

A Case Report Of Bilateral Pulmonary Embolism Presenting To Komfo Anokye Teaching Hospital, Kumasi.

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Citation

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Abstract

Venous thrombo-embolism has traditionally been considered rare in West Africa. Patients who present with unilateral leg swelling are therefore less likely to be evaluated for deep vein thrombosis; and this may lead to fatal consequences. The following case report illustrates a middle-aged man who developed unilateral left leg swelling and was wrongly treated initially. He was later admitted to the teaching hospital in Kumasi, and treated for bilateral pulmonary embolism.

INTRODUCTION

Pulmonary embolism is a common and often fatal disease¹⁻³. Acute pulmonary embolism is a common cause of death in hospital patients⁴. It is one of the commonest non-diagnosed or mis-diagnosed disease. Many of the symptoms and signs detected in patients with pulmonary embolism are common among patients without pulmonary embolism, making the diagnosis of pulmonary embolism difficult.

The difficulties in establishing an accurate diagnosis of pulmonary embolism is a great challenge. It is therefore confirmed ante-mortem in only approximately 30 percent of patients with the remaining 70 percent of cases diagnosed by autopsy. However, with thorough clinical examination and economic utilization of available investigative tools, the diagnosis of pulmonary embolism should be made ante-mortem.

Pulmonary embolism (PE) is associated with a mortality rate of approximately 30 percent without treatment, primarily the result of recurrent embolism. However, accurate diagnosis followed by effective therapy with anticoagulants decreases the mortality rate to 2 to 8 percent³⁻¹⁰.

It is estimated that 65 to 90 percent of cases of pulmonary embolism arise from thrombi in the deep venous system of the lower extremities; however, they may also originate in the pelvic, renal, or upper extremity veins and in the right heart. Most calf vein thrombi resolve spontaneously and only 20 to 30 percent extend into the proximal veins if untreated. Ilio-femoral thrombi are the source of most clinically recognized pulmonary embolism⁷⁻⁹. It is estimated

that 50 to 80 percent of iliac, femoral, and popliteal vein thrombi originate below the popliteal vein and propagate proximally.

After travelling to the lung, large thrombi may lodge at the bifurcation of the main pulmonary artery or the lobar branches and cause hemodynamic compromise. Smaller thrombi continue travelling distally and are more likely to produce pleuritic chest pain, by initiating an inflammatory response adjacent to the parietal pleura. Only about 10 percent of emboli cause pulmonary infarction, usually in patients with pre-existing cardio-pulmonary disease. Most pulmonary emboli are multiple, with the lower lobes being involved in the majority of cases⁶.

The clinical severity of pulmonary embolism can be highly variable, ranging from asymptomatic to severe hypoxemia and shock. As a result, therapy varies from patient to patient and requires considerable clinical judgment. For a patient in whom there is a high clinical suspicion of pulmonary embolism, empiric anticoagulant therapy should be initiated in addition to supportive care^{3,4,10,11}.

Patients in whom anticoagulation was initiated during resuscitation should remain anticoagulated during the diagnostic evaluation. Long-term anticoagulation is indicated if pulmonary embolism is confirmed.

Anticoagulation should be discontinued if pulmonary embolism is excluded. Thrombolysis should be considered once pulmonary embolism is confirmed. If thrombolysis is chosen, anticoagulation should be temporarily discontinued during the thrombolytic infusion. Patients should be

considered for embolectomy if thrombolysis is contraindicated or unsuccessful. Inferior vena caval filter placement should be considered if anticoagulation is contraindicated, fails, or causes complications such as severe bleeding.

HISTORY

On the 19th of July, 2010, a 46-year old research fellow presented to the medical emergency unit at the Komfo Anokye Teaching Hospital, Kumasi, Ghana, with two days history of shortness of breath at rest, He had developed a swelling of the left leg and foot three months earlier and had been treated at a private clinic with antibiotics. Some weeks before the admission he noticed an increasing easy fatigability, without chestpain, cough or haemoptysis.

