DEEP Vein Thrombosis

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10.1 Definition of Deep Vein Thrombosis

Deep vein thrombosis (DVT) occurs when thrombus forms in the deep veins of the limbs, and although many of these cases are asymptomatic, most (88%) typically involve the proximal veins, with 96% occurring in the pelvis and lower limb while 4% are found in the upper extremities [1]. Pulmonary embolism, the most feared complication, occurs when thrombus migrates into the lung accounting for the major cause of mortality [2].

10.2 History of Deep Vein Thrombosis

Thrombosis was known to ancient civilizations, appearing in Chinese writings in 2650 BC and was described as a pathological hemostasis [3]. The European entry to this landscape involves the Greek philosophers Plato (428–347 BC) who suggested that blood had "fibers" and Hippocrates, who observed that the blood of wounded soldiers on the battlefield "congealed" as it cooled (400 BC). The word "Thrombus" appears in the seventeenth-century texts, a transliteration of the Greek word θρομβος which is "clot" designated in the 1850s by Virchow [4]. He demonstrated that a clot is formed when blood semi-solidifies ex vivo and a thrombus, in vivo.

Eventually, van Leeuwenhoek discovered the presence of red cells as a component of blood, previously thought to be fluid, and Marcello Malpighi (1628–1694) described the flow of blood in the capillaries of the lung (1661) and the effect of stagnation in these capillaries. John Hunter (1728–1793) noted the coagulated blood had a malevolent association with the veins which were afflicted with

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"inflammation of the internal walls." Baillie observed that obstruction of the inferior vena cava led to thrombosis and Cruveilhier correlated the changes in the lung, thought initially to be lobar pneumonia, noting clots in the pulmonary arteries. He astutely observed that when these lesions were found in the lung, "others could be found somewhere else within the venous system." Hunter concluded that DVT was a venous occlusion caused by clots [5] and performed venous ligations above the clots [6] to prevent fatal venous thromboembolism to the lungs [7].

Around 1846, Virchow indicated that pulmonary emboli arose from thrombi in the pelvic and femoral veins (quoted by Jorpes, 1946) and that these formed from eddies in pockets of larger valves of the veins [8]. By 1856, he had put forward the theory that there was a triad of interactions between decreased blood flow in the veins, vein wall injury, and abnormality in the blood and concluded that **thrombi** and **emboli** formed from moving, not static blood.

Unconfirmed reports are that the first clinical description of DVT came from the ancient Indian surgeon Sushruta [9] who was regarded as the "Father of Surgery" in India, and his influential treatise, the *Sushruta Samhita*, contains the first known description of basic surgical procedures. Phlegmasia alba dolens, seen historically during pregnancy and puerperium with extensive DVT compromising arterial flow, was described in 1784 and Phlegmasia cerulea dolens, the painful "blue" leg, due to obstruction in the deep as well as in the superficial venous system, in 1857. Homans sign, pain in the calf on dorsiflexion of the foot to clinically demonstrate DVT, was described in 1944 [10].

10.3 Epidemiology of Deep Vein Thrombosis

The reported incidence of DVT in the Western world is approximately 50–80/100,000 population with increases during winter and increasing age [11]. Studies in the USA and Sweden show that the weighted mean occurrence of a first-time DVT in the general population was 50.4/100,000 people with a 20–30/100,000 per year increase in the 30-49-year age group, up to 200/100,000 per year in the over 70 age group [12]. Women had a higher relative risk of DVT than men in the >65-year age group [13] and men a twofold increase above 75.5 years [14].

Ethnicity and variations by regions have a great influence on incidence. The incidence of DVT and venous thromboembolism (VTE) in the population of California, USA, considered variable in ethnicity was high among Caucasians (230 per 100,000) and African Americans (293 per 100,000) but had a low incidence in Latinos (139 per 100,000) and Asian-Pacific Islanders (60 per 100,000) [15]. In the Black Caribbean population, the overall annual incidence of DVT was 11.0 per 100,000 person years with a sharp rise with age (both genders), but rare in pregnancy [16].

Independent studies showed that people of European ancestry generally have a DVT rate of between 45 and 117 per 100,000 population [14, 17] with Britain, Denmark, Norway, and France showing varied incidences of 40, 65, 93, and 124 per 100,000 population [18–20].

The incidence in Asian Indian patients was thought to be lower than in the West, but one study found that Indian patients undergoing major lower limb surgery had a rate of 43.2%, comparable to Western figures [21], and in Southern India, researchers found 194.9 cases per 100,000 admissions over a 6-year period [22]. It is possible that VTE is quite similar in incidence to that in the West [9].

In other Asian populations, the incidence was also considered to be low, but in Singapore, the frequency of DVT at one hospital was found to be 158/100,000 admissions [23], while in Malaysia patients undergoing orthopedic procedures exhibited a high incidence (62.5%) of radiographically diagnosed DVT [24].

Japan showed an incidence of 19.2 per 100,000 population and a doubling of the incidence from 2006 [25]. This was explained by the change from a traditional Japanese to a more Westernized lifestyle, increased obesity, more diagnoses of malignancy, and liberal use of the oral contraceptive pill [26, 27].

10.4 Pathophysiology of Deep Vein Thrombosis: Mechanism and Pathology

Deep vein thrombosis (DVT) occurs because of the triad of venous stasis, vessel injury, and hypercoagulability [28] (Virchow 1856). Although venous stasis is considered to be the most prominent [29], by itself, it may not be capable of promoting thrombosis.

Thrombosis appears where there is altered blood flow in relation to the "pockets" next to the valves of the deep veins [30, 31]. It is thought that a drop in the velocity of blood causes stasis and hypoxia [32], and these two combine with existing inflammatory changes to initiate thrombosis [33, 34]. Many of the vessel-based anti-thrombotic agents like thrombomodulin [35] and endothelial protein C receptor (EPCR) are physically expressed on the valves [36].

It is also believed that hypoxia can lead to stimulation of procoagulant factors [32] which exist in the endothelium and promote cellular aggregation [34]. The presence of tissue factor (TF) lends to favorable conditions [37]since cellular elements containing TF are known to play a major role in thrombus formation [38]. Active malignancies appear to have fragment pieces (membrane particles) which have procoagulant activity [39, 40], one of which is TF [41]. In addition, some substrates possess sites for carbohydrate-binding adhesion molecules called Selectins, and one of these, P-Selectin, interacts with immunologic cells containing TF. P-Selectin has procoagulant properties and appears to be driven here by hypoxia [42], but it also binds to mucin cells produced by malignant tumors [43], and this interaction is thought to cause thrombosis in these patients.

This risk increases with age because the quantity of procoagulants increases with age, but this is not complemented by a corresponding increase in naturally occurring endothelial anticoagulants such as protein C [44]. This also decreases when there are extended periods of illness and immobilization [45].

10.5 Sites of Deep Vein Thrombosis

The most common site recorded in three large studies was the **femoral-popliteal** segment - 74% [46], 42% [47], 34% [48], followed by the **ilio-femoral segment** - 58% [46], 38% [48], 23% [49], and the **calf segment** - 69% [50], 40% [46], 32% [49].

Labrapoulos et al. [51] found that 40% of "normal" color-coded duplex scans had acute isolated calf DVT when the muscular veins were imaged. When the calf veins were independently assessed, the peroneal vein 81% [50], 41% [51], the posterior tibial vein 69% [50], 37% [51], and the soleal 39% and gastrocnemius 29% [51] were the most common non-muscular and muscular calf vein sites to demonstrate thromboses. Yoshimura et al. also found that muscular calf veins were the commonest site for DVT [52].

Unusual sites for DVT included the deep femoral vein (0.31%), the deep external pudendal vein (0.04%), and veins in Klippel-Trenaunay syndrome (0.12%): the muscular thigh branch, lateral thigh branch, and sciatic vein [53].

10.6 Pathology of the Edema of Deep Vein Thrombosis

Edema of the lower limb can be a significant finding in DVT. Its cross-sectional area, estimated by computerized tomography, is approximately 300 mls, 50% of which appears in the extracellular fluid [54]. In DVT, there is an increase of about 200 mls, most residing in this extracellular fluid [55].

The pathology of this edema appears to have its basis in the transcapillary forces at work in the limb. Under normal circumstances, these forces keep the fluid balance in dynamic equilibrium; however, this is disrupted in the presence of thrombosis [56].

Venous pressure at the foot in the upright patient exceeds 100 mmHg and at this pressure, transcapillary filtration is increased, which increases the fluid in the interstitium [57]. This increase in capillary filtration is thought to be the major cause of edema in DVT, but there is also a corresponding decrease in the resorption of fluid at the venous end of the capillaries [58]. The crucial factor usually preventing this is the calf muscle pump mechanism, which normally empties the sinusoids on contraction, e.g., during walking [59], and on relaxation the veins expand lowering the pressure significantly. However, this activity is severely impaired in DVT, since the deep venous pump system is not intact and may be paralyzed.

The lymphatic system also plays a role in edema of patients with DVT [60]. There is a reduction in the clearance of injected ^{131}I-labeled albumin from the subcutaneous tissue in patients with lymphedema, and there is an observed decrease in cutaneous lymphatic flow which is an important factor in the production of edema in DVT [61].

10.7 Risk Factors Associated with Deep Vein Thrombosis

Age and ethnicity are major risk factors, with DVT increasing with age for both male and females [62] with Afro-Americans and Caucasians having a greater risk of DVT than Hispanic and Asian-Pacific Islanders [15]. Surgery is an independent risk factor with the incidence of postoperative DVT increasing with patient age, duration of surgery [63], and type of surgery [64]. DVT also remains a life-threatening complication after major trauma [65], and at presentation, 25% of trauma patients show evidence of hypercoagulability [66]. Immobility brought about by prolonged bed rest, nursing home confinement [67], air travel [68], or hospitalization poses a greater than 100-fold risk of thrombotic events than in the community [69].

There is an increased risk of DVT in active malignancy [70] with first-time idiopathic DVT in normal individuals being a marker for malignant disease [71]. In the year which precedes this diagnosis, there is usually an unexplained rise in VTE associated with advanced disease [72] and also a sharp increase in the diagnosis of malignancy in the following years, persisting for up to 10 years after initial admission for DVT [73].

Medical disorders capable of inducing thromboses include chronic kidney disease, inflammatory bowel diseases (1.98-fold) [74], clinically proven HIV (2- to10-fold) [75], and heparin induced thrombocytopenia [76]. Diabetes mellitus (type 1 and 2), with diminished fibrinolysis, showed a tendency toward thrombosis twice that of the nondiabetic population [77]. Congestive cardiac failure patients are considered at risk of developing DVT, and the longer the hospital stay, the greater the risk [78]. Women have increased risks in pregnancy and the puerperium [79, 80], with the pill, hormonal treatment, and hormone replacement therapy [81, 82].

Hematological diseases such as polycythemia vera, leukemia, Hodgkin's and other lymphomas, myeloproliferative disorders [73, 83, 84], and relapsed and refractory multiple myeloma all have an increased risk of DVT [85, 86]. Anticoagulant factor deficiency in antithrombin III, proteins S and C [87], and the lesser known protein Z [88] also produce increased risks of DVT. The factor V Leiden thrombophilic gene mutation [89], prothrombin G2021OA gene mutation [90], and the novel prothrombin variant C20209T [91] are seen in patients with DVT and a strong family history of thrombophilia [92]. In addition, elevated levels of factor VIII carry a high risk of DVT [93] as seen in Afro-Caribbean populations where thrombin production is elevated [94].

The risk of incident DVT among varicose vein patients [95] appears due to increased inflammatory and hypercoagulation factor markers in their plasma [96]. Klippel-Trenaunay syndrome, due to venous stasis [97] and May-Thurner syndrome by venous compression [98] also produce severe deep vein thromboses. Superficial venous thrombosis (SVT) considered a "benign" disease is now regarded as part of the paraneoplastic syndrome [99] since there is a clear association between migratory thrombophlebitis and malignancy [100], based on the observations of Trousseau [101]. SVT clearly is not always benign nor self-limiting as previously thought [102].

Table 10.1 Risk factors for deep vein thrombosis

High Risk
➢ Major surgery >3 h: Orthopedic, transplant, cardiovascular, trauma ➢ 75 yrs. old, current smoker, major trauma, # pelvis/femur/tibia ➢ Active malignancy +/− chemotherapy, oral contraceptive pill ➢ Acute spinal cord injury; neurological disease with leg paresis ➢ Stroke <1 month, BMI >50 kg/m^3
Medium Risk
➢ Major surgery (arthroscopic + laparoscopic surgery) >30 min ➢ 60–74 yrs old, confined to hospital bed >3 days with acute illness ➢ Recent VTE (DVT, PE), family history VTE, superficial venous thrombosis (SVT), varicose veins, venous malformations, venous compression ➢ Positive factor V Leiden and prothrombin 20210A, +ve lupus anticoagulant, elevated anticardiolipin antibodies, heparin-induced thrombocytopenia (HIT); other thrombophilia: proteins S, C, Z, and antithrombin III deficiencies. ➢ Central venous access, BMI ≥35 kg/m^3, non-contraceptive estrogen+progestins, immobilizing plaster cast, long-distance travel ➢ Inflammatory bowel disease, congestive cardiac failure, acute myocardial infarct <1 month, sepsis <1 month, pregnancy/ puerperium
Low Risk
Age >40 years, minor surgery, elective abdominal and thoracic surgery <30 min, no other risk factors
Age <40 years, minor surgery, uncomplicated abdominal or thoracic surgery, no other risk factors.

Caprini [103], Agarwal et al. [104],Grant et al. [105]

10.8 Diagnosis of Deep Vein Thrombosis

The diagnosis of a deep vein thrombosis (DVT) requires consideration of clinical findings, hematological investigations, and imaging. Additional consideration must be given to the level and extent of thrombus in the deep veins, whether the thrombus is completely or partially occlusive and if there are complications.

The diagnosis should be entertained in patients who present with acute unilateral leg swelling, erythema, warmth, tenderness, and engorged superficial veins [Fig. 10.1]. Since the clinical presentation of DVT is variable, scoring systems have been established to make a definitive diagnosis. The Wells score is a useful tool in evaluating and guiding management of DVT [Table 10.2]. The probability scores estimate the likelihood of DVT by incorporating signs, symptoms, and risk factors. According to the NICE guidelines, the sensitivity and specificity for DVT ranged from 77% to 98% and 37% to 58%, respectively [106].The Wells score clearly boasts of good sensitivity but lacks specificity and should not be used as a sole diagnostic modality.

10.9 D-Dimer

Following thrombus formation, the fibrinolytic response is activated resulting in the generation of plasmin and the release of fibrin degradation products including D-dimer. The D-dimer level is elevated in patients with acute DVT and can be

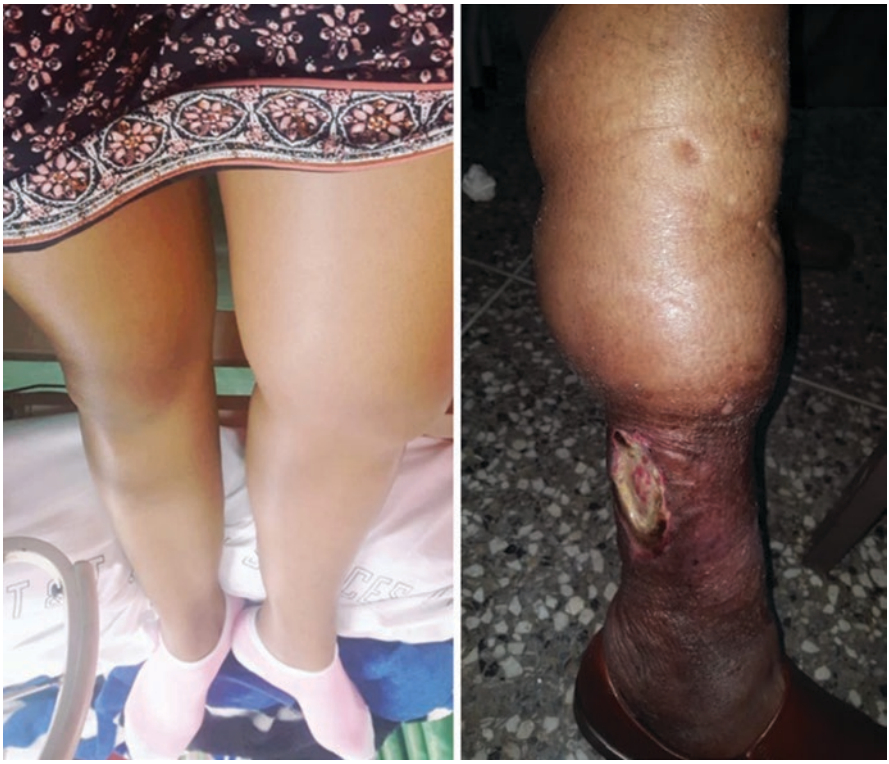

Fig. 10.1 Left: Acute unilateral limb edema with ilio-femoral DVT. Right: Post-thrombotic syndrome - lipodermatosclerosis with venous ulcer and proximal unilateral leg edema

Table 10.2 The Wells score [107]

Clinical Feature	Points
Active cancer (on treatment, treated in the last 6 months or palliative)	1
Paralysis, paresis, or plaster immobilization of the lower limb	1
Bedridden for 3 days or more, or major surgery in the past 12 weeks requiring general or regional anesthesia	1
Localized tenderness along the distribution of the deep venous system	1
Entire leg swollen	1
Calf Swelling 3 cm larger than the symptomatic side	1
Pitting edema confined to the symptomatic leg	1
Collateral superficial veins (non-varicose)	1
Previous DVT	1
Alternative diagnosis is at least as likely as DVT	−2
Clinical probability simplified score	Points
DVT likely	2 points or more
DVT unlikely	1 points or less

quantitatively assessed. It can also be elevated in conditions such liver disease, pregnancy, malignancy, and post-surgery. The sensitivity and specificity for D-dimer tests ranged from 75% to 100% and 26% to 83%, respectively, implying that this test is not suitable for confirming the presence of DVT, but can assist in eliminating it [106].

10.10 Ultrasound (US)

US is preferred as a first-line imaging modality for the diagnosis of DVT, and diagnostic criteria include non-compressibility, increased intraluminal echogenicity, absence of flow augmentation, and reduced or absent blood flow. US examination has high sensitivity and specificity for the diagnosis of symptomatic proximal lower extremity DVT when compared to conventional venography [Fig. 10.2]. The diagnostic performance is consistent in the femoral and popliteal veins, but less in the ilio-caval region and below the knee [108, 109]. It has improved diagnostic accuracy and reproducibility and is the preferred first-line diagnostic test for patients with suspected chronic DVT [110, 111].

10.11 CT Venography

Since ultrasonography may be influenced by body habitus and operator dependence, CT venography can be complementary in achieving diagnostic accuracy, proximal extent, and characterizing extrinsic compression in the case of pelvic masses and May-Thurner's syndrome [112–114]. CT venography can also be incorporated into an examination that includes pulmonary CT angiography for evaluation of both PE and proximal DVT [115]. In patients with suspected PE, a recent meta-analysis found that CT venography for the diagnosis of proximal DVT has a high sensitivity (95.9%) and specificity (95.2%) [Fig. 10.3] [116].

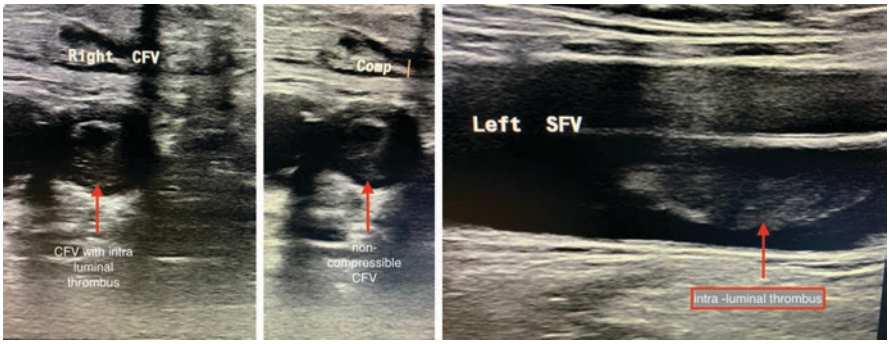

Fig. 10.2 (Right) Thrombus in common femoral vein will fail to compress on ultrasonography. (Left) Partially occlusive thrombus in superficial femoral vein

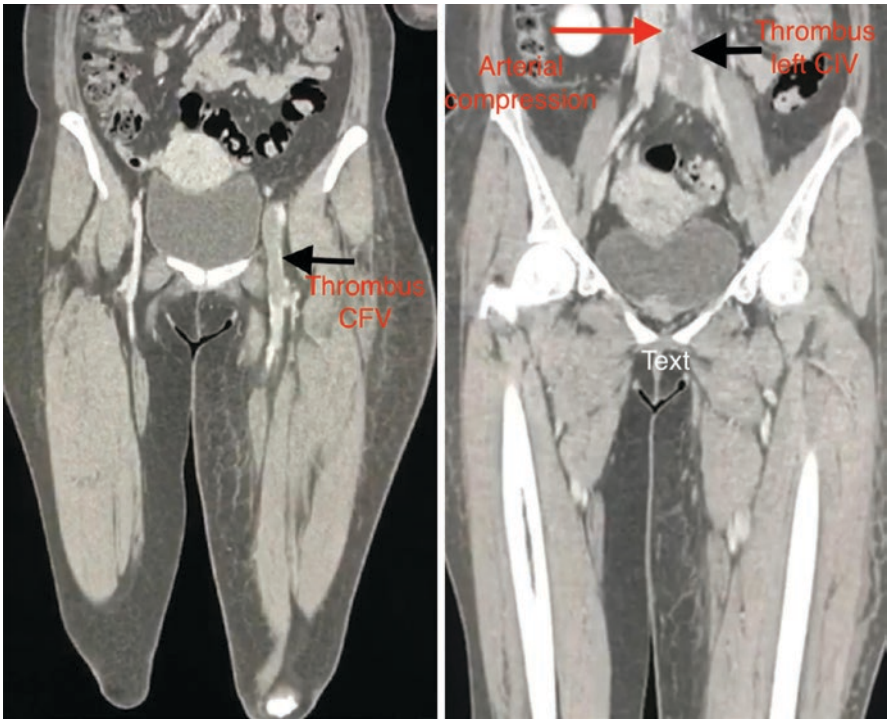

Fig. 10.3 (Right) CT venogram demonstrating thrombus in left CFV extending to the CIV. (Left) Diagnosis of May-Thurner's syndrome

10.12 Magnetic Resonance Venography

Magnetic resonance (MR) venography shares many of the clinical advantages of US, such as preventing exposure to ionizing radiation or iodinated contrast media. It also has the advantage of cross-sectional imaging for delineation of extravascular anatomy and identification of extrinsic venous compression, which may be an underlying cause of lower extremity DVT.

10.13 Venography

Ascending venography can accurately identify post-thrombotic changes in the deep venous system, the collateral patterns, and status of ilio-caval veins. It is useful for determining whether endovascular or surgical intervention is needed and which procedure is preferable. Descending venography can determine the extent of the reflux and may be useful for determining whether deep venous reconstructive surgery is needed and what type of surgery is feasible [117]. Imaging with intravascular

ultrasound (IVUS) with cross-sectional views of the vein and adjacent structures has high diagnostic accuracy for ilio-caval thrombus burden or ilio-caval compression. This may influence the therapeutic options in terms of the accuracy and efficacy of endovascular venous techniques (Fig. 10.4) [118, 119].

10.14 Hematological Investigations

Thrombophilia is an acquired or inherited predisposition to venous thrombosis, and inherited thrombophilia includes deficiencies in one of the three natural anticoagulants—antithrombin III, protein C, and protein S, which have been linked with familial venous thrombosis.

According to the International Consensus Statement (2005), screening for thrombophilia should be performed in (1) all patients with a first episode of spontaneous VTE; (2) patients with VTE under the age of 50 years even with a transient predisposing factor; (3) patients with VTE whose only risk factor is oral contraceptive therapy, estrogen replacement therapy, or pregnancy; and (4) patients with recurrent VTE irrespective of the presence of risk factors. Genetic thrombophilia testing is not routinely recommended in all patients with DVT. [121]

