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A CASE OF UPPER LIMB DEEP VEIN THROMBOSIS IN A KNOWN HIV PATIENT WITH NON HODGKINS LYMPHOMA

Dr. Abolarin Ademola Toyosi,MBBS, FMCPATH[1*], Dr. Olufemi-Aworinde Kehinde Joyce, MBBS, FWACP, FMCPATH[1], Dr. Olutogun Toluwalase, MBBS, FWACP, FMCPATH[1]

[1]Department of Haematology and Blood Transfusion, Bowen University Teaching Hospital, Ogbomosho, Oyo State, Nigeria.

ABSTRACT

This is a case of upper limb deep vein thrombosis in a HIV positive patient who had also been diagnosed of Non-Hodgkins Lymphoma. This case highlights the importance of thromboprophylaxis and thrombotic risk assessment in all HIV positive as well as cancer patients particularly in low resource setting which are at risk of increased morbidity and mortality.

Keywords: Thrombosis; Non Hodgkins lymphoma; HIV/AIDS

***Correspondence to Author:**
Dr. Abolarin Ademola Toyosi, MBBS, FMCPATH
Department of Haematology and Blood Transfusion, Bowen University Teaching Hospital, Ogbomoso, Oyo State, Nigeria.
Phone: +2347032691801

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Case Presentation

The patient was a 45-year-old man who presented at the surgical outpatient clinic in our facility with a 2-month history of a right sided jaw swelling and a month's history of swelling on the anterior wall of the right axilla which measured 10cm by 8cm on examination. Initial workup showed a packed cell volume of 39%, an ESR of 63 mm/hr and retroviral screening done was reactive. Patient had a Lymph node biopsy for histology done following which he was referred to the HAART clinic where he was immediately commenced on Tenofovir / Lamivudine / Doltegravir combination. Initial CD4 count of 334copies/ml.

Four weeks later patient was referred to the Haematology clinic on account of a histology report which showed effacement of nodal architecture by malignant lymphoid cells which had also infiltrated perinodal fat in the right axillary biopsy taken. A diagnosis of Non Hodgkins Lymphoma was made. Examination at the haematology clinic showed normal vital signs and a rock hard swelling of the right axilla and anterior chest wall measuring about 16 x 21cm with swelling and pain along the entire right upper limb.

Investigations showed a packed cell volume of 37%, a WBC of 6.4 x 109/l and a platelet count of 236 x 109/l. Other significant findings were elevated uric acid and creatinine levels of 16.6mg/dl (3.5 – 7.2mg/dl) and 1.7mg/dl (0.6 – 1.5mg/dl) respectively, low calcium levels of 7.1 mg/dl (8 - 11mg/dl) and elevated phosphate levels of 7.4mg/dl (2-4mg/dl). The liver function test and fasting blood glucose levels were essentially normal.

The patient was admitted and commenced on chemotherapy with CHOP regimen (Cyclophosphamide, Adriamycin, vincristine and prednisolone) and Allopurinol. He was also placed on prophylactic antibiotics.

Outcome and Follow up

After completion of the first cycle of CHOP regimen, and following reassessment, a diagnosis of primary chemoresistant NHL was made based on a progressive enlargement of the neck and axillary masses. He was changed to second line EPOCH regimen and following two cycles of chemotherapy, there was a reduction of the axillary mass by 90% and complete disappearance of the cervical mass. On day 84 of chemotherapy regimen, he presented with ulceration of overlying skin, pain and upper limb lymphedema. An upper limb Doppler ultrasound was requested based on a suspicion of upper limb deep venous thrombosis. He was also commenced on SC Clexane 40mg daily and the Doppler USS results confirmed an upper limb deep venous thrombus. Report showed multiple thrombi in the right median cubital, basilic and brachial veins. SC clexane was immediately increased to 100mg daily (in 2 doses) and warfarin 5mg nocte was added due to financial constraints leading to an anticipated shift to oral anticoagulants. 6 days later, Clexane was stopped and patient was continued on warfarin with an initial Prothrombin time of 20 seconds and an INR of 1.6. By day 15 of warfarin therapy, the right upper limb swelling had reduced by 65% and the pain had subsided significantly. The right axillary mass had also resolved significantly. Doppler USS done after 21 days on warfarin showed that the basilica and median cubital veins were free of thrombi, but the right brachial vein thrombus was still present, with incomplete filling of its lumen. This suggested a progressive resolution of the Upper limb DVT.

Discussion

We present a patient with a diagnosis of Non-Hodgkins lymphoma who while on chemotherapy developed upper extremity DVT. Venous thromboembolic disease in Non-Hodgkins lymphoma has been studied in an effort to throw light on the incidence and predisposing factors in the development of venous thrombo-embolism in Non-Hodgkin's lymphoma. [1] The aim is to determine the utility of risk stratification and assessment for VTE risk in order to administer prophylaxis. Current VTE prophylaxis is not part of routine management in non-Hodgkin's lymphoma. [2] Maneval et al carried out a prospective study on patients with Non-Hodgkin's lymphoma in whom a central venous line was used (peripherally inserted

central venous cannula). [2] They found a 2-fold risk of developing upper extremity DVT. Many incidents of upper limb thrombosis occurred within the first three months of diagnosis. African Americans were also found to have a higher likelihood of developing upper limb DVT post chemotherapy use in NHL. A prospective cohort study was conducted where they identified the risk factors in NHL for thromboembolic phenomenon which could help in risk stratification. [3] In the study, they determined that the strongest risk factor for developing VTE was initiation of chemotherapy. Patients with Non-Hodgkins lymphoma were at increased risk of deep vein thrombi at commencement of chemotherapy and the risk dropped as chemotherapy progressed. Most developed by the end of the third month on chemotherapy. Other strong risk factors included the presence of B symptoms, tumor burden or stage at diagnosis and the use of doxorubicin.3This patient had multiple risk factors that predisposed him to development of deep venous thrombosis; He was in the 84th day of chemotherapy, his chemotherapy contained doxorubicin as a component (CHOP and EPOCH) chemotherapy. He had no stage B symptoms, no central catheter inserted and he was not at an advanced clinical stage.

Upper-extremity DVT accounts for 5-10% of all cases of deep vein thrombosis. The axillary and subclavian arteries are the most common sites affected. There are two forms called primary and secondary upper extremity deep vein thrombosis based on aetiopathogenesis. Secondary upper extremity deep venous thrombosis is mostly caused by lymphoma. [4] Risk factors like cancer-induced prothrombotic states and/or the venous stasis resulting from vein compression or infiltration are the predisposing factors in the generation of thrombi in cancer patients even without catheter implantation. [5]

While this patient did not have a central venous line inserted, he was given bolus chemotherapy through a peripheral venous access line which may cause microtrauma to the intima of the axillary vein, compounding a prothrombotic state, increased his risk for development of upper extremity deep venous thrombi. Secondly, the tumor compressed his axillary vein. Because thrombosis can complicate and interrupt treatment, as well as increasing medical costs, morbidity and mortality, [6] thromboembolism has been implicated as a leading contributor to death in patients with cancer. Any patient with cancer (even those without evidence of metastasis) is 47 times more likely to die from the venous thromboembolism compared with the general population without cancer. [7] Cancer related thrombosis is also the leading cause of death in ambulatory patients receiving active therapy without evidence of metastasis. [7] Patients with Non-Hodgkin's lymphoma should therefore all be assessed for risk of developing venous thromboembolism. Due to the amount of risk factors for thrombosis found in these patients, primary thromboprophylaxis should also be considered routinely in all patients diagnosed with Non-Hodgkin's lymphoma.

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