He is not a known patient with hypertension, diabetes mellitus or dyslipidaemia but has been smoking about two packs of cigarette for many years and takes alcohol occasionally. He sits behind the computer working for long hours, in most days of the week.

EXAMINATION

On physical examination, he looked ill, dyspnoeic at rest, afebrile, not pale, and acyanotic. His left leg was swollen and slightly tender. The respiratory rate was 36 cycles/minute, the radial pulse was 96 beats/minute, regular and of small volume, and the blood pressure was 100/66 mmHg. The jugular venous pressure was not raised and the apex beat was at the 5th left intercostals space in the mid-clavicular line. The heart sounds were normal and no murmurs were heard. The chest, abdomen and the central nervous system were also normal.

INVESTIGATIONS

Figure 1 shows the electrocardiogram (ECG) of the patient on admission. It showed sinus rhythm with a heart rate of 93/minute, and the typical sign of S-wave in lead I, Q-wave in lead III, and inverted T-wave in lead III pattern.

Table 1 shows the patient’s blood biochemistry and haematology results. D-dimer assay was positive.

The patient’s chest X-ray and the chest CT scan are shown in figures 2 and 3 respectively. Serial axial enhanced chest CT scan showed complete filling defect of low attenuation in the left main pulmonary artery, and partial filling defects in the other proximal arteries. On the right there were partial marginal and central filling defects in the proximal

pulmonary arteries.

Doppler ultrasound of leg veins showed hypoechogenic mass filling the lumen of the left femoral vein at the level of the saphenofemoral junction (with stenosis of about 98%) through to the popliteal vein (72.7% stenosis). Both iliac veins were normal as well as the right femoral and popliteal veins.

Echocardiography showed normal cardiac dimensions and function.

Figure 1

Table 1. Patient’s blood biochemistry and haematology results on admission

Biochemistry	Haematology
D-dimer : >500ng/ml (positive)	Prothrombin time(PT): 21 (10-16 secs)
Blood Urea: 6.8 mmol/L	Control prothrombin time: 15
Creatinine: 111 µmol/L	Ratio: 1.40
Fasting blood sugar: 5.4 mmol/L	INR : 1.5
Fasting Total cholesterol: 6.5mmol/L	HB: 13.6 g/dl
Fasting Triglycerides: 1.1mmol/L	Wbc :6.15 k/u1
Fasting HDL-cholesterol: 1.2mmol/L	Neu : 49.5%
Fasting LDL-cholesterol: 4.8mmol/L	Lym: 34.7%
Liver Function Tests : Normal	Mon : 11.4%
	Eos: 2.94%
	Baso: 1.42%
	ESR: 10mmfall/Hour

