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Unexplained Recurrent Multiple Thrombosis; The Role of Nurse Practitioner in the Prevention of Complications Through Early Investigations and Diagnosis. A Case Study

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ABSTRACT

Introduction: Thrombosis is the development of a blood clot in the vessel that stops blood flow. Thrombosis is of two types; arterial and venous thrombosis. a clot in the vein is venous thrombosis or a clot in an artery is arterial thrombosis. Thrombosis in arteries or veins can result in a heart attack, ischemia, or stroke.

Purpose: Enhance knowledge of Nurse Practitioners (NPs) through overview, pathophysiology, etiology, clinical symptoms, comprehensive assessment, diagnosis process, and management of patients with unexplained recurrent thrombosis.

Method: A case study (Patient history, investigations, and management) and review of the literature.

Conclusion: Unexplained recurrent MI and thrombosis is a life-threatening condition and different etiologies and pathologies are involved in the occurrence of this condition. Time management and early diagnosis prevent patients from complications.

Implication for practice: By providing proper knowledge regarding the cause and pathology involved in unexplained thrombosis, a nurse practitioner can properly asses and diagnosed the disease on time and can manage it on time, and can prevent complications in such patients. Without knowing the cause behind thrombosis it occurs again and again with complications and leads to mortality and disability.

Keywords: Recurrent thrombosis; DVT; Venous thrombosis; Arterial thrombosis

INTRODUCTION

Thrombosis is the formation of a blood clot in the vessel that prevents blood flow. Thrombosis may occur in arteries or veins locally**,** which can lead to heart attack, ischemia of different parts or organs of the body, and stroke. Thrombosis is of two types; arterial and venous thrombosis. a clot in vein is venous thrombosis or a clot in an artery is arterial thrombosis.[1] Causes of venous thrombosis are surgery (injury), immobility, fracture, obesity, medicines, autoimmune, coagulation abnormality, and inherited. The artery-hardening condition known as arteriosclerosis may contribute to arterial thrombosis (Atherosclerosis). The walls of the arteries thicken as a result of calcium or fatty deposits. As a result, artery walls may begin to accumulate fatty material (known as

plaque). When this plaque ruptures, a blood clot may ensue. The arteries are capable of developing thrombosis (coronary arteries) and leads to heart attack. A stroke may result from arterial thrombosis in a brain artery.[2] Myocardial infarction or heart attack is a condition when blood flow is compromised to the heart and the heart muscle is unable to pump properly. The blockage of the coronary arteries is due to occlusion or thrombosis or plaque in the artery which prevents the flow of blood in the heart vessels.[3] The majority of deaths related to thrombosis in the US are caused by myocardial infarctions and cerebrovascular accidents (CVAs).

Thrombosis occurs mostly in 1st month of surgery due to the activation of cells responsible for healing and clotting. Thrombosis may occur everywhere in the vessels of the body. It has different symptoms and signs depending on the area of involvement e.g. pulmonary embolism, myocardial infarction, and stroke. Thrombosis reoccurs in patient who have more risks or with unknown causative agents.

CASE STUDY

A 52-year-old male known case of ischemic heart disease (IHD) and DVT, presented to the Emergency department with a complaint of chest pain that started one day back. The pain was moderate and radiating to the shoulder, back, and to jaws. Associated with mild nausea and sweating. The pain was increased with exertion and decreased with resting. The patient had experienced chest pain in September 2022 and was diagnosed before as CAD (S/P CABG) and was also diagnosed in 2009 with DVT and pulmonary embolism (treated with anticoagulants).

Pathophysiology

Early thrombosis in vessels can be due to technical problem (such as graft damage during harvesting, anastomotic inadequacies), mismatch preexisting graft pathology, or extrinsic factors (such as hypercoagulability producing acute thrombosis) injury to the vessel or immobilization. Endothelial denudation and smooth muscle cell (SMC) harm occur as a result of mechanical stresses and ischemia-reperfusion injury during harvesting and storage. The extracellular matrix is made visible by de-endothelization, which also causes the tissue factor to activate the extrinsic coagulation cascade. Vasoconstriction and stasis are brought on by decreased prostacyclin and nitric oxide (NO) bioavailability, which in turn encourages fibrin deposition, activated platelet and leukocyte adhesion to the luminal surface, and thrombus formation.[4]

Within months of CABG, a condition known as "arterialization" known as SVG intimal hyperplasia develops as an adaptation to high arterial pressure. Though infrequently, it can result in considerable early stenosis and modest lumen decrease. Numerous cytokines (such as interleukin-1 and interleukin-6) and growth factors (such as platelet derived growth factor and transforming growth factor beta) are secreted by activated platelets to aid in the proliferation of SMCs. In parallel, thrombin is produced as a result of coagulation activation, and finally, polymerized fibrin is deposited. Both directly and through the production of PDGF by platelets, thrombin promotes SMC growth. Over a layer of platelets and fibrin, neo-endothelium starts to grow from the wounded zone's boundaries. SMC proliferation peaks four days after graft implantation, and the SMCs of the medial layer undergo phenotypic modification, changing from a dormant contractile state to a synthetic stage that resembles

fibroblasts and migrates to the intima. Extracellular matrix, which is made up of elastin, collagen, glycoproteins, and proteoglycans, is secreted and causes the intima to thicken even more. High proliferative adventitial fibroblasts go to the intima and transform into myofibroblasts, which aids in the thickening of the intima. The aforementioned processes initially begin at the anastomotic sites and then spread across the whole SVG. The development of intimal hyperplasia also involves innate immune system cells such mast cells and natural killer cells and thus thrombosis occur in the vessels.[4] The hyper coagulation state is responsible for thrombosis in the body by the same pathophysiology in which plaque is formed in the vessels due to fibrin formation also known as Red thrombi.[5]

History

Past medical history showed that the patient had a Pulmonary Embolism (PE) and Deep Venous Thrombosis (DVT) in 2009. Had Ischemic heart disease (triple vessel coronary artery disease) in September 2022**.** Patient did not have other comorbidities. Past surgical history showed that Coronary Artery Bypass (CABG) was done in September 2022.

Patient family history was not significant for any disease. Patients walk half an hour daily. Diet; sleep was normal, no any change in weight. Patient did not have any allergy to drug and food.

Physical examination

The patient had a heart rate of 80 beats/minute, Blood pressure of 120/70 mmHg, Respiratory rate 18 breaths/minute, SPO2 99% on room air, and was Afebrile.

Central Nervous System was intact with GCS of 15/15. Abdomen was soft non tender.

The cardiovascular assessment was normal (S1+S2+0). No edema was seen. While the respiratory assessment revealed bilateral equal entry, No cyanosis, No Short of breath.

Investigations

Full Blood Count, APTT, PT/INR, LFTs, RFTs, Serum Electrolytes, Blood Sugar and HbA1c were in the normal range. Although Troponin I was in the high range of 9,564.

ECG showed T wave inversion in leads I, II, AVF, V1 to V3 and ST depression in V3-V6. Echo result revealed EF 45%, mild MR. Coronary Angiography showed that LAD (SVG) 50%, and PDA, RCA (SVG) was 100% occluded/thrombosed, SVG-OM Normal, while the native three coronary arteries were fully occluded. Chest X- ray was unremarkable.

After physical examination and investigation, the patient was diagnosed with inferio-posterior wall MI as a result of graft thrombosis.

Treatment

Pharmacological management: Patient was treated as acute coronary syndrome because of thrombus in the venous graft and was loaded with aspirin/ clopidogril 300+300mg and Injection Heparin IV 5000units stat.[6] Routine medication was started including tab glyceryl Trinitrate 2.6mg BD was given to relive chest pain as result of vasodilation. Tab Clopidogril 75mg BD and Tab Aspirin 150mg OD was given as antiplatelet to prevent further thrombosis or embolism.[7] Tab Bisoprolol 2.5mg OD was given as heart rate limiting drug to treat angina. Tab Rosovistatin 20mg HS was started as anti-lipid also for anti-inflammatory effect. Tab diltiazem 30mg BD started for prevention of angina and also for arrhythmia post MI.[8]

Non-pharmacological management: Patient left heart catheterization was done which showed 2 vessels graft thrombosis so percutaneous coronary intervention was done to the native right coronary artery.[9]

DISCUSSION

Thrombosis can lead to some complication and can cause obstruction in any vein or artery and can cause ischemia.[1]The patient was diagnosed in 2009 with Deep venous thrombosis (DVT) and pulmonary embolism and was discharged from the hospital by anticaugolents including, tab warfarin 5mg, but the patient was having poor compliance to medication and had stopped the warfarin by self. Poor compliance in such patients can lead to re-thrombosis.[10] Following DVT and PE, patient developed coronary artery thrombosis, so in September 2022 patient was diagnosed as triple vessel coronary artery disease as a result of thrombosis in coronary arteries so due to triple vessel disease coronary artery bypass was done and the patient was discharged on dual antiplatelet to prevent from thrombosis re-occurrence. Dual anti platelets have better outcome and patient have less risk of thrombosis as found by meta-analysis and randomized control trails.[11] Patient was having a mild rectal bleed and stopped the antiplatelet so few days after stopping the antiplatelet patient presented to the emergency department with chest pain and investigation showed that the patient had developed graft thrombosis and had MI (Inferior-posterior NSTEMI). LHC was done and graft study showed thrombosis in two grafts. The patient who had great risk of thrombosis could continue the dual antiplatelet, until unless had high risk of bleeding to prevent reoccurrence of thrombosis.[12]

Patient who are at risk of thrombosis should not stop any blood thinner without cardiologist advice because it is very important to prevent recurrent event of thrombosis.[13] Poor compliance to anticoagulants and blood thinner in such patient can lead to further complication and recurrent thrombosis.

Multifaceted approach is required for diagnosis of thrombosis include physical examination/assessment and diagnostic tests.[14] Rule out the cause in such patients are very important because by treating the actual cause we can prevent the further damage and complication. Coagulation profile is very important in such patient in which other risks are not present as this patient was not having any other risk of thrombosis. The blood coagulation profile may responsible for thrombosis.[15] In the current case the patient coagulation profile and blood factor were checked for hyper coagulation status on discharge from hospital on third event and the patient was referred to the hematology, upon checking the blood coagulation profile the patient LA SCREEN (45.9) and LA RATIO (1.2) were positive, which is risk for recurrent thrombosis.[16] It is important in such cases to rule out

the cause otherwise patient presents with thrombosis recurrently and also with complications. Anticoagulation is highly indicated in patients diagnosed with thrombosis to reduce progression of disease and post thrombotic syndrome.

In current case the patient had positive marker for coagulation which needed anticoagulation therapy so patient education is very important regarding proper teaching about medication adherence and proper follow up. In the current case the patient has stopped the anticoagulants and antiplatelet without physician advice and he developed re-thrombosis. The patient had DVT, PE, then Coronary artery thrombosis and now presented with graft thrombosis. Life style modification including proper exercise, avoid long standing and sitting, use of compression stocking regarding teaching should be given in such patients to prevent from further complication and reoccurrence of thrombosis.[17]

CONCLUSION

Thrombosis can lead to life threatening condition including MI, stroke, Pulmonary embolism and ischemia. Time management, early prevention, diagnoses is very important to prevent from further disease progression. Patient with recurrent thrombosis can be investigated for the underlying cause because without knowing the cause in such cases the patient can lead to further complications. Patient and family should be educated about the disease process and compliance and adherence to the medical therapy also should educate for life style changes. Because the poor compliance and non-adherence to medicine can increase morbidity and mortality in such patients.

Disclosure

Patient identification not disclosed and the patient permission was taken for publication.

Conflict of interest

The author has no conflict of interest.

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