Core Curriculum

Diagnosis and Treatment of Spontaneous Coronary Artery Pseudoaneurysm: Rare Anomaly with Potentially Significant Clinical Implications

Subrata Kar,1* DO and Richard R. Webel,2 MD

Spontaneous coronary artery pseudoaneurysm (PSA, false aneurysm) is an extremely rare occurrence with the precise incidence unknown. It is defined as an outwardly bulging monolayer or double layer within the coronary artery that lacks all 3 layers (intima, media, and adventitia) of the arterial wall. Coronary PSA commonly occurs from arterial dissection or perforation induced by catheter intervention, infection, pregnancy, or trauma. Traumatic dissection or perforation of the coronary artery after a percutaneous coronary intervention (PCI) remains the most common cause. Such cases may progress to myocardial ischemia, acute myocardial infarction, or acute coronary artery rupture causing death from cardiac tamponade. Intravascular ultrasound or cardiac computed tomography may aid in the diagnosis. Treatment options include PCI with a covered stent, bare or drug-eluting stent, coil embolization, or conservative management with vigilant clinical follow-up. In this review, we sought to describe the diagnosis, etiology, treatment, and the limited literature on spontaneous coronary artery PSA.

Key words: coronary pseudoaneurysm; coronary aneurysm; intravascular ultrasound; percutaneous coronary intervention; cardiac stent

INTRODUCTION

A coronary artery aneurysm is a focal dilation of a coronary artery segment whose diameter exceeds that of the adjacent normal coronary artery by a maximal luminal diameter of at least 50% [1,2]. Coronary ectasia is also defined as a coronary artery diameter of more than 1.5 times the adjacent normal coronary artery, but it is a diffuse dilation of >50% of the vessel length, as opposed to a coronary aneurysm which is a focal expansion of <50% of the vessel length [3,4]. Coronary artery aneurysms are further classified as true or false. The wall of a true aneurysm contains all three vascular layers (i.e., intima, media, and adventitia). A false aneurysm or coronary artery pseudoaneurysm (PSA) lacks at least one of these layers of the arterial wall and consists of an outwardly bulging monolayer or double layer, which develops from loss of vessel wall integrity, typically with disruption of the external elastic membrane [1,2,5].

Intravascular ultrasound (IVUS) may provide superior definition of a coronary PSA versus a true coronary aneurysm compared with angiography [5]. On IVUS, a coronary artery PSA is large, thin-walled, saccular structure with the absence of distinct vascular wall layers. It communicates with the arterial lumen usually

1Division of Cardiovascular Medicine, Texas Tech University Health Sciences Center, Paul L. Foster School of Medicine, El Paso, Texas
2Department of Medicine, University of Missouri School of Medicine, Columbia, Missouri

Disclosures: None

Conflict of interest: Nothing to report.

*Correspondence to: Subrata Kar, D.O., Assistant Professor of Medicine, Division of Cardiovascular Medicine, Texas Tech University Health Sciences Center, Paul L. Foster School of Medicine, 4800 Alberta Ave, El Paso, TX 79905. E-mail: skar762@aim.com

Received 15 November 2016; Revision accepted 28 January 2017

DOI: 10.1002/ccd.26997
Published online 00 Month 2017 in Wiley Online Library (wileyonlinelibrary.com)

© 2017 Wiley Periodicals, Inc.
Coronary PSA or true coronary aneurysm should be considered the cause of ischemia and subsequent MI. Cavity and adjacent blood vessel contributing to thrombus formation, rupture, thrombosis, distal embolization. Platelet aggregation within the PSA may cause ischemia by engendering endothelial dysfunction, impaired coronary flow, slow flow, altered flow dynamics, or platelet aggregation within the PSA cavity and adjacent blood vessel contributing to thrombosis inducing coronary ischemia and subsequent MI. Coronary PSA or true coronary aneurysm should be considered if a young patient with connective tissue disease develops an acute MI [12]. Coronary PSA may also develop after a coronary dissection, but the incidence is probably low.

**Differentiation between a Coronary Aneurysm and PSA**

True coronary artery aneurysms usually remain stable in size or enlarge slowly which engenders a favorable prognosis so it can be monitored for enlargement or development of symptoms [15]. Table I lists the characteristics of true coronary aneurysms versus coronary PSAs. The natural history and optimal treatment of coronary PSA is unknown [16]. If a true coronary aneurysm is suspected as cause of myocardial ischemia then invasive treatment may be necessary; however, it is more common that an associated stenosis is the cause of ischemia or MI, not the aneurysm [16]. If a patient has no evidence of myocardial ischemia or is asymptomatic, then close observation may be warranted. Cardiac CT may be used for clinical follow-up of a true aneurysm. If the aneurysm is large with the risk of rupture, then percutaneous or surgical intervention may be necessary. However, no precise definition exists for size and it’s relation to rupture in coronary PSA. It may be postulated that a large PSA with thrombus formation, large PSA, which is expanding during clinical follow-up, or a patient who is clinically symptomatic may pose a greater risk of rupture and merit either percutaneous or surgical intervention. Anti-coagulation may be useful since many large true coronary aneurysms contain thrombus [16].

**Etiology of True Coronary Aneurysms and PSAs**

True coronary aneurysms may be atherosclerotic (most common), congenital, inflammatory, infectious, metastatic tumors, blunt chest trauma, spontaneous or induced by blunt chest trauma [5–11]. Inflammatory causes include Kawasaki disease, Takayasu arteritis, lupus erythematosis, or polyarteritis nodosa [9,10]. Infectious causes include endocarditis or syphilis [1]. True coronary aneurysms occur in 1.5% of patients discovered during cardiac catheterization or autopsy [12]. True coronary aneurysms occur more often in the right coronary artery (RCA, 40–61%) followed by the left anterior descending coronary artery (LAD; 15–32%) and left circumflex artery (15–23%). The left main coronary artery is rarely affected (0.1–3.5%) [2].

Coronary PSAs may be caused by arterial dissection or rupture during catheter interventions (balloon angioplasty or stent implantation), infection, trauma, or during pregnancy [13,14]. Of these causes, traumatic coronary dissection or perforation after a percutaneous coronary intervention (PCI) remains the most common cause [14]. In rare instances, stent implantation may cause an iatrogenic coronary PSA. PSA occurring after a PCI may induce ischemia and lead to an acute myocardial infarction (MI) or rupture leading to death from cardiac tamponade [1,15]. True aneurysm or PSA may possibly cause ischemia by engendering endothelial dysfunction, impaired coronary flow, slow flow, altered flow dynamics, or platelet aggregation within the PSA cavity and adjacent blood vessel contributing to thrombosis inducing coronary ischemia and subsequent MI. Coronary PSA or true coronary aneurysm should be considered if a young patient with connective tissue disease develops an acute MI [12]. Coronary PSA may also develop after a coronary dissection, but the incidence is probably low.

**TABLE I. Characteristics and Treatment of True Coronary Artery Aneurysm versus Coronary PSA**

<table>
<thead>
<tr>
<th>Type</th>
<th>Cause</th>
<th>Symptoms</th>
<th>Course</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aneurysm (focal coronary artery dilation at least 50% larger than an adjacent coronary segment; contains all 3 layers of the vascular wall)</td>
<td>Atherosclerotic (most common), congenital, inflammatory, infectious, metastatic tumors, blunt chest trauma, spontaneous</td>
<td>Chest pain, ischemia, fatigue, dyspnea, palpitations, syncope, most may be asymptomatic</td>
<td>Stable in size or enlarges slowly, fistula formation, rupture, distal embolization</td>
<td>Spontaneous resolution, stent, CABG</td>
</tr>
<tr>
<td>PSA (lacks at least 1 layer of the vascular wall and contains an outward bulging mono-layer or double layer)</td>
<td>Arterial dissection or rupture (common cause), Post-PCI, atherosclerotic, infection, trauma, pregnancy, spontaneous</td>
<td>Chest pain, ischemia, MI, death</td>
<td>May expand rapidly and rupture, cardiac tamponade, thrombosis, distal embolization</td>
<td>Stent, CABG, possible coil embolization</td>
</tr>
</tbody>
</table>

CABG, coronary artery bypass graft surgery; PCI, percutaneous coronary intervention; MI: myocardial infarction.
Patients with aneurysmal CAD had a greater incidence of MI (52.9% vs. 47.0%, \( P < 0.001 \)) and triple vessel CAD compared with controls (42.3% vs. 34.2%, \( P < 0.001 \)). The risk factors for CAD among patients with and without aneurysmal CAD were similar. When accounting for the combination of the number of diseased vessels, the number of proximal diseased vessels, and left ventricular score, no statistically significant independent survival difference was found suggesting that conservative management of true coronary aneurysms may be acceptable. Table II lists the various reported cases of coronary artery PSA and their treatment.

However, coronary artery PSAs which develop after PCI may be less likely to resolve if untreated. PSA formation from vessel trauma during PCI could possibly lead to endothelial dysfunction, release of inflammatory mediators, platelet adhesion and aggregation, which can promote thrombosis with distal embolization. Moreover, PSA may rapidly enlarge and rupture causing cardiac tamponade [15]. The natural history of spontaneous coronary PSA remains poorly defined since the incidence is

### TABLE II. Summary of Reported Cases of Coronary Artery PSA

<table>
<thead>
<tr>
<th>Case</th>
<th>Location of PSA</th>
<th>Etiology</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Spontaneous PSA</td>
<td>Mid left main coronary artery</td>
<td>Spontaneous (gravid)</td>
<td>CABG, left main coronary artery ligation</td>
</tr>
<tr>
<td>Rahman et al. [21]</td>
<td>Proximal RCA</td>
<td>Spontaneous</td>
<td>Medtronic stent</td>
</tr>
<tr>
<td>Aqel et al. [5]</td>
<td>Proximal LAD</td>
<td>Spontaneous coronary artery dissection medically treated evolving into a PSA</td>
<td>Four everolimus-eluting stents</td>
</tr>
<tr>
<td>Furuichi et al. [42]</td>
<td>Mid LAD</td>
<td>Spontaneous</td>
<td>2 covered stents (PTFE)</td>
</tr>
<tr>
<td>Kawano et al. [29]</td>
<td>Proximal RCA</td>
<td>Spontaneous coronary artery dissection medically treated evolving into a PSA</td>
<td>CABG, resection and repair of PSA</td>
</tr>
<tr>
<td>Izutani et al. [18]</td>
<td>Mid LAD</td>
<td>Spontaneous coronary artery dissection medically treated evolving into a PSA</td>
<td>3 overlapping drug-eluting stents</td>
</tr>
<tr>
<td>Dai et al. [28]</td>
<td>Proximal LAD</td>
<td>Spontaneous</td>
<td>CABG, PSA excision</td>
</tr>
<tr>
<td>Wang et al. [43]</td>
<td>Mid RCA</td>
<td>Spontaneous connective tissue disease</td>
<td>CABG, PSA ligation</td>
</tr>
<tr>
<td>Kumar et al. [12]</td>
<td>Mid RCA</td>
<td>Spontaneous</td>
<td>CABG, ligation of PSA</td>
</tr>
<tr>
<td>Frischknecht et al. [23]</td>
<td>Mid RCA</td>
<td>Post-partum spontaneous coronary artery dissection medically treated evolving into a PSA</td>
<td>CABG, ligation of PSA</td>
</tr>
<tr>
<td>Post-partum PSA</td>
<td>Mid RCA</td>
<td>Post-partum spontaneous coronary artery dissection medically treated evolving into a PSA</td>
<td>Covered stent (PTFE)</td>
</tr>
<tr>
<td>Dhakam et al. [14]</td>
<td>Mid left main coronary artery</td>
<td>Post-partum spontaneous coronary artery dissection medically treated evolving into a PSA</td>
<td>2 Paclitaxel-eluting stents</td>
</tr>
<tr>
<td>Chabrot et al. [20]</td>
<td>Distal left main coronary artery</td>
<td>Post-partum spontaneous coronary artery dissection medically treated evolving into a PSA</td>
<td>CABG and PSA excision</td>
</tr>
<tr>
<td>Post-PCI PSA</td>
<td>Proximal LAD</td>
<td>Post-PCI</td>
<td>CABG</td>
</tr>
<tr>
<td>Kapoor et al. [35]</td>
<td>Proximal LAD</td>
<td>Post-PCI</td>
<td>CABG and PSA resolution</td>
</tr>
<tr>
<td>Chen et al. [34]</td>
<td>Proximal LAD</td>
<td>Post-PCI</td>
<td>Debridement of PSA, excision of infected everolimus-eluting stent, SVG to LAD and LCx</td>
</tr>
<tr>
<td>Schobel et al. [30]</td>
<td>Mid LAD</td>
<td>Postangioplasty</td>
<td>SVG to LAD and LCx</td>
</tr>
<tr>
<td>Lim et al. [36]</td>
<td>Mid LAD</td>
<td>Infected PSA post-PCI</td>
<td>2 Paclitaxel-eluting stents</td>
</tr>
<tr>
<td>Shintani et al. [25]</td>
<td>Mid LAD</td>
<td>post-rotational atherectomy</td>
<td>Bare-metal stent</td>
</tr>
<tr>
<td>Cabarrus et al. [26]</td>
<td>Proximal RCA</td>
<td>Postangioplasty</td>
<td>CABG and PSA excision</td>
</tr>
<tr>
<td>PSA of SVG</td>
<td>Proximal SVG to LAD</td>
<td>Spontaneous</td>
<td>Coil embolization</td>
</tr>
<tr>
<td>Kim et al. [33]</td>
<td>Proximal SVG to LAD</td>
<td>Spontaneous</td>
<td>Covered PTFE stent</td>
</tr>
<tr>
<td>Flecher et al. [39]</td>
<td>Mid SVG to LAD</td>
<td>SVG rupture</td>
<td>SVG ligated, PSA excised, redo CABG</td>
</tr>
<tr>
<td>Puri et al. [38]</td>
<td>Mid SVG to obtuse marginal of LCx</td>
<td>Spontaneous</td>
<td>Urgent thoracotomy (expired)</td>
</tr>
<tr>
<td>Davey et al. [40]</td>
<td>Mid SVG to RCA</td>
<td>Spontaneous</td>
<td>PSA excised and SVG ligated</td>
</tr>
<tr>
<td>El-Jack et al. [31]</td>
<td>Mid SVG to RCA</td>
<td>Spontaneous</td>
<td>PTFE stent</td>
</tr>
<tr>
<td>Le Breton et al. [22]</td>
<td>Distal SVG to LAD</td>
<td>Spontaneous</td>
<td>3 bare-metal stents</td>
</tr>
<tr>
<td>Hameed et al. [44]</td>
<td>Distal SVG to LCx</td>
<td>Spontaneous</td>
<td>PTFE stent</td>
</tr>
<tr>
<td>Rogers et al. [45]</td>
<td>SVG and radial graft</td>
<td>Spontaneous</td>
<td>CABG</td>
</tr>
<tr>
<td>Trauma induced PSA</td>
<td>Posterior descending artery of distal RCA</td>
<td>Blunt chest trauma</td>
<td>Aneureysmectomy</td>
</tr>
</tbody>
</table>

PSA, pseudoaneurysm; LAD, left anterior descending artery; RCA, right coronary artery; CABG, coronary artery bypass surgery; PTFE, polytetrafluoroethylene; PCI, percutaneous coronary intervention; SVG, saphenous vein graft; LCx, left circumflex artery.
extremely rare. Whereas true coronary artery aneurysms may regress spontaneously or demonstrate no significant mortality difference as noted by the CASS registry [17], coronary PSAs generally do not resolve. PSAs are potentially more malignant and thought to require either PCI or CABG with ligation or resection to prevent expansion with possible rupture and mortality. Therefore, it is vital to differentiate between true and false aneurysms to provide optimal patient care.

Diagnosis of Coronary PSA

Invasive coronary angiography most commonly detects a coronary PSA. It is visualized as an outward bulging of the coronary lumen. Figure 1 displays a suspected spontaneous coronary PSA discovered on coronary angiography for a patient who presented with an ST segment elevation myocardial infarction (STEMI). It is important to note that invasive coronary angiography only shows the lumen of the aneurysm and adjacent vessel, not the arterial wall, yet a PSA can be suspected based on the morphology of the dilated segment, especially the width of the neck of the aneurysm compared with the diameter. Specifically, the presence of a narrow neck which communicates with the true lumen is suggestive of a coronary PSA on angiography.

IVUS can be used to diagnose coronary PSAs or differentiate them from true coronary aneurysms [18]. Since IVUS demonstrates characteristics which suggest a coronary PSA including the loss of vessel wall integrity, adventitial and perivascular tissue damage, and an abrupt transition from a 3 layered wall to a monolayered outward bulging of the vessel wall, this may be a more accurate discriminator than angiography alone [5]. In cases of true coronary aneurysm or PSA larger than 10 mm, IVUS might have significant limitation in imaging the layers at such extreme distances from the catheter, which remains in the main vessel. No literature on optical coherence tomography (OCT) has been reported for diagnosing coronary PSAs. However, it may provide more detail than IVUS, although with less depth of penetration. Moreover, OCT might have even more difficulty imaging the vessel wall in larger sizes. It may help differentiate a true coronary aneurysm from a PSA when the wall can be seen. Furthermore, OCT has been shown to enhance the detection of spontaneous coronary artery dissection [19]. Therefore, it may prove useful to detect coronary PSA.

Cardiac computed tomography (CT) may also be used for diagnosis [14,20,21]. Figure 2 displays the cardiac CT of a coronary PSA of the proximal LCx in the same patient whose angiogram is shown in Fig. 1. Figure 3 portrays the coronary PSA visualized on a volume-rendered cardiac CT. A cardiac CT shows the wall of the PSA while a coronary angiogram shows the lumen only, without clear definition of the arterial wall. Rahman et al. [21] describes a case report of a patient who developed a spontaneous coronary dissection followed by PSA formation, which was detected by cardiac CT. The patient was 34 weeks pregnant and presented with an acute MI which was initially managed medically followed by subsequent CABG.
transesophageal echocardiography actually detected thrombus formation within the PSA cavity visualized as an echodensity in the left main coronary artery.

A coronary PSA may also present as a hilar or mediastinal mass visualized on routine chest x-ray, which may then lead to a definitive diagnostic test [22]. Thus, it should be considered in the differential diagnosis in the appropriate clinical setting. Discovering a coronary PSA prior to rupture may allow initiation of prompt lifesaving treatment.

Moreover, true coronary aneurysms or PSAs may also mimic cardiac masses (intracardiac and myocardial) which may not be accurately detected via echocardiography, trans-esophageal echocardiography, angiography, or cardiac magnetic resonance imaging [23]. If echocardiography or trans-esophageal echocardiography detects an abnormal echodensity in the coronary artery or a cardiac mass, this may provide a clue to the possible existence of a PSA. Subsequently, this should be investigated via coronary angiography or cardiac CT. Cardiac CT provides a non-invasive modality to follow a PSA in asymptomatic patients. In rare cases of large PSAs with thrombus formation mimicking a large mass, echocardiography or transesophageal echocardiography may allow clinical follow-up for a patient who is managed medically.

**Treatment Options**

Since coronary PSAs are a rare disorder, no standard treatment recommendations exist and guidelines for optimal management have not been established [1]. Medical management with spontaneous resolution or vigilant observation and clinical follow-up, covered stents, drug-eluting stents, bare-metal stents, coil embolization, or surgery involving CABG followed by resection or ligation of the PSA are possible treatment options [1,24]. Bare-metal or drug-eluting stents may diminish blood flow into the PSA allowing its exclusion. Neointimal proliferation within the stent struts may decrease blood flow into the PSA and activate thrombus formation allowing its eventual closure [25]. No randomized controlled or prospective trials have been conducted to evaluate the superiority of one treatment option compared with another. Surgical treatment may be the best alternative if coexisting coronary or valvular heart disease is present.

Occasionally, close observation with medical follow-up may be a suitable option if patients have no evidence of myocardial ischemia or are asymptomatic. Antianginal treatment with beta blockers, calcium channel blockers, or long acting nitrates may provide angina relief in patients with ischemic symptoms. Furthermore, coronary PSAs may also induce thrombosis or distal embolization for which antithrombotic or antiplatelet therapy may be beneficial. However, no standard medical regimen is recommended for coronary PSAs. Cardiac CT may also be utilized for PSA monitoring during clinical follow-up. Cabarrus et al. [26] reports a giant coronary PSA with daughter aneurysm formation after coronary perforation during angioplasty. The patient was initially diagnosed with a pericardial hematoma and managed conservatively. After 16 years, the patient developed worsening dyspnea, new left ventricular dysfunction, and an increase in the size of the pericardial mass. Cardiac magnetic resonance imaging and cardiac CT diagnosed the mass as a thrombosed coronary PSA with a daughter aneurysm. This was surgically evacuated followed by PSA ligation and CABG. Izutani et al. [18] reports a case of a spontaneous coronary PSA, which was treated by surgical resection followed by CABG. In cases of left main, proximal LAD, or proximal LCx PSA, it may be difficult to ligate such coronary territories secondary to inadequate visualization. Consequently, PCI may be a reasonable alternative in symptomatic patients. Therefore, a patient specific approach based on the size, location, and clinical symptoms should be considered for optimal treatment. Figure 4 provides a suggested treatment algorithm for spontaneous coronary PSAs.

**PCI of Coronary PSA**

A case report by Chabrot et al. [20] describes treatment of a post-partum coronary PSA with paclitaxel-eluting stents [20]. Dhakam et al. [14] reports a case of...
a spontaneous left main coronary artery dissection in a post-partum female (day eleven) who presented with an acute anterior STEMI. The coronary dissection was treated medically. However, it eventually progressed into a large left main coronary artery PSA at 1 month, which was successfully treated with a covered stent [14]. Afterwards, the patient was treated with aspirin, clopidogrel, and warfarin (INR target 2-3 for 3 months). Covered stents are composed of polytetraflouroethylene (PTFE) which can prevent plaque protrusion, thereby sealing an aneurysm or PSA [11]. PTFE can form a multilayer and expand up to 4-5 times its original diameter without laceration or shrinkage. Moreover, the negative charge of the PTFE prevents blood-protein coagulation on tissue surfaces, which mitigates platelet activation and thrombus formation [11]. Caruso et al. [27] describes a patient who developed a large RCA PSA several months after treatment of a non-STEMI with a bare

![Algorithm for Coronary Artery PSA Management](image-url)
metal stent to the RCA. The PSA was successfully treated with 2 PTFE covered stents. Dai et al. [28] recently reported a case of a giant RCA PSA, which was successfully treated with multiple overlapping covered stents.

A PSA which develops after PCI probably merits treatment since they have an associated risk of progressing and rupturing into the pericardium causing cardiac tamponade [27]. However, spontaneous resolution of post-PCI PSAs may also occur [28]. Nevertheless, the feared complication is rupture, which can lead to death from cardiac tamponade. Such a case was successfully treated with two expanded PTFE covered stents for PSA rupture with tamponade [29].

Coronary PSA of Saphenous Vein Grafts (SVGs)

Le Breton et al. [22] reports a PSA of a SVG to the RCA with fistula formation between the SVG and the right atrium, which was surgically treated. Schobel et al. [30] reports a case of a coronary PSA, which developed 2 weeks after perforation of the LAD during balloon angioplasty for an acute MI. The patient was successfully treated with CABG [30]. El-Jack et al. [31] reports a PSA of a SVG to the RCA, which occurred 13 years after CABG. The patient was successfully treated with 3 PTFE covered stents and 2 bare metal stents overlapping into the covered stents.

Microcoil Embolization of Coronary PSA

Microcoil embolization of a coronary PSA is another alternative [32]. Ducksoo et al. [33] reports a PSA of the SVG to the LAD (9 years post-CABG) which was successfully treated with transcatheter coil embolization using 6 mm Gianturco steel coils. Le Breton et al. [22] also reports a PSA of a SVG to the LAD which was successfully treated with percutaneous coil embolization. However, sparse literature on microcoil embolization is available to elaborate on this treatment option.

Iatrogenic PSA Post-PCI

Chen et al. [34] reports a coronary PSA formation at 1 month after paclitaxel-eluting stent implantation into the proximal LAD, which resolved spontaneously. It is an extremely rare phenomenon for PSA to occur after stent implantation [34]. Another case by Kapoor et al. [35] describes a coronary PSA of the LAD, which developed 8 weeks after an initial PCI for angina. The patient developed recurrent angina and was subsequently treated with CABG. Lim et al. [36] reported a case of an infected coronary PSA from everolimus drug-eluting stents after repeated PCI with bare-metal and drug-eluting stents. The patient was successfully treated with CABG.

Complications of Coronary PSA

Coronary PSA can cause thrombosis, ischemia, and vessel rupture leading to death. A SVG may rarely develop a PSA, which requires urgent surgical intervention [37,38]. Flecher et al. [39] reports a SVG PSA, which ruptured requiring emergent PCI with a PTFE-covered stent.

However, fatality may result if the diagnosis is not promptly recognized. Davey et al. [40] describes a rupture of a spontaneous PSA of a SVG leading to death during emergent surgery. Moreover, emergent surgery carries a much higher mortality risk during the postoperative period [41]. Therefore, PCI, coil embolization, or CABG, are all viable options for successful treatment of a ruptured PSA, but prompt recognition and treatment is crucial.

CONCLUSIONS

Coronary PSAs are extremely rare, but if present may cause mortality. Therefore, detection and urgent treatment may be essential to mitigate morbidity and mortality. No randomized controlled trials or retrospective studies have evaluated the superiority of either PCI, CABG, or medical therapy. Therefore, no consensus or guidelines exist for optimal treatment and management so it should be decided on a case-by-case basis. Vigilant detection and treatment with either PCI or CABG can be considered, if feasible, after a meticulous plan has been formulated with consideration of the individual anatomical and clinical circumstances.

ACKNOWLEDGMENTS

The authors thank William P. Fay, MD, for reviewing the manuscript and providing helpful suggestions.

REFERENCES


Catheterization and Cardiovascular Interventions DOI 10.1002/ccd. Published on behalf of The Society for Cardiovascular Angiography and Interventions (SCAI).