Figure 2

Figure 1. ECG of the patient on admission

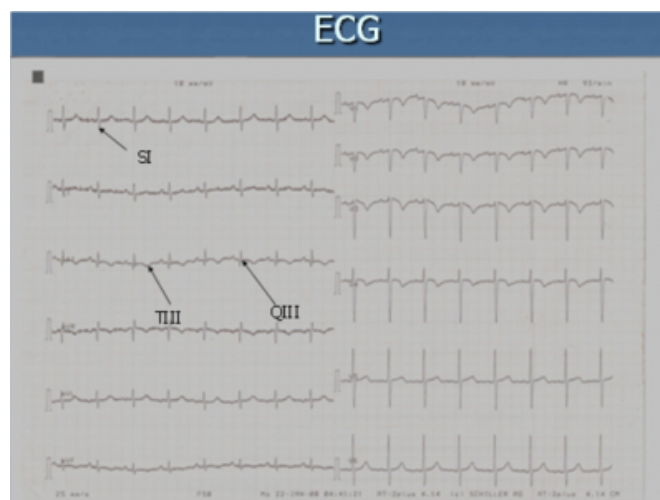


Figure 3

Figure 2. Chest X-ray of the patient on admission

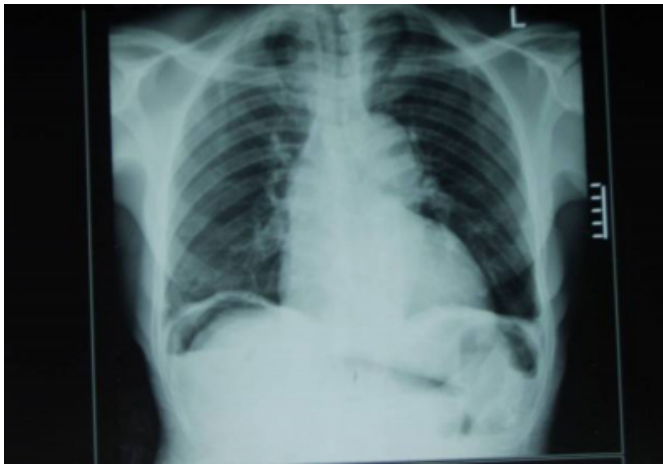
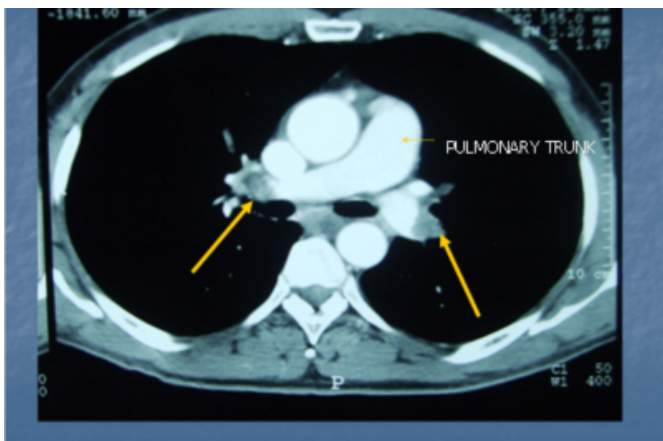


Figure 4

Figure 3. Chest CT scan of the patient



DIAGNOSES

1. Bilateral Pulmonary Embolism
2. Left femoro-popliteal vein thrombosis

TREATMENT

The patient was admitted, oxygen and anticoagulation with enoxaparin were started immediately. Thrombolysis with streptokinase was given after the diagnosis of pulmonary embolism was confirmed; streptokinase 250,000 units was given intravenously over 30 minutes; a repeat dose of 100,000 units was given after 1 hour.

The patient's symptoms subsided about 24 hours after the thrombolysis, he stayed on the ward for some few days and was discharged. He is now on long-term anticoagulation with warfarin.

DISCUSSION

Venous thrombo-embolism refers to deep vein thrombosis and pulmonary embolism. Reports from Africa^{1,2,12-16} and Asia^{17,18} show that that venous thrombo-embolism is common in the developing world. Unfortunately most cases are diagnosed at post-mortem^{12,15}.

The patient developed the deep vein thrombosis probably from prolonged immobilisation as a result of his habit of sitting behind his computer for hours. He was not started on thrombo-prophylaxis to prevent the pulmonary embolism because the initial doctor failed to diagnose the deep vein thrombosis. This resulted in the fatal consequence of bilateral pulmonary embolism.

The clinical diagnosis of deep vein thrombosis and pulmonary embolism is difficult as the symptoms and signs are non-specific. In the developing world, this misery is further compounded by the presence of heavy patient load, limited modern diagnostic investigative tools, and financial constraints on patients. However, early and accurate diagnosis of venous thrombo-embolism is extremely important as a range of effective therapeutic options are available.

Literature shows that most clinically important and fatal pulmonary embolism occurs from proximal than distal deep vein thrombosis in the leg. Pulmonary embolism has been found to occur in 50 percent of patients with proximal deep vein thrombosis¹⁹ while asymptomatic thrombosis of the leg veins has been observed in 70 percent of patients with pulmonary embolism²⁰.

