiotics prescribed for pneumonia by an internist failed to prevent the exacerbation of the symptoms. The internist subsequently referred the patient to a cardiologist, who subjected him to an exercise stress test (to rule out coronary artery disease), during which he developed non-sustained ventricular tachycardia. The following day, he

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suffered a one-minute syncope while talking a walk in his yard. He was, therefore, once more referred to our hospital by the internist to evaluate ventricular tachycardia as a cause of syncope. When the patient arrived at our hospital, he had anxiety and had a respiratory rate of 30/min, pulse rate of 120/min, and supine blood pressure of 100/60 mmHg. Additionally, the patient's lungs were clear and his heart sound and lower limb were normal. Chest X-ray revealed mild cardiomegaly, dilated right descending pulmonary artery, and oligemia in the right lung (Figure 1).

On echocardiography, the left ventricle was normal; however, the right heart was dilated and trabeculated and there was also right ventricular dysfunction. The pulmonary artery pressure was elevated to 45 mmHg. The patient denied any history of deep vein thrombosis and mentioned only a six-hour car travel more than three weeks earlier. His family history for venous thromboembolism was weakly positive and one of his nephews had a history of pulmonary thromboembolism.

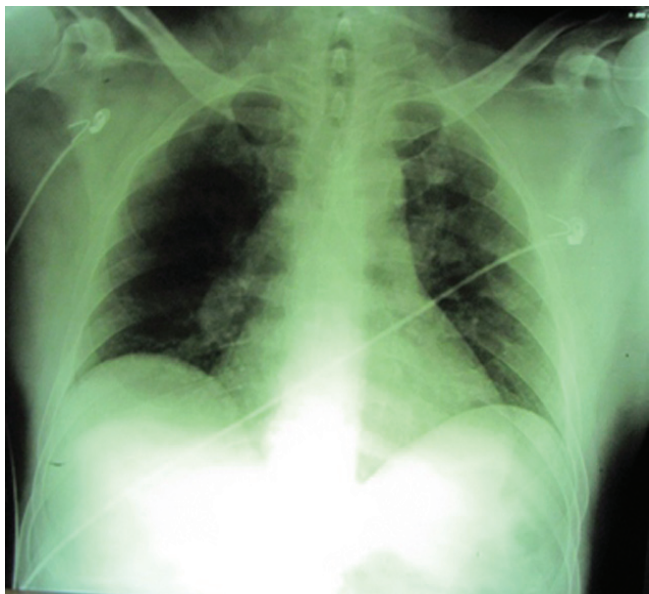


Figure 1. Chest X-ray revealing mild cardiomegaly, dilated right descending pulmonary artery, and oligemia in the right lung

At this point, lung CT angiography was performed to rule out pulmonary embolism as the main cause of acute right ventricular enlargement. The CT angiography demonstrated bilateral pulmonary artery thrombosis from the first division of the right and left pulmonary arteries to the distal ones (Figures 2 & 3).

With a diagnosis of pulmonary embolism, the patient underwent thrombolytic therapy with recombinant tissue plasminogen activator (r-TPA) 100mg over a 90-minute period, which brought about immediate improvement in the patient's condition. The following day, his symptoms improved dramatically, his respiratory rate decreased, his  $O_2$  saturation rose from 85% to 98%, and his blood pressure

remained stable in different positions.

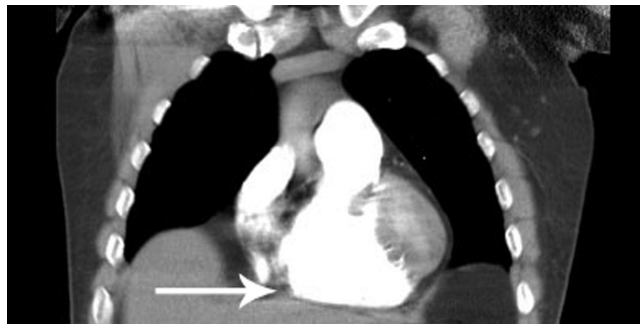


Figure 2. Dilation of right ventricle and pulmonary arteries (arrow)

