

Case study**Vitamin B12 deficiency, an independent risk factor for thromboembolism**

Author

Dr Omar Ataallah Mukhlif. MBChB. MRCP (UK)**Introduction**

Thromboembolic events are increasingly common and serious life threatening incidents. Among many risk factors, vitamin B12 deficiency is becoming more recognized. We report a case of pulmonary embolism with no risk factors other than vitamin B12 deficiency.

Case Report

54 year old gentleman presented to the Emergency Department early in the morning with sharp left side chest pain and shortness of breath for 1 hour duration. He is normally fit and active and has no significant medical history. However, he reports reduced exercise tolerance over the last month, dyspnoea on exertion, and dry cough. On examination, he looks pale and uncomfortable because of the pain, but otherwise normal. He has no tachypnoea or tachycardia, his calves are soft and not tender, and his spo₂ was within normal range.

Routine investigation was done and reported as follows:

ECG normal sinus rhythm

CXR normal

HB 111 g/LWBC 7.02 10⁹/LPLT 214 10⁹/L**MCV 143 fl**

INR 1.0

APTT 0.93

Blood film: Moderate anisocytosis, marked macrocytosis, neutrophils appear right shifted along with hypesegmented neutrophils suggest check B12 and folate level

NA 138 mmol/L

K 4.1 mmol/L

Urea 6.1 mmol/L

Creatinine 6.1 μmol/L

ALT 28 u/L

Alk.Phos. 55 u/L

Total Bilirubin 37 μmol/L

Total Protein 77 g/L

Albumin 47 g/L

Glucose 6.4 mmol/L

Total Calcium 2.34 mmol/L

Adjusted Calcium 2.30 mmol/L

Troponin T 4.5 ng/L

eGFR 88.5

CRP 21 mg/L

Due to The finding of macrocytic anaemia, additional tests were requested

L.D.H. 1597 u/L (240-480)**Vitamin B12 <50 ng/L (191 – 663)**

Folate 14.5 ug/L (3.7 – 18.7)

Ferritin 455 ug/L (30 – 400)

He was diagnosed with megaloblastic anaemia caused by vitamin B12 deficiency as a cause for his breathlessness, and his chest pain was thought to be musculoskeletal. He was referred to the department of medicine to adjust his replacement therapy.

The patient was seen in the Emergency Department in preparation for his discharge. He was still breathless and pale, and still complaining of left pleuritic chest pain. On further questioning, he did not report any risk factor for pulmonary embolism or deep vein thrombosis. On examination, heart rate was 85, blood pressure was 144/85, respiratory rate was 20, spo2 was 96% on room air, chest clear, with only mild local tenderness over the lateral chest wall, normal heart sounds, and abdomen soft and not tender, no signs of deep vein thrombosis. However, the diagnosis of pulmonary embolism was still suspected and D-Dimer level test was requested.

D Dimers 1.82 mg/L

CT- pulmonary angiogram was requested accordingly and reported as positive for bilateral pulmonary emboli in the right lower and left upper lobes with no right heart strain.

The patient was treated with novel oral anticoagulant (Rivaroxaban) for 6 months, and IM hydroxocobalamin 1 mg every 3 days for 2 weeks, then 1 mg every 2 months. He was discharged with an echocardiography appointment after 6 months and to review his anticoagulant at that time. We sent a letter to his general practitioner to follow vitamin B12 level. He was referred to the gastroenterology team to exclude pernicious anaemia and malabsorption.

Discussion

Thromboembolism is a serious and common disease which may affect multiple body organs. There multiple risk factors including environmental and genetic factors, however, vitamin B12 deficiency is often neglected, especially in cases of pulmonary embolism where the breathlessness is frequently attributed to anaemia alone. Over a period of time many

studies have exhibited that hyperhomocysteinemia is one of the factors. Cardiovascular risk rises with either folate or vitamin B12 deficiency because folate and vitamin B12 (cyanocobalamin) are closely connected with the metabolism of homocysteine and methionine⁽¹⁾. Vitamin B12 deficiency is observed in an important percentage of patients with thrombosis. In many patients with thrombosis and Hyper Hcy, the increase in Hcy is caused by vitamin B12 deficiency, suggesting that parenteral vitamin B12 with or without folic acid should be administered to correct vitamin B12 deficiency⁽²⁾. In 2015, a study was done on 90 patients with homocysteine cerebral infarction with DVT. It was found that Hcy was found to be negatively correlated with folic acid and vitamin B12. Folic acid and vitamin B12 can reduce the recurrence rate of thrombosis in patients with lower extremity limb deep venous thrombosis and in patients with Hcy disease suffering from high Hcy levels complicated by lower limbs deep venous thrombosis, maybe by reducing the levels of Hcy to prevent the recurrence of thrombosis⁽³⁾.

Summary Points

Vitamin B12 deficiency is well known to cause many cardiovascular and neurological problems, and the risk of thromboembolism and a significant addition to these risks. Fortunately, its diagnosis and treatment is readily available in community. Such risk need to be explained to both the patient and health practitioners to raise the awareness about the possibility of thromboembolism as a significant differential diagnosis for wide range of presentations.

References

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