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Spontaneous Coronary Artery Dissection: Review of Current Management Strategies

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Abstract

Pregnancy-associated spontaneous coronary artery dissection (SCAD) often causes worse outcomes, larger infarctions, and lower ejection fractions (EF). We report the case of a 33-year-old post-partum patient with acute coronary syndrome (ACS) secondary to SCAD who required a temporary percutaneous left ventricular assist device (LVAD) and was placed on the heart transplant list. The LVAD was upgraded to a higher flow assist device, and goal-directed medical therapy and cardiopulmonary rehabilitation was started, resulting in recovery of left ventricular EF to >45% and removal from the transplant list. Similar treatment plans may allow hospitals without heart transplant capabilities better manage SCAD patients.

Keywords: Spontaneous coronary artery dissection (SCAD); Acute coronary syndrome (ACS); Percutaneous coronary intervention (PCI); Fibromuscular dysplasia; Obstetric anesthesia; Postpartum; Mechanical circulatory support; Cardiomyopathy

Introduction

Spontaneous coronary artery dissection (SCAD) is a non-traumatic, non-iatrogenic, and non-atherosclerotic condition. It is an important cause of acute coronary syndrome (ACS), myocardial infarction, and sudden death in people of all ages [1-6]. Using diagnostic criteria to rule out other causes of ACS such as trauma, atherosclerosis, iatrogenic injury; SCAD is responsible for 1-4% of ACS overall [1-5].

The distribution of which arteries are affected most often involves the left anterior descending artery (LAD) in 32-46% of SCAD cases [4,6-8]. In many cases, SCAD affects the mid to distal areas of the coronary arteries, with fewer than 10% involving the proximal LAD, circumflex, right coronary, or left main arteries [6]. In over 90% of cases, SCAD primarily presents with chest pain [9,10]. The cardiovascular exam is essential for early suspicion of SCAD. Physical exam may also be notable for elevated jugular venous pressure, pitting edema, pulmonary edema, delayed capillary refill, and decreased mentation. Serious complications like ventricular arrhythmia, cardiogenic shock, and sudden cardiac death can also occur [1,11,12].

We report the case of a 33-year-old female patient who was post-operative day (POD) 8 for repeat cesarean section who presented with ACS leading to further diagnosis of SCAD. The patient rapidly developed cardiogenic shock, had an Impella CP placed at the outside hospital, and was then transferred for emergent CABG. The CABG was aborted because all distal targets were dissected and unable to be anastomosed. The patient was placed on conservative guideline-directed medical therapy (GDMT) and cardiopulmonary rehabilitation with subsequent recovery of her overall heart function while awaiting heart transplant.

Case Presentation

Physical exam was unremarkable except for new onset bradycardia (44 bpm). An EKG was performed which displayed bradycardia (47 bpm) with anterior ST depressions concerning for NSTEMI (Figure 1). Mild hypokalemia, hyperchloremia, normal creatinine, BUN, BNP, and elevated troponin I 191.4 pg/mL (normal 0-45.2 pg/mL) were noted on initial lab values. CT angiography of the chest showed no evidence of pulmonary embolism or aortic dissection. Cardiomegaly without pulmonary effusions or infiltrates was additionally noted. A transthoracic echocardiogram (TTE) revealed normal EF of 63%. No regional wall motion abnormalities were present. At this time, doppler findings suggested mild pulmonary hypertension.

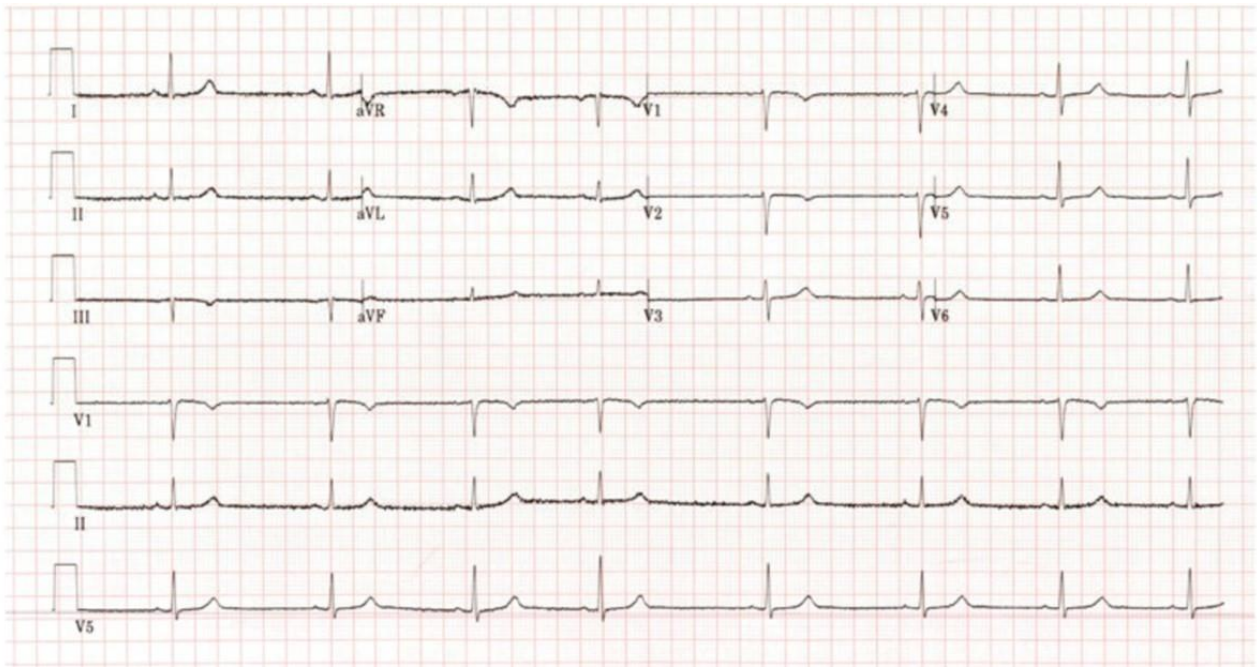

Figure 1: Sinus bradycardia with findings consistent with anterior NSTEMI noted in leads II, V1, V2.

Troponins continued to trend up, peaking at 700.7 pg/mL, and aspirin was administered due to a concern for type I NSTEMI vs. SCAD, given the postpartum timeline. A left heart catheterization and coronary angiogram (Figure 2) without percutaneous coronary intervention (PCI) were performed. Anticoagulation was started using a heparin drip. During these diagnostic procedures, a right dominant coronary artery system was appreciated with a notable spontaneous coronary artery spiral dissection, as demonstrated by multiple radiolucent lumens or arterial wall contrast staining which extended from the left main to the left anterior descending (LAD) and left circumflex artery. This pattern signified type I SCAD. Of note, LVEDP was found to be elevated at 18 mmHg.

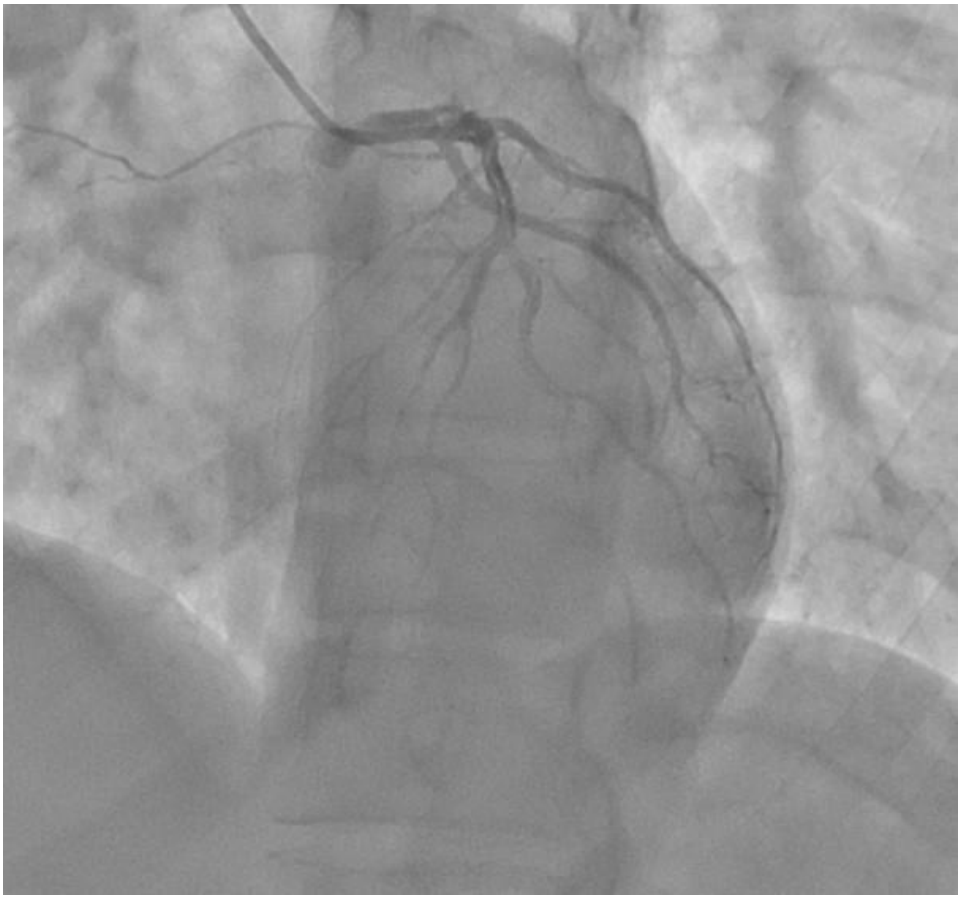

Figure 2: Coronary angiography in LAO Cranial projection, demonstrating coronary dissection extending from the left main into the proximal LAD and diagonal artery.

Following coronary angiogram, the patient subsequently developed severe chest pain and became hemodynamically unstable. Nitroglycerin was started without remission of symptoms. Blood pressure dropped to 88/58 mmHg, and the patient became somnolent, concerning for cardiogenic shock. The patient was intubated and placed on mechanical ventilation, and emergent mechanical circulation was achieved via intra-aortic balloon bump that was upgraded to an Impella CP after monitors continued to show significant ST elevations. The patient was emergently transferred to Hospital 2, for immediate cardiothoracic surgery evaluation and coronary artery bypass grafting (CABG).

The patient arrived at Hospital 2 with continued bradycardia. Upon initial evaluation, all extremities were warm to touch, trace lower extremity edema, and a capillary refill <3 seconds on physical exam signifying adequate tissue perfusion. Upon arrival into the operating room (OR), adequate vascular access for hemodynamic monitoring and volume resuscitation was achieved. The patient underwent intraoperative TEE which indicated hypokinesis of the basal anterior wall and akinesis of mid papillary and apical anterior and lateral walls of the left heart. The patient's EF was 15-20%. The right ventricle was mildly enlarged with a right ventricular systolic pressure (RVSP) of 54 mmHg. Central venous pressure was noted to be 28 mmHg. As cardiothoracic surgery commenced with sternotomy and left saphenous vein harvest, it was determined that the entire distribution of left coronary arteries was completely dissected down to the cardiac apex with partial thrombosis intermittently throughout.

An obtuse marginal graft was attempted, however, due to the severe dissection and thrombosis, there was no viable vessel portion to graft upon. The graft was not attempted on the LAD or diagonal do to similar friable tissue and lack of adequate targets. The patient continued having significant arrhythmias despite cross-clamp removal. The OSH-placed Impella CP was then upgraded to an Impella 5.5 with modest inotropic support in the form of milrinone and norepinephrine infusions. A request was sent for transfer to Hospital 3 for cardiac transplant admission. Meanwhile, the sternotomy wound was closed. Cardiopulmonary bypass (CPB) time was 111 minutes. Within 30 minutes of arrival to the cardiac intensive care unit (CICU) at Hospital 2, the care team was notified that Hospital 3 accepted the transfer of care request.

Following the aborted CABG procedure, the patient was transferred to Hospital 3 for urgent cardiac transplant due to severe heart failure secondary to SCAD. The patient was slowly titrated off inotropic support and weaned off Impella dependence by POD 20 from initial presentation. GDMT was initiated which included aspirin, metoprolol, Farxiga, Entresto, spironolactone, and furosemide. TTE performed on POD 15 showed an EF of 45% and by POD 30, TTE demonstrated similar EF with improving apical kinesis, global LV myocardial longitudinal function, and strain pattern. The patient was weaned off Impella 5.5 after approximately three weeks. With marked improvements in overall cardiac function, Hospital 3 declined investigation into potential genetic causes and the patient was subsequently discharged home with scheduled cardiology follow up and enrollment into a cardiopulmonary rehabilitation course.

The following three-month course was uneventful. During follow up cardiology visits, the patient stated dizziness and lightheadedness were improving. The patient experienced mild dyspnea when going upstairs, in addition to mild bilateral lower extremity swelling. Despite this, the patient stated they were able to perform activities of daily living without issue. They denied further chest pain episodes.

Further imaging using computed tomography angiography (CTA) of the head, chest, abdomen, and pelvis was performed 3 months after hospital discharge to workup for fibromuscular dysplasia. A 1 cm pseudoaneurysm of the ascending aorta was noted as well as focal dissection of the left lateral wall of the aortic arch. Nearly 5 months post SCAD diagnosis, the patient presented back to an OSH ER for concerns of headache with visual aura and associated diaphoresis and nausea. On physical exam, a left carotid bruit was noted. ESR and CRP were within normal limits. CTA of the head showed beaded appearance of bilateral internal carotid arteries (ICAs) with near occlusion of the right ICA due to dissection at C1-C2 and a similar appearance of the vertebral artery (VA) with 90% diameter reduction of the left vertebral at V3 level. A follow up MRI/MRA confirmed these findings. The patient was then transferred to Hospital 2 for neurology direct admission.

On hospital stay day 2, neurointerventional radiology performed a DSA with endovascular stenting of the right ICA dissection. Evidence of fibromuscular dysplasia was noted in bilateral ICAs, and possibly in the left vertebral artery at the V3 segment, with no significant stenosis. Apparent type I fibromuscular dysplasia of the middle third of the cervical segment of the left ICA without any significant flow compromise was also noted. There were extensive traumatic injury and dissection of the cervical segment of the right internal carotid artery (Biffl Grade II), with prominent downstream flow reduction, successfully treated by telescopic reconstructive stenting. At this time, an updated TTE was performed with a noted EF of 50-55%. The remainder of the patient's hospital stay was uneventful, and the patient was discharged on post-admission day 3 with prescriptions for atorvastatin and clopidogrel. Metoprolol was stopped due to continued dizziness and lower end of normal blood pressure prior to discharge.

Two weeks post-discharge, the patient began experiencing mild bilateral neck pain (3/10 on pain scale) aggravated by laying down or with leg movements. However, the patient waited until the fourth day of continued symptoms before presenting to her PCP, who recommended returning to the local OSH ER. Repeat imaging was notable for spontaneous dissection of right common carotid artery with flow limitation, patent stent in right ICA cervical segment, and fibromuscular dysplasia of left internal carotid artery cervical segment. The patient was again transferred to Hospital 2 as a neurology direct admit. CTA of the head was again performed for concern of stenosis of proximal right common carotid artery (CCA). The previous right ICA stents were noted to be patent. The dissection of the proximal right CCA was noted to have a procedural stretch injury with pseudoaneurysm (Biffl Grade III). Stenting of the right CCA was performed with resolution of the stenosis. Type I fibromuscular dysplasia of the cervical segment of the left ICA was also prominent. The patient was discharged home the following day. Follow up imaging has since demonstrated stabilized patent vessels. The patient is currently doing well and being closely monitored by follow up with outpatient cardiology, neurology, and rheumatology for her diagnosis of SCAD likely secondary to fibromuscular dysplasia.

Discussion

The typical SCAD patient is a young, otherwise healthy, white female in her 40-50s with no previous significant atherosclerotic or cardiovascular risk. Other case reports have described SCAD occurring in a woman from their thirties to fifties who present with retrosternal chest pain and describe acute coronary syndrome like symptoms [13-16]. In these reports, as with our case of SCAD, the patient's initial physical exam did not demonstrate initially significant clinical findings. EKG findings often found ST elevation and increased troponin levels [13-16]. The initial LV EF has been reported to often be around 50% [17]. Following other literature, most cases are diagnosed utilizing coronary angiography [13-16]. Cases that involved the mid to distal vessels are managed conservatively with favorable outcomes using only medications [14-16]. When more proximal vessels are involved or the patient becomes hemodynamically unstable, case reports describe using a more invasive approach involving stenting or CABG surgery [13,16].

SCAD was previously thought to be largely idiopathic, however, with increased screening and recognition of risk factors that number has been decreasing [5,6]. The cardiac injuries due to SCAD are caused by the formation of intramural hematoma or intraluminal thrombus leading to epicardial coronary artery obstruction [9]. One mechanism thought to be the possible pathophysiology behind SCAD is intimal dissection caused by a tear in the intimal layer of the artery causing blood to enter the intimal space from the endoluminal space, thus forming a false lumen [5]. Another theoretical mechanism is intramural hematoma which is caused by a hemorrhage of the vasa vasorum that supplies the walls of the arteries. After rupture, blood can build up in the intramural space [5]. The false lumen goes on to extend and compress the true lumen leading to myocardial ischemia. There is discussion on whether the intimal tear in the first mechanism is caused secondary to increasing pressure in the false lumen leading to rupture as a tear is not always identifiable [9].

Advances have been made in recognizing and diagnosing SCAD since it was first described in 1931. Case reports have proven the high mortality diagnosis of SCAD much more common, although still rare, condition than previously thought [9]. However, it is important to note that this condition is often underdiagnosed or misdiagnosed, leading to patients potentially being poorly managed as if to suffer from atherosclerotic ACS [9]. Due to lack of clinician experience with the condition and coronary angiographic imaging techniques available, it can be a missed diagnosis, even in the presence of typical symptoms and patient presentation [9]. Recognizing and accurately diagnosing SCAD is crucial for optimal patient health outcomes.

A timely diagnosis is critical when allocation of resources is needed to provide supportive care, as well as determining accommodations required for invasive procedures and transfer partnerships for higher levels of care [9].

SCAD is an important diagnosis with a complex and unclear clinical course. Coronary angiography is widely accepted as the gold standard initial diagnostic method and should be performed as soon as SCAD is suspected due to it being readily available at most hospitals and its preference in early management of ACS [5,9,18]. Per literature review, the mindset of SCAD treatment has shifted towards conservative modalities. The in-hospital mortality rates of SCAD are low, however, up to 14% of cases treated conservatively require subsequent revascularization via PCI or CABG [6,8,9,19]. PCI may include wiring only, balloon angioplasty, or stent placement.

PCI and other less conservative management strategies have shown higher rates (around 50%) of technical failure in patients with SCAD compared to atherosclerotic ACS [6,17]. There are associated poor success rates, extension of dissections, iatrogenic dissections, and need for emergency CABG after unsuccessful PCI when treating SCAD [8,17,20]. Revascularization also has not been shown to be protective against future dissection as the dissections often occur in new locations [17,19]. When reviewing previously reported SCAD cases, PCI has been more often used when patients presented with STEMI, higher rates of vessel occlusion, and larger diameter vessels [17]. However, most SCAD cases have a favorable outcome when managed conservatively, and late vessel healing often occurs in the following weeks to months [5,6,8,9,17,19,20]. On medical therapy, patients are sent home with a regimen typically consisting of a beta blocker, aspirin, and an ADP antagonist/clopidogrel [5,8,19].

Antiplatelets and anticoagulants are typically started per ACS management guidelines when a patient with SCAD comes in with chest pain. However, there is recommendation to stop anticoagulation after SCAD is diagnosed with no other indication for anticoagulation due to the potential to worsen the intramural hematoma [5,9]. This is still an evolving area for research and discussion as early anticoagulation may reduce thrombus burden [5,9]. Long term aspirin is supported in SCAD for at least 1 year, possibly indefinitely [5,6,9]. Patients who undergo PCI with stenting should be on dual antiplatelets for one year, but the role for antiplatelet use for those who do not undergo stenting is not well established. Beta blockers are supported and should always be prescribed due to lowering the risk of recurrent SCAD by reducing arterial wall stress on coronary arteries comparable to their benefit in aortic dissection [6,21]. ACEi and ARBs have previously been reserved for MI that is complicated by LV systolic dysfunction [18]. There is not a clear role for statins, since SCAD, by definition, is not due to atherosclerosis or plaque rupture. However, after presentation, these medications are often used, in keeping with current guidelines, to prevent further atherosclerotic disease progression [9]. Nitrates, calcium channel blockers, and ranolazine have also previously been used to help avoid recurrent hospitalizations for chest pain after SCAD that may be due to coronary vasospasm, microvascular diseases, or non-cardiac in origin [10]. Cardiac rehabilitation is a holistic area of SCAD management that should be prescribed, involving patient education on the importance of aerobic exercise and avoiding rapid return to isometric exercise, high-intensity exercise, or prolonged Valsalva maneuvers. This form of rehab offers patients a role in their own care, making them active participants on their journey to healing and allows closer monitoring for signs of psychiatric diseases, such as depression, which are common after a life-altering diagnosis.

There are no clear guidelines on when invasive management should be pursued in SCAD. Historically, it has been left up to the clinical team to decide based on ongoing ischemia, left main artery dissection, or hemodynamic instability whether to utilize urgent intervention of PCI or CABG procedures. In our case the patient's multivessel and proximal vessel involvement and rapid decline into cardiogenic shock led to the attempted CABG procedure, abortion of CABG and transferal to a heart transplant capable facility. While being treated conservatively while awaiting a viable organ for transplant, the patient's ejection fraction increased and symptoms subsided, allowing her to return home without further invasive intervention.

Our interventions followed commonly practiced management for SCAD. The patient was rapidly transferred to our facility from outside hospital once angiography recognized the dissection and the patient became hemodynamically stable. At our hospital, it was determined to need an invasive intervention due to her decompensation and available literature on SCAD once the dissection leads to instability. However, due to the patient's severe dissection and extensive involvement of multiple arteries, the aborted CABG procedure resulted in utilizing an additional ventricular assist device to support the patient. This is a different approach than most other SCAD cases have previously taken. Cardiogenic shock occurs in only 2-5% of all cases of SCAD, but in 25% of pregnancy associated SCAD cases [9]. There is currently limited literature on cardiogenic shock during SCAD and the uses of mechanical circulatory support [22]. There have been cases of pregnancy associated SCAD that were managed via intra-aortic balloon pump or Impella. There is potential to use these devices to help bridge the patients to CABG, heart transplant, or recovery [18,22]. The assist device alongside supportive medical therapy utilized in our case allowed time for the previously described healing of the coronary vessels without invasive repair. This showcases the use of ventricular assist devices and medical therapy in a case that has previously been more likely to be treated with stenting, CABG, or heart transplant. The favorable outcome our patient had in the presence of a proximal and multivessel coronary artery dissection offers a unique patient clinical and management course.

Challenges and limitations that were encountered during diagnosis of this patient were in relation to hospital logistics. This patient initially presented to a small local hospital where they were diagnosed via coronary angiogram. However, after the extensive involvement of the vessels and the development of cardiogenic shock, the patient had to be transported with a life-threatening dissection to our hospital. This brings to light an issue that smaller hospitals may face when treating SCAD due to limited access to cardiothoracic surgeons or onsite anesthesiologists, perfusionists, and support staff. This was a further problem later in the patient's clinical course when the patient's status necessitated a heart transplant. The patient underwent an additional procedure for Impella placement to bridge her through transport to the third hospital for heart transplant. Other challenges that may be faced may include timing from symptomatic onset to diagnosis, severity of the dissection and provider experience. Additionally, patient demographics, comorbidities, and family history can lead to different initial differential diagnoses and treatment offered.

SCAD is an overall uncommon disease of primarily young women. The variable presentations and level of dissections complicate its diagnosis and treatment further. This case allowed us to learn more about conservative management in synchronous use with a ventricular support device during pregnancy associated SCAD. Our case demonstrates the importance of timely diagnosis and power of conservative management alongside mechanical support devices in severe dissections while awaiting definitive treatment. In our case, conservative medical management utilizing GDMT and an Impella device allowed vessel healing following proximal and multivessel involvement.

Expectations based on our experience were that the patient would require invasive revascularization via CABG procedure. When that was not a viable option, heart transplant was the expected management strategy. Future studies may be possible to investigate further if conservative management with simultaneous use of mechanical support devices could be useful in more severe cases of SCAD than previously discussed. Further improvements in this area of SCAD management may allow hospitals like ours, who do not have heart transplant capabilities or previously would have had to transfer patients out, to continue their care and allow time for previously described innate vessel healing.

Acknowledgements

Informed Consent Declaration

All procedures were performed in compliance with relevant laws and institutional guidelines, and this case report has been approved by the University of Missouri-Columbia Institutional Review Board. The privacy rights of human subjects have been observed and explicit informed consent from the patient was obtained prior to data collection.

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Alexis DeTienne: Conceptualization, Investigation, Writing – Original Draft.

Brooke Scieszinski: Conceptualization, Writing – Review and Editing, Supervision.

Jared Olson: Writing - Review and Editing, Visualization.

Paige Spencer: Writing - Review and Editing.

Bradford Cardonell: Writing – Review and Editing, Supervision, Project Administration.

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