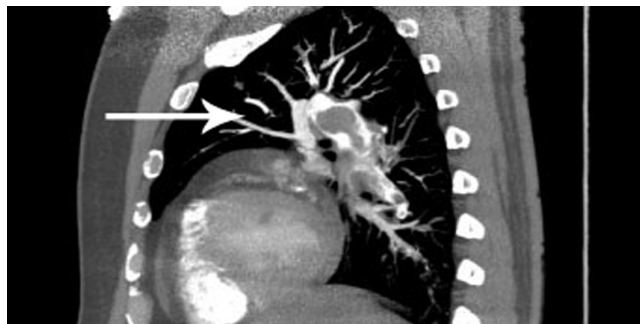


Figure 3. Obstruction of pulmonary arteries from proximal portion (arrow)

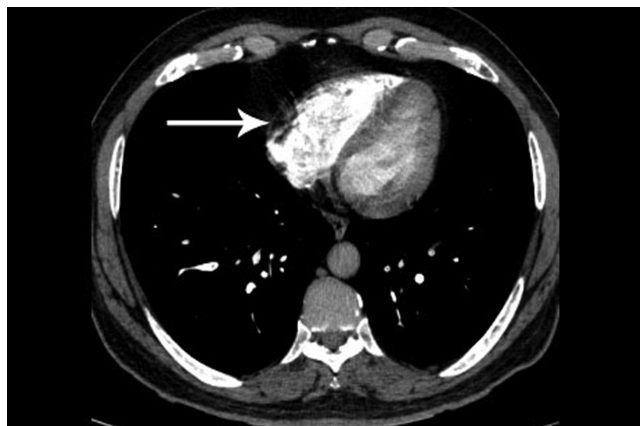


Figure 4. Decrease in the right ventricle size (arrow)



Figure 5. Decrease in pulmonary arteries size (arrow)



CT angiography showed improved right ventricular function and decreased right ventricular size and pulmonary arteries (Figures 4 & 5).

## Discussion

The diagnosis of acute pulmonary embolism is amongst the most challenging problems encountered in clinical practice.

Despite the awareness amongst physicians of the risks associated with a delay in the diagnosis of pulmonary embolism, it was not initially considered in this case because of the following reasons:

Firstly, syncope is an uncommon presentation of pulmonary embolism, occurring in only 14% of patients. The most common symptoms are dyspnea (82%), pleuritic chest pain (49%), and cough (20%).<sup>3</sup>

Secondly, the patient had no history of deep vein thrombosis. The exact number of the symptoms and signs of deep vein thrombosis in patients with a diagnosis of pulmonary embolism is not clear, but one study reported deep vein thrombosis in 62% of patients with pulmonary embolism.<sup>4</sup>

And thirdly, our patient had severe right ventricular enlargement with severe trabeculations. The occurrence of non-sustained ventricular tachycardia in the exercise test gave rise to other diagnoses, including arrhythmogenic right ventricular dysplasia, which justifies the syncope and right ventricular dilation.

Although arrhythmogenic right ventricular dysplasia is more common in younger patients, we considered it as one possible diagnosis because of the patient's having developed non-sustained ventricular tachycardia during the exercise test and his dilated right ventricle with trabeculations.<sup>5</sup> The patient experienced three episodes of presyncope in the CCU, but his rhythm during these episodes was sinus bradycardia. We concluded that ventricular tachycardia was not the mechanism of syncope/presyncope and performed CT angiography to confirm pulmonary embolism as the culprit for the syncope.

The patient had a weak family history of pulmonary embolism, hinting at the presence of hypercoagulable states such as activated PROTEIN C (aPC) resistance, factor V leiden, anti thrombin III deficiency, and protein C and S deficiency. For the evaluation of hypercoagulable states, family history remains the most rapid and cost-effective method of identifying a predisposition to venous thrombosis.<sup>6</sup>

Whereas management with anticoagulants alone is typically sufficient for low-risk patients, more aggressive treatments such as thrombolysis, embolectomy, and inferior vena cava (IVC) filters are recommended for higher-risk patients. Thrombolytic therapy should be considered in all patients with massive pulmonary embolism and hypotension

associated with deep vein thrombosis in the popliteal area or higher.<sup>7</sup> The main indications for thrombolytic therapy include ongoing hypoxia, respiratory distress, pulmonary hypertension, and right heart failure because thrombolytic therapy often achieves an impressive and almost an immediate clinical benefit in these clinical settings.<sup>8</sup>

