



A CASE REPORT ON DRUG INDUCED DVT IN A 12 YEARS FEMALE CHILD

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ABSTRACT: Deep vein thrombosis(DVT) is a life threatening condition characterised by formation of thrombus in the deep veins of pelvis or legs including calf , femoral and popliteal veins which leads to swelling of one (or) both legs associated with throbbing pain usually in calf or thigh, swollen veins. Males are mostly affected than females, it increases with age and accounts 1 in 10000 persons per year.DVT is mostly caused due to hereditary (mutations in the prothrombin gene), acquired (oral contraceptives secondary to cancer, pregnancy, haemophilic treatment) and idiopathic. Apart from these elements, factors that can cause DVT are hypercoagul ability, stasis and endothelial injury.DVT was diagnosed by USG, D-Dimer, MRI, Contrast venography and treated with LMWH,Vit-K antagonists, Thrombolytics. A 12years old female child with chief complaints of left lower limb swelling since 10days associated with pain, unable to walk since morning, H/O trouble in walking from past 10 days and is progressive in nature.H/O using tab ovral-L since 3months for puberty menorrhagia. Child underwent left lowerlimb Doppler study, impression was found to beacute left lower limb DVT extending proximally up to the bilateral common iliac veins. Varicose veins in great saphenous territory. Mild subcutaneous oedema in the thigh, leg, foot and ankle regions. Patient Hb and RBC were below normal, INR was increased to 1.18(0.92-1.09).child was treated with LMWH, antibiotics, antiemetics, analgesics, antacids, foot elevation. The patient lower limb swelling was reduced within 5 days and got discharged.

KEYWORDS: DVT-Deep vein thrombosis, LMWH- Low molecular weight heparin, endothelial injury,stasis, hypercoagul ability.

I. CASE PRESENTATION–

HISTORY OF PRESENT ILLNESS

A 12 years old female child brought to hospital with chief complaints of left lower limb swelling since 10 days associated with pain, unable to walk since morning.

H/O of trouble in walking from past 10 days and is progressive in nature.

PAST HISTORY:

H/O of using Tab. OVRAL – L (Ethinylestradiol (0.03mg)+Levonorgestrel (0.15mg)) - since 3 months

MENSTRUAL HISTORY:

Age of menarche – 11 years

Menstrual periods – Irregular

Early menses + (for every 15 days)

Menstrual cycle - 7/20-25 days – with heavy flow

SOCIAL HISTORY:

No significant social history.

FAMILY HISTORY:

No significant family history.

PERSONAL HISTORY:

Diet – mixed diet

Sleep – adequate

Appetite – adequate

Bowel and bladder – normal

GENERAL EXAMINATION:

Pt – c/c

Temp – Afebrile

BP – 110/60 mmhg

PR – 78 bpm

RR – 23 /min

CVS – S1+S2+

RESP – BAE +

CNS – NAD+

P/A - Soft

LABORATORY INVESTIGATIONS:

CBP

Hb – 10.6 gm% ↓ (12-14gm %)

RBC – 3.08 million/cmm ↓ (4-6M/cmm)

WBC – 9,700cells/cmm (4,000 – 11,000)

PLT – 5.21 lakhs/cells (1.4 lakhs – 4.4lakhs)

RENAL FUNCTION TESTS:

Sr.creatinine – 0.88 mg/dl (0.7 – 1.2 mg/dl)

Blood urea – 19.53 mg/dl (7-30 mg/dl)

COAGULATION PROFILE:

PT – 15.7 (N – 13.0 – 15.9 sec)

CONTROL – 13.0

INR – 1.18 ↑ (N – 0.92 – 1.09)

APTT – 36.0 (N – 34.7 – 37.6 sec)

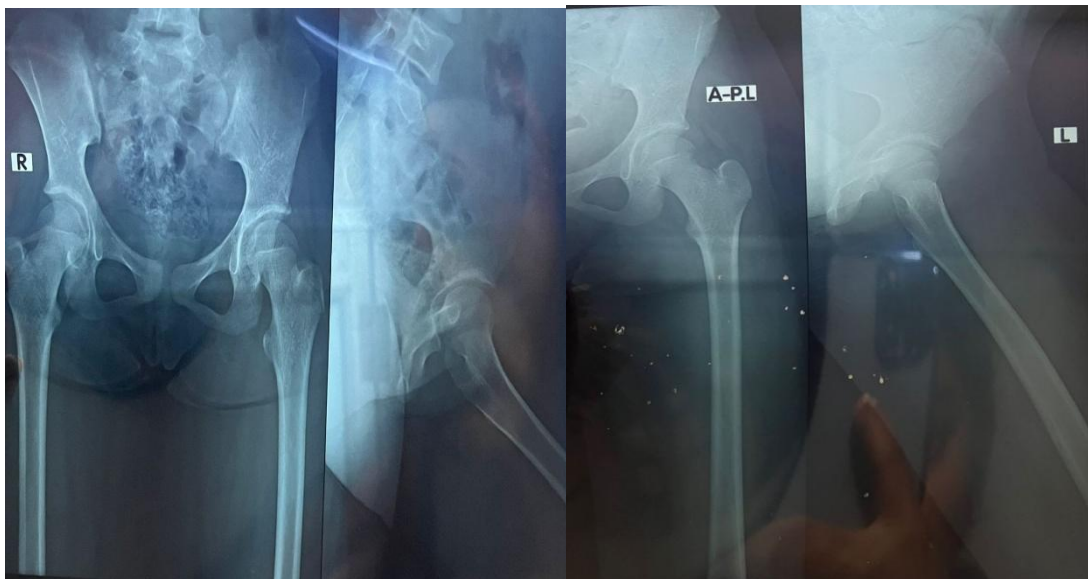
CONTROL – 34.0

LEFT LOWER LIMB DOPPLER STUDY:

Acute left lower limb DVT extending proximally upto the bilateral common iliac veins.

Varicose veins in great saphenous territory.

Mild subcutaneous oedema in the thigh, leg, foot and ankle regions.



TREATMENT:

IVF 20ml NS

INJ. MONOCEF 1gr in 100ML NS BD IV

INJ. PAN 40 mg OD IV

INJ. ZOFER 3mg BD IV

INJ. TRAMADOL 1amp in 100ml NS IV

INJ. ENOXAPARIN 40 mg BD SC

TAB. CHYMORAL FORTE BD PO

TAB. OROFER-XT – 1tab OD PO

FOOT ELEVATION

II. DISCUSSION

DEFINITION:

Deep vein thrombosis (DVT) is the formation of blood clots (thrombi) in the deep veins. It typically affects the deep veins of the pelvis or the deep veins of the legs, including the calf, femoral, and popliteal veins. It is a potentially dangerous condition that can lead to preventable morbidity and mortality.^[1]

EPIDEMIOLOGY:

Incidence of venous thrombosis, which includes pulmonary embolism (PE) and deep vein thrombosis (DVT), is roughly 1 in 1000 in adult populations each year. Males have slightly greater rates than females.^[2]

Venous thrombosis is an aging-related condition that starts slowly at 1 in 10,000 cases per year before the fourth decade of life, increases quickly after 45, and exceeds 5–6 cases per 1000 cases by the time an individual is 80 years old. In one of the study it states that individuals 85 years of age and above was 13 times higher than for those 45 to 55 years of age, with an absolute rate of 7 per 1000 annually.^[2]

ETIOLOGY:

The etiology of DVT can be divided in three main groups: idiopathic, hereditary and acquired.

Idiopathic acquires around 50% of cases.

Hereditary is primarily caused by mutations in the prothrombin gene, specifically G20210A and Factor V Leiden, which are relatively uncommon in Asian and African people but frequent in healthy white cultures. DVT risk increases significantly with hyperhomocysteinemia, a genetic illness that affects homocysteine metabolism, vitamin B6 or B12 or folic acid insufficiency, renal failure, hypothyroidism, etc., is added to one of these conditions.^[3]

Acquired is caused mainly due to Secondary to Cancer (Ovarian, Colon, Lung, Brain) Pregnancy and puerperium, Oral contraceptives, Secondary to hemophilic treatment (F VIII elevated), Secondary to surgery or major trauma, Acquired blood conditions: antiphospholipid antibodies (lupus anticoagulants, anticardiolipin antibodies) hyperhomocysteinemia thrombocytosis heparin associated thrombocytopenia.^[3]

Aside from these elements According to few studies, dvt may also result from^[4]

1. 'hypercoagulability', either systemic or local
2. 'stasis' of the venous blood; and
3. Endothelium injury to the vein wall intima.

CLINICAL FEATURES^[6]:

- Throbbing pain in 1 leg (rarely both legs), usually in the calf or thigh, when walking or standing up
- Swelling in 1 leg (rarely both legs)
- Warm skin around the painful area
- Red or darkened skin around the painful area – this may be harder to see on brown or black skin
- Swollen veins that are hard or sore when you touch them

PATHOGENESIS^[7]

Inappropriate thrombus formation is a disruption of homeostasis and may result from an alteration in any of the factors listed below. The dominant influence, and the one factor that by itself can lead to thrombosis, is endothelial injury.

Endothelial Injury: Endothelial injury causes subendothelial collagen exposure and platelet adherence, among other changes; many factors can contribute to the injury, including hypertension, vasculitis, scarred valves, bacterial endotoxins, cholesterolemia, and chemicals from cigarette smoke.

Abnormal Blood Flow: Endothelial damage can result from changes in normal blood flow, such as stasis and turbulence. Aneurisms (dilated aortic and artery dilations) produce localised turbulence; when atrial fibrillation is present, a dilated atrium is a region of substantial stasis that may initiate the formation of thrombus. Other illnesses including sickle cell anaemia and polycythemia, which both increase a patient's risk of thrombosis, are linked to stasis.

Hypercoagul ability: Any modification to the coagulation pathway that puts the patient at risk for thrombosis is referred to as hypercoagulability. Primary disorders (e.g., mutations in factor V, allelic variations in prothrombin levels) and secondary disorders (e.g., tissue damage from surgery, fractures, burns, myocardial infarction, cancer, heparin-induced thrombocytopenia [HIT]) are the two types of hypercoagulability states. An increase in platelet aggregation and a decrease in prostacyclin, a powerful vasodilator and inhibitor of platelet aggregation generated by the endothelium, are thought to be the causes of hypercoagulability linked with ageing.

PATHOPHYSIOLOGY^[8]

The use of oral contraceptives has been associated with a higher risk of thrombovascular disease. Their impact on the haemostatic system may act as a mediator in this. Using pills causes coagulation Factors VII, X, and fibrinogen to become more active. The oral contraceptives progesterone and estrogen components are both necessary for elevated Factor VII levels. Some, but not all, of the investigations have also shown a decrease in antithrombin III levels. Oral contraceptive users have also been demonstrated to have increased fibrinolysis, which should counteract the coagulation pathway alterations. A decrease in plasminogen activator inhibitor I levels along with an increase in plasminogen (tissue plasminogen activator antigen) levels is assumed to be the cause of the increase in fibrinolytic potential.

TAB. OVRAL-L:

Composition -EthinylEstradiol (0.03mg) + Levonorgestrel (0.15mg)

Mechanism of action:-

Ovral -1 is a combination of (ethinylestradiol and levonorgestrel) oral contraceptive pill. It acts by suppressing gonadotropin hormone due to its estrogen and progesterone activity. The primary effect of estrogen is to inhibit the secretion of Follicle Stimulating Hormone (FSH) and progesterone action is to inhibit Luteinizing Hormone, thereby suppress ovulation.^[10]

Adverse effects:-^[11]

The Ovral L contraceptive pill's potential adverse effects are:

- Heart attacks, brain strokes, venous thromboembolism and myocardial infarctions can result from blood clotting in arteries and veins throughout the body.
- An increased risk of developing breast cancer, cervical cancer (of the mouth, uterus, or womb), and hepatocellular carcinoma, or liver cancer. Hepatic adenoma (benign liver tumors), gallstones, inflammation of the gall bladder, bile stasis, jaundice, and pancreatic inflammation
- Tendency for the production and accumulation of harmful cholesterol in blood arteries.

RISK FACTORS

- Age
- Lack of movement
- Injury or surgery

- Pregnancy
- Oral contraceptives or birth control pills
- Obese
- Smoking
- Cancer
- Heart failure
- Inflammatory bowel disease

DIAGNOSIS:

Compression ultrasonography is the most preferred imaging test to diagnose DVT. The diagnostic criteria is the compressibility of a venous segment; however, Doppler (including colour flow) can be added to properly identify vessels and validate the compressibility of a specific segment.^[5]

D-dimer - A blood clot composed of cross-linked fibrin breaks down to produce D-dimer. Patients with acute venous thromboembolism and a number of nonthrombotic conditions (such as recent major surgery, bleeding, trauma, pregnancy, or cancer) often have increased D-dimer levels.^[5]

Levels of D-dimer can be popularly measured using three types of assay:^[1]

- Enzyme linked immunosorbent assay (ELISA).
- Latex agglutination assay.
- Red blood cell whole blood agglutination assay.

Contrast venography - the gold standard for diagnosing deep vein thrombosis (DVT), it is not frequently performed due to the greater suitability and accuracy of noninvasive tests such as D-dimer and venous ultrasonography for treating acute episodes of the condition. It entails cannulating a pedal vein and injecting a contrast agent (such as Omnipaque, which is typically noniodinated). A large volume of Omnipaque diluted with normal saline results in better deep venous filling and improved image quality.^[1]

Magnetic resonance (MRI) - This method is highly sensitive in identifying upper extremity venous thromboses as well as DVTs in the pelvis and calf. It is also relevant in ruling out differential diagnoses in patients suspected of DVT.

TREATMENT^[9]

Fondaparinux or low-molecular-weight heparin for five days or until the INR is more than two for twenty-four hours (unfractionated heparin for individuals with elevated risk of bleeding and renal failure)

Antagonists of vitamin K for three months

Low-molecular-weight heparin anticoagulation for six months should be considered for cancer patients.

If the patient has spontaneous DVT, vitamin K antagonists should be considered after three months.

The FDA and NICE recently approved rivaroxaban, an oral factor Xa inhibitor that is appealing because it does not require routine INR monitoring.

Change from heparin to fondaparinux, which is not linked to heparin-induced thrombocytopenia, if the platelet count falls to less than 75,000.

FOOT ELEVATION: Foot elevation helps in reducing pressure and enhancing circulation to prevent vein complications from arising.

III. CONCLUSION

In my case report, development of DVT is may be due to the usage of oral contraceptive pills. The patient was treated with low-molecular weight heparin and she recovered within 3 weeks.

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