Hong Kong College of Physicians
Case report for Interim Assessment
Specialty Board of Advanced Internal Medicine (AIM)

For AIM Training, case reports should be submitted in the prescribed format together with the application form for Interim Assessment at least EIGHT Weeks before the date of Interim Assessment

Name of candidate (print and sign): Luk Wing Lam Fion
Hospital and Unit: PWH M&T Specialty: ID and AIM
Name of supervisor (print and sign): Prof. Grace Wong
Date(s) and place (hospital) of patient encounter: 12 Feb 2023 PWH
Date of report submission:4 March 2023

Case report

Note: Failure to follow the prescribed format (including the number of words) results in a FAILURE mark (score between 0 and 4) for the Case Report.

Title: A patient with bilateral pulmonary embolism presented as loss of consciousness

Case history:

A 66-year-old male patient presented to the hospital with loss of consciousness preceded by 3-day history of left calf pain and swelling. His past medical history included hypertension on amlodipine, generalized anxiety disease on fluoxetine and benign prostate hyperplasia on Terazosin. He denied any chest pain, palpitation, shortness of breath or hemoptysis preceding loss of consciousness and was all along afebrile. He regained consciousness spontaneously after few seconds without any limb twitching or incontinence. He was mobile all along without any preceding immobilization.

On admission, his blood pressure was 168/82 mmHg, pulse 91 beats per minutes, oxygen saturation 99% on 3L of oxygen. He was afebrile. Examination showed no audible murmur and no sign of heart failure. Neurological exam was unremarkable. Patient's left calf was swollen when compared to right calf. Hemoglobin level on admission was 11 g/dL similar to baseline. White cell count, platelet count, liver and renal function was unremarkable. Troponin T was raised from 61.0 ng/L to 595 ng/L (reference <14ug/L). Creatine kinase was normal. Arterial blood gas was not

suggestive of respiratory failure. D-dimer was >10000ng/ml (reference <500ng/ml). Electrocardiogram (ECG) showing sinus rhythm 79 beats per minute, with S1Q3V3 pattern and right bundle branch block, there was no axis deviation. Serial ECG with no ischemic changes. Echocardiogram showed dilated right ventricle with flattened intraventricular septum. Left ventricular ejection fraction was 65%. In view of clinical presentation of desaturation, S1Q3T3 pattern on ECG and elevated D-dimer level, computed tomography pulmonary angiogram (CTPA) was performed. CTPA showing extensive filling defects in both lungs, predominantly in the segmental and subsegmental pulmonary arteries of right upper, right middle, right lower and left upper lobes, as well as subsegmental pulmonary arteries of left lower lobe. Features are suggestive of bilateral pulmonary embolism. No filling defects in the main pulmonary arteries or pulmonary trunk. Enlarged right ventricle is worrisome of right heart strain. (Figure 1) A diagnosis of pulmonary embolism was made.

Catheter directed thrombolysis was discussed but the patient refused due to financial issue as this is a self-finance item under Hospital Authority service. Patient was put on low molecular weight heparin (enoxaparin) for anticoagulation. Doppler ultrasound of bilateral lower limb showing no evidence of above-knee deep vein thrombosis in both lower limbs. Further investigation showed elevated prostate specific antigen (PSA) 6.1 ug/l (reference <4 ug/l) with normal alpha-fetoprotein (AFP) and carcinoembryonic antigen (CEA). Anti-cardiolipin and Anti-B2GP1 were not elevated. Anti-cytoplasmic antibody and anti-nuclear antibodies were negative. Thyroid function test was normal. Patient remained haemodynamically stable and was able to wean off oxygen. Left calf swelling improved. Referral for urology regarding elevated PSA was made. Ultrasound of abdomen and pelvis was arranged. Enoxaparin was switched to Apixaban upon discharge. Patient was stable and no bleeding symptoms on discharge.

Discussion and literature review

This case illustrated a case of bilateral pulmonary embolism. Pulmonary embolism (PE) occurs when there is occlusion of blood flow in the pulmonary artery. Thrombus formation is contributed by 3 factors: venous stasis, intravascular wall damage and hypercoagulability (Virchow triad). PE is typically preceded by a thrombus formed in lower limb vein (deep vein thrombosis(DVT)). 70-80% of PEs are preceded by DVT.[1] The mortality rate of venous thromboembolism (PE + DVT) in China is 2.1%.[2] PE should be considered in patients presented with acute chest pain, shortness of breath, hemoptysis, DVT or syncope. The prevalence of syncope in PE patients ranged from 4% to 17%, and usually syncope is not the only presentation. The cause of syncope is often due to transient hypotension, which may indicate a high clot load of PE in patient presented with syncope.[3] A high degree of pulmonary artery occlusion and related vasoconstriction will lead to higher pulmonary vascular resistance, which then contributes to a higher right ventricular afterload, leading to a lower left ventricular preload and then lower cardiac output. Patient with PE presented with syncope is considered as intermediate to high risk as it is associated with a higher mortality a higher prevalence of hemodynamic instability.[4]