In conclusion, venous thromboembolism should be recognized as worldwide health problem not only in the western countries but also in West African sub-region.

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References

1. Awotedu AA, Igbokwe EO, Akang EE, Aghadiuno PO. Pulmonary embolism in Ibadan, Nigeria: five years autopsy report. *Cent Afr J Med*. 1992 nov; 38(11): 432-435.
2. Igun GO. A 10-year review of venous thrombo-embolism in surgical patients seen in Jos, Nigeria. *Niger Postgrad Med J*. 2001 Jun; 8(2): 69-73.
3. Horlander KT, Mannino DM, Leeper KV. Pulmonary embolism mortality in the United States, 1979-1998: an analysis using multiple-cause mortality data. *Arch Intern*

Med. 2003; 163: 1711.

4. Dismuke SE, Wagner EH. Pulmonary embolism as a cause of death. The changing mortality in hospitalized patients. *JAMA*. 1986; 255:2039.

5. Carson JL, Kelly MA, Duff A, et al. The clinical course of pulmonary embolism: One year follow-up of PIOPED patients. *N Engl J Med*. 1992; 326: 1240.

6. Moser KM. Venous thrombo-embolism. *Am Rev Respir Dis*. 1990; 141: 235.

7. Kistner RL, Ball JJ, Nordyke RA, Freeman GC. Incidence of pulmonary embolism in the course of thrombophlebitis of the lower extremities. *Am J Surg*. 1972; 124: 169.

8. Moser KM, LeMoine JR. Is embolic risk conditioned by location of deep venous thrombosis? *Ann Intern Med*. 1981; 94: 439.

9. Weinmann EE, Salzman EW. Deep-vein thrombosis. *N Engl J Med*. 1994; 331: 1630.

10. Dalen JE, Alpert JS. Natural history of pulmonary embolism. *Prog Cardiovasc Dis*. 1975; 17: 257.

11. Anderson FA, Wheeler HB, Goldberg RJ et al. A population-based perspective of the hospital incidence and case-fatality rates of deep vein thrombosis and pulmonary embolism. *Arch Intern Med*. 1991; 151: 933.

12. Kane A, Mboup MC, Diao M, et al. Pulmonary embolism: autopsy study of 73 cases in Senegal. *Dakar Med*. 2008; 53(2): 136-141.

13. Kinque S, Tagny D, Binam F, et al. Venous thrombo-

embolism in Cameroon (report of 18 cases). *Med Trop. (Mars)* 2002; 62(1): 47-50.

14. Akinmoladun VI, Arotiba JT, Fasola OA, et al. The use of thrombo-embolic prophylaxis by surgeons: A multicentre Nigerian Study. *Niger Postgrad Med J*. 2007 Dec; 14(4): 330-335.

15. Sotunmbi PT, Idowu AT, Akang EE, Aken'Ova YA. Prevalence of venous thrombo-embolism at post-mortem: a cause for concern. *Afri J Med Sci*. 2006 Sep; 35(3): 345-348.

16. Akanmu AS, Nnodu OE, Giwa SO, et al. Efficacy and safety of enoxaparin, a low molecular weight heparin in the prevention of deep vein thrombosis in Nigerian patients after orthopaedic surgery. *Afr J Med Med Sci*. 2004 Dec; 33(4): 335-340.

17. Lee AD, Stephen E, Aqarwal S, Premkumar P. Venous thrombo-embolism in India. *Eur J Vasc Endovasc Surg*. 2009 Apr; 37(4): 482-485.

18. Narani KK. Deep vein thrombosis and pulmonary embolism - Prevention, management, and anaesthetic considerations. *Indian J Anaesth*. 2010 Jan; 54(1): 8-17.

19. Plate G, Ohlin P, Eklöf B. Pulmonary embolism in acute iliofemoral venous thrombosis. *Br J Surg*. 1985; 72: 912-915.

20. Hirsh J, Hoak J. Management of deep vein thrombosis and pulmonary embolism. *Circulation*. 1996; 93: 2212-2245.

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