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Clinical Presentation and Diagnostic Work Up of Suspected Pulmonary Embolism in a Tertiary Care Hospital Emergency Care

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Abstract

The diagnosis of pulmonary embolism (PE) is challenging to make and is often missed in the emergency centre. The diagnostic work-up of PE has been improved by the use of clinical decision rules (CDRs) and CT pulmonary angiography (CTPA) in high-income countries. CDRs have not been validated in the South African environment where HIV and tuberculosis (TB) are highly prevalent. Both conditions are known to induce a hyper-coagulable state. The objective of this study was to describe the clinical presentation and diagnostic workup of suspected PE in our setting and to determine the prevalence of HIV and TB in our sample of patients with confirmed PE. This study was a retrospective chart review of patients with suspected PE who had CTPAs performed between October 2023 and October 2024 at sree mookambika institute of medical sciences. Data were collected on demographics, presenting signs and symptoms, vitals, bedside investigations, HIV and TB status. A Revised Geneva score (RGS) was calculated retrospectively and compared to the CTPA result. The median age of patients with confirmed PE was 45 years and 68% were female. The CTPA yield for PE in our study population was 32%. The most common presenting complaint was dyspnoea (83%). Deep venous thrombosis (DVT) was present in 29%. No sign or symptom was observed to be markedly different in patients with confirmed PE vs no PE. Among patients with confirmed PE, 37% were HIV positive and 52% had current TB. RGS compared poorly with CTPA results. PE remains a diagnostic challenge. In our study, the retrospectively calculated CDR was not pre-dictive of PE in a population with a high prevalence of HIV and TB. Emergency physicians should be cautious when making a clinical probability assessment of PE in this setting. However, further studies are needed to develop a predictive CDR for the local environment.

INTRODUCTION

Pulmonary embolism (PE) is a potentially fatal disease with a widely variable clinical presentation. The true incidence of PE is difficult to establish in the general population. In high-income countries (HIC) the incidence rate is estimated to be 0.5-2 per 1000 person years^[1,2]. In low-and middle-income (LMIC) countries such as South Africa, no reliable data are available on the incidence of PE. Un-diagnosed and therefore untreated PE is associated with significant morbidity and mortality^[3]. The mortality rate for untreated PE can be as high as 30%^[1,4-6]. The use of Clinical Decision Rules (CDRs) and increased availability of CT pulmonary angiography (CTPA) have improved the diagnostic workup of PE. However, the currently used CDRs have never been validated in the South African environment, in which both HIV and tuberculosis (TB) are highly prevalent. This study aimed to describe the clinical presentation and diagnostic workup of patients who presented to a district hospital's emergency centre in South Africa with suspected PE. Secondly, the study sought to determine whether CDRs were being used by clinicians, for assessment of pre-test probability, prior to requesting CTPAs. Revised Geneva scores (RGS) were calculated retrospectively and compared to the CTPA results. The study also sought to determine the prevalence of HIV and TB in the sample population with confirmed PE.

MATERIALS AND METHODS

This study was done at the emergency centre at Mookambika College of Medical Sciences from the period of 2024-2025. The decision to send a patient for CTPA is made after discussions with the Emergency Medicine consultant, for patients who are deemed to be at high risk of having a PE. It was not known whether CDRs were used or documented, or whether decisions were made by clinical gestalt alone. CTPA scans are only available on site from 8 am to 4pm on week-days. After-hours or on weekends, patients with high clinical probabilities are anti-coagulated while awaiting CTPA. If unstable, patients are anti-coagulated and sent to the tertiary hospital for CTPA. A 16-slice multi-detector CT scanner is used for CTPA and is reported on by the in-house radiologist. A retrospective chart review was done. The study population included all patients over the age of 18 who had CTPAs performed for suspected PE at Mitchell's Plain Hospital over a period of 24 months. Patients were excluded if they had CTPAs performed for other indications (e.g. thoracic trauma), repeat CTPAs (in patients already diagnosed with PE on initial CTPA), chronic PE or if the patients' electronic notes and/or physical folders could not be found. 160 patients met the inclusion and exclusion criteria, due to missing patient notes the final sample size was 127. An a priori sample size was not calculated.

