


The fibroblastic sleeve, the neglected complication of venous access devices: A narrative review

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Abstract

The presence of a vascular access device (or of any intravascular foreign body) inside the bloodstream is often associated with the formation of a connective tissue sleeve around the catheter (often named—erroneously—“fibrin sleeve”). Such sleeve is usually a physiological phenomenon with little or no clinical relevance, but its pathogenesis is still unclear, so that it is frequently confused with venous thrombosis; also, its relationship with other major catheter-related complications, such as venous thrombosis and bloodstream infection, is uncertain. This narrative review tries to convey in a systematic form the current knowledge about pathogenesis, incidence, clinical manifestations, diagnosis, and management of this phenomenon.

Keywords

Fibroblastic sleeve, fibrin sleeve, fibrin sheath, catheter-related thrombosis, venous access devices, catheter-related complications

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Background

The topic of this narrative review is one of the most neglected and misinterpreted of the many phenomena associated with the clinical practice of inserting venous access devices (VADs). While a vast literature exists about the pathogenesis, diagnosis, prevention, and treatment of catheter-related infections, catheter-related thrombosis (CRT), lumen occlusion, and so on, the studies that have addressed the topic of the present review are quite scarce in the literature.

The actual clinical impact of this phenomenon is unknown, to the point that some clinicians, even if working in the field of VADs, may ignore its existence or have very vague ideas about it.

The term itself that identifies this “entity” is uncertain, and sometimes inappropriate or misleading. During the last five decades, several terms have been used (see Table 1): “fibrin sleeve,” “fibrin sheath,” “catheter sleeve,” “catheter-related sheath,” “fibroblastic sleeve,” and so on.

This uncertain terminology reflects the lack of knowledge about the pathogenesis and the histological structure of the phenomenon. While the morphological concept of “sleeve” or “sheath” is quite appropriate, since it represents the special feature of creating an “envelope”

that “wraps” the catheter, the term “fibrin” is certainly erroneous, since the material of the sleeve is not “fibrin” but a structured cellular tissue, more specifically a connective tissue, which is developed by fibroblasts; although fibrin may initially happen to be part of this tissue, the main non-cellular component in the mature sleeve is collagen. However, in recent papers of the last few years (see Table 1), the term “fibroblastic sleeve” seems to prevail; considering that this term describes appropriately the pathogenesis of the sleeve/sheath, we think it is adequate and we will adopt it in this review.

At its first appearance in the medical literature, the fibroblastic sleeve (FS) was erroneously baptized “fibrin sleeve”. This occurred in 1964, in a French study by Motin and coworkers; in 154 polyethylene central catheters, a sleeve was found and named “fibrin sleeve,” in the presumption that it may be a phenomenon somehow related to thrombosis.¹

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Table 1. Main studies on fibroblastic sleeve.

Author	Term	Type of article	Model	Type of VAD
Motin et al. ¹	“Fibrin sleeve” (article in French)	Postmortem study	Human	Polyethylene catheters
Hoshal et al. ²	Fibrin sleeve	Postmortem study	Human	Catheters in different materials: polyethylene, Teflon, nylon, silicon, heparin-bonded polyethylene, graphite-benzalkonium chloride-heparin-bonded polyethylene
Peters et al. ³	Fibrin sheath	–	–	–
Borow et al. ⁴	Fibrin sheath	Experimental study	Dog	Catheters in different materials: hydromer-coated polyurethane, heparin hydromer-coated polyurethane, polyvinylchloride, silicon, Teflon, polyurethane
Lloyd et al. ⁵	Fibrinous sheath	Experimental study	Rat	Silicon catheter
Hombrouckx et al. ⁶	Fibrin sheet	Clinical study	Human	Dialysis catheters: 12-Fr silicon and 8-Fr polyurethane
Raad et al. ⁷	Thrombin sheath	Postmortem study	Human	Non-tunneled catheters in polyurethane or silicon.
Cardella et al. ⁸	Fibrous sheath	Case report	Human	3-Fr single-lumen silicon PICC
O’Farrell et al. ⁹	Fibrin sheath	Experimental study	Rat	Silicon catheters
Crain et al. ¹⁰	Fibrin sheath	Pictorial essay	Human	Double-lumen dialysis catheter; Hickman-type single-lumen catheter
Xiang et al. ¹¹	Catheter sleeve	Experimental study	Rat	Silicon catheters
Xiang et al. ¹²	Catheter sleeve + sleeve-related thrombosis	Experimental study	Rabbit Rat	4.2-Fr Broviac single-lumen catheters
Mehall et al. ¹³	Fibrin sheath	Experimental protocol	Rat	2.7-Fr silicon catheters
Oguzkurt et al. ¹⁴	Peri-catheter sleeve	Prospective study	Human	11.5-Fr polyurethane dialysis catheters
Keller et al. ¹⁵	Peri-catheter sheath	Experimental study	Rat	2.7-Fr silicon catheters
Forauer et al. ¹⁶	Catheter-related (fibrin) sheath	Experimental study	Swine	6.6-Fr single-lumen silicon catheters; 7-Fr dual-lumen silicon catheters
Sabbaghian et al. ¹⁷	Catheter “cast”	Case report	Human	9.6-Fr implantable port
Sinno and Alam ¹⁸	Fibrinous sheath/ catheter related sheath/fibrin sheath	Case report	Human	Cuffed tunneled catheters; implantable ports
Percarpio et al. ¹⁹	Catheter-related sheath (CRS)	Book chapter	–	–
Krausz et al. ²⁰	Fibrin sheath	Retrospective study	Human	Tunneled catheters; implantable ports; PICCs
Boddi et al. ²¹	Fibrin sheath	Prospective study	Human	Implantable ports
Sylvia et al. ²²	Fibroblastic sleeve	Experimental study	Ovine	Polyurethane catheters with different coating: chlorhexidine, fluoro-oligomers, poly(2-methoxy-ethyl acrylate)
Tanabe et al. ²³	Catheter sleeve	Experimental study	Rabbit	Poly(2-methoxy-ethyl acrylate) catheter
Baciarello et al. ²⁴	Fibroblastic sheath	Case report	Human	6-Fr implantable ports
Hill et al. ²⁵	Central venous access device-related sheaths	Prospective study	Human	Implantable ports

VAD: venous access devices; PICCs: peripherally inserted central catheters.