Diagnosis of PE consist of 3 modalities: first with evolution of clinical probability, second with D-dimer checking when it is indicated, third with chest imaging namely CTPA or ventilation/perfusion lung scintigraphy (V/Q scan). Wells score has been validated for evaluating clinical probability of PE. It includes subjective clinical suspicion of DVT, tachycardia, immobilization, previous history of DVT or PE, hemoptysis, cancer status and if other alternative diagnosis exists. Risk of PE is determined to be "unlikely" if Wells score is 0-4 points, and one should consider high sensitivity d-dimer testing, to stop workup if d-dimer is negative, or proceed to chest imaging if d-dimer is positive. Risk of PE is determined to be "likely" if Wells score >4 points and should directly go for CTPA/ V/Q scan.[5] Other investigation includes bedside echocardiography to detect any non-specific signs of PE namely right ventricular dilatation, or bulging intraventricular septum. It may also rarely detect a thrombus inside ventricles or pulmonary artery.

Treatment choice for PE is based on the risk for in-hospital mortality. For low-risk patient with stable hemodynamic status, anticoagulation with low molecular weight heparin (LMWH) followed by warfarin was suggested. Recently, more studies showing direct oral anticoagulants (DOACs) were not

inferior to warfarin for PE recurrence. DOACs were also associated with a lower risk of bleeding.[6] For patient with hemodynamic instability, defined as systolic blood pressure <90mmHg or mean blood pressure <65mmHg, or evidence of end-organ hypotension, thrombolytic therapy with recombinant tissue type plasminogen activator (rtPA) is suggested. However, for patient with active bleeding or high risk for bleeding, rtPA is not suggested. These patients may be selected for percutaneous catheter-directed treatment or surgical embolectomy. In patient that developed cardiac arrest, venous-arterial extracorporeal membranous oxygenation should be started.[1] For patient with contraindication to put on anticoagulation, inferior vena cava filter should be considered. A study showing placement of inferior vena cava was associated with lower recurrence of PE (absolute risk reduction 5%; 95% CI 2%-8%), however no effect on mortality was shown.[7] The optimal anticoagulation treatment duration for PE patient is still unclear. A report of data analysis showing the rate of 2-year PE recurrence did not differ significantly in patient who received 3 months of anticoagulation with warfarin compare with those received 6 months.[8] For patient with persistent risk factors, such as cancer and thrombophilia, longer course of anticoagulation should be considered.

Our patient presented with syncope and features of right heart strain, with hemoglobin level 11g/dL on 3L of oxygen, with intermediate to high risk of mortality, catheter directed thrombolysis was suggested. As with the refusal of our patient, patient was given oral apixaban 10mg twice daily for 7 days, followed by 5mg twice daily. For thrombophilia screening, Anti-cardiolipin, Anti-B2GP1, anti-cytoplasmic antibody and anti-nuclear antibodies were not elevated. Further investigation for malignancy workup would be needed to determine the duration of anticoagulation therapy.

Tables and figures (where applicable) (no more than two figures)

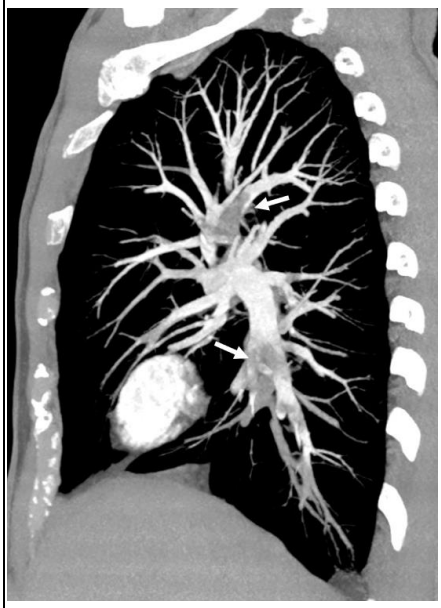

Figure 1 Multiple filling defects in the right upper and lower lobe pulmonary arteries (arrows)

Reference (not more than 10)

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No of words in Case History and Discussion (excluding references):___1131_____

(should be between 1000-2000)

Declaration

I hereby declare that the case report submitted represents my own work and adheres to the prescribed format. I have been in clinical contact with the case selected. The case report has not been submitted to any assessment board or publication and it is NOT related to my second specialty(ies), if any. My consent is hereby given to the College to keep a copy of my case report, in written and/or electronic, at the College Secretariat and allow the public to have free access to the work for reference.

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(signature of Trainee)

Endorsed by Supervisor *

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(signature of Supervisor)

* Supervisors must go over the Case Report with the Trainees, advise Trainees whether further amendments are necessary, review the Originality/Similarity Report prepared by Trainees, adherence to the required format, sign on the report and remind Trainees on issues related to copyright and plagiarism.