The patient's right ventricular failure and progressive course prompted us to candidate him for thrombolysis. The other option in management would have been embolectomy, which is performed either with a catheter or during open heart surgery. Embolectomy via open surgery is reported to have improved the survival rate to 89% in twenty-nine emergent pulmonary embolism cases.<sup>9</sup> Although interventional catheter-based catheter fragmentation and suction embolectomy are also available for pulmonary embolectomy in some institutions, open surgical embolectomy is indicated in patients who have contraindications to thrombolytic therapy, persistence of thrombi in the right heart or pulmonary arteries after pulmonary embolism, or severe hemodynamic compromise with cardiovascular collapse. Early surgical treatment must also be considered in patients whose course deteriorates in spite of aggressive medical therapy depending on the series. The overall mortality rate after pulmonary embolectomy varies from 16% to 46%, with a mean mortality rate of 26%. These findings suggest that earlier surgical intervention may result in improved survival.<sup>10</sup>

We can conclude that surgical embolectomy is warranted in patients with severe hemodynamic instability on the basis of clinical impression, after other causes of hemodynamic collapse have been ruled out. An experimental catheter embolectomy used in cardiac catheterization appears promising.<sup>11</sup> IVC filters are an appropriate option for patients with high bleeding risks. The insertion rate of IVC filters has increased in recent years; this increase, however, has occurred without proven improved safety and efficiency.<sup>12</sup>

Another important point in the management of this patient was the optimal duration of anticoagulation. Despite the fact that the patient had a weak family history of hypercoagulable states, a proper course of action required his management as a case of idiopathic venous thromboemboli and with an indefinite duration of oral anticoagulant (warfarin) therapy.<sup>13</sup>

## References

1. Maguire A. Pulmonary embolism. In: Kochar S, ed. *Clinical Medicine for Students*. 5th ed. Boston: Lippincott Williams & Wilkins; 2008. p. 1357-1389.
2. Kline JA, Runyon MS. Pulmonary embolism and deep venous thrombosis. In: Marx JA, Hockenberger RS, Walls RM, eds. *Rosen's Emergency Medicine Concepts and Clinical Practice*. 6th ed. London: mosby; 2006. p. 1368-1382.
3. Goldhaber SZ, Visani L, De Rosa M. Acute pulmonary embolism: clinical outcomes in the International Cooperative Pulmonary Embolism Registry (ICOPER). *Lancet* 1999;353:1386-1389.



4. Köhn H, Mostbeck A, Bachmayr S, Eber O. <sup>99m</sup>Tc-DTPA aerosol for same-day post-perfusion ventilation imaging: results of a multicentre study. *Eur J Nucl Med* 1993;20:4-9.
5. Dalal D, Molin LH, Piccini J, Tichnell C. Clinical features of arrhythmogenic right ventricular dysplasia/cardiomyopathy associated with mutations in plakophilin-2. *Circulation* 2006;113:1641-1649.
6. Samuel Z, Goldhaber SZ. Pulmonary embolism. In: Libby P, Bonow R, Mann D, Zipes D, eds. *Braunwald's Heart Disease*. 8th ed. Philadelphia: Saunders; 2008. p. 1863-1879.
7. Linn BJ, Mazza JJ, Friedenberg WR. Treatment of venous thromboembolic disease. A pragmatic approach to anticoagulation and thrombolysis. *Postgrad Med* 1986;79:171-180.
8. Gallus AS. Thrombolytic therapy for venous thrombosis and pulmonary embolism. *Baillieres Clin Haematol* 1998;11:663-673.
9. Aklog I, Williams CS, Byrne JC, Goldhaber SZ. Massive pulmonary embolism. *Circulation* 2006;113:577-582.
10. Meyer G, Tamisier D, Sors H, Stern M, Vouhé P. Pulmonary embolectomy a 20-year experience at one center. *Ann Thorac Surg* 1991;51:232-236.
11. Kucher N, Windecker S, Banz Y, Schmitz-Rode T, Mettler D, Meier B, Hess OM. Percutaneous catheter thrombectomy device for acute pulmonary embolism: in vitro and in vivo testing. *Radiology* 2005;236:852-858.
12. Jaff MR, Goldhaber SZ, Tapson VF. High utilization rate of vena cava filters in deep vein thrombosis. *Thromb Haemost* 2005;93:1117-1119.
13. Ost D, Tepper J, Mihara H, Lander O, Heinzer R, Fein A. Duration of anticoagulation following venous thromboembolism: a meta-analysis. *JAMA* 2005;294:706-715.