Data collection was performed using three databases: radiological imaging (CTPAs) from the local Picture Archiving and Communication System (PACS), electronic patient notes from Enterprise Content Management (ECM) and laboratory data from the National Health Laboratory Service (NHLS). Initially, patients who had CTPAs within the study period were identified on PACS. These patients' emergency centre and in-patient notes were traced on ECM and data were collected and entered onto a pre-designed Excel spreadsheet (Microsoft Excel, USA). Statistical analysis was done using the statistical package for social sciences (SPSS). Different statistical methods were used as appropriate. Mean±SD was determined for quantitative data and frequency for categorical variables. The independent t-test was performed on all continuous variables. The normal distribution data was checked before any t-test. The Chi-Square test was used to analyze group difference for categorical variables. A p-value<0.05 was considered significant.

RESULTS AND DISCUSSIONS

Except for recent hospitalisation (36%), age >65 years (10%), being post-partum (10%), having a previous VTE (6%) and immobilisation (5%), there were very few individual risk factors in our inpatients with confirmed PE on CTPA, 68% had one or more co-morbidities, compared to 80% in those without PE. 46% of patients with confirmed PE had current or previous lung pathology e.g. active TB, previous TB, TB bronchiectasis or chronic obstructive pulmonary. The most prevalent vital sign abnormalities on presentation were tachycardia, tachypnoea and hypoxaemia (Table 2). Patient-reported dyspnoea was the most common presenting symptom (83%), followed by cough and chest pain. Less than 40% of patients reported a sudden onset of symptoms. Physical examination revealed 'clear' or no findings on chest examination in 33%, but clinicians documented signs of pulmonary hypertension in 25% of patients with PE. Twenty-eight patients with suspected PE and leg pain/swelling suggesting DVT also received compression ultrasonography. Fifteen patients were diagnosed with DVT of which twelve also had PE confirmed on CTPA. This means that in our sample of confirmed PE patients, 29% presented with clinical signs of DVT. Vital signs and clinical features between patients with confirmed PE and those with no PE. No sign or symptom was observed to be statistically significant at the 95% level between patients with confirmed PE and those without PE. The most common abnormalities on ECG in patients with confirmed PE were sinus tachycardia (68%), T wave inversion in the precordial leads (51%) and non-specific ST segment or T wave changes (43%). With regards to arterial blood gas measurements, there were very small differences in PaO₂, PaCO₂ and oxygen saturation between patients

with confirmed PE and no PE. The Wells score was documented in the notes in only 13% of patients with suspected PE. The Revised Geneva score was not documented in any of the notes. The Revised Geneva score contains only objective variables compared to the Wells score that includes a heavily weighted subjective criterion. The simplified dichotomised Revised Geneva score categorises patients into 'PE likely' (score >2) or 'PE unlikely' groups (score ≤ 2). This was calculated retrospectively on the collected data and the categories compared poorly with the CTPA result (Fig. 1). In our study population, the RGS had a sensitivity of 29%, 95% CI [16.1-45.5], specificity of 83% [72.9-89.9], positive predictive value (PPV) of 44% [29.2-60.8] and a negative predictive value (NPV) of 71% [66.3-75.3]. The positive likelihood ratio was 1.68 [0.87-3.25] and negative likelihood ratio was 0.86 [0.69-1.07]. 55% of the Revised Geneva scores were equal to 2, which is just below the cut-off point for the dichotomised rule. The CTPA yield for PE in our study population was 32% [24.2-40.4] (n=41). The anatomic positions included saddle emboli, left and right main pulmonary arteries/lobar arteries/segmental arteries and sub-segmental arteries. In 61% of positive CTPAs, the pulmonary embolism was found simultaneously at different levels and/or included both lungs.

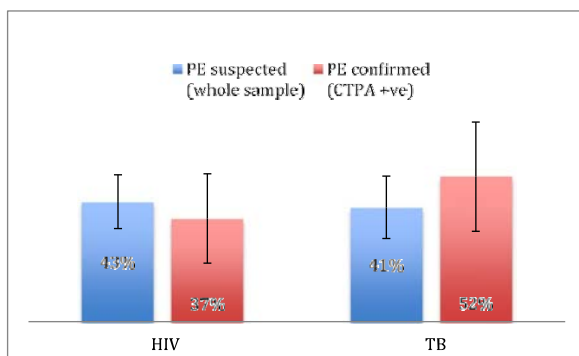


Fig. 1. Comparison: Revised Geneva Score vs CTPA.
Note: CTPA, CT pulmonary angiography

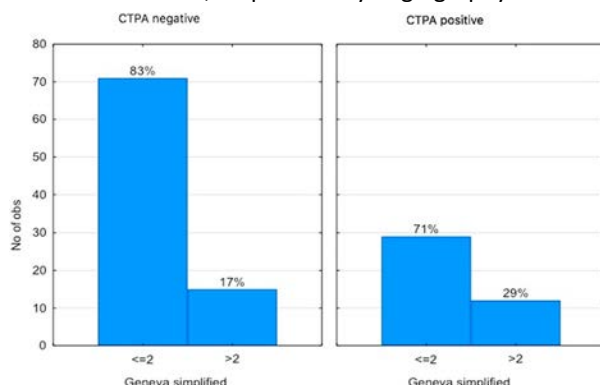


Fig. 2: Prevalence of HIV and TB in suspected PE vs confirmed PE.

Note: PE, pulmonary embolism., CTPA, CT pulmonary angiography., HIV, human immunodeficiency virus., TB, tuberculosis.

above the cut-off value ($>500 \mu\text{g/ml}$) in 76%. Ordering of D-dimer tests did not correlate with documented CDR score (Wells) or retrospective CDR score (Revised Geneva). In our study population of patients with suspected PE, the pre-valence of HIV was 43% and that of TB was 41% (Fig. 2). It must be noted that 20% of HIV results were missing from the data (either not done or not documented) and in 42% of patients the TB status was unknown (no laboratory testing done). Among those patients with confirmed PE, 37% were HIV positive and 52% had current TB (Fig. 2). Of the 52% (n=13) with current TB and PE, twelve patients also had previous TB and eight of those had been diagnosed with TB bronchiectasis. The prevalence of having both HIV and TB was 35% in the study population. 31% also had documented previous TB. In HIV positive patients with suspected PE, the CTPA was positive in 30% (compared to 38% in HIV negative patients). In patients with active TB and suspected PE, 43% had confirmed PE on CTPA (compared to 27% in TB negative patients). In the study population, no data on the incidence of PE could be found in the literature. Still, PE is a condition that often remains undiagnosed, untreated and leads to significant morbidity and mortality. Mortality data from Statistics SA in 2014 found 2525 deaths 'attributable to diseases of the pulmonary circulation'^[7]. This number includes deaths from PE but grossly underestimates the burden of disease. In our study population, the mean age at diagnosis of PE was 45 years and only 10% of patients were over the age of 65. This could be related to the total life expectancy in South Africa, which was 62.5 years at the 2015 mid-year population estimate^[8]. The most common clinical presentations in our study were shortness of breath, cough and chest pain. This is congruent with what has been found in studies in HICs where the two most common symptoms of PE are chest pain and shortness of breath^[3]. These are also known to be the two most common symptoms presenting to emergency centres around the world^[9]. The clinical diagnosis of PE is difficult as it spans a spectrum of medical presentations from asymptomatic to cardiovascular collapse and death^[3]. In our study population, no sign or symptom was observed to be markedly different between patients with confirmed PE and those without. A systematic review and meta-analysis by West, *et al.* concluded that no feature in isolation could be used to rule a PE in or out^[10]. Although tachycardia, tachypnoea and hypoxaemia were the most frequent vital sign abnormalities in our patients with confirmed PE, they were similarly frequent in those without PE. Even in the multi-centre United States (US) study, no differences in vital signs were detected between the PE positive and PE negative groups^[11]. A possible explanation is that patients presenting with a significant cardio-respiratory complaint will all have some degree of

