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Review

Inconsistent Use of Terminology and Different Treatment Outcomes of Venous Adventitial Cystic Disease: A Proposal for Reporting Standards

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Adventitial cystic disease (ACD) is a very rare condition characterized by the accumulation of a cyst filled with gelatinous substance in the adventitia of a vessel adjacent to the joint area. The cyst usually compresses the vessel lumen, causing claudication or leg swelling. The disease usually affects the popliteal artery. However, several cases of venous ACDs particularly in the common femoral or external iliac vein have been reported. The definition, diagnosis, and optimal treatment of ACD remain controversial because of its rarity and the inconsistent use of terminology. The heterogeneity of the reported cases is more prominent in venous ACD. Herein, the accurate terminology of cysts correlated to the joint (synovial cyst, ganglion cyst, and adventitial cyst) and the pathogenesis, anatomy, and optimal therapy of venous ACD are discussed in detail to establish reporting standards for future studies.

Key Words: Adventitial cystic disease, Veins, Synovial cyst, Ganglion cysts, Joints

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INTRODUCTION

In a recent issue of *Vascular Specialist International*, Lim et al. [1] presented a surgical strategy that can reduce the recurrence of adventitial cystic disease (ACD) after treatment. ACD is a very rare disease characterized by the accumulation of a cyst filled with gelatinous mucinous substance, similar to the joint fluid, particularly in the adventitia of a vessel adjacent to the joint area [2]. The growing cyst compresses the vessel lumen, causing stenosis or occlusion of the affected vessel. The disease usually affects the popliteal artery. However, several cases of venous ACDs particularly in the common femoral vein (CFV) and external iliac vein (EIV) have been reported [3].

However, the definition, diagnosis, and optimal treatment of ACD remain controversial because of its rarity and the inconsistent use of terminology. Herein, the accurate terminology of cysts correlated to the joint and the pathogenesis, anatomy, and optimal therapy of venous ACD are discussed in detail to establish reporting standards for future studies.

TERMINOLOGY OF CYSTS AND PSEUDOCYSTS

A cyst is a closed sac with a distinct membrane and division compared with the adjacent tissues. Hence, it is a cluster of cells that have grouped together to form a sac. Meanwhile, a pseudocyst is a cyst-like structure that lacks epithelial or endothelial cell linings. A pseudocyst or pseudoaneurysm commonly indicates a rupture of or leakage in a certain anatomic structure contained by fibrous connective tissues. For cysts around the joints, the term pseudocyst is seldom used. Instead, synovial cyst (SC) or

ganglion cyst (GC) is used interchangeably in several studies.

DIFFERENTIAL DIAGNOSIS OF CYSTIC DISEASES AROUND THE JOINT

A cyst compressing the artery or vein adjacent to the joint is not a pathognomonic sign of an ACD. Several cysts of different origins can compress the vessel near the joint because of the confined space adjacent to the joint area. A differential diagnosis is important for the optimal treatment and prevention of recurrence.

1) Synovial cyst

SC is a juxta-articular fluid-filled collection lined by synovial cells, indicating a true cyst. It represents a focal extension of joint fluid that may communicate with the joint via a stalk [4]. The prototype of SC is Baker cyst in the knee joint. SC may be caused by herniation of the synovium into the surrounding tissue or displacement of the synovium in the embryonal stage [5]. SC is usually associated with joint diseases, such as osteoarthritis, rheumatoid arthritis, and meniscus tear. Moreover, it is filled with synovial fluid ranging from normal to inflammatory synovial fluid, which is dependent on the underlying joint disease. The term bursae or bursitis can be used for some SCs arising from the bursa itself with no communication with the joint, particularly SC of the popliteal area in children.

2) Ganglion cyst

Previously, GC was believed to be caused by myxomatous degeneration of certain fibrous tissue structures [4]. However, GC is most likely caused by a flaw in the joint capsule or tendon sheath, and this allows the joint fluid to leak from the joint space, bulge, and be surrounded with fibrous connective tissues similar to other pseudocysts or pseudoaneurysms (Fig. 1) [5]. SC and GC differ primarily in terms of content and histologic features. That is, GC is a pseudocyst and is delineated by dense fibrous connective tissue, without any synovial lining. Moreover, it is filled with a more viscous content of hyaluronic acid and mucopolysaccharides similar to the synovial fluid but at a higher concentration.

On ultrasonography, sono-palpation (direct pressure applied over the structures of interest using the probe) and sono-compressibility are used to differentiate SC and GC [6]. Normally, SC is easier to compress, and GC is significantly harder to compress due to its thick viscous content and dense fibrous capsule.

3) Intraneural ganglion cyst

Intraneural ganglion cyst (NGC) is a variant of GC that affects the nerves causing peripheral neuropathy. A cyst arises from the synovial joint and propagates along the perineurium of the articular neural branch into the main parent nerve, which can result in pain or paralysis of the affected nerve territories. Its content and cyst wall composition are similar to those of GC.

4) Adventitial cystic disease

ACD is a variant of GC in which the cyst extends along the vessel wall adventitia, with a mechanism similar to that of NGC. Due to the strong fibroelastic nature of the adventitia, the cyst extends proximally or distally along the vessel until it grows large enough to compress the lumen in a concentric or eccentric manner (Fig. 2) [7]. Because the cyst extends in the confined area of the adventitia, it can easily compress the vessel lumen even when it is small. The extension can be cranial, caudal, transverse, or spiral along the vessel axis, which makes complete cyst excision extremely difficult. Because the cyst and the vessel share

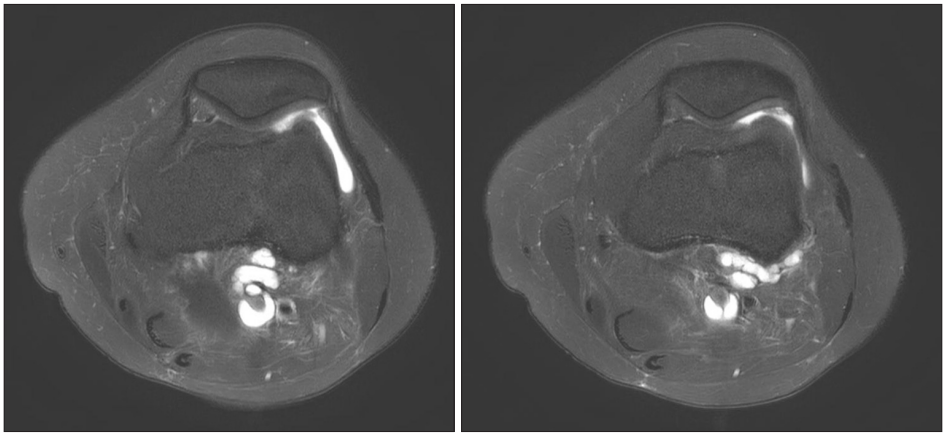

Fig. 1. Ganglion cyst encasing and compressing the popliteal vein. A joint connection was clearly observed.

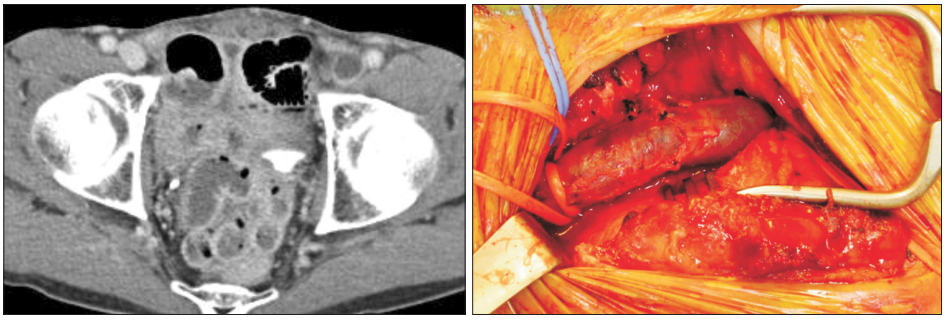

Fig. 2. Adventitial cystic disease compressing the left external iliac vein. Intraoperative image showing the adventitial collection of gelatinous material. Adapted from the article of Kang and Choi (J Korean Soc Vasc Surg 2009;25:163-166) [7].

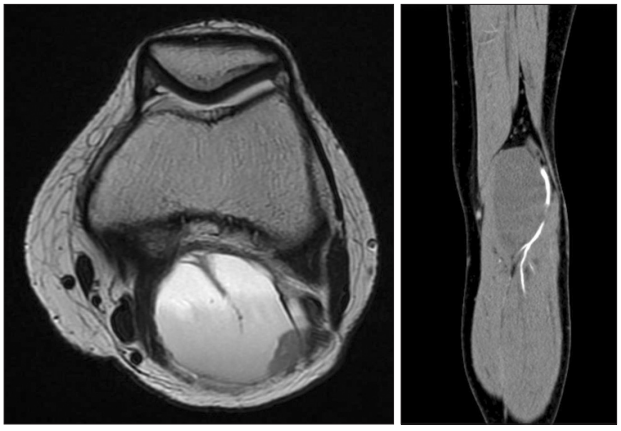

Fig. 3. Synovial sarcoma displacing and compressing the popliteal vessels.

the adventitia, it is immensely challenging to remove the cyst completely without causing vessel tear and aneurysmal dilation of the vessel wall.

5) Synovial sarcoma

Synovial sarcoma (SS) is a malignant soft tissue neoplasm and is easily misconceived as a sarcoma originated from the synovium. Because there is no evidence showing that SS originates from the synovial tissue, it is believed to arise from undifferentiated or pluripotent mesenchymal cells with a dual differentiation capacity (both epithelial and mesenchymal). In addition, it frequently presents as a biphasic tumor containing epithelial and spindle cells with various patterns. However, there is significant morphologic heterogeneity and overlap with a variety of other neoplasms, which can cause diagnostic challenges [8]. It is often observed in young adults during the 3rd and 5th decades of life and can arise from almost any site. However, the most common site is the lower extremities, particularly around the knees. Sometimes, its appearance is similar to that of a cyst with a thick wall compressing the popliteal vessels (Fig. 3).

CLINICAL CHARACTERISTICS OF ARTERIAL ACD

Arterial ACD is a rare type of non-atherosclerotic peripheral vascular disease and is characterized by the accumulation of mucinous substances in the arterial adventitia. These cystic lesions can cause stenosis or obstruction of the involved artery and symptoms of intermittent claudication at a relatively young age. In 1947, Atkins and Key first reported ACD of the external iliac artery [9]. Desy and Spinner [2] conducted a literature review of ACD, including 729 cysts in 724 patients. Results showed that 660 (91%) patients presented with arterial ACD. Moreover, lower extremity ACD was observed in 87%, bilateral involvement in 0.7%, joint connection in 17%, and recurrence in 9% of the patients. The most commonly involved vessel and vein were the popliteal artery (80.5%) and CFV (4.7%), respectively.

LITERATURE REVIEW OF VENOUS ACD

Venous ACD is an extremely rare condition, and an extensive literature review revealed that only 53 cases have been reported [2]. The condition is often misdiagnosed as deep vein thrombosis (DVT), femoral aneurysm, venous tumor, or lymphadenopathy. Bascone et al. [3] performed a collective review of 45 cases of venous ACD for 50 years (from 1963 to 2016). Based on the results, the most common site was the femoral vein (56%), followed by the EIV (24%), saphenous vein and others (13%), and popliteal vein (7%). The mean age of the patients was 47 (range, 5-75) years. Moreover, 50% were men, and 97.7% of cases involved the lower extremity. However, there was no case of bilateral involvement. The most common treatment was cyst evacuation and excision (66.7%), followed by vein resection with graft interposition (15.6%) and cyst aspiration with drainage (13.3%). The recurrence rate was highest after cyst aspiration and drainage (83.3%), followed by cyst excision (20%) and vein resection with interposition graft (14.3%).

Only 12 cases of venous ACD among Korean patients

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Table 1. Literature review of Korean patients with venous adventitial cystic disease

Case	Year	Reference	Age (y)	Sex	Side	Vein	Imaging	Size (mm)	Joint connection	Operation	Recur	2nd therapy	Follow-up exam (mo)
1	2005	Cho et al. [10]	68	M	L	EIV	CT, DUS	19	ns	Marsupialization			DUS; NED (8)
2	2005	Cho and Shin [11]	52	M	L	CFV	DUS	20	ns	CE			VG; NED (8)
3	2009	Kang and Choi [7], Seo et al. [12], Ann et al. [13]	69	M	L	EIV/CFV	DUS	23	ns	CE, patch repair	Yes	Sclerotherapy	ns; NED (36)
4	2011	Kwun and Suh [14]	40	F	L	CFV	DUS, CT, VG	ns	ns	CE			DUS; NED (6)
5			68	F	R	CFV	DUS, CT, VG	ns	ns	CE			DUS; NED (6)
6	2013	Park et al. [15]	50	F	L	EIV/CFV	DUS, MRI	44	Yes	CE			ns; NED (12)
7			32	F	L	EIV	DUS, MRI	21	Yes	CE, patch repair	Yes	CE	Occluded (10)
8	2016	Kim et al. [16]	50	F	L	CFV	DUS, CT	27	ns	Partial CE			ns
9	2019	Lim et al. [1]	39	F	R	CFV	DUS, CT	37	Yes	CE	Yes	CE	ns; NED (38)
10			33	F	L	CFV	DUS, CT, MRI	21	Yes	CE			
11			39	M	L	CFV	DUS, CT	12	No	CE, patch repair	Yes	CE	ns; NED (1)
12			55	F	L	Pop	DUS, CT	25	Yes	CE			

M, male; L, left; EIV, external iliac vein; CT, computed tomography; DUS, duplex ultrasonography; ns, not stated; NED, no evidence of the disease; CFV, common femoral vein; CE, cyst excision; VG, conventional venography; F, female; R, right; MRI, magnetic resonance image; Pop, popliteal vein.

have been reported based on 14 articles found in PubMed and KoreaMed [1,7,10-21]. The mean age of the patients was 49.6 (range, 32-69) years. Moreover, the condition was more commonly found in women (66%) and left-side dominance was observed (83%). The most common site was the CFV (58%), followed by the EIV (17%), both CFV and EIV (17%), and popliteal vein (8%). Approximately 41.6% of the patients had joint connections. However, in other cases, the presence of any joint connection was not noted. The patients were treated with complete cyst excision (n=7, 58%), cyst excision and patch angioplasty (n=3, 25%), and partial cyst excision (n=2, 17%). Four patients developed recurrence after 1, 19, 36, and 38 months after the surgery. Interestingly, three-fourths of these recurred patients were initially treated more aggressively with cyst excision and patch angioplasty, and only one patient who underwent complete cyst excision had recurrence. All patients treated with excision and patch angioplasty presented with recurrence (Table 1) [1,7,10-16]. This paradox raises serious questions doubting the heterogeneity of terminology and dubious diagnosis in the reported cases.

Considering that ACD is a cyst inside the adventitia with a connection from the nearby joint space, venous adventitial cyst shares the adventitia with the affected vein, and complete removal of the cyst wall compressing the vein is extremely difficult and commonly results in vein tearing or venous aneurysmal change after cyst resection. This usually leads to vessel replacement with a patch or an interposition graft. The joint connection should be eliminated to prevent recurrence. Some hypotheses can explain the discrepancies in severity, extent of the surgery, and recurrence rate. Recurrent cases even after vein excision may be true ACD. The joint connections were not eliminated even after patch angioplasty because ACD commonly develops at the CFV and EIV junction just below the inguinal ligament, which is extremely difficult to approach using either the inguinal or retroperitoneal approach. The lower recurrence rate after simple aspiration or evacuation of the cyst can be explained by the short follow-up period or misdiagnosis of SC or GC as venous ACD.

THEORIES ON THE POSSIBLE ETIOLOGY AND PATHOGENESIS OF ACD

1) Repeated trauma theory (microtrauma theory)

Because of repeated microtrauma, the vessel adventitia undergoes cystic degeneration caused by stretching and distortion of the vessel near the joints. However, a literature review showed that only 4% of patients had a history of trauma [2]. If ACD is caused by trauma, then a higher num-

ber of ACD cases will be reported, more athletes will present with the condition, and the incidence should be positively correlated to age. However, these were not observed in the literature. Moreover, this theory cannot explain the occurrence of ACDs in the radial, ulnar, and external iliac vessels. Therefore, there is no compelling evidence showing that trauma can be an etiologic factor of ACD.

2) De novo adventitial degeneration theory (systemic disorder theory)

Synovial mucinous degeneration in systemic disorders, including connective tissue disorder, can cause ACD. However, lack of consistent association with systemic disease in the literature, and very rare cases of bilateral or multiple involvement of ACD failed this theory to gain substantial support.

3) Developmental theory (embryological cellular inclusion theory)

Remnant mesenchymal mucin-secreting cell rests during development incorporate within the vessel adventitia, and can eventually cause mucin-filled cyst in the vessel adventitia [17]. However, the histologic markers for synovium are absent in ACD. This theory cannot explain cyst recurrence after complete cyst excision and within bypass grafts and rapid recurrences within a few months. Furthermore, the mean age of the patients was reported to be 47 years [3], and it is hard to believe that the ectopic synovial cells suddenly activate and secrete mucin after a long silent period of over 40 years. And this disease was observed only in one patient from the pediatric age group. Thus, the developmental theory cannot be considered [18].

Some advocates of the developmental theory insisted that the presence of joint connection could support this theory [19,20]. However, they failed to show the presence of synovial cells in the cyst. Moreover, the connection was misinterpreted as the pathway of the synovial cell migration during embryogenesis. However, the joint fluid, not the cells, migrates through the connection.

4) Articular theory (synovial theory, ganglion theory)

Desy and Spinner [2] reported that ACD begins with a capsular rent or defect that leads to the tracking of synovial fluid along a vascular articular branch. Vanhoenacker et al. [21] reported that they favored the articular theory, rather than the developmental theory, due to several reasons. First, the cyst commonly had communication with the adjacent joint along with the capsular branch of the affected artery. Second, the onset of ischemic symptoms during middle age in most patients supports the herniation theory. Third, the fact that the histologic composition of the cyst differs from that of the normal synovium is not against the synovial theory. The presence of the check-valve mechanism in the communicating stalk only allows unidirectional flow toward the cyst. Furthermore, the synovial theory provides a broader explanation of the origin of all types of para-articular cysts, including intraneural GC and ACD. Spinner et al. [22] presented numerous data with magnetic resonance imaging (MRI) findings to support the notion that ACDs, analogous to NGC, are also connected to the joints, and the middle genicular artery is a conduit through which the joint fluid pass from the knee joint synovium to the adventitia of the popliteal artery.

5) Is the same theory applicable for arterial and venous ACD?

Several theories on the etiology of arterial ACD have been applied in discussions on the etiology of venous ACD. However, Paravastu et al. [23] argued against the equivalence of the two processes based on epidemiologic factors. Arterial ACD and venous ACD have different clinical manifestations, such as the location of the most commonly affected vessels (popliteal artery vs. CFV) and male-to-female ratio (5:1 vs. 1.6:1). In contrary, Desy and Spinner [2] mentioned that it seems illogical that arterial and venous ACD have different etiologies as suggested according to Occam's razor. Thus, we support the hypothesis that arterial ACD and venous ACD have a similar etiology.

CLINICAL PRESENTATION OF CYSTIC DISEASES AROUND THE JOINTS

While arterial ACD presents with intermittent claudication or other ischemic symptoms, venous ACD presents with painless unilateral swelling of the involved extremity by compressing the vein. However, a cystic mass compressing the vein is not always an ACD, and a large SC, GC or cystic tumor can compress the vessels. Colasanti et al. [24] reviewed 27 cases of vessel compression caused by a SC of the hip joint. The mean age of the patients was 62 (35-80) years. Approximately 60% were women, and 55% presented with joint disorders, such as osteoarthritis and rheumatoid arthritis.

DIAGNOSIS OF ACD

A simple X-ray is effective in assessing the presence of joint disorders. Duplex ultrasonography (DUS) can facilitate

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the differentiation of SCs from femoral aneurysms or DVT. DUS can reveal a hypoechoic fluid-filled cyst accompanied by a posterior acoustic window.

Computed tomography (CT) or angiographic finding of ACD include an hour-glass sign, scimitar sign, and scalloped appearance. CT can reveal the site and extent of the obstruction. Hence, it is considered the best diagnostic modality because it can identify the cyst and its content, adjacent vessels and compression, presence of collateral vessels, and possible communication between the cyst and adjacent joint. The cystic structure can be evaluated on not only cross-sectional images but also coronal or sagittal images, which can be used to easier tract the joint connection.

On MRI, a cyst can be observed as a lesion with homogeneous low signal intensity on T1-weighted images and multiloculated high signal intensity adjacent to the vessels on T2-weighted images. MRI is helpful because of its increased tissue definitions, greater likelihood of finding a joint connection, and aid with surgical planning. Maged et al. [25] reported that high spatial resolution MRI can help achieve an accurate preoperative diagnosis and can detect very small communications between the ACD and the joint space, thus, it can decrease the incidence rate of ACD recurrence after surgery. Sagittal CT scan or MRI are considered optimal imaging techniques because they can validate clinical suspicion and identify the relationship between the mass and the surrounding structures [2].

However, current imaging studies cannot differentiate ACDs from SC or GC. ACD can be diagnosed wholly based on operative findings (sharing common wall with the vessel) and its pathology (no synovial lining). The more critical role of preoperative imaging is that it can detect the presence and location of the joint connection, which can help establish a proper surgical plan and decrease recurrence. Maged et al. [25] emphasized that multiplanar imaging, three-dimensional reconstructions, and high spatial resolution combined with the judicious use of a contrast agent (intra-articular and intravenous) can further assist in the visualization of the joint connection and other anatomic relationships. Neto and Nunnes [26] revealed that the role of delayed arthrography is particularly valuable in obtaining a differential diagnosis between atypical GCs and cystic tumors. Either via radiography, CT scan, or MRI, 1 to 2 hours after the intra-articular injection of water-soluble contrast agent, delayed arthrography improves the sensitivity of identifying a cyst–joint communication, which is detectable in less than 50% by standard DUS or MRI.

TREATMENT OF CYSTIC DISEASES AROUND THE JOINTS

Several surgical techniques have been applied for treating arterial or venous ACD.

1) Percutaneous image-guided needle aspiration

Colombier et al. [27] advocated for percutaneous CT-guided aspiration as the initial treatment option for small cysts. However, they recommended close long-term follow-up to detect recurrence, and the recurrence rates are high due to viable mucin-secreting mesenchymal cells left in situ or remnant joint connection of the cyst wall [3]. Therefore, simple aspiration is not recommended.

2) Aspiration and sclerotherapy

Johnson et al. [28] reported a case of recurrent ACD of the femoral vein after simple aspiration, which was successfully treated with aspiration, and ethanol sclerosis without recurrence for 18 months. Moreover, Ann et al. [13] presented a case of recurrent ACD at the iliofemoral vein after cyst excision and polytetrafluoroethylene patch angioplasty, which was treated with aspiration and ethanol sclerotherapy, and recurrence was not observed for 3 years. A minimally invasive therapy can decrease the risks of open surgery, including complications of general anesthesia, hemorrhage, infection, anastomotic failure, and DVT. However, only two cases were reported thus far, and a larger study must be conducted in the future to assess the efficacy of sclerotherapy. Theoretically, the complete evacuation of multiloculated cysts with thick mucin contents is not easy, and a weak adventitia can result in hemorrhage or inadvertent ethanol injection into the normal vein.

3) Transadventitial or transluminal cyst drainage with cyst wall excision

A previous study has shown good immediate outcomes with flow restoration after cyst drainage [29]. Because the cyst is usually located in the posterior wall of the vein using the femoral or popliteal approach, two venotomies in the anterior and posterior walls are sometimes required [29]. However, recurrence is common when the wall of the cyst is not completely excised or the joint connection is not obliterated.

4) Complete excision of the cyst wall or exarterectomy

Considering that the recurrence rate of ACD is 20%, Bas-

cone et al. [3] emphasized the importance of thorough cyst wall resection to prevent the cyst-lining mesenchymal cells to be left behind in the adventitia and to secrete enough mucin causing recurrence. However, this is partly true in SC with synovial cell lining. GC and ACD have no synovial cell lining, and the remaining cyst wall has no cells that can produce mucin causing recurrence. Hence, the incomplete removal of the joint connection may be the actual cause of recurrence.

5) Vein wedge resection and patch angioplasty

After removing the cyst wall in the adventitia, the vein wall becomes weak, which results in aneurysmal dilation. In this case, vein wedge resection and patch angioplasty can be performed. This method is more extensive than exarterectomy. However, several cases of recurrence after these surgeries were reported [1,30]. In iliofemoral ACD, the joint connection is usually located on the posterior wall beneath the inguinal ligament. Longitudinal inguinal incision or retroperitoneal incision alone may not be enough to approach the cyst ends in the EIV and CFV, respectively. This can cause recurrence by missing the joint connection at the end of the incision. The role of preoperative localization of the joint connection and optimal surgical approach must be discussed. Thus, disobliteration of the joint connection may be the key to prevent recurrences in iliofemoral venous ACD [1].

6) Vein resection and interposition graft

If a cyst, such as SC or bursae, has synovial cell lining, the cyst wall should be removed completely. If the cyst, such as GC or ACD, has no synovial lining, the removal of the joint connection is more important. During surgery, the joint connection is found deep to the diseased vein with a cyst in the opposite wall, and the stalk with joint connection cannot be easily identified. Therefore, vein resection and interposition graft should be the choice of operation to prevent recurrence by removing the joint connection with the cyst.

However, Bascone et al. [3] reported that the recurrence rate after vein resection and interposition was 14.3%. This result indicates that there is still communication with the joint space. Hence, the longer vessel segment should be resected. Because most surgical approaches to the cyst are used in the opposite direction of the joint communications, the connections are located below the vein hidden from the surgeon's direct vision. Thus, joint connections can be easily missed during surgery, if not detected by preoperative imaging. Interestingly, in two patients, cyst recurrences occurred within the saphenous vein bypass grafts in arterial ACDs, and a knee joint connection was identified during revision surgery, which supports the articular theory and the importance of completely removing any joint connections [31,32]. Desy and Spinner [2] showed that in cases of ligation of the joint connection as part of the treatment, no cyst recurrence was found. Considering that the arterial ACD follows the branch of the middle genicular artery, all branches behind to the direction of the joint should be ligated.

7) Role of endovascular therapy on the treatment of ACD

Angioplasty and stenting are not recommended for the treatment of ACD because they cannot deal with the extrinsic compression material and can eventually result in recurrence and treatment failure. Stenting across the joint and in the extremity vein is usually contraindicated.

Considering the mechanism of ACD, the primary goal is to relieve vein compression caused by the cyst contents. This can be achieved by percutaneous aspiration, open drainage or marsupialization, cyst excision, or vein excision with the cyst. However, to prevent recurrence caused by the re-accumulation of the viscous mucinous fluid, removing the source of the synovial fluid is the key for curative surgery.

Needle aspiration/puncture can decrease the size and symptoms of the cyst and is easier to perform and less invasive than surgery, and the contents of a cyst can help in obtaining a diagnosis. Thus, considering the minimally invasive nature of aspiration, some studies recommend this procedure as the initial treatment option [5]. If the involved vessel is not occluded, cyst aspiration can be performed first without causing a scar, followed by mandatory follow-up imaging. If the patient had symptomatic recurrence, re-aspiration or curative surgery can be performed after a complete discussion with the patient. Therefore, the treatment modality should be tailored after weighing the degree of invasiveness and the risk of recurrence.

RECOMMENDED REPORTING STANDARDS FOR VENOUS ACD

The terminology of the diagnosis, ACD or cystic adventitial disease (CAD), is used interchangeably in the literature. We propose to unify the term as ACD, because it is a cystic disease of the adventitia, rather than an adventitial disease of the cyst. In addition, CAD often stands for coronary or carotid artery disease, which can cause confusion.

To define the risk factors of ACD, the possible etiologic details, including age, sex, history of trauma, occupation

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involving repetitive or heavy exercise, family history, and any medical history of vasculitis or connective tissue disorder, should be presented.

Preoperative imaging studies, including, DUS, CT, and MRI, should be presented in detail. We recommend to perform DUS as a screening test to identify a cystic disease in the vessel wall. Then, a cross-sectional study of CT/MRI should be conducted to confirm the finding. The diameter and length of the cyst, location and direction of the cyst compressing the vessel wall, and presence of joint connection should be evaluated and reported.

Operative findings should include the incision site particularly for iliofemoral ACD, length of the cyst compressing the vessel, degree of difficulty separating the cyst and the vessel wall, bulging of the vessel wall after cyst removal, presence or obliteration of any joint connection, and content of the cyst. The operative technique should include simple drainage, drainage and cyst excision, partial vessel excision with the cyst and patch angioplasty, and vessel excision with the cyst and interposition graft. The pathology should include the presence of epithelial lining of synovial cells in the cyst wall.

The interval, duration, and imaging modality of postoperative follow-up should be reported because early recurrences were reported within 6 months [19,29-32]. We recommend serial imaging follow-up at 3, 6, and 12 months after the surgery, and follow-up with DUS at least after 12 months is necessary to identify whether the treatment was successful without recurrence. If any recurrence developed, the timing, imaging studies, and treatment should be reported as described before.

CONCLUSION

When we report a cyst compressing the vein around the joint, we need to distinguish ACD from SC, GC, or SS. For future collective review of the disease, we recommend to follow the reporting standards described above, including imaging studies of axial or sagittal views, surgical approach and methods, nature of the cyst material, presence of any connection, pathologic outcome of the cell lining on the cyst wall, and timing and pattern of recurrences.

CONFLICTS OF INTEREST

The authors have nothing to disclose.

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Concept and design: SKM. Analysis and interpretation: SKM. Data collection: AH, YJP. Writing the article: SKM. Critical revision of the article: AH, SM. Final approval of the article: all. Statistical analysis: none. Obtained funding: none. Overall responsibility: SKM.

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