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"A STUDY ON DEEP VEIN THROMBOSIS INDUCED BY EXCESS ORAL CONTRACEPTIVE USE AND ITS APPROACH THROUGH HOMOEOPATHY"

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Abstract

Deep vein thrombosis (DVT) is an obstructive disease with a hindering venous reflux mechanism. DVT usually involves the lower limb venous system, with a clot formation originating in a deep calf vein and propagating proximally.[12]
It is estimated that approximately 1 million people are diagnosed with DVT each year. Approximately 3,00,000 dies of venous thromboembolic complications, which exceeds that of acute myocardial infraction or acute stroke.[3]
The estimated incidence in the population at risk, adjusted for age and sex, is higher in men (130 events on 1,00,000 subjects) than in women (110 on 1,00,000 subjects). In fact, in younger populations, women have higher incidence of thromboembolic events linked to hormonal fluctuations that change the ratio between estrogen and progesterone, as occurs during pregnancy and puerperium.[1]
Birth control pills can slightly increase the risk of developing blood clots, including DVT.
The risk of developing a blood clot is highest in the first year of oral contraceptive pill (OCP) use. The risk decreases after the first year but remains until the pills are stopped.[14] women taking OCCP have higher risk of developing deep vein thrombosis (DVT), usually in the legs, and this may lead to pulmonary embolism, a serious complication. For every
1,00,000 women aged 15-44 years not taking pill, approximately 5-10 are likely to develop
blood clot in 1 year and this risk increases 3-4 times in those using second-generation OCCP and 6-8 times in those using third-generation OCCP.

Keywords: *Deep vein thrombosis, oral contraceptives, Virchow triad, homoeopathic approach, Trousseau syndrome, Features of DVT. Homoeopathic management, Homoeopathic remedies for DVT.*

BACKGROUND

Thrombosis of deep leg veins accounts for more than 90% of cases of thrombophlebitis and phlebothrombosis. In deep venous thrombosis (DVT) of the legs, prolonged immobilization resulting in venous stasis is the most important risk factor. This can occur with extended bed rest or even just sitting during long plane or automobile trips. The postoperative state is another independent risk factor for DVT, as are congestive heart failure, pregnancy, oral contraceptives use, malignancy, obesity, male sex and age over 50 years. Inherited defects in coagulation factors often predispose affected individuals to development of thrombophlebitis venous thrombi also can result from elaboration of procoagulant factors from cancers; the resulting hypercoagulable state can manifest as evanescent thromboses in different vascular beds at different times, so-called "migratory thrombophlebitis" or "Trousseau syndrome".[4]

Conventional treatment for DVT would be rest, elevation of limb, bandaging the entire limb with crepe bandage, and anticoagulants such as heparin/low molecular weight heparin, warfarin, phenindione.[5]

In homoeopathy the treatment would be based on individualization and holistic approach with remedies like Vipera, Apies, hamamelis, Secale cor, Lachesis etc., are prescribed for venous stasis, thrombotic tendencies, aiming to restore balance rather than suppressing the pathology.

VTE is an important concern for women's health. Pregnancy is a well-recognized risk factor for VTE. In addition, the combination of oral contraceptive pills (odds ratio [OR], 1.1-4.8) and hormone replacement therapy has been associated with an elevated risk of VTE. The increased estrogen state is primarily responsible for the increased VTE risk, although those that contain the progestin agent drospirenone have been associated with increased risk as well.[6]

AIM

To study the development of deep vein thrombosis (DVT) as a complication of excessive intake of oral contraceptive pills and to evaluate its therapeutic management through homoeopathy.

OBJECTIVE

1. To identify the risk factors that increase the chances of OCP-induced DVT (e.g. smoking, obesity, immobility, genetic thrombophilia).
2. To evaluate the clinical signs and symptoms observed in patients developing DVT after OCP use.
3. To highlight the complications of untreated DVT (such as pulmonary embolism).
4. To evaluate the scope of homoeopathy in the management and prevention of complications of DVT.

INTRODUCTION

A group of genetic traits has been identified that increase the risk of venous thromboembolism and, collectively may account for up to 70% of patients with deep vein thrombosis or pulmonary emboli. These mutations also heighten the risk of thrombosis in patients who are pregnant, use oral contraceptives, have malignancy, or taking certain medications**. (1) antithrombin (AT) deficiency:** this was the first group of disorders to be identified. It is an autosomal dominant trait and occurs in approximately 1 in 2000 individuals. Most patients with AT deficiency will develop symptoms of DVT or PE before 30years old. Relatives of patient with known AT deficiency should be tested and, if they carry the mutation, should avoid oral contraceptives and receive prophylaxis with elective surgery.

(2) protein S and C deficiency: these two proteins, similar to coagulation factors II, VII, IX, and X, are synthesized in the liver and require a posttranslational modification (gamma carboxylation of specific glutamic acids) for biological activity. Protein S acts as a high- molecular-weight cofactor and forms a complex with protein C and thrombomodulin to facilitate the inactivation of factors V and VIII. It exists in two forms: an active fraction that is free in plasma, and as inactive fraction bound to a steroid-binding globulin. Pregnancy and use of oral contraceptives can increase the level of this protein and thereby induce or exacerbate protein S deficiency. This reduction in protein S, when combined with another mild defect such as the factor V Leiden or prothrombin gene mutation, may account for the increase in DVT/PE in pregnancy and in users of oral contraceptives who were previously asymptomatic. **(3) Factor V Leiden:** this mutation R506Q is present in 5% of the Caucasian population but is uncommon in Africans, Asians, and Latinos. This mutation modifies one of the two protease-sensitive sites in factor V that are cleaved by activated protein C and thereby results in excess thrombin generation. Carrying the mutation increases the life-time risk of DVT/PE on oral contraceptives or during pregnancy often have this mutation. Homozygosity at this locus (inheritance of two defective genes) increases the risk of DVT/PE 30-fold to 80- fold, to 1 in 12 individuals. **(4) prothrombin gene:** the prothrombin gene mutation G20210A occurs in the 3' untranslated region of the gene rather than in the coding sequence. It stabilizes prothrombin mRNA levels and thereby increases the steady state level of prothrombin in plasma by 25% to 30%. This results in increased thrombin generation. The clinical course is quite similar to factor V Leiden.[6]

MATERIALS AND METHODS

Literature review was done from reference articles, authenticated text books, web sites focusing on deep vein thrombosis due to excessive use of oral contraceptives.

REVIEW OF LITRETURE INTRODUCTION

Deep vein thrombosis (DVT) is called as phlebothrombosis. It is a semisolid clot in the vein which has got high

tendency to develop pulmonary embolism and sudden death. Common
site of beginning of thrombus is soleal veins which later propagate proximally, often getting detached to cause acute massive pulmonary embolism or moderate sized emboli can cause pyramidal/wedge shaped pulmonary infarcts.[5]
Venous thromboembolism [VTE] is defined by the presence of deep vein thromboembolism [DVT] and/or pulmonary embolism [PE]. Venous thrombosis indicates the presence of clots within the vein, while phlebitis refers to the presence of inflammation within a vein. PE arises from DVT or intracardiac clots that embolize to the pulmonary arterial system.

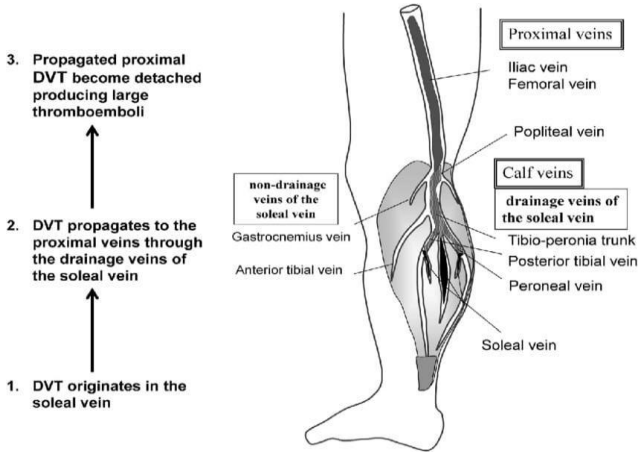

Image source: https://share.google/images/8jlCMVnH9l353f4J5 Venous thrombosis is classified as deep [proximal or distal] or superficial

- **Lower extremity proximal** DVTs occur in or superior to the popliteal vein, whereas distal lower extremity DVTs occur inferior to the popliteal vein
- **Upper extremity proximal** DVTs occur in the axillary vein or more centrally, whereas **distal upper extremity** DVTs occur in the brachial vein or more peripherally.

Superficial venous thrombosis [SVT] occurs in superficial vein near the surface, including the cephalic and brachial upper extremity veins and the lower extremity axial veins that include the great saphenous, accessory saphenous, and small saphenous veins.
PE may occur in **central** [main, lobar, or segmental pulmonary arteries] or **distal [**subsegmental or smaller pulmonary artery branches].[7]

The factors responsible for venous thrombosis in the absence of direct vein wall injury have been of interest for more than a century. In the year 1856, Rudolf Ludvig Karl Virchow, the
father of cellular pathology, proposed his classic triad elucidating the aetiology of venous thrombosis. He indicated that changes in blood elements (hypercoagulability), reduced blood flow velocity (stasis) vein wall injury (endothelial damage) combined to produce an environment promoting thrombus formation.[3]
Blood vessel damage or other events activate primary (platelet plug) and secondary hemostasis (coagulation cascade) to cause thrombosis. Fibrin strands stabilize the thrombus (converting fibrinogen to fibrin). Factor XIIIa cross-links the fibrin strands. Solidification of fluid blood into an aggregation of blood cells that is entangled within long fibrinous chains of molecules (fibrin) leads to thrombus. SVT may propagate into the deep vein system. Untreated DVTs may propagate proximally. Without treatment, half of the patients with proximal lower extremity DVT develop PE. DVTs that occur in upper extremities, often due to indwelling catheters, may also cause PE.[7]
Although the deep veins of the lower extremity are the most common location for DVT, thrombosis may also form in the veins of the upper extremities and pelvis or, less commonly, the splanchnic and cerebral veins damage from DVT may lead to dysfunction of valves of the deep venous system and, ultimately, the postthrombotic syndrome. Chronic lower extremity edema and calf discomfort characterize the postthrombotic syndrome and are associated with reduction in quality of life and impaired functional status that can last for months or years.
Postthrombotic syndrome is also associated with an increased risk of recurrent VTE.

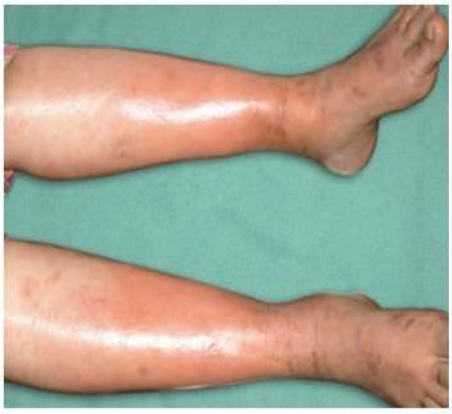

Fig. 1.398: Deep vein thrombosis (DVT) in both legs. 30% cases of DVT are bilateral.

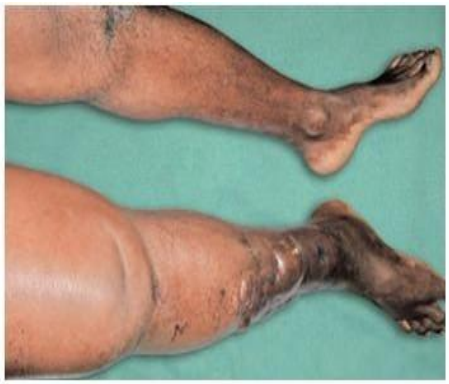

Fig. 1.399: Right leg venous gangrene. Note the discolouration, blebs and oedema.

Image source: Bhat M Sriram. SRB's Manual of Surgery. 5th ed. New Delhi: Jaypee Brothers Medical Publishers; 2016. Page no 223

Determining a patient's risk factors for VTE is critical because they determine the risk of recurrence, use of prevention, treatment duration. Patients are categorized as provoked when a cause is found; or unprovoked, when no cause is found. In 20% - 50% of cases, no
identifiable cause is determined.[6]

ORAL CONTRACEPTIVE PILLS

Definition:

Oral contraceptives of the combined type are almost 100 per cent effective in preventing pregnancy. They provide the best means of ensuring spacing between one childbirth and another. More than 65 million in the world are estimated to be taking the "pill" of which about 9.52 million are estimated in India.[8]

Types

1. Combined pill
2. Progesterone only pill (POP)
3. Post-coital pill
4. Once-a-month (long-acting) pill
5. Male pill

Historically oral contraceptives were introduced in the early 1960's. during the first decade of their use, investigations focused on the benefit of pregnancy prevention and risk of abnormal cycle bleeding. During 1970's, following their widespread use it became apparent that the oral contraceptives have some adverse effects principally on the cardiovascular system (e.g., myocardial infarction, deep vein thrombosis, etc.) and that these effects were associated with the estrogen component of the pill.[8]

Combined oral contraceptive (COC) pills are available in many formulations. Most formulations contain estrogen ethinyl estradiol, in doses of 35, 30, or 20 micrograms. There is also currently one formulation available with 10 micrograms of ethinyl estradiol and one with estradiol valerate. Oral contraceptive pills may be monophasic (same dose of estrogen and progesterone throughout the cycle) or multiphasic (varying doses throughout the cycle). Standard use of oral contraceptive pills is one hormonal pill for 21 days followed by 7 days of placebo pills.[6]

Early oral contraceptives [OCPS] contained higher dose of estrogen than what are currently used, and dose of 50 micrograms or higher are no longer recommended. All combined oral contraceptive methods are increasing the risk of venous thromboembolic disease [VTE]. The risk of VTE is greater in pills with 50 micrograms of ethinyl estradiol than in pills with 20 to 35micrograms.[6]

Women who had used the pill were reported to have a 40 per cent higher death rate than women who had never taken pill. Virtually, all the excess mortality was due to cardiovascular causes, that is myocardial infarction, cerebral thrombosis and venous thrombosis, with or without pulmonary embolus. [8]

Complications

1. Cardiovascular effects – women who had used the pill were reported to have 40 per cent higher death rate than women who had never taken the pill. Virtually all the excess mortality was due to cardiovascular causes, that is myocardial infarction, cerebral thrombosis, and venous thrombosis, with or without pulmonary embolus.
2. Carcinogens – however, the multicenter case-control study on the possible association between the use of hormonal contraceptives and neoplasia indicated a trend towards increased risk of cervical cancer with increasing duration of use of oral contraceptives.
3. Metabolic effects – these have included the elevation of blood pressure, the alteration of serum lipids with a particular effect on decreasing high-density lipoproteins, blood clotting and ability to modify carbohydrate

metabolism with the resultant elevations of blood glucose and plasma insulin.

4. Other adverse effects –

(i) liver disorders – the use of pill may lead to hepatocellular adenoma and gall bladder disease. Cholestatic jaundice can occur in some pill users.

(ii) lactation – preparations containing a relatively high amount of estrogen adversely affect the quantity and constituents of breast milk, and less frequently cause premature cessation of lactation.

(iii) subsequent fertility – in general, oral contraceptive use seems to be followed by a slight delay in conception.

(iv) ectopic pregnancies – these are more likely to occur in women taking progestogen-only pills, but not in those taking combined pills.

(v) fetal development – oral pills taken inadvertently during pregnancy might increase the incidence in birth defects of the fetus.

Common unwanted effects

(i) breast tenderness – breast engorgement, and fullness are said to be dependent on progesterone; pain and tenderness are attributed to estrogen.

(ii) weight gain – about 25 per cent of users complain of weight gain.

(iii) headache and migraine

(iv) bleeding disturbances. [8]

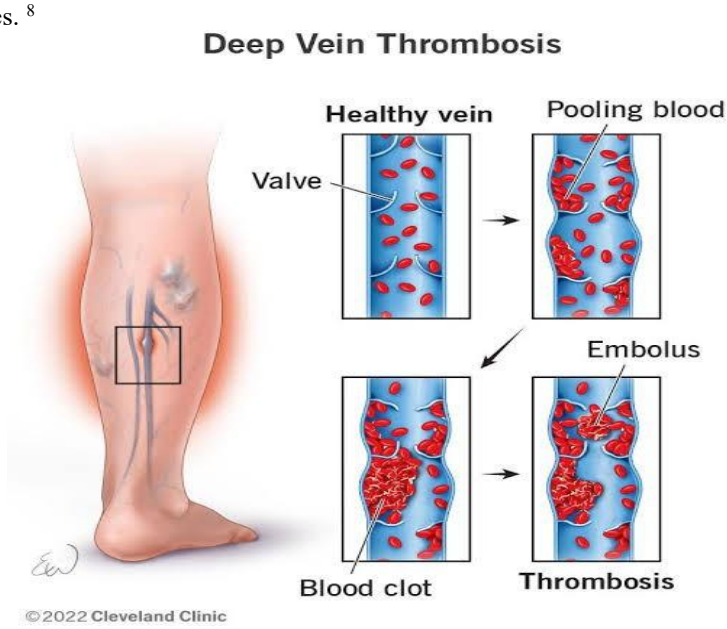

Image source: https://share.google/images/yOg7R7c6lDo1Tx1Yy

Risk factors associated with VTE, include both acquired and hereditary risk factors through promotion of **Virchow's triad** [blood stasis, endothelial injury, and hypercoagulability].

Acute illnesses and the associated immobilization.[6]

VTE are an important women health concern. Pregnancy is a well-recognized risk for VTE.

In addition, the combination of oral contraceptive pills and hormone replacement therapy has been associated with an elevated risk of VTE. The increased estrogen state is primarily

responsible for the increased VTE risk, although those contain the progestin agent drospirenone have been associated with increased risk as well. Along the same lines, tamoxifen, an antiestrogen hormonal therapy used in hormone-receptor positive breast cancer, also poses an increased risk.[6]

Features of DVT- commonly it is asymptomatic-60%; fever-most common; tender, tense, warm, pale or bluish, shiny swelling calf; positive Mose's sign (gentle squeezing of lower part of calf from side to side is painful. Gentleness is very important otherwise it may

dislodge a thrombus to form an embolus); Homan's sign (passive forceful dorsi flexion of the foot with extended knee will cause tenderness in the calf); Neuhof's sign (thickening and deep tenderness elicited by palpating deep in calf muscles); features of pulmonary embolism.[5]

INVESTIGATIONS - Duplex venous sonography is the initial imaging test of choice in the evolution of suspected lower and upper extremity DVT. No compressibility of a vein is diagnostic of DVT. Alternative imaging modalities for assessment of patient with suspected DVT, including CT, MR and contrast venography, may be warranted when ultrasonography is inadequate, such as when acute-on-chronic thrombosis is suspected.[6]

HOMOEOPATHIC APPROACH

1. Vipera

When the limb is allowed to hang down it seems as if it burst, it is so full of blood. Veins swollen, tense, sensitive; phlebitis; thrombosis; deep venous inflammation.[9] Thrombosis of deep veins, with swelling, bluish-black appearance,

and intense soreness. Cannot bear to let the limb hang down; if feels as it would burst.[10] venous system affected; varices, ulcers, phlebitis, purpura, tendency to thrombosis. The part becomes bluish, swollen, extremely painful, worse from letting the limb hang down.[11] Vipera causes great swelling of the affected limb, with bursting sensation on allowing it to hang down. Phlebitis; septic conditions; blood poisoned, tendency to form clots.[12]

2. Hamamelis

Venous engorgements, varicose veins, phlebitis, hemorrhages of dark, passive blood, never active, always passive.[9] Venous hemorrhages; blood dark, fluid, non- coagulable. Varicose veins, with soreness; phlebitis; bruised soreness of affected parts.[10] venous congestion; varicose veins and ulcers. Phlebitis; passive venous hemorrhage. Great soreness of affected parts, as after injuries.[11] Hamamelis acts especially on the veins, producing relaxation and engorgement. Hemorrhages from any part, passive, venous, dark blood; phlebitis, varicose, traumatic bruising.[12]

3. Secale cornutum

Tendency to gangrene from thrombosis; blood stagnates in the capillaries; skin shriveled, dry, cold, but the patient does not to want to be covered.[9] Passive hemorrhages, thin, fetid, dark, oozing; gangrene from thrombosis of small vessels. Limbs icy cold, yet cannot bear wearing.[10] produces contraction of unstriped muscular fibers, causes anemic condition, passive hemorrhages, tendency to gangrene. Thrombosis of small vessels with dry gangrene.[11] Secale acts powerfully on the circulation; causes contraction and thrombosis of vessels. Hemorrhages passive, thin, black, gangrene, with icy coldness, yet aversion to covering.[12]

4. Lachesis mutus

The venous system is disturbed, varicose veins, phlebitis, deep seated thrombi, tendency to clots. Hemorrhages dark, decomposed, offensice.[9] Septic states; purpura; hemorrhages black, fluid, non-coagulable, thrombosis, especially left-sided affections, with great sensitiveness of skin and intolerance of pressure.[10] septic conditions; disorganization of blood, hemorrhages of dark, decomposed blood. Venous affections, varices, ulcers, gangrene, phlebitis.[11] Acts on the blood, disorganizing it, produces hemorrhages and thrombi. Venous system profoundly affected; septic, left-sided, hemorrhagic conditions.[12]

5. Bothrops lanceolatus

Tendency to clot formation in the blood; thrombosis of cerebral vessels, hemiplegia, aphasia, great prostration and hemorrhages.[9] Hemiplegia from thrombosis; inability to articulate though conscious hemorrhagic diathesis; tendency to clots.[10] hemorrhages, from tendency to clots. Thrombosis; hemiplegia from thrombosis of cerebral vessels. Aphasia.[11] Bothrops has special affinity for the blood, causing in coagulability, but also thrombosis. Hemiplegia and aphasia from cerebral thrombosis are keynote conditions.[12]

6. Apis mellifica

Edematous swelling, pitting up on pressure, waxy, pale or transparent, inflammation of veins, phlebitis, erysipelatous conditions, with stinging pain.[9] Oedema, erysipelatous inflammation; dropsical swelling after scarlatina phlebitis. Burning, stinging pains; soreness, intolerance of heat.[10] acts on cellular tissue, causing oedema of skin and mucous membranes edematous swellings, erysipelatous inflammations. Dropsies; phlebitis; meningitis serosa.[11] Apis causes edematous swellings, erysipelas, phlebitis, serous inflammations. Thrombosis may follow its venous involvement; stinging pains, better cold applications.[12]

7. Pulsatilla

Venous constitutions; veins are full and turgid; varices, phlebitis, inflamed veins, tendency to form clots. Passive congestion, sluggish circulation, blue appearance.[9]

Adapted to venous constitutions, female complaints; varicose veins, phlebitis, stasis of blood. Hemorrhages from venous congestion, blood dark.[10] venous system affected; varicose veins, phlebitis, inflamed veins, tendency to thrombosis. Patient mild, yielding, thirstless, worse in warm room, better in open air.[11] Acts strongly on venous circulation. Venous stasis, varices, phlebitis tendency to thrombosis. Hemorrhages from passive congestion; veins blue, distended.[12]

8. Arnica montana

Traumatic states; phlebitis after injuries; extravasations; thrombosis following mechanical causes. Sore, bruised felling in my whole body.[9] After injuries; trauma to soft parts; phlebitis and thrombosis from mechanical causes. Bruised soreness, as if beaten, bed feels too hard.[10] effects of injuries, falls, blow. Venous congestion, phlebitis, ecchymoses thrombosis after trauma. Sore, lame, bruised condition.[11] Arnica affects the circulation by causing extravasations and venous stasis, phlebitis and thrombosis after mechanical injuries are well covered by it.[12]

9. Sulphur

Congestion of blood to single parts, especially to venous system; tendency to venous stasis, varices, phlebitis, hemorrhoids, ulcers, passive hemorrhages.[9] Congestion, especially venous; hemorrhoids, varices, venous stasis burning sensation everywhere. Passive hemorrhages from venous engorgement.[10] acts on the venous system producing congestion; varices, phlebitis, hemorrhoids. Hemorrhages, passive, dark blood. Chronic tendency to venous stasis.[11]

Sulphur has a marked action on venous circulation, producing passive congestion, varicosities, hemorrhoids, venous inflammations and ulcers, blood dark, venous, passive.[12]

10. Arsenicum album

Septic states, blood disorganized; tendency to gangrene from venous thrombosis. Hemorrhages dark, thin, offensive; great prostration, burning pains, restlessness.[9] Passive hemorrhages; blood thin, putrid, offensive. Gangrene from thrombosis; burning pains, restlessness, prostration, unquenchable thirst for small sips.[10] Great prostration, restlessness, burning pains. Septic states, tendency to gangrene from thrombosis; passive hemorrhages, blood thin, dark, offensive.[11] Arsenic profoundly alters the blood, producing septic, hemorrhagic states. Thrombosis, gangrene, passive hemorrhages; intense anxiety and restlessness accompany all symptoms.[12]

11. Carbo vegetabilis

Passive venous congestion; blood stagnates in the capillaries, surface bluish, cold, collapse states. Thrombosis in low fevers, with tendency to gangrene.[9] Venous stasis; skin bluish, cold; hemorrhages of dark, fluid blood. Collapse from blood- poisoning. Thrombosis, especially in debilitated states.[10] venous stasis; stagnant capillary circulation, cold surface, bluish skin. Passive hemorrhages, blood dark, fluid.

Collapse, gangrene, thrombosis.[11] Carboveg profoundly affects the venous system. Venous stasis, stagnation of blood in capillaries, coldness, blueness, tendency to putrescence and thrombosis.[12]

12. Sepia

Venous congestion, pelvic stasis, varices of limbs, phlebitis in women. Hemorrhages passive, dark, suited ton sluggish circulation.[9] Venous stasis, varicose veins, especially of female pelvis and lower limbs. Passive hemorrhages. Circulation sluggish; coldness, yet hot flushes.[10] acts especially on venous circulation of women; varices, phlebitis, passive, dark hemorrhages, stasis.[11] Sepia affects the venous system producing stasis, varices, phlebitis, passive hemorrhages. Suited to women with pelvic congestion and sluggish circulation.[12]

13. Natrum sulphuricum

Chronic effects of suppressed malaria; venous congestion of liver and portal system. Varices, hemorrhoids, sluggish circulation, tendency to thrombosis.[9] Complaints from damp weather; venous congestion of liver and portal system. Hemorrhoids, varices, phlebitis. Tendency to form clots in venous stasis.[10] acts on venous system; sluggish portal circulation; hemorrhoids, varices. Venous congestion, worse damp weather. Thrombosis in hepatic and portal circulation.[11] Natrum sulph. Acts chiefly on the venous side of circulation, causing stasis, varices, hemorrhoids, passive hemorrhages. Tendency to clot formation in portal system.[12]

14. Belladona

Belladonna produces congestion of blood in veins and arteries; intense inflammation; vascular engorgement. Thrombosis and phlebitis may arise with heat, redness, throbbing, and sensitiveness.[9] Congestion, especially vascular; blood rushes to head and chest. Sudden suppression of discharges lead to vascular stasis, venous engorgement, phlebitis.[10] belladonna acts powerfully on the vascular system. Active congestion, redness, swelling, throbbing pains. Phlebitis, varices, sudden inflammatory attackes.[11] Belladonna causes congestion and inflammation of venous system. Suppression of menses often results in vascular engorgement, phlebitis, or thrombosis.[12]

15. Nux vomica

Venous stasis in sedentary people; portal congestion; hemorrhoids, phlebitis. Thrombosis from sluggish circulation aggravated by allopathic drugging.[9] Hemorrhoids, constipation, venous congestion, phlebitis in sedentary or dissipated persons. Effects of drugs and stimulants producing venous stasis.[10] acts on portal system; venous congestion, hemorrhoids, varices, phlebitis, sluggish circulation, after debauchery or excessive drugging.[11] Nux vomica powerfully affects the venous system. Congestion of liver and portal veins, hemorrhoids, phlebitis. Thrombosis and venous obstruction after drugging.[12]

DISCUSSION

Deep vein thrombosis [DVT] is one of the most common vascular disorders, occurring when a blood clot forms in the deep veins, most often in the lower limbs. The development of DVT is influenced by three main factors, collectively known as Virchow's traid, sluggish blood flow [stasis], damage to the vessel wall [endothelial injury], and increased blood clotting tendency [hypercoagulability]. Among women, the use of oral contraceptive pills is a well-recognised risk factor because the oestrogen component in these pills promotes clot formation by increasing coagulation factors and reducing natural anticoagulants. Although modern pills contain lower hormone doses, the risk persists, particularly among women with inherited clotting disorders, obesity, sedentary life style, or a family history of venous thrombosis.

Conventional management of DVT includes rest, limb elevation, compression therapy, and use of anticoagulants like heparin or warfarin to prevent the clot from enlarging or travelling to the lungs, which could cause pulmonary embolism. While these measures are essential for acute management, they often do not address the person's constitutional susceptibility or long-term prevention.

Homoeopathy offers a holistic and individualised approach by treating the person as a whole rather than the disease alone. Medicines such as vipera, Hamamelis, Lachesis, and Bothrops act strongly on the venous system, improving

circulation and relieving symptoms such as swelling, heaviness, and bursting pains in the limbs. In chronic or complicated cases with septic or gangrenous tendencies, remedies like Secale cornutum, Arsenic album, and Carbo vegetabilis can help improve tissue vitality and prevent further degeneration.
When homoeopathic treatment is combined with preventive lifestyle measures such as avoiding prolonged immobility, staying hydrated, exercise regularly, and assessing hormonal use carefully, it not only helps in faster recovery but also reduces recurrence. Thus, homoeopathy can serve as a valuable supportive therapy alongside conventional management for better venous health and overall well-being.

CONCLUSION

All contraceptives that contain estrogenic compounds induce hypercoagulability of the blood in a dose-dependent fashion, increasing the risk of deep venous thrombosis, myocardial infarction, and stroke. The homoeopathic approach to DVT associated with oral contraceptive use is centered on the individualization of treatment, considering both the acute pathology and the patient's constitutional tendencies. Homoeopathic remedies like hamamelis virginiana, Vipera, Bothrops, and Lachesis are frequently indicated for acute venous statis, bursting pains, congestion, and hemorrhagic tendencies.

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