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Pregnancy Provoked Quiescent and Dormant Vascular Revamping: Pathological Inclination for Sparking Off Postpartum Spontaneous Coronary Artery Dissection

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Abstract

Introduction*: Postpartum SCAD is a rare form of acute coronary syndrome presenting in women of childbearing age. It is typically characterized by vessel occlusion and flow limitation in the coronary arteries, secondary to intimal tears, blood accumulation, and hematoma formation. It occurs in the absence of atherosclerotic vascular disease or trauma. Pregnancy-related hormonal changes, vascular remodeling, vasculitis, and inflammation are the causative factors of SCAD. Coronary artery dissection leads to myocardial infarction and cardiogenic shock.*

Clinical Case*: We present a clinical case where a 33y/o F who is s/p cesarean-section G4P4 presented 2 weeks after postpartum with chest pain, dyspnea, and elevated blood pressure. Cardiac workup revealed NSTEMI. Subsequently, she was referred to the cardiology service and planned for a coronary angiogram with percutaneous intervention.*

Treatment*: She underwent a left heart catheterization, along with an intravascularultrasound, which revealed spontaneous LAD dissection. Consequently, drug-eluting stents were considered and implemented. She was later discharged with beta-blockers, statin and dual antiplatelet therapy and advised close follow-up with cardiology.*

Take-home message*: Postpartum SCAD presents with signs and symptoms of acutecoronary syndrome in women within 6 weeks of delivery. Often, it is misdiagnosed in young women with no cardiac risk factors, who would be likely candidates. This would result inthe disease process advancing to a more clinically severe stage by the time of diagnosis, after which clinical interventions become futile. Without early diagnosis and treatment, there is a higher probability of morbidity and mortality. A high degree of clinical suspicion and prompt attention to these cases will be lifesaving, while simultaneously decreasing mortality & morbidity and improving survival rates. Unfortunately, even with aggressive interventions, the risk of complications and disease progression occurs. Keeping in mind the increased recurrent rate in susceptible women, counselling and education of these women to avoid further pregnancies should be considered.*

Keywords: *Postpartum, acute coronary syndrome, chest pain, PCI, CABG, heart transplantation.*

Introduction

Postpartum SCAD is the narrowing of vessel diameter of coronary arteries secondary to intimal rupture, development of false lumen, blood accumulation and hematoma formation, all of which synergistically are instrumental in sparking off sudden myocardial infarction. Importantly, the relevant risk factors including coronary atherosclerosis, aortic dissection, drug use, hypertension or fibromuscular dysplasia are excluded from risk factors[1]. It is initially recognized by Pretty et al in 1931, when a young Caucasian women presented with chest pain and sudden cardiac arrest following pregnancy[2]. The typical clinical profile of postpartum SCAD is multiparous women of 33 years presenting with chest pain or shortness of breath within 25 days of pregnancy[3]. The highest risk of postpartum SCAD unfolds within first 2 weeks after pregnancy[4].

Clinical Case

33y/o F who is 14 days s/p cesarean-section G4P4, with no significant past medical history, presented to ED with chest pain for 1 day. Chest pain is retrosternal and radiates to her back. It is constant and associated with dyspnea. Chest pain and dyspnea are worse with exertion. She denies family hx of cardiovascular disease. She denies tobacco, EtOH, or illicit drug use. The patient presented to the hospital 2 weeks postpartum with chest pain, dyspnea, and elevated blood pressure. Patient was found to have a NSTEMI and LAD dissection (Figure 1&2). Patient had a left heart catheterization with successful revascularization of mid LAD. The patient was treated with Dual anti-platelet therapy, a beta-blocker, and a statin. The patient was diagnosed with ischemic cardiomyopathy secondary to LAD lesion

secondary to SCAD. Patient underwent LHC, which revealed a significant mid-LAD lesion likely secondary to spontaneous coronary artery dissection. The lesion was successfully wired, followed by revascularization with a drug-eluting stent, with excellent TIMI-3 flow and 0% residual stenosis.

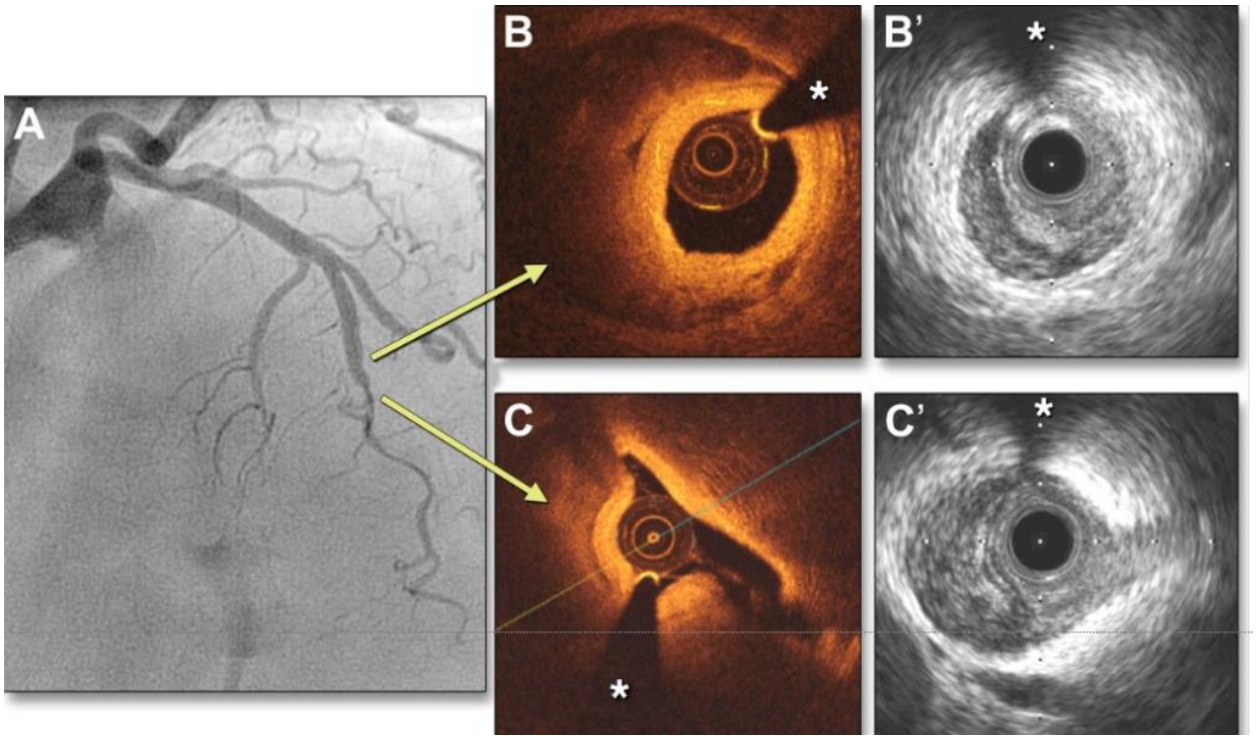

Figure-1: Intravascular ultrasound of LAD.

IVU is commonly used to supplement left heart catheterization. It showing false lumen, accumulation of blood in false lumen, intravascular hematoma and narrowing vessel lumen.

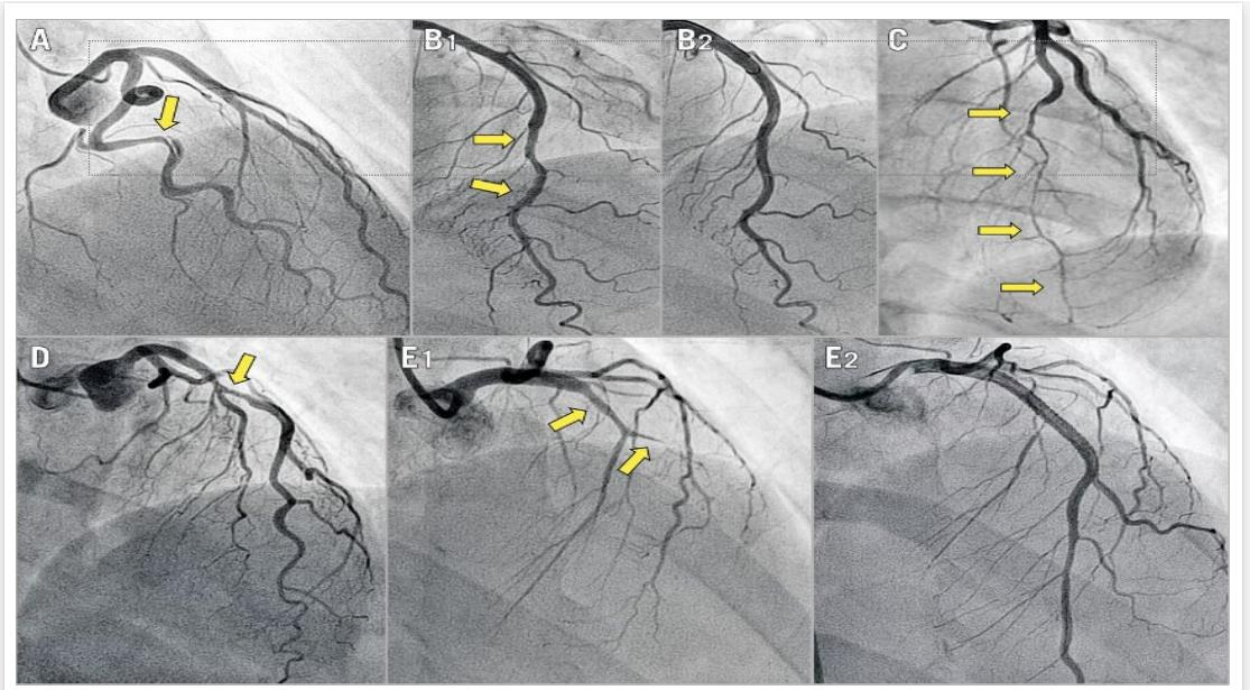

Figure-2: Left heart catheterization of LAD showing coronary dissection.

Discussion

We present a clinical where a 33y/o F who is 14 days s/p c-section G4P4 presented 2 weeks after postpartum with chest pain, tachycardia and hypertension, which upon evaluation revealed NSTEMI secondary to spontaneous LAD dissection. Subsequently, she underwent a left heart catheterization, revascularization and drug eluding stents was placed. She was later discharged with beta-blockers, statin and dual antiplatelet therapy.

The incidence and prevalence of postpartum SCAD are reported to be 0.5-1.5% and 0.7-2.5% [5-7]. The percentage of cases that can be reported during late pregnancy, peripartum, and

postpartum period are estimated to be around 26-38%[8]. Reports indicate that postpartum SCAD can be encountered as rare as one case per 20,000-30,000 deliveries[9]. For every 100,000 pregnant women in United States, 1.8% of them are more prone to develop SCAD[10]. Out of 20,000-30,000 deliveries, one women is likely to be effected by postpartum SCAD[11].

Fibromuscular dysplasia, family history of ischemic heart disease, hypertension, hypercholesterolemia, smoking, advanced age and multiparity are common risk factors that increased the cardiovascular risk for giving rise to postpartum SCAD [8, 12]. However, more importance is given to gestational hypertension, gestational diabetes, preeclampsia, oral contraceptives, and previous fertility treatment in postpartum SCAD[13]. Antiphospholipid syndrome, bromocriptine, immunosuppressive drugs, type IV Ehler Danlos syndrome, Marfan Syndrome, and cocaine use are some other risk factors that can also grease the wheels for transpiration of post-partum SCAD [8, 14-23]. Furthermore, emotional/physical stress, in-vitro fertilization, fertility treatments and menses related chest pain are speculated be possible risk factors that increase the proclivity for developing postpartum SCAD[24].

The onset of Postpartum-SCAD can occur within third semester pregnancy and usually within the time-frame of 6 months after pregnancy[25]. An important hallmark of postpartum SCAD is embroilment of physiologically normal coronary arteries as compared to atherosclerotic coronary dissection that supervenes during myocardial infarction[9]. Reports point to fact that there is innate proclivity towards engagement of left main left main descending (64%), and right coronary arteries (23%) in this clinical disorder[9, 26].

Sudden cardiac death may be the initial clinical presentation in 46-80% of cases and short term-term mortality can be as high as 28% [27, 28]. The most common presenting symptoms of women with postpartum SCAD includes chest pain, back pain, hypertension, fatigue, headache, dizziness, sweating, vomiting and shortness of breath [10, 29]. Mostly, due to normal vital signs and ECG findings, it is not uncommon for misdiagnosis as well as delayed diagnosis as younger women are less likely to have cardiovascular risk factors [10, 30, 31].The amount of troponin elevation in SCAD is dependent of extent of cardiac muscle injury, albeit extensive troponin spiking in postpartum SCAD is contingent upon involvement of proximal lesions, multi-vessel disease and presence of hemodynamic instability[10, 24]. EKG changes including ST elevation (46%), ST depression (6%) and T-wave inversion (22%) are commonly seen[29]. Although most cases of postpartum-SCAD are managed conservatively, some cases progress rapidly to culminate in sudden death due to severe cardiac arrhythmias[25].

Researchers have speculated a few hypotheses for crystallization of SCAD in postpartum women. The materialization of SCAD in the postpartum period can be a culmination of various events acting together or separately. Hemodynamic changes including increased cardiac output and plasma volume during pregnancy can put forward a structural strain on the layers of coronary vasculature, hence making them increasingly prone to intimal rupture[32].

Hormonal changes during pregnancy including increasing estrogen, relaxin, progesterone and prostaglandins might provoke drastic changes in the cardiovascular system not limited to cardiac output, vascular smooth muscles, systemic vascular resistance and pulmonary vascular resistance [33, 34]. Estrogen has been shown to induce the synthesis of various mediators such as MMP-2&MMP-9 in the coronary circulation, thence amplifying the risk of vascular remodeling. Progesterone incited changes including disintegration of elastic fibers and derailment of collagen architecture potentiates the friability in the layers of the vascular wall, thus impelling the risk for flourishment of SCAD[8]. On top of these, loss of acid mucopolysaccharides, cystic medial necrosis, media degeneration, periadvential inflammation, fibrocystic dysplasia and mucinous microcystic changes in the media are also sometimes implicated for inciting coronary dissection [16, 35, 36]. This aforