

REVIEW

Clinical Significance of Superficial Vein Thrombosis

L. Leon,¹ A.D. Giannoukas,² D. Dodd,² P. Chan² and N. Labropoulos^{1*}

¹Loyola University Medical Center, 2160 South First Avenue, Maywood, IL, USA; and ²Sheffield Vascular Institute, Northern General Hospital, Herries Road, Sheffield S5 7AU, UK

Objective. To evaluate the clinical implications of superficial thrombophlebitis (STP) including its demographic characteristics, distribution, risk factors, relationship with deep vein thrombosis (DVT), pulmonary embolism (PE), diagnosis and management.

Methods. Data were collected from relevant papers using a MEDLINE search and an extensive bibliography review. Studies were considered only when they contained pertinent material to STP. Thirty-seven papers were analysed.

Results. The diversity of patients and methods used in the different studies made the comparison among them difficult. STP is a common condition with an underestimated prevalence. There are many risk factors associated with STP but the strongest relation was seen with hypercoagulable states. Malignancy may be another important factor but the strength of this association remains unknown. Coexistence with DVT was found in 6–53%. PE occurred in 0–33.3%. Propagation to DVT ranged from 2.6 to 15%. Treatment has not been standardised and may include elastic compression, anti-inflammatory drugs, anticoagulation and surgery.

Conclusion. The limited number of prospective randomised studies on STP does not allow strong recommendations to be given. Although STP most often is perceived as benign, it can coexist with or progress to DVT, and even give rise to PE. It is also associated with hypercoagulability and malignancy.

Keywords: Superficial vein thrombosis; Duplex ultrasonography; Hypercoagulable states; Venous thromboembolism.

Introduction

Thrombosis in the superficial veins has been termed superficial thrombophlebitis (STP) or superficial vein thrombosis. It is most often found in the veins of the lower extremities but it has been reported in many other locations. It has been considered benign, and it is usually treated conservatively with elastic stockings (ES), ambulation, antibiotics, and non-steroidal anti-inflammatory drugs (NSAIDs).^{1–4} Patients with thrombosis in proximity to the deep veins have been treated with ligation or anticoagulation.

STP presents with pain, erythema, and swelling around a superficial vein that becomes solid and on palpation feels like a cord. It is most frequently found in patients with varicose veins, but many other factors have been associated with this condition.^{5–10} Although

its diagnosis was based solely on clinical assessment, the use of duplex ultrasound imaging (DUS) has enabled the accurate detection of its extent and progress.^{6,11–14}

There is evidence in the literature suggesting that STP is not always benign. It may precipitate or be associated with deep venous thrombosis (DVT), and it could cause pulmonary embolism (PE). The strength of these associations has not been adequately studied, and there are only a few prospective studies dealing with this condition. The purpose of this manuscript is to review the aetiology, characteristics, natural history and management of STP since such analysis in the literature is scarce.

Methods

Literature search

A MEDLINE search from 1966 until 2003 (National

* Corresponding author. Nicos Labropoulos, Associate Professor of Surgery, Department of Surgery, Loyola University Medical Center, 2160 South First Avenue, Maywood, IL 60153-3304, USA.
E-mail address: nlabrop@lumc.edu.

Library of Medicine and OVID) and an extensive manual search were carried out using bibliographies from relevant published papers. The main terms for inclusion were superficial vein thrombosis, thrombophlebitis, saphenous thrombosis, varicose vein thrombosis and other similar terms. Other search terms relevant to this topic included thromboembolism, malignancy, hypercoagulable states, pregnancy and management. As there were very few prospective studies it was decided to also include retrospective papers. Case reports and very small series ($n < 15$ patients) were excluded. Papers were chosen by their cohesion and relevance of data. A total of 37 studies were included in the analysis. The data were analysed for associated conditions such as DVT, PE, malignancy, hypercoagulable states and management related to STP.

Limitation

The wide diversity of patients and methods used in the published reports made comparisons between them difficult. The small number of prospective natural history and randomised studies did not allow robust conclusions to be made or strong recommendations to be given. Because of these limitations, the authors considered that pooled analysis and statistical manipulation of the data would not be appropriate. Interpretation and analysis was confined to a descriptive report of the selected studies.