In 1971, in a postmortem study examining the subclavian veins of 55 patients who had died with a central venous catheter, Hoshal et al.² described the presence of a “sleeve” around VADs made of different materials (polyethylene,

polytetrafluoroethylene, nylon, silicon plastic). The authors noted that the sleeve surrounding the catheter was already evident 24 h after VAD insertion and that some materials may be less affected by this phenomenon; more

specifically, the sleeve formation around polyethylene catheters bonded with graphite-benzalkonium chloride-heparin (GBH) was less evident if compared to other materials. Hoshal and coworkers hypothesized that the development of the sleeve might start in contact points between the catheter and the vein wall, that is, at the entrance site and where the tip touches the endothelium. They assumed that such contacts would cause a deposition of fibrin over the catheter. Obviously, this hypothesis was not supported by any evidence. Interestingly, in the same study, 2 of the 55 patients had evidence of a CRT: one patient had CRT and pulmonary embolism, while the other patient had a thrombus contaminated by *Staphylococcus aureus*. On this basis, the authors concluded (quite inappropriately) that the “fibrin sleeve” might be a potential cause of infection, thrombosis, and pulmonary embolism.

In the 90s, FS was described as a possible source of catheter malfunction (typically because of difficult blood aspiration) in tunneled-cuffed dialysis catheters and its composition was thought to consist of “fibrin and thrombocytes”.⁶ The authors noted that FS—that they called “fibrin sheet”—was evident at VAD removal as a gray sleeve, 1-mm thick and 30-mm long, wrapped around the catheter.

Crain et al.¹⁰ described the FS as a thin protein layer that developed early 24h after VAD insertion and was developed over the full length of the catheter after 3–7 days.

In 1996, O’Farrell et al.⁹ studied FS experimentally in rats, by histological examination of veins cannulated with catheters; they finally described that the sleeve was not made of fibrin. In their experimental model, the sleeve developed approximately 7 days after catheter insertion, as an aggregate of whitish material surrounding the device; although in an initial phase fibrin was one of the components of the sleeve, the final structure had the appearance of a connective tissue. The authors hypothesized that such connective tissue might be the final evolution of a thrombotic event.

The most important experimental studies on FS were carried out few years later by Xiang et al.^{11,12} at the Catholic University of Leuven (Belgium). In a series of studies on animal models (rats and rabbits), these studies demonstrated that the FS is a cellular tissue, made of smooth muscle cells covered by a layer of endothelial cells.

In an experimental study on rats, silicon catheters were inserted through the posterior facial vein and the external jugular vein into the superior vena cava, and the sleeve was examined histologically by optical microscopy and by electronic microscopy.¹¹ The authors found the early formation of a cellular tissue made of smooth muscle cells, starting at the site of entrance of the catheter into the vein, where the endothelium had been damaged; approximately 7 days later, this cellular tissue had become a sleeve surrounding the catheter, consisting of smooth muscle cells and collagen, but covered with endothelial cells. Some evidence of the migration of smooth muscle cells from the

vein wall to the sleeve was also found after 2 weeks, by electronic microscopy and optic microscopy with immunohistochemical staining, as “bridges” of cells between the two structures.

This was the best confirmation that the mature FS is not a “fibrin” sleeve, but a structured connective tissue made of collagen and cells, coated by endothelial cells.

In a study conducted in 2001, the same group of researchers¹² described three different potential types of CRT. The first type of thrombus would occur at the site of entrance of the catheter and connect the vein wall with the device: they hypothesized that the FS would develop from this kind of thrombus, after migration of smooth muscle cells along the catheter. The second type of CRT would be the thrombus at the tip. The third type would be the “classic” thrombosis arising from the vein wall. Although this description/classification of CRT is now obsolete or—at least—inconsistent with the recent clinical studies on thrombosis, the differentiation between “sleeve” and “thrombus” is interesting and still valid today.

In 2005, Kira et al.²⁶ described a yellowish “fibrin-like” mass surrounding the tip of the catheter, macroscopically visible, hard but friable.

In 2006, the development and the histological structure of the FS were studied in an experimental study on pigs, after cannulation of the internal jugular vein.¹⁶ The anatomic samples were examined after different lengths of catheterization (7, 14, 30, and 45 days). The sleeve was studied by standard optic microscopy and the different cell populations were identified by immunohistochemical procedures. In this study, the sleeve—usually coating 33%–100% of the length of the catheter—was found in 100% of cases. After 7 days, the sleeve had a mixed composition, both cellular and acellular, with evidence of both smooth muscle cells and endothelial cells. On day 14, the cellular component was predominant. On day 30 and 45, the sleeve was a structured connective tissue, made of collagen and smooth muscle cells, coated by a neo-formed endothelium; several small bridges between the vein wall and the sleeve were evident, as previously described by Xiang et al.¹¹

Very recently, two experimental studies have readdressed another relevant issue, that is, the potential effect of different types of material on favoring or decreasing sleeve formation.

In an experimental study on sheep, Sylvia et al.²² have measured sleeve formation, 14 and 30 days after VAD insertion, utilizing different types of polyurethane catheters: non-treated, treated with chlorhexidine, treated with low-molecular-weight fluorine-oligomers, or treated with an amphiphilic poly(2-methoxy-ethyl acrylate) (PMEA)-based technology. The authors found that the chlorhexidine-treated catheters were characterized by a thinner and shorter sleeve if compared to the others, even though such difference was not statistically significant.