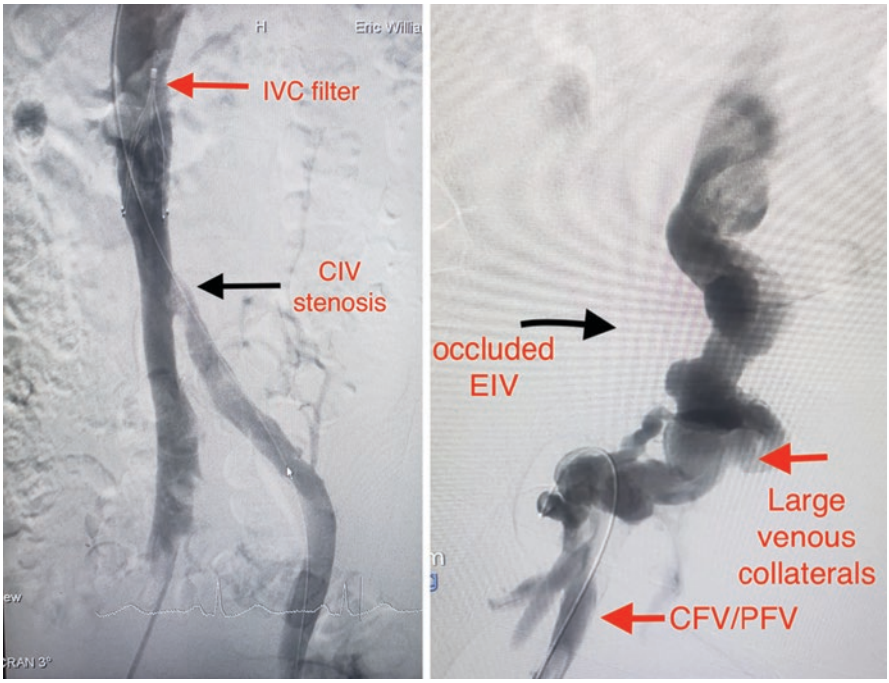

Fig. 10.4 (Right) Ascending venogram with IVC filter in situ filled with thrombus and left CIV stenosis. (Left) Chronically occluded external iliac vein with large collaterals

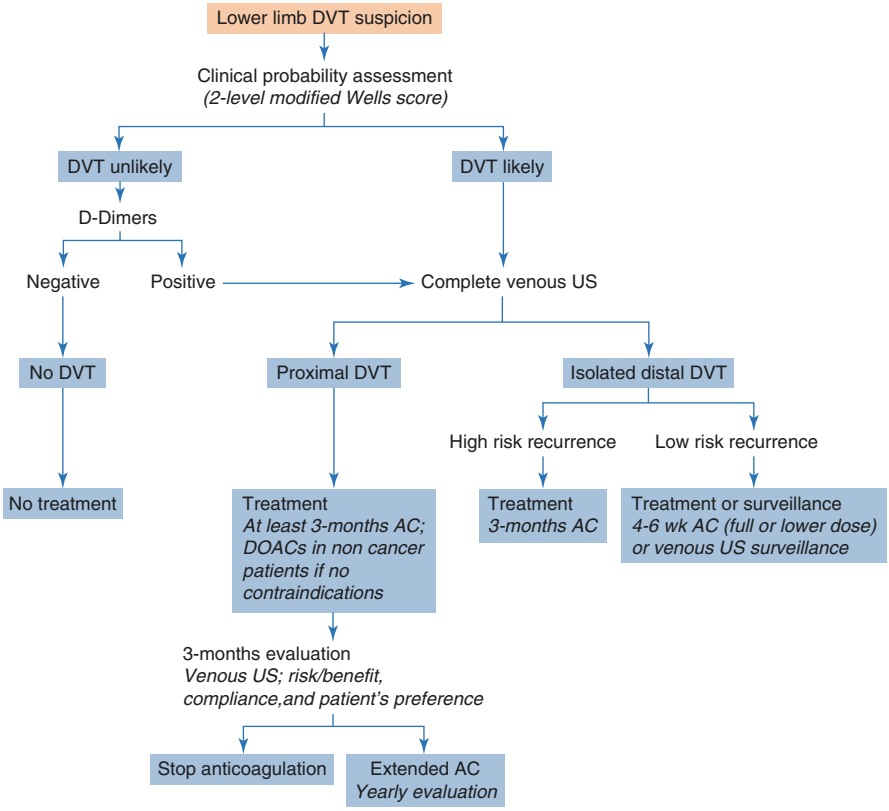

Fig. 10.5 Algorithm for the diagnosis of DVT. In patients with a low or unlikely clinical probability score, further diagnostic tests, such as the D-dimer test and duplex ultrasonography, can be performed to rule out DVT [120]

10.15 Treatment Strategies

Treatment of lower limb deep vein thrombosis can be considered and consists of three phases [122]:

- Initial treatment (5–21 days following diagnosis)
- Long-term treatment (first 3–6 months)
- Extended treatment (beyond 3–6 months)

10.16 Anticoagulation

In the initial phase of treatment, the patient receives parenteral therapy transitioning to vitamin K antagonists (VKA) or high-dose direct oral anticoagulants (DOAC). In patients with severe renal failure (creatinine clearance <30 mL/min), unstable renal

function, or high bleeding risk, intravenous unfractionated heparin (UFH) may be preferable due to its short half-life and protamine sulfate reversibility. However, UFH is associated with dosing variability requiring laboratory monitoring and regular adjustments. Additionally, it is associated with high risk of heparin-induced thrombocytopenia [122]. For these reasons, low-molecular-weight heparin (LMWH) is the treatment of choice since they are as effective as UFH and probably safer [123]. Fondaparinux can also be used as parenteral agent [124].

Direct oral anticoagulants (DOACs) have emerged as valid options for long-term DVT treatment [122]. A meta-analysis (27,023 patients) showed similar VTE recurrence rates in patients receiving DOACs or conventional therapy (2.0% vs 2.2%, RR 0.90). Major bleeding (RR 0.61), fatal bleeding (RR 0.36), intracranial bleeding (RR 0.37), and clinically relevant non-major bleeding (RR 0.73) were significantly lower in DOAC-treated patients. DOACs as standalone therapy for DVT is gaining popularity [106, 125].

The risk of recurrence following discontinuation of anticoagulation after a first episode of DVT remains consistently around 30%. The risk is significantly increased in unprovoked DVTs (annual rate >7%) versus those with provoked DVT. Additionally, the risk of recurrence is higher for unprovoked DVTs than unprovoked PE [120].

For proximal DVT and/or PE, 3-months anticoagulation is the best option if transient and reversible risk factors were present. In all other patients, prolonging anticoagulation protects from recurrence (70–90%) but exposes to risk of unpredictable bleeding complications. Decision to discontinue anticoagulation should be individually tailored and balanced against bleeding risk, taking also into account patients' preferences and the associated cost.

10.17 Compression Therapy

PTS is a seriously burdensome complication of DVT that develops in 25–50% of patients [126, 127]. Its clinical features range from minor limb swelling and discomfort to severe leg pain, edema, skin changes, and even ulceration. Venous claudication and eventually lymphedema can severely affect quality of life of patients who develop PTS [128]. Compression therapy was thought to prevent PTS by reducing venous hypertension and reflux and can be used to treat leg complaints [129].

Kahn and colleagues reported the results of a multicenter randomized placebo-controlled trial of active treatment (knee-length stockings with 30–40 mmHg compression) versus placebo compression stocking (knee-length sham stockings without therapeutic compression) for 2 years in 806 patients to prevent PTS after a first proximal lower extremity DVT. The cumulative incidence of PTS was 14.2% for active compression stockings versus 12.7% for placebo compression stockings. These findings do not support routine wearing of compression stockings after DVT [130].

The efficacy of compression stockings in terms of reducing the incidence of PTS in patients with lower extremity DVT remains controversial; however, compression therapy associated with early mobilization and walking exercises has been effective in the relief of acute DVT symptoms and as such the decision for its early usage should be individualized. However, compression stockings may be required by patients to control their PTS symptoms beyond 2 years. This is more as a treatment for PTS rather than prevention of PTS.

10.18 Thrombolysis and Thrombectomy

Anticoagulation therapy is the main treatment option for acute femoropopliteal DVT, but anticoagulation in extensive ilio-femoral DVT is often not sufficient to prevent later development of chronic venous insufficiency and post-thrombotic syndrome [131–133].

Therefore, early thrombus removal is desirable. Surgical venous thrombectomy is recommended in selected patients who are candidates for anticoagulation but in whom thrombolytic therapy is contraindicated or not available [134]. In patients with limb-threatening venous ischemia, such as venous gangrene or phlegmasia cerulea dolens, aggressive thrombus removal should be considered if the patient's general condition is acceptable [135, 136]. Endovascular techniques for early thrombus removal for DVT consist of catheter-directed thrombolytic therapy (CDT) and/or percutaneous mechanical thrombectomy (PMT) [137]. Endovascular maneuvers at thrombus removal should be considered for patients with symptomatic iliofemoral DVT who have symptoms of less than 14-days duration, good functional status, a life expectancy of 1 year or more, and a low risk of bleeding. When phlegmasia cerulea dolens or venous gangrene is present, aggressive endovascular thrombus removal should be performed on an emergency basis for limb salvage [132, 138, 139].

The threshold for thrombus removal strategies in acute femoropopliteal DVT should be higher than that for iliofemoral DVT. Multicenter registries have suggested a less favorable outcome for femoropopliteal than iliofemoral DVT treated with thrombolytic therapy [140].

CDT achieves early lysis of the blood clot, resulting in rapid relief of the symptoms and improved venous patency rates. The results of randomized trials and meta-analyses suggest that CDT improves quality of life without an unacceptable increase in bleeding [141–143]. Major bleeding complications have been reported at rates of 2–4% [144–146]. CDT is noted to preserve venous valvular function and decreased occurrence of PE and PTS [137].

Although CDT may be associated with asymptomatic radiographic evidence of PE, symptomatic PE appears to be a relatively rare complication of CDT. The incidence of clinical PE during CDT does not appear to exceed that in patients who receive anticoagulation therapy alone. According to the Prevention du Risque d'Embolie Pulmonaire par Interruption Cave (PREPIC) study, which was the first

reported multicenter randomized controlled trial, the incidence of DVT at 2 years after inferior vena cava (IVC) filter placement was significantly higher than that in the no IVC filter group. In contrast, the overall survival rate was not significantly different between the two groups [147]. Therefore, routine placement of permanent IVC filters in patients undergoing CDT for DVT is not recommended in terms of short-term efficacy and long-term complications [138, 147, 148]. However, the introduction of retrievable filters has contributed to their increased overall usage. Filter Implantation to Lower Thromboembolic Risk in Percutaneous Endovenous Intervention (FILTER-PEVI) trial reported that IVC filter implantation during pharmaco-mechanical-thrombectomy (PMT) reduced the risk of iatrogenic PE eightfold (1.4% vs. 11.3%), without reducing mortality [149]. The use of periprocedural IVC filter may be considered in patients at high risk of PE, such as those with free-floating thrombus extending into the IVC, with marked limited cardiopulmonary reserve, or with mechanical thrombectomy alone [132, 149, 150].

Open surgical venous thrombectomy can be performed in selected patients when endovascular facilities are not available, such as those with a first episode of acute iliofemoral DVT, symptoms <14 days in duration, a low risk of bleeding, ambulatory with good functional capacity, and an acceptable life expectancy. In cases where these criteria are not met, conventional anticoagulation may be preferred over high-risk surgery (Fig. 10.6 [134]).

10.19 Surgical Treatment of Chronic Lower Extremity DVT

Despite therapeutic anticoagulation and elastic compression stocking therapy, a significant proportion of DVT patients may develop post-thrombotic sequelae. Because the iliac vein rarely recanalizes, patients with chronic iliofemoral DVT develop significant symptoms of PTS associated with valvular reflux and persistent venous obstruction [35, 46, 151]. Endovascular strategies at recanalization of the iliofemoral venous segments play an important role in improving presenting symptoms, reduction of venous disability, and/or healing of existing venous ulcers [138]. Recanalization was technically successful in 83% of occluded iliac veins with the 4-year primary and secondary patency rates approximately 35% and 72%, respectively [152, 153]. Although there are no high-level randomized controlled data, recanalization of chronic occlusive DVT can be performed safely and successfully and provide significant improvement in venous flow, ultimately leading to symptom relief and improvement in the quality of life.

Patients with chronic DVT who are not candidates for endovascular repair or those who failed attempts of endovascular revascularization can undergo open surgical reconstruction. Successful surgical bypass for iliofemoral venous occlusion refractory to endovascular therapy has been reported [154]. It has also been suggested that open procedures should be performed in cases of unsuccessful stenting attempts, stent failure, and long occlusions in which stenting may not be feasible [155].

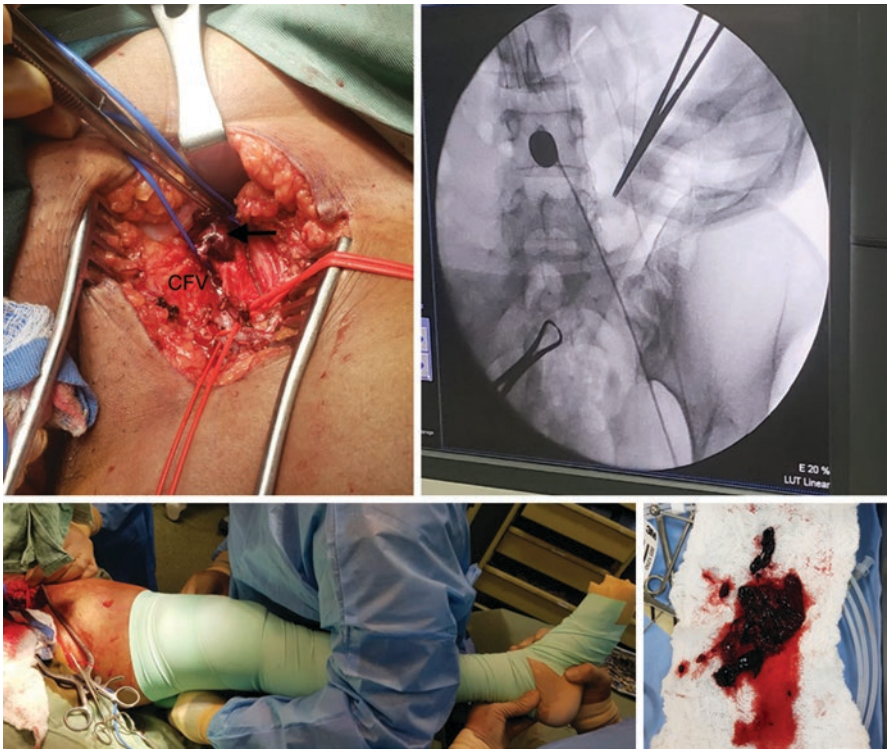

Fig. 10.6 Technique of open surgical venous thrombectomy - CFV exposure with proximal thrombus removal using embolectomy catheter/distal thrombus removal with the aid of an Esmarch bandage followed by completion venogram

Common open procedures for chronic DVT include a crossover bypass procedure (Palma-Dale procedure), in-line bypass surgery, and endo-phlebectomy. Endo-phlebectomy is an open surgical technique which demonstrated significant improvement in venous scores postoperatively. The venous clinical severity score was decreased from 17 to 9.8 after the operation and the Villalta scale was decreased from 13.6 to 6.0 postoperatively [156]. This technique can be combined with an endovascular recanalization of an occluded iliac venous segment as a hybrid technique with good long-term results [157, 158].

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