Demographic characteristics

The incidence of STP has been reported to be 3–11% in the general population.¹⁵ Coon *et al.*¹⁶ reported a rate of 123,000 patients per year for clinically recognised STP. Seventy-eight percent of those patients were female. Both of these epidemiological studies are old and the diagnosis was clinical. Its prevalence is probably underestimated because in many patients, the symptoms of STP can be minor, and patients may not present for medical attention. Most papers revealed a preponderance of female sex with a ratio of 6–4,^{17–22} possibly because of the increased prevalence of varicose veins in pregnancy.²² Risk factors that have been cited in association with STP include previous thromboembolic episodes, long-haul flight, pregnancy, oral contraceptives, hormone replacement therapy, immobilization, obesity, recent surgery, trauma and sclerotherapy. Often, two or more risk factors have to be present for thrombosis to occur although age is an additional factor; the older the

patient, the fewer factors are needed.²³ The mean age of presentation is approximately 60 years.^{20,21,24–28}

Varicose veins, location and STP

Thrombosis in the saphenous veins and their tributaries is the most common location followed by the cephalic and basilic vein in the upper extremities. Thrombosis in superficial veins in other parts of the body is uncommon.²⁰ The prevalence of STP in large series of patients with varicose veins has a wide range varying from 4 to 59%.^{6,18,22} The great saphenous vein (GSV) is involved in 60–80% of cases, and the small saphenous vein (SSV) in 10–20%. No predilection for either lower extremity is found.⁶ Bilateral cases are reported in 5–10%^{6,20} and in one report were associated with a high number of complications (8%).⁶ Patients with GSV thrombosis had more complications than thrombosis in other locations.⁶ STP was more frequently found in varicose tributaries rather than in the saphenous trunk.¹⁸ There may also be localised defects in the processes of fibrinolysis and platelet aggregation.²⁹ The current evidence is not definitive and further work is needed.

Hypercoagulable states

STP confined to varicose tributaries is a complication of varicose vein disease. However, saphenous trunk thrombosis is often a more significant thromboembolic process.^{12,24} Abnormalities of coagulation have been reported to be associated with saphenous thrombosis including deficiencies of antithrombin III, anticardiolipin antibodies, heparin cofactor 2, protein C and S, abnormal fibrinolytic activity, Hageman trait and the gene mutations of factor V Leiden and factor II (prothrombin) G20210A.^{8,9,24,30} In the absence of varicose veins, autoimmune diseases or malignancy the risk of STP was found to be 6-fold for the Factor V Leiden mutation, 4-fold for the factor II G20210A mutation and 13-fold for antithrombin III, protein C and S deficiencies taken together.⁷ It appears that anticardiolipin antibodies and increased levels of factor VIII are more prevalent in patients with recurrent STP.^{30,31} There are no large studies evaluating the prevalence of hypercoagulable states in different cohorts of patients with STP. However, it is clear in multiple studies as shown in Table 1^{7,8,24,30–35} that patients with STP have high prevalence of hypercoagulable states. Arguably, patients with spontaneous STP without varicose veins, or extending to the main trunk of the GSV, should be screened for hypercoagulability.

Table 1. STP and hypercoagulable states

Author	Patients	State	Prevalence (%)	Odds ratio (95% confidence intervals)	STP
Engesser 1987 ³²	71	Protein S deficiency	72		
Lohr 1992 ³³	29	Abnormal coagulation profile	65	–	Isolated
Pabinger 1996 ³⁴	230	Coagulation inhibitor deficiency	↑ in Protein C and S deficiency	–	First episode and recurrent
De Moerloose 1998 ⁸	112	Factor V Leiden	14.3	2.51 (1.04–6.24)	First episode
		Factor II A20210	3.6	3.28 (0.46–36.84)	
Hanson 1998 ²⁴	17	ATIII	17.6	–	First episode
		ATIII + Protein S	5.9		
		Protein S	5.9		
		Protein S + APCR	5.9		
Martinelli 1999 ⁷	63	Factor V:A1691	15.9	6.1 (2.6–14.2)	First episode
		Factor II G20210A	9.6	4.3 (1.5–12.6)	
		ATIII, Prot C or S deficiency	10.2	12.9 (3.6–46.2)	
de Godoy 2001 ³¹	45	Anticardiolipin antibodies	33.3	6.64 (2.48–17.82)	Recurrent
de Godoy 2003 ³⁵	36	Protein S deficiency	5.2		Recurrent (≥2)
Schonauer 2003 ³⁰	45	High Factor VIII	24% (<i>p</i> =0.004)	4 (2.0–8.6)	Recurrent VTE

VTE, venous thromboembolism; ATIII, antithrombin III deficiency; APCR, activated protein C resistance.

Pregnancy

There is limited information on the relation of pregnancy and STP. In one retrospective study involving 30,040 pregnancies, 14 cases (0.05%) were diagnosed by ultrasonography, mostly presenting within 48 h of delivery.³⁶ McColl *et al.*¹⁰ found an incidence of 0.068% [49/72,200 deliveries; 95% confidence intervals (CI) 0.048–0.088]. Ten cases occurred prior to delivery and the rest of cases (*n*=39, 0.054%, 95% CI 0.037–0.071) within 7 days postpartum. Of the patients with STP only 24 were tested for thrombophilic abnormalities, and only one was positive for a Factor V Leiden mutation. Both studies have probably underestimated the prevalence of STP because they evaluated only symptomatic patients.

Malignancy

Patients presenting with significant venous disease may have an underlying neoplasm. A literature review on vascular disorders that preceded the diagnosis of malignancy revealed a weak relationship between STP and cancer.³⁷ This association was mainly based on data from two papers from the early 60s, but did not include two further studies, which also reported an association. Among 106 limbs with STP the incidence of malignancy was 13%; in 11 cases the diagnosis was known at the time of onset of STP, and in three the diagnosis was made within 1 month.¹³ In a large study of 398 limbs with STP in the GSV or SSV, ascending superficial thrombosis was detected in 56. Ten (18%) of

these had malignant disease.³⁸ However, apart from these retrospective studies there is no other evidence supporting an association between cancer and STP. In our practice occasionally we find patients with STP and cancer and usually the latter is diagnosed first. The strength of this association remains unknown and needs to be further studied, preferably in cohorts with defined malignant disease.

DVT and STP

The evidence on concomitant STP and DVT and/or PE is controversial. Most of the studies that we found are retrospective, and some include very small numbers of patients. We chose only papers that included at least 20 patients for analysis. The presence of DVT in association with saphenous thrombosis ranges from 6 to 53% (Table 2).^{6,14,17,18,21,22,25,39–51} Thrombus propagation can occur in a contiguous and in a non-contiguous fashion. From 53 patients with clinical manifestations of STP and diagnosed with DVT, evidence of direct contiguous propagation was found in 40 cases (75.5%), and the rest was non-contiguous calf involvement at the posterior tibial and soleal levels.⁶ Contiguous extension of the thrombotic process from the superficial into the deep veins can occur in three ways.⁴¹ The most common involves extension from the GSV into the femoral vein.^{6,41} Less often, the thrombus extends from the SSV into the popliteal vein through the saphenopopliteal junction (SPJ). Extension through perforating veins can also occur to several deep venous structures. From 186 DUS scans with evidence

Table 2. STP and concomitant venous thromboembolism (VTE)

Author	Patients (n)	DVT (%)	PE (%)	Diagnosis	Treatment
Gervais 1956 ³⁹	64	6	–	Surgery*	Surgery
Gjores 1962 ⁴⁰	40	32	5	–	Surgery
Zollinger 1962 ²¹	335	–	10.1	Clinical	–
Hafner 1964 ⁴¹	133	17	–	Surgery*	–
Lofgren 1981 ¹⁸	163	8	–	Clinical	Surgery
Husni 1982 ²²	139	7 [†]	–	–	Surgery
Plate 1985 ⁴²	28	14	–	VNG, VQ	Surgery
Bergqvist 1986 ⁴³	56	16	–	VNG	–
Skillman 1990 ⁴⁴	42	12	–	VNG, SG, DUS	Medical
Lutter 1991 ⁵	186	28	4	DUS	–
Prountjos 1991 ⁴⁵	57	20	–	VNG	–
Pulliam 1991 ⁴⁴	20	30	0	DUS	Surgery
Lohr 1992 ⁴⁶	43	53	–	DUS	Surgery
Jorgensen 1993 ⁴⁷	44	23	–	DUS	–
Ascer 1995 ²⁵	20 [‡]	40	0	DUS	Medical
Blumenberg 1997 ⁴⁸	213 (232 limbs)	8.6	0.93	DUS	Medical
Bounameaux 1997 ⁴⁹	551	5.6 (95% CI 3.8–7.9)	–	VNG, DUS	–
Verlato 1999 ⁵⁰	21	–	33.3	DUS, VQ, CXR	–
Murgia 1999 ⁵¹	85	25.3	–	DUS	–
Unno 2002 ¹⁷	51	11.8	7.8	DUS	–

DUS, Duplex ultrasound; VNG, venography; SFJ, saphenofemoral junction; VQ, ventilation perfusion lung scan; CXR, chest X ray.

* Free-floating thrombus in the common femoral vein extending from the SFJ was found during surgery.

† These thrombi were found in the perforator veins and nothing was mentioned about the deep veins.

‡ These patients were selected to have SFJ thrombosis.

of STP, isolated perforating vein involvement was never found.⁶ It is possible that thrombosis can extend from the deep veins to the superficial ones, but this has not been evaluated in any study.

Chengelis *et al.*²⁶ reported that 30 (11%) of 263 patients with saphenous thrombophlebitis had documented extension of thrombus into the deep veins. In 21 patients, the thrombus extended from the thigh GSV into the common femoral vein (CFV). In three patients, thrombus was extended from the thigh GSV into the femoral vein through thigh perforators, three patients had extension of a below knee saphenous thrombus to the popliteal vein, and in another three patients below-the-knee saphenous thrombi extended via calf perforators to the tibioperoneal veins.

The prevalence of DVT in the presence of varicose veins was reported in two small studies at 13% (5/39) and 24.5% (10/41).^{44,47} DVT was not detected in the absence of varicose veins but there were only three limbs without them in each study. Bergqvist and Jaroszewski⁴³ performed ascending venography in 56 patients with clinical evidence of STP, and DVT was found in nine patients. Eight of those nine patients did not have varicose veins. Therefore, the prevalence of DVT in patients with varicose veins was 2.6% in contrast to 44% in those without ($p < 0.01$). No patients with varicose veins developed a malignancy on follow-up whereas two patients without varicosities were subsequently diagnosed with breast cancer and polycythemia vera, respectively.

STP confined to above knee segment of the GSV was

associated with 17–19% incidence of DVT and 4–5% when STP did not extend above the knee.^{26,43} However, in another study the site of STP did not point to the presence or absence of DVT.⁴⁴ Propagation of STP to the deep veins by serial DUS has been documented in three studies (Table 3).^{14,26,48} These data indicate that most patients with STP should have their deep veins evaluated even if we accept the lowest DVT prevalence of 5.6% shown by Bounameaux *et al.*⁴⁹

PE and STP

Given the low prevalence of this association, most of the studies available in the literature include a small number of patients. Gjores⁴⁰ described five cases of PE where the embolus originated from the GSV and no evidence of DVT was found. Two of them were fatal episodes after prostatectomy and the rest were small and non-fatal. Zollinger *et al.*²¹ found a 1.5% rate of fatal PE in patients with STP in his series. PE cases have been reported with SSV involvement as well. An 18% rate of PE was reported when the thrombotic process was located at the above-the-knee location in the GSV; a 4% rate for SSV involvement and a 7% propagation rate to the popliteal vein.⁶ The only prospective study by Verlato *et al.*⁵⁰ evaluated 21 consecutive patients with STP in the thigh segment of GSV by DUS, chest X-ray and perfusion lung scanning, regardless of their symptoms. They found seven patients with high probability of PE (33%; 95% CI, 14.6–57). Only one of those patients was symptomatic.

Table 3. STP and development of venous thromboembolism

Author	Patients (n)	DVT (%)	PE (%)	Diagnosis	Treatment
Pulliam 1991 ¹⁴	20	15	-	DUS	Surgery
Chengelis 1996 ²⁶	263	11.4	≥2.3	DUS; VQ	Medical
Blumenberg 1997 ⁴⁸	232 limbs	2.6	-	DUS	Medical

DUS, Duplex ultrasound; VQ, ventilation perfusion lung scan.

Unno *et al.*¹⁷ enrolled 51 consecutive patients with STP, chosen among 710 patients referred for treatment of varicose veins, in a risk assessment study. A 7.8% rate of PE was found, all cases having involvement of the GSV or SSV.

It is unclear whether PE associated with STP arises from extension into the deep veins or from clot that detaches while still in the superficial venous system. This may be an important distinction, as surgical ligation could prevent the latter case, but monitoring with DUS should suffice if the former is more common.

Diagnostic approach of STP

The diagnosis of STP can be made on basis of clinical or DUS data. STP is often easy to diagnose due to its superficial location. Clinical features include the presence of a warm, tender, palpable cord or nodule-like structure following the course of a superficial vein. But the diagnosis should not be limited to clinical grounds only. The correlation between clinical exam and surgical findings is poor. Clinical exam does not reveal the true extent of STP; surgical exploration shows often extension of the thrombotic process 5–10 cm higher than the level that was clinically diagnosed.⁴⁰ We commonly find in our practice that DUS often identifies a more proximal extent of STP compared to clinical exam. DUS is recommended for confirmation of diagnosis, for estimation of the extent of thrombosis and for follow-up.^{11,20,26} Systematic application of DUS has been criticized because of the low incidence of DVT diagnosed. Bounameaux *et al.*⁴⁹ retrospectively analysed a large number of medical records of patients with STP over a 6-year period. This study showed a 5.6% association of DVT in patients with STP (31/551; 95% CI 3.8–7.9%), of which 26 cases had proximal vein involvement. This paper suffers the limitations of a retrospective design, and it may have had important loss of data in follow-up.

STP cases can be divided in three broad categories, based on DUS and clinical examination: those of a short segment not associated with varicose veins; short segment associated with varicosities; and extensive saphenous thrombophlebitis.¹² That stratification portends important differences with regard to diagnostic

workup and management. Patients within the first category often will have an underlying systemic disease that should be investigated. Patients in the second group need no further workup and their treatment is symptomatic and surgery for varicosities if needed. Clinical and DUS follow-up may still be needed to confirm the absence of thrombus propagation to the saphenous trunk. Patients in the third group have an important association with DVT and PE (Table 2)^{6,14,17,18,21,22,25,39–51} and the management needs to be tailored accordingly.¹²

Thrombophilia screening has been recommended to identify patients at risk for developing thromboembolic complications.^{7,8,17,30,52} Some authorities perform screening selectively based on the presence or absence of risk factors.^{52–54} Once STP develops it is important to differentiate whether this is confined to varicose saphenous tributaries within the context of pre-existing varicose vein disease or if it involves the saphenous trunk, particularly the thigh segment. The former situation seems not to require further investigation, as it appears to be a localized event complicating varicose vein disease. However, clinical and DUS follow-up may still be needed to confirm the absence of thrombus propagation to the saphenous trunk. In contrast, the latter situation seems to be more serious. Most of the literature supports investigation by DUS in order to accurately evaluate the extent of thrombus and to exclude the presence of DVT.^{6,11,13,14,25,26,44–48,55} We recommend that the clinician should consider the possibility of hypercoagulable states and malignancy in patients without varicose veins who present with STP.⁴³

Considerations in the management of STP

The treatment options available for STP include ambulation, ES, anti-inflammatory agents, anticoagulation and surgery (Table 4).^{18,19,21,22,24,25,27,28,34,40,41,46,56–58} The evaluation of medical treatment for STP in the literature yields conflicting results. This is due to the lack of consistency with regards to the measured end points, inadequate follow-up, limited evaluation to rule out venous thromboembolism (VTE), small number of patients studied and mainly due to the retrospective nature of most of the studies.

Table 4. STP treatment

Author	Patients (n)	Treatment	Measured outcome	Results	Follow-up (days)
Gjores 1962 ⁴⁰	40	Surgery	VTE	8% PE	–
Zollinger 1962 ²¹	335*	Bed rest + elevation ± Abx Anticoagulation Surgery ± anticoagulation	Ambulation	19% ≤ 3 days 18% ≤ 3 days 60% ≤ 3 days	–
Williams 1964 ⁵⁶	92	Surgery	VTE	0	
Hafner 1964 ⁴¹	324	Surgery (133) Medical (191)	VTE	PE 2.3% DVT 10.5%	No follow-up
Husni 1982 ²²	221	Medical (82) Surgery (139)	PE	12%† 0	8–28
Lofgren 1981 ¹⁸	163	Surgery	VTE	1.2% PE; 4.3% recurrent STP	365–4380 (median 1825)
Plate 1985 ³⁴	28	Surgery	VTE, signs and symptoms	14.3% Had persistent symptoms	
Lohr 1992 ⁴⁶	41	Surgery	VTE	one recurrent STP; one with PE; two with contralateral DVT	Minimum 120
Titon 1994 ⁵⁷	117	LMWH NSAIDS	Signs and symptoms	Greater improvement with LMWH	56
Ascer 1995 ²⁵	20	Anticoagulation	DUS outcome of thrombosis and PE	N=13; 92.3% improvement; 7.7% without change	420
Hanson 1998 ²⁴	17	Medical (NSAIDS, anticoagulation, ES)	DUS thrombus progression	N=13; 46.2% unchanged; 46.2% worse; 7.6% improved	?
Belcaro 1999 ¹⁹	444	ES, surgery and anticoagulation in six combinations	DVT rate Thrombus extension	No difference Higher with ES; lowest with surgery	180
Beatty 2002 ²⁷	17	Emergent SFJ division	VTE	5.9% popliteal DVT; no PE	60
Marchiori 2002 ⁵⁸	60	UFH low vs. high dose (±NSAIDS)	VTE	20 vs. 3.3%, respectively (p=0.05)	180
STENOX 2003 ²⁸	436	LMWH (two doses) NSAIDS ES	STP extension and VTE	Best	90

LMWH, low molecular weight heparin; NSAIDS, non-steroidal anti-inflammatory drugs; ES, elastic stockings; SFJ, saphenofemoral junction; VTE, venous thromboembolism.

* These patients were known to have STP by clinical examination but the number of patients with concomitant DVT was not known.

Surgery ± anticoagulation improved patients' signs and symptoms. However, nowadays almost all patients ambulate and this does not seem a good end point for evaluation. Five patients (1.5%) died from PE.

† Unknown whether these patients had concomitant DVT.

Bed rest, elevation and antibiotic treatment compared unfavourably to anticoagulation in one trial, but early ambulation was the measured end point. Furthermore, antibiotics have no role in the management of STP. Bacteriologic studies of thrombi obtained in surgery that revealed a low incidence of microorganisms and a low rate of wound infection reported in most series speak against an infectious origin for STP.⁴¹ The role of aspirin and NSAIDS in VTE are not well defined. They decrease local pain and may add anti-inflammatory benefits in STP. Non-steroidal anti-inflammatory agents orally or as topical treatment, local application of hirudoid and agents with enzymatic action have been used. These may have some effect in the alleviation of pain and local inflammatory signs but their efficacy is controversial.^{59–63}

Anticoagulation includes unfractionated heparin (UFH), low molecular weight heparin (LMWH), coumadin and recently pentasaccharides. LMWH proved to be at least as effective and safe as UFH in the treatment of DVT. Therefore, it could be a reasonable alternative.⁶⁴ The only randomised trial enrolled 436 patients and compared ES, NSAIDS and two doses of LMWH for 10 days. LMWH showed the most favourable trend but it was only significant for thrombus extension and not significant for development of VTE.²⁸

Interventional options include local thrombectomy, vein ligation, excision or stripping and sclerotherapy. Papers that analyse outcomes after surgery for STP revealed a PE rate between 0 and 8% (Table 4).^{18,19,21,22,24,25,27,28,34,40,41,46,56–58} This rate may be underestimated

given that in most of those studies the diagnosis of PE was made clinically in symptomatic patients only. The reported rate of recurrent STP varies from 2 to 4.3%, again probably underestimated due to presentation bias. Selection of patients who have favourable risk/benefit profiles for surgery is a problem as there are no randomised control trials against anticoagulation.

ES should be used if tolerated in all cases of STP as an adjunctive treatment regardless of the associated conditions. Its use alone was associated with more thrombi extension and VTE episodes when compared with anticoagulation.²⁸ In cases of STP located away from the SFJ or the SPJ, or STP at lower levels, without evidence of DVT, conservative management using ES, NSAIDs or aspirin often suffice. In cases of concomitant STP and DVT anticoagulation should be started. When a thrombus is found in the SPJ or SFJ and extends as free-floating in the CFV or popliteal veins, removal of the thrombus during surgery is an equally good treatment. If the thrombus is adherent or located non-contiguously, anticoagulation is mandatory.

We emphasise that patients with STP require follow-up, either clinical or with DUS. DUS should be performed at about 7–10 days after the original diagnosis to assess the extent and progression of STP. If the symptoms worsen or if there is DUS or clinical evidence of progression, anticoagulation or surgery may be needed following the criteria described above.

Final remarks and conclusions

STP is not always a benign condition. Although generally benign if confined to varicose tributary veins, there is evidence that involvement of the GSV or SSV may be associated with more serious conditions, notable DVT and PE. There is also a significant association of STP with hypercoagulable states. A relationship between STP and malignancy has been reported but its strength has not been determined. It is important to exclude concomitant DVT at presentation. Therefore, its diagnosis should not rely only on clinical evaluation but DUS should be included.

Therapy for STP affecting varicose tributaries should be conservative. For more serious STP affecting the truncal GSV or SSV, we recommend anticoagulation or early saphenous ligation. However, there are no randomised studies to prove the superiority of any of these options. In addition, the optimal length of the anticoagulation treatment and the type of the anticoagulant drug are unknown.

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