In an experimental study in rabbits, Tanabe et al.²³ have compared standard polyurethane catheters versus catheters coated with PMEA technology, with or without an internal stylet (so to test also the effect of the rigidity of the catheter on the formation of FS). After 14 days, sleeve formation was more evident around the catheters with stylet and in particular around the uncoated catheters. The authors concluded (a) that the increased mechanical stimulation over the vein wall—as exerted by the stiff catheters with the stylet—would increase sleeve formation and (b) that the PMEA coating would be able to decrease it.

Current definition and pathogenesis

The FS is an adequate though imperfect term for describing the connective tissue that envelops the VAD in its intravascular tract; it can be regarded as a kind of “foreign body” reaction of the blood tissue, caused by the presence of the catheter, and it probably occurs in most catheters that remain in place for more than 7 days.

The FS develops very quickly, as its formation starts very early, even 24h after the insertion of the VAD and is completed within 2 weeks.^{22,27} Although the fine mechanisms are not perfectly defined, it appears that the external surface of the foreign body (i.e. the catheter) attracts a circulating protein—fibronectin—produced by the liver. According to the most credited theory,²⁸ fibronectin attracts blood macrophages, which stick to the catheter and differentiate into smooth muscle cells and fibroblasts, starting the production of collagen, creating a connective tissue that slowly enwraps the catheter.

Although still there are some doubts about this pathogenesis—according to some evidence,^{11,12} smooth muscle cells also migrate from the site of the vein wall damaged by the entrance of the catheter—it is evident that FS is a phenomenon completely different from venous thrombosis.

As a “foreign body reaction,” some degree of FS develops not only around venous catheters but also on the external surface of any intravascular device, as long as it stays in contact with the bloodstream for a few days.

Incidence

For several reasons, the reported incidence of FS is extremely variable in the literature.^{2,3,14,21,29,30} FS formation is reported to occur in 10%–56%^{30,31} of central venous catheters in radiology series, but the rate is as high as 100% in some experimental studies.^{5,13,22}

Some authors have hypothesized a higher incidence of FS in short venous catheters, in central VADs with a tip malposition, and—in particular—in VADs with very long dwelling time such as tunneled-cuffed catheters for chronic hemodialysis. Such variability is related to the heterogeneity of the studies (experimental studies on animal models or on cadaver vs clinical studies) and to the criteria for diagnosis and definition of FS. In a clinical study on 57

oncological patients with central VAD who had no clinical signs of venous thrombosis, FS was detected by venography in 78% of cases.³² More recently, Boddi et al.²¹ have studied FS in 400 patients. Using ultrasound, the authors described FS as a hyper-echogenic tissue around the catheter, at least 20-mm long, with no relationship with the vein wall. They found FS in 12.8% of cases in the first month after VAD insertion. They commented that FS appears to be more frequent than CRT and that, in most cases, FS was not associated with catheter malfunction or other adverse events.

Whether the development of FS may be related to the presence of concomitant comorbidities such as diabetes or hypercholesterolemia is still a matter of debate. A small randomized study investigating the incidence of FS and of CRT in tunneled-cuffed dialysis catheters did not find a clear significant association between these events and pre-existing morbidities.³³

Clinical manifestations

In patients with VADs, the development of FS is a frequent but usually harmless pathophysiological phenomenon.

Should clinical manifestations occur, they typically occur late, when the connective sleeve around the catheter is almost complete. This may cause a mechanical obstacle to the function of the VAD, in particular when the tip of the catheter is enwrapped. The FS enveloping the tip may act as a unidirectional valve, causing different types of catheter malfunction—difficulty in infusion and/or difficulty in aspiration. A malfunction typically caused by FS is the so-called “persistent withdrawal occlusion” (PWO), characterized by a non-transitory impossibility of aspirating blood, though infusion remains feasible. While the mere difficulty in infusion is likely to be related most frequently to an occlusion of the lumen (due to clots, lipid aggregates, or drug precipitates), a PWO might arise the suspicion of FS. VADs with a closed end and a distal valve (Groshong valve) are more prone to be affected by the sleeve, since the connective tissue which surrounds the tip may easily interfere with the functioning of the valve.

However, PWO might be theoretically associated with other five causes (pinch-off syndrome, with compression of the catheter between the clavicle and the first rib; a malposition, with the tip of the catheter stuck into the vein wall or in a small secondary vessel; the presence of a Groshong valve, which is often associated with difficult opening of the valve in aspiration, even in absence of a sleeve; a CRT that has occurred at the tip of the VAD; and the presence of a clot at the tip of the catheter, obstructing it with a ball-valve mechanism). The percentage of catheter malfunction that is actually related to FS is unknown, due to a lack of uniform definition of the FS. In tunneled-cuffed dialysis catheters in silicone, some malfunction potentially related to FS was reported in 13%–57% of cases.³⁴

When the FS has completely wrapped the catheter tip, it may cause a complete obstacle: the infusate may not find its way into the bloodstream, but it may be pushed back into the cleavage between the catheter and the inner surface of the FS, with the risk of extravasation at the exit site (for non-tunneled VADs), inside the tunnel (for tunneled VADs), or inside the pocket of the reservoir (for totally implantable VADs). Such extravasation may be associated with severe tissue damage, if the infused drugs are vesicant.^{35,36} The incidence of extravasation or infiltration due to FS has not been defined. However, this event is probably more likely to occur with short catheters (5–10 cm) which stay in place for 2 weeks or more, such as long peripheral venous catheters (so called “mini midline”) in adults and children, or centrally inserted central catheters (CICCs) in pediatric patients.³⁷

Recently, FS has been held responsible for cases of difficult removal of peripherally inserted central catheters (PICCs) in adult patients. PICCs usually are not affected by catheter malfunction due to FS, probably because they are very long, and the sleeve takes many weeks to be completed. On the other hand, when removing a PICC, the sleeve may roll up around the catheter, in particular, in the tract where the catheter is in the small veins of the arm, and create some resistance to the removal, typically after the first 10–15 cm of withdrawal. In such cases, the maneuver of inserting a 0.018” floppy straight-tip guidewire into the PICC and rotating catheter and guidewire together usually is successful, since it breaks possible adhesions between the catheter and sleeve.

Sleeve versus catheter-related infections

The formation of FS is a phenomenon that occurs in probably the vast majority of VADs (in 100% according to some authors), while catheter-related infections are a relatively rare complication. Nonetheless, many studies have searched a possible correlation between FS and bacterial colonization, or between FS and infection.

A few old experimental studies^{38,39} have postulated that fibronectin, which plays a major role in the pathogenesis of FS, might attract bacterial adhesions, but the results were inconclusive. On the other hand, another study in rats suggested that the presence of FS might protect the catheter from bacterial colonization and from bloodstream infection after intravenous inoculation of *S. aureus*.⁵ More recent experimental studies have suggested a positive correlation between FS and staphylococci biofilm formation.^{13,40} In a study on silicon catheters implanted in rats, the intravenous inoculation of *Enterobacter cloacae* and *Staphylococcus epidermidis* was associated with a higher rate of positive blood cultures in catheters with FS (84%) versus catheters without FS (21%).¹³ In an experimental study by the same group, the administration of heparin was

apparently associated with a reduction in catheter colonization, which the authors explained as possibly related to the reduced formation of FS.¹⁵

In a recent experimental study comparing the effect of different PICC materials on FS formation in sheep,²² there was no significant association between FS and bacterial colonization: only one animal had FS colonized by bacteria.

Clinical studies are also inconclusive in this regard. Some old clinical studies showed some beneficial effects of urokinase⁴¹ and of heparin-bonded catheters⁴² on the prevention and treatment of catheter-related infections; in both cases, these effects were hypothesized to be secondary to the reduction of FS formation, since at that time, FS was considered a special type of thrombosis. However, current evidence shows that FS is not likely to be affected either by thrombolytic or by anticoagulant drugs. The benefit from urokinase might have been related to an indirect effect on infection, due to the reduction of intraluminal clots and/or venous thrombosis. In fact, in a clinical study testing the hypothesis that FS would favor bacterial adherence to the catheter,⁷ the authors found that the catheter-related infection was associated with CRT rather than FS.

In a retrospective clinical study on tunneled-cuffed dialysis catheters published in 2013, Shanaah et al.⁴³ found that the incidence of FS formation was 47%; considering all maneuvers of catheter replacement, there was no difference in the subsequent risk of infection between replacement of catheters with FS versus without FS.

However, there are a few cases reported in the literature where FS was suspected to be colonized by bacteria, causing a bacterial infection.^{18,44,45} In all these reports, there are some doubts about the exact diagnosis of FS as opposed to CRT, since the criteria of differential diagnosis by transesophageal echocardiography (TEE) are uncertain.

In a recent study by Hill et al.,²⁵ patients with isotope-highlighted sheaths experienced higher incidence of infective complications, particularly when the isotope uptake was high; the study is inconclusive, as it is possible that the isotope uptake was dependent on the actual presence of previous bacterial colonization.

Sleeve versus CRT

Although FS and CRT are quite different phenomena in terms of pathogenesis, many authors have often considered FS as a particular type of thrombosis.^{4,7,29,46,47} However, the incidence of FS is consistently reported as more frequent than CRT.^{7,14,21,30} Also, it appears to develop independently from the material of the catheter.^{4,30}

The confusion between FS and CRT has also affected the validity of clinical studies evaluating the possibility of reducing CRT using catheters made of special material, such as heparin-bonded catheters.³

Table 2. Main studies on correlation between fibroblastic sleeve and catheter-related thrombosis.

Author	Diagnosis	Catheter type	FS incidence (%)	CRT incidence
Brismar et al. ³⁰	Venography	CVC for parental nutrition	42	8%
Raad et al. ⁷	Postmortem examination	Non-tunneled CVC	100	38%
Oguzkurt et al. ¹⁴	Venography	Short-term dialysis catheters	56	28%
Alomari and Falk ⁴⁸	Venography	Tunneled dialysis catheters	76	24% of FS
Boddi et al. ²¹	Ultrasound	CVC for chemotherapy	13	1.25%

FS: fibroblastic sleeve; CRT: catheter-related thrombosis; CVC: central venous catheter.

Considering those clinical studies where FS and CRT were clearly differentiated, the following conclusions can be drawn:

- FS is far more frequent than CRT (see Table 2).
- The undesired clinical effects of FS (catheter malfunction, infection) seem to be associated with the concomitant presence of CRT.⁴⁹ It is known that some catheters may get fully “wrapped” in a calcified FS and may stick to the vessel wall: these catheters can break during removal attempts, leaving a visible remnant in situ, without other problems.
- There are no clinical or experimental data suggesting that FS formation may increase the risk of CRT;²¹ only one study—focused exclusively on tunneled dialysis catheters—has described thrombus formation in 24% of FS.⁴⁸

Further studies are necessary to clarify the relationship between FS and CRT. It is unknown whether the evidence of a thick or extended FS might be an indication to antithrombotic prophylaxis. This might be particularly relevant in oncological patients with high thrombotic risk (Khorana score of 2 or higher prior to starting a new systemic chemotherapy regimen), who—according to some authors⁵⁰—deserve anticoagulation.

Diagnosis

For many years, the only method for an appropriate diagnosis of FS has been the line-o-gram, that is, a series of X-rays taken after injection of contrast medium (5–20 mL) through the catheter itself; such method can visualize the FS only at its final stage, when the envelope around the catheter is complete and the contrast medium does not get into the bloodstream but gets back around the device, with a typical image of “double contour”.^{10,51} This method is also effective for ruling out other causes of PWO (ball-valve occlusion of the tip, malfunction of the distal valve of the device, pinch-off syndrome, tip malposition, and thrombosis around the tip). However, pinch-off syndrome and tip malposition can be detected with X-ray imaging without contrast as well. Moreover, malposition of the catheter may be suspected if its tip is not visualized by echocardiography. On the other hand, the “line-o-gram”

cannot detect an incomplete FS, since it cannot properly visualize those parts of the FS attached to the catheter wall but not associated with catheter malfunction.

In this regard, ultrasound is now playing a role more and more relevant, since it is capable of detecting parts of FS around the catheter, as long as the tract of vasculature where the catheter lays can be explored by ultrasound. Furthermore, ultrasound scan is a harmless, inexpensive, repeatable, “real-time” methodology that can be performed at bedside.

Although the use of ultrasound is widely diffused in the world of venous access, this has not been associated with an increased knowledge of the phenomenon of FS, since many operators commonly using ultrasound for the diagnosis of catheter-related complications still adopt the term “peri-catheter thrombosis” when they see echogenic material around the catheter. In other words, to use ultrasound devices and to be able to visualize the vein and the catheter is not enough: the operator needs clear criteria for an appropriate ultrasound-based diagnosis of FS. This is a very relevant issue from the clinical point of view, since the erroneous diagnosis of “peri-catheter thrombosis” may yield an inappropriate treatment with anticoagulant drugs.

The typical ultrasound image of an FS is a thin layer (>1 mm) of echogenic material located all around the catheter wall, as a sort of sleeve, with a regular surface and little or no relationship with the vein wall; the sleeve may be detected in any tract of the catheter, and sometimes may be so long to cover completely the catheter in its full length. Contrary to the venous thrombosis, this sleeve has no consistent adhesions with the vein wall, though there may be some “bridges” between the two structures (Figure 1).

The degree of echogenicity of the sleeve is very variable and may change in the course of time. While venous thrombosis has a typical pattern of echogenicity, moving slowly from anechogenic (fresh thrombus) to hyper-echogenic (old thrombus with fibrosis) in the course of some weeks, the echogenic density of FS is more difficult to predict. A hypo-echogenic sleeve can be more easily identified using color Doppler⁵² (Figures 2 and 3). Most FS become quite hyper-echogenic so that they can be clearly detected floating inside the vein after VAD removal (so-called “ghost of the catheter”; Figure 4).

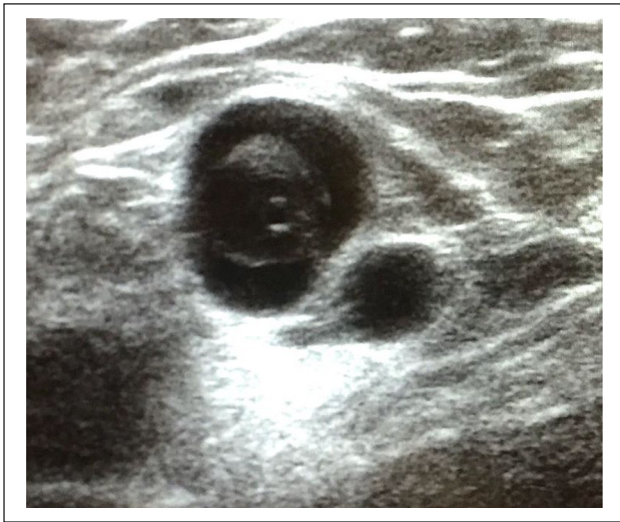


Figure 1. FS with hypo- and hyper-echogenic areas, completely surrounding the catheter. In this, as in the following figures, images were obtained using the same ultrasound devices, Philips Epiq 5 (3- to 12-MHz linear probe) and Philips iU22 (5- to 17-MHz linear probe).

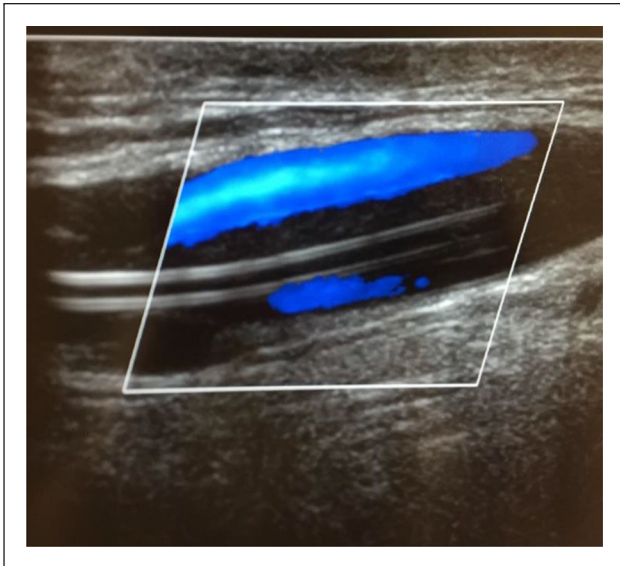


Figure 2. Longitudinal view of hypo-echogenic FS, with blood flow (in color) between FS and vein wall.

A proper ultrasound training of the operator is necessary to differentiate FS from CRT. As already stressed, such differential diagnosis has great clinical relevance, since CRT may require an anticoagulant treatment, while FS requires no pharmacological therapy; in fact, if the FS is not causing any functional problem, it needs no intervention at all.

The differential diagnosis between FS and CRT is often relatively easy, considering that the following: (a) as already stressed, FS develops around the catheter, with minimal attachment to the vein wall, at least initially,

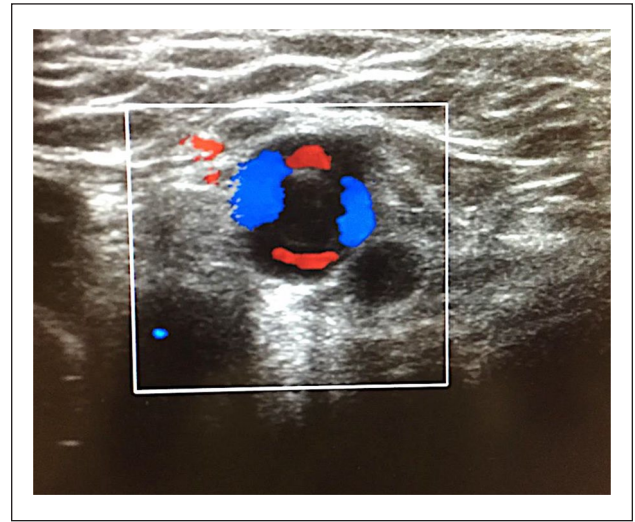


Figure 3. Transversal view of hypo-echogenic FS, with blood flow (in color) between FS and vein wall (so-called “circle sign”).



Figure 4. Residual FS—after catheter removal—floating inside the vein.

while CRT is a pathophysiological phenomenon that typically starts from the vein wall; (b) FS is almost never associated with local symptoms, while CRT can be both symptomatic or asymptomatic; and (c) FS may have different levels of echogenicity, while CRT—in its early stages—is anechogenic or hypo-echogenic; in other words, a hyper-echogenic image close to the catheter within few weeks after insertion will strongly suggest a sleeve rather than a CRT.

Nonetheless, in the asymptomatic patient, when hypo-echogenic material is detected around the catheter but also in contiguity with the vein wall, the differential diagnosis between FS and CRT may be difficult. In such cases, a strict follow-up (every 48–72 h) of the echographic findings and/or the dosage of D-dimers in plasma may be helpful,⁵³ though D-dimers have a high sensitivity but a low specificity in the diagnosis of venous thrombosis.⁵⁴

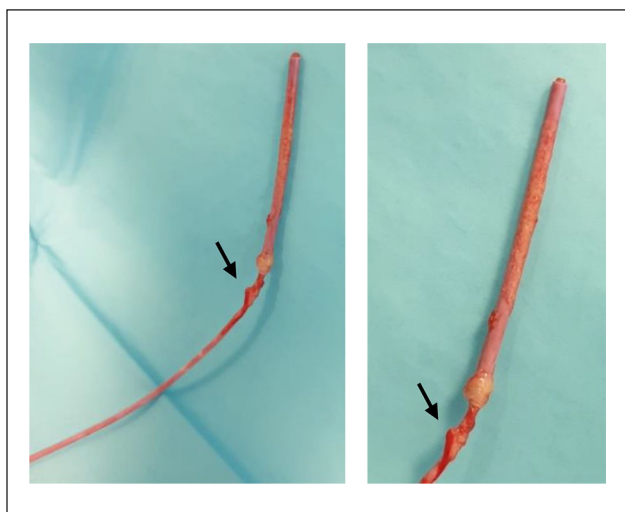


Figure 5. FS still adherent to the catheter wall, after removal of the VAD.

Proper ultrasound scan of the veins where the catheter is located may be important also before VAD removal or before replacement of the VAD over a guidewire. The detection of a probably recent CRT is a contraindication both to removal and to replacement over a guidewire because of the risk of accidental embolization of thrombotic material during the maneuver, with resulting pulmonary embolism. On the other hand, the detection of an FS may be a contraindication to replacement over a guidewire—since the new catheter will enter the pre-existing sleeve—but not to VAD removal.

Treatment

The fortuitous detection of an FS around a catheter does not require any treatment. In case of catheter malfunction, if there is good evidence that the malfunction is related to the FS (i.e. after a line-o-gram that has identified the sleeve and excluded other causes of malfunction), the best treatment is usually the removal of the VAD (Figure 5).

Needless to say, since the sleeve is made of connective tissue, heparin or thrombolytic drugs have no role, neither as prevention strategies nor as pharmacological treatment. Nonetheless, several old clinical studies have claimed the effectiveness of urokinase or recombinant tissue plasminogen activator (rt-PA) in treating an FS-related catheter malfunction:^{36,55–58} the bias of all these clinical experiences is that the authors could not exclude whether the malfunction was caused by intraluminal clots or venous thrombosis, accidentally associated to FS.

In long-term central VADs, several interventional radiology procedures for removal or disruption of the sleeve have been described.

Stripping of the sleeve using a snare^{59–66} has proven to be useful to restore function in ports with

FS-related malfunction, though its cost-effectiveness may be questionable. Endoluminal dilatation by ballooning (disruption of the sleeve by stretching) has been introduced by Hong⁶⁷ and later used by other authors,^{68,69} not only for FS removal but also for removal of adhesions between the vein wall and catheter or between the vein wall and transvenous pacemaker leads.⁷⁰ Disruption of the FS by angioplasty during an over-the-wire catheter exchange procedure has also been used in tunneled-cuffed dialysis catheters.^{71–73} Some authors have suggested to consider catheter exchange with or without balloon disruption of the FS in all malfunctioning tunneled-cuffed catheters for dialysis,⁷⁴ obviously when the malfunction is proven to be caused by the sleeve.

Unfortunately, all the techniques of interventional radiology for removing the FS without sacrificing the VAD (FS removal by stripping, by ballooning, and so on) are quite complex and expensive, and associated with a high risk of FS recurrence, so that their cost-effectiveness, at least in the field of non-dialysis VADs, is questionable.

Furthermore, there might be a possible association between percutaneous transluminal angioplasty balloon disruption of FS and late-onset central venous stenosis.⁷⁵

What happens to the sleeve after VAD removal?

The observation that the FS might persist for sometime inside the vasculature after VAD removal is very old. Rockoff et al.⁷⁶ visualized the FS in computed tomography (CT) scans obtained in children with previous long-term catheters.

FS can be easily visualized in the axillary or subclavian or internal jugular or brachio-cephalic vein after removal of a CICC, by a simple infra- or supra-clavicular ultrasound scan with a linear probe. FS will be even more easily seen in neonates and children, where the ultrasound examination is particularly easy, and/or utilizing good-quality linear probes with >10 MHz. A persistent FS might also be an accidental finding during TEE,¹⁸ appearing as a thin and long hyper-echogenic image floating in the superior vena cava. In some rare occasions, the persistent FS might be very thick and dense, and even calcified, and mistakenly interpreted as a retained catheter fragment at chest X-ray, CT scan, trans-thoracic echocardiography (TTE),^{26,77} or TEE.^{17,44,45,78–80}

Of course, the sleeve around the catheter will persist also in its extravascular tract (Figure 6).

As already mentioned, much of the clinical studies on FS come from the area of long-term dialysis catheters, which often remain in place for years. In this condition, the sleeve becomes quite structured and thick, so that it can be easily detected after VAD removal while it floats in the vasculature, often resembling the shape and length of the catheter (so-called “ghost of the catheter”). In

1994, Hombrouckx et al.⁶ reported the persistence of the FS inside the vein for sometime after VAD removal, commenting that this event was harmless. However, they also hypothesized that repeated episodes of sleeve

formation might be associated with a subsequent partial obstruction of the vein. This contention was not supported by evidence.

In an experimental model, FS was found to persist within the vein even 10 months after catheter removal.¹¹ However rarely, a persisting FS may undergo calcification: a calcified FS was detected 7 years after catheter removal.⁷⁷

In a retrospective study on 147 patients who had recently removed a central VAD, Krausz et al.²⁰ visualized the persistent FS at CT scan in 20 cases (13.6%); in 9 of the 20 cases, the sleeve was apparently calcified.

Calcification of the FS may be particularly seen in chronic renal failure patients with abnormalities of calcium and phosphate metabolism.^{81,82}

A calcified residual FS may be erroneously interpreted as a residual (or retained) catheter fragment.^{78,79,81} The differential diagnosis between FS and catheter fragment may require echocardiography^{18,83} or even CT scan.^{37,78}

There have been rare reports of removal of residual FS,^{26,77} though the actual usefulness of this maneuver is questionable.

There are no reports that may associate the persistence of the FS inside the vein with any risk of pulmonary embolism. In a clinical study on dialysis catheters,¹⁴ FSs were

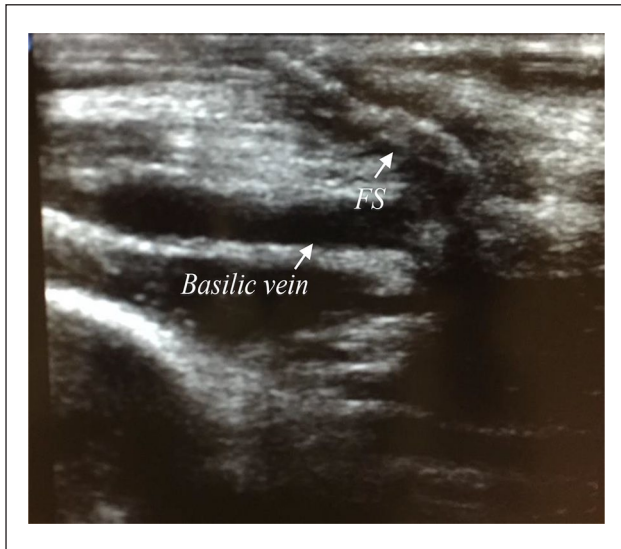


Figure 6. Residual FS (partially calcified) still evident in the extravascular tract of the catheter, after removal of the VAD.

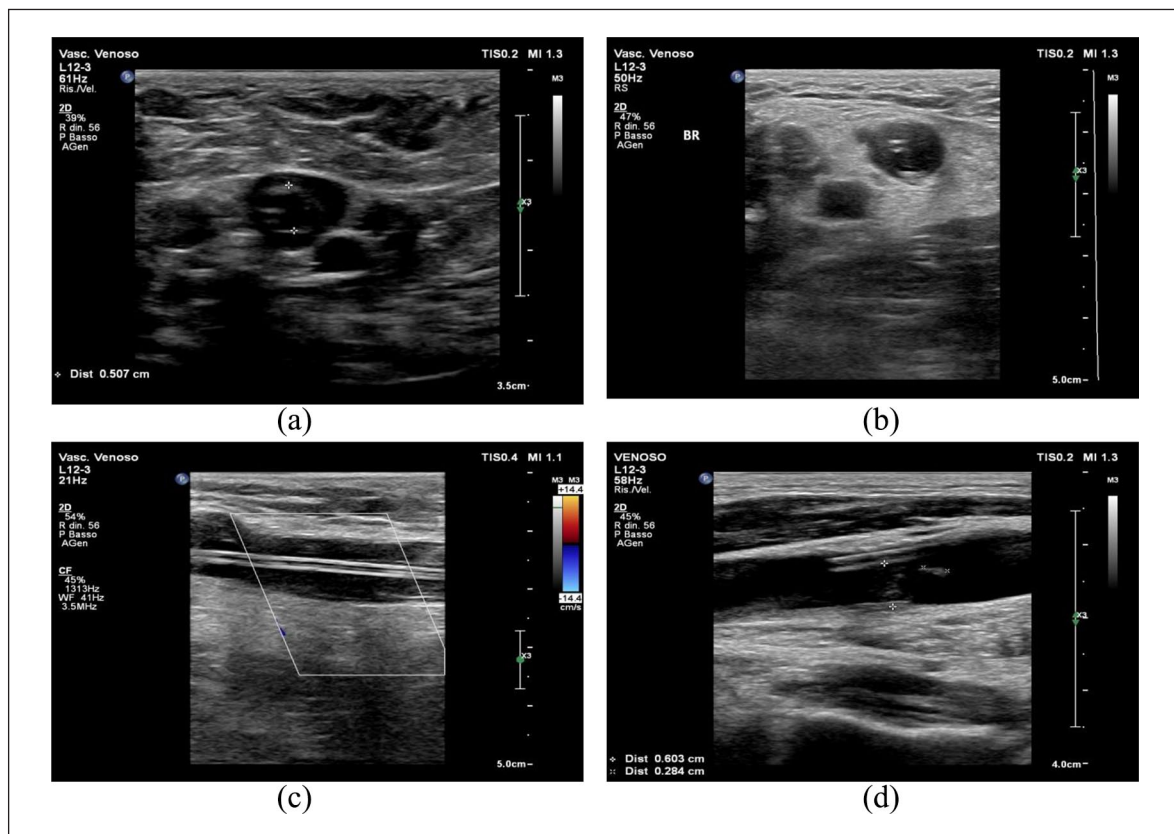


Figure 7. Different ultrasound images of (a) fibroblastic sleeve, (b) catheter-related thrombosis, (c) occlusive catheter-related thrombosis, and (d) non-occlusive (“mural”) catheter-related thrombosis.

Table 3. Main differences between catheter-related thrombosis (CRT) and fibroblastic sleeve (FS).

	CRT	FS
Etiopathogenesis	Endothelial damage	Foreign body reaction
Molecular trigger	Tissue thromboplastin	Fibronectin
Type of tissue	Thrombus	Connective tissue
Location	At the site of vein wall damage	Around the catheter
Evolution	Fibrosis/reabsorption	Reabsorption (?)
US imaging	Mass obstructing the vein Anechoic, and then hypo-echoic Mainly attached to the vein wall	Sleeve all around the catheter Hypo- or hyper-echoic Mainly attached to the catheter
Clinical manifestation	Signs and symptoms of venous obstruction + risk of catheter malfunction	Catheter malfunction
Risk of pulmonary embolism	Yes	No
Need for VAD removal	Rare (not responsive to therapy)	Rare (irreversible catheter malfunction)
Preventable with anticoagulants	Yes (not consistently)	No
Sensitive to thrombolysis	Yes (in the initial phase)	No
Pharmacological management	LMW heparin	None

VAD: venous access devices; US: ultrasound; LMW: low-molecular-weight.

consistently described after catheter removal and detected as floating inside the vasculature, without any event of pulmonary embolism.

Previous case reports of suspected FS-related pulmonary embolism^{30,84} are probably due to CRT, considering the confusion existing between FS and CRT in the 20th century.

On the other hand, the real problem of the persistent FS is that it may be erroneously interpreted as a thrombus, or as residual catheter fragment. The image of the persistent FS might be deceiving both during ultrasound examinations (preprocedural scan before VAD insertion, echocardiography, and so on) and during X-ray procedures (CT scan, fluoroscopy, and so on).

Considering the widespread use of ultrasound for vascular access, and in particular the use of ultrasound for preprocedural evaluation of the available veins before VAD insertion, the unexpected visualization of the sleeve of a previous central catheter is now a frequent event.

Conclusion

The FS is inevitable for any VAD, as long as it stays in place for at least 1 week, but it is almost always harmless and requires no treatment.

The sleeve has no relationship with CRT, if not for the fact they are both generated by an endothelial damage and by the presence of a foreign body in the vasculature (see Table 3 and Figure 7). The sleeve is a connective tissue, with both cellular and acellular components (smooth muscle cells, fibroblasts, endothelial cells, collagen), and is never associated with venous obstruction, or with local inflammation or with pulmonary embolism.

Although the sleeve may sometime cause catheter malfunction or extravasation, such complications occur only when the sleeve has completely wrapped the catheter for its whole length, typically in very short catheters

with at least 2 weeks of duration (long peripheral venous catheters or CICC in pediatric patients, or short multiple-lumen CICC in adult patients, with the tip above the tracheal bifurcation) or in catheters that remain in place for months and even years (tunneled-cuffed catheters for chronic dialysis, long-term VADs for home parenteral nutrition, and so on).

The most clinically relevant issue with FS is that, very frequently, it is erroneously interpreted as venous thrombosis, with obnoxious effect on the overall management of the patient. The appearance of a thin layer (≥ 1 mm) of echogenic material around a venous catheter, in particular if it has little or no contact with the venous wall, should be correctly interpreted as an innocuous connective tissue sleeve and not as “peri-catheter thrombosis.”

Author contributions

This narrative review was conceived by all authors (G.P., M.P., and A.L.G.). The literature was collected by G.P., through an electronic search of PubMed, MEDLINE, and Google Scholar without time restriction until April 2020. Search terms were (“fibrin sheath” OR “fibrin sleeve” OR “fibroblastic sleeve”) AND (“catheter” OR “thrombosis” OR “infection” OR “treatment”) in title, abstract, and keywords. The bibliographies of all located papers were examined and cross-referenced for further relevant literature. All types of articles were included. No unpublished or gray literature was searched. Papers in English or with English abstracts were included in the search. A critical appraisal of the collected studies was conducted by M.P. and A.L.G., evaluating the relevance and the consistency of the data. A final set of literature was evaluated by all authors. All authors contributed to the writing and revision of the final manuscript.

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