tachycardia, ta-chypnoea and lower oxygen saturation. This also presents the problem of looking at vital signs in patients with co-morbidities and suspected PE. Many symptoms of PE also mimic those of other cardiopulmonary diseases such as congestive heart failure and chronic obstructive pulmonary disease (COPD)^[3]. In our study, 68% of confirmed PE patients had one or more co-morbidities and 46% had current or previous lung pathology. In South Africa, there is also a higher burden of infectious diseases (e.g., pneumonia, TB) with a steady rise in lifestyle-associated diseases such as congestive cardiac failure and COPD. The EMPEROR study, which described the clinical presentation of PE in patients presenting to multiple emergency centres in the US, found dyspnoea, chest pain and extremity swelling suggesting DVT to be the most common presentations^[11]. We found a DVT by compression ultrasonography in 15 of 28 patients with suggestive leg pain/swelling. Of the 15 patients diagnosed with a DVT, twelve also had PE confirmed on CTPA. This means that in our sample of confirmed PE patients 29% presented with clinical signs of DVT. This may be lower than documented in other studies, but in our setting, compression ultrasonography for DVT is not routine practice in patients with suspected PE and was only performed if DVT was also suspected clinically. The current evidence-based approach to the diagnosis of PE is a non-invasive sequential use of different modalities: clinical probability assessment (by use of clinical gestalt or CDR), followed by D-dimer measurement or CTPA^[12]. These have been combined into a diagnostic algorithm, using the Revised Geneva Score in this example (Fig. 3). The use of a validated diagnostic algorithm has been found to lower healthcare costs and also decrease complication risk^[13]. However, despite the overwhelming evidence to support the use of diagnostic algorithms, adherence in clinical practice even in HICs is poor^[14]. In our setting, no written diagnostic algorithm or guideline exists. As seen in our study, only 13% of patients with suspected PE had a documented clinical probability assessment by a CDR (the Wells score was used by our clinicians). It appears that most of our patients deemed to be high risk enough to be sent for CTPA were dependent on the clinicians' unstructured estimate of their pre-test probability (i.e. clinical gestalt). The purpose of the clinical probability assessment is to categorise the patient into pre-test probability categories: 'PE likely' (score >2) or 'PE unlikely' groups (score=2) by using the dichotomised Revised Geneva score in this example^[16]. The categories correlate with the patient's estimated risk of PE and guide the next step in the diagnostic algorithm^[17]. (Fig. 3) In our study population, the RGS had a sensitivity of 29% and specificity of 83%. The sensitivity compares poorly, but the specificity is improved when compared to the values described by Lucassen *et al.* in a meta-analysis (91% and 37%,

respectively)^[18]. The latter study also noticed an increase in sensitivity and a decrease in specificity when prevalence was increased, which is in contrast to our findings. A higher prevalence, as found in our sample population, is known to increase the positive predictive value of a test, PPV 44% vs 32%. In our study, 71% of patients with confirmed PE (CTPA positive) would have been incorrectly categorised as 'PE unlikely' using the RGS. Therefore, if the decision to perform CTPAs in our population was based on the RGS, CTPA would have not been performed and 71% of PEs would have been missed (Fig. 1). The diagnostic yield of the 'PE unlikely' group could have been improved by the addition of a D-dimer test (Fig. 3), however it was only performed in 21 patients (17%). The low use of D-dimer testing is a reflection of the low use of CDRs by clinicians (13%) in our study. This shows not only the need for a predictive CDR for our environment but also an improved adherence to using a diagnostic algorithm combining CDR, D-dimer and CTPA (as shown in Fig. 3). Multi-Detector Computed Tomography Pulmonary Angiography (MD-CTPA) is the imaging modality of choice for the investigation of suspected^[3]. In HICs, the increased availability and advancing technology of CT scanners has led to overuse of this modality (14-fold increase in the US)^[19]. This is associated with an increased risk of harm due to radiation and unnecessary expense. A study evaluating the appropriateness of CTPA use in emergency centre patients found that one third of CTPAs performed for suspected PE were avoidable and recommended the use of diagnostic protocols or guidelines to lower the number of inappropriate^[20]. In LMICs, such as South Africa, CTPA is often only available at large tertiary hospitals and some secondary-level hospitals. Its use is also limited by cost and radiological expertise. It would make sense that the implementation of diagnostic algorithms/guidelines in our setting would improve the utilisation of a scarce and costly resource. The CTPA positivity rate in our study population was 32% (n=41). International studies performed since 2001 reported that the yield of CTPA in emergency centre patients differs widely and produced rates of between 5.7% and 37%^[21]. Only three of those studies had rates above 20%, however these followed an ideal workup of patients instead of actual clinical practice^[21]. A recent study concluded that adhering to a diagnostic protocol increased the yield of CTPA and reported a yield of 29.6%^[22]. The high yield in our study could be explained by the fact that only patients who were clinically assessed as high risk for PE were sent for CTPA, even though a few could have been missed as there was no diagnostic protocol. According to the World Health Organization (WHO), South Africa is a high-HIV, high-TB burden country^[23]. The 2015 mid-year statistics estimate for the prevalence of HIV in adults aged 15-49 was 16.6%^[8]. The WHO Global TB Control Report 2015

estimated the South African TB prevalence rate at 696/100,000 and the prevalence of HIV infection in TB patients at 61%^[23]. Although prolonged hospitalisation and traditional risk factors play a role, examination of risk factors in HIV positive patients revealed an increased risk in patients younger than 50 years old, the presence of concomitant infections (e.g. cytomegalovirus), low CD4 counts (<200/ mm³), or a diagnosis of AIDS^[24]. A Kenyan study reported a 10.9% prevalence rate of HIV in a group of PE patients^[25]. In South Africa, the incidence of PE in HIV-infected patients is unknown. However, a study reviewing the risk factors for DVT found that in patients with confirmed DVT, 64.4% were HIV-in-fected, 56.5% had TB and 43.3% were co-infected^[26]. The above compares to our study of patients with confirmed PE where 37% were HIV-infected, 52% had TB and 35% were co-infected. Tuberculosis also induces a hyper-coagulable state and adults with active tuberculosis have an increased risk of VTE^[6]. One review found that more than half of TB patients diagnosed with VTE had no apparent risk factor except for^[27]. Our study showed that in patients with active TB and suspected PE, 43% had confirmed PE on CTPA (compared to 27% in TB negative patients). This shows that TB patients, in whom the clinical suspicion of PE was high, had confirmed PE in >40% of cases. This illustrates the importance of not discarding PE as a diagnosis in patients with active TB. This retrospective chart review was subject to limitations concerning missing data. The lack of data influences the analysis and interpretation of the results of our sample. As the sample population was drawn from patients that had been sent for CTPA, it includes a higher than average risk population. It is therefore not representative of the undifferentiated emergency centre population.

REFERENCES

1. Cushman M., A.W. Tsai, R.H. White, S.R. Heckbert, W.D. Rosamond, P. Enright and A.R. Folsom., 2004. Deep vein thrombosis and pulmonary embolism in two cohorts: The longitudinal investigation of thromboembolism etiology. *The Am. J. Med.*, Vol. 117: 10.1016/j.amjmed.2004.01.018.
2. NAESS I.A., S.C. Christiansen, p. romundstad, s.c. cannegieter, f.r. rosendaal and j. hammerstrøm., 2007. Incidence and mortality of venous thrombosis: A population-based study. *J. Thrombosis Haemostasis*, Vol. 5: 10.1111/j.1538-7836.2007.02450.x.
3. Ouellette D.W. and C. Patocka., 2012. Pulmonary Embolism. *Emergency Med. Clin. North Am.*, Vol. 30: 10.1016/j.emc.2011.12.004.
4. White R.H., 2003. The Epidemiology of Venous Thromboembolism. *Circulation*, Vol. 107: 10.1161/01.CIR.0000078468.11849.66.
5. Bibas M., G. Biava and A. Antinori., 2011. HIV-Associated venous thromboembolism. *Mediterr. J. Hematol. Infect. Dis.*, Vol. 3: 10.4084/mjh.2011.030.
6. Dentan C., O. Epaulard, D. Seynaeve, C. Genty and J.L. Bosson., 2014. Active Tuberculosis and Venous Thromboembolism: Association According to International Classification of Diseases, Ninth Revision Hospital Discharge Diagnosis Codes. *Clin. Infect. Dis.*, Vol. 58: 10.1093/cid/cit780.
7. S.S.A., 2015. Mortality and cause of death in South Africa 2014: Findings from death notification. Pretoria., Vol. 3.
8. S S.A., 2015. Mid-year population estimates 2015. Pretoria., Vol.
9. Mc Caig L.F. and E.W. Nawar., 2006. National Hospital Ambulatory Medical Care Survey: 2004 emergency department summary. *Advance data.*, 372: 1-29.
10. West J., S. Goodacre and F. Sampson., 2007. The value of clinical features in the diagnosis of acute pulmonary embolism: Systematic review and meta-analysis. *QJM*, Vol. 100: 10.1093/qjmed/hcm113.
11. Pollack C.V., D. Schreiber, S.Z. Goldhaber, D. Slattery and J. Fanikos *et al.*, 2011. Clinical characteristics, management, and outcomes of patients diagnosed with acute pulmonary embolism in the emergency department: initial report of EMPEROR (Multicenter Emergency Medicine Pulmonary Embolism in the Real World Registry). *J. Am. Coll. Cardiol.*, Vol. 57: 10.1016/j.jacc.2010.05.071.
12. Raja A.S., J.O. Greenberg and A. Qaseem, et al., 2015. Evaluation of patients with suspected acute pulmonary embolism: best practice advice from the clinical guidelines committee of the American College of Physicians. *Ann Intern Med.*, Vol. 163: 10.7326/M14-1772.
13. Huisman M.V. and F.A. Klok., 2013. Diagnostic management of acute deep vein thrombosis and pulmonary embolism. *J. Thrombosis Haemostasis*, Vol. 11: 10.1111/jth.12124.
14. Smith C., A. Mensah, S. Mal and A. Worster., 2008. Is pretest probability assessment on emergency department patients with suspected venous thromboembolism documented before Simpli RED D-dimer testing? *Cjem.*, 10: 519-523.
15. Klok F.A., I.C.M. Mos, M. Nijkeuter, M. Righini, A. Perrier, G.L. Gal and M.V. Huisman., 2008. Simplification of the Revised Geneva Score for Assessing Clinical Probability of Pulmonary Embolism. *Arch. Internal Med.*, Vol. 168: 10.1001/archinte.168.19.2131.

16. van Belle A., H.R. Buller, M.V. Huisman, P.M. Huisman, K. Kaasjager and P.W. Kamphuisen, *et al.*, 2006. Effectiveness of managing suspected pulmonary embolism using an algorithm combining clinical probability, D-dimer testing, and computed tomography. *Jama.*, Vol. 295: 10.1001/jama.295.2.172.
17. Bounameaux H., A. Perrier and M. Righini., 2010. Diagnosis of venous thromboembolism: An update. *Vasc. Med.*, Vol. 15: 10.1177/1358863X10378788.
18. Lucassen W., G.J. Geersing, P.M.G. Erkens, J.B. Reitsma, K.G.M. Moons, H. Büller and H.C. van Weert., 2011. Clinical Decision Rules for Excluding Pulmonary Embolism: A Meta-analysis. *Ann. Internal Med.*, Vol. 155: 10.7326/0003-4819-155-7-201110040-00007.
19. Smith-Bindman R., D.L. Miglioretti, E. Johnson, C. Lee and H.S. Feigelson *et al.*, 2012. Use of Diagnostic Imaging Studies and Associated Radiation Exposure for Patients Enrolled in Large Integrated Health Care Systems, 1996-2010. *JAMA*, Vol. 307: 10.1001/jama.2012.5960.
20. Venkatesh A.K., J.A. Kline, D.M. Courtney, C.A. Camargo and M.C. Plewa *et al.*, 2012. Evaluation of pulmonary embolism in the emergency department and consistency with a national quality measure: quantifying the opportunity for improvement *Arch. Internal Med.*, Vol. 172: 10.1001/archinternmed.2012.1804.
21. Costa A.F., H. Basseri, A. Sheikh, I. Stiell and C. Dennie., 2014. The yield of CT pulmonary angiograms to exclude acute pulmonary embolism. *Emergency Radiol.*, Vol. 21: 10.1007/s10140-013-1169-x.
22. Walen S., E. de Boer, M.A. Edens, C.A.J.V. Worp, M.F. Boomsma and J.W.K.V. Berg., 2016. Mandatory adherence to diagnostic protocol increases the yield of CTPA for pulmonary embolism. *Insights into Imaging*, Vol. 7: 10.1007/s13244-016-0509-2.
23. WHO., 2015. Global TB Report. Geneva., Vol. 2015.
24. Kiser K.L. and M.E. Badowski., 2010. Risk Factors for Venous Thromboembolism in Patients with Human Immunodeficiency Virus Infection. *Pharmacother.*, Vol. 30: 10.1592/phco.30.12.1292.
25. Ogeng'o J.A., M.M. Obimbo, B.O. Olabu, P.M. Gatonga and D. Ong'era., 2011. Pulmonary thromboembolism in an East African tertiary referral hospital. *J. Thrombosis Thrombolysis*, Vol. 32: 10.1007/s11239-011-0607-4.
26. Alsherhi M.F., A. Kropman and H. Geduld., 2013. Risk factors for deep vein thrombosis in a South African public hospital: Open UCT collection.
27. Sharif-Kashani B., B. Bikdeli, A. Moradi, P. Tabarsi and E. Chitsaz *et al.*, 2010. Coexisting venous thromboembolism in patients with tuberculosis. *Thrombosis Res.*, Vol. 125: 10.1016/j.thromres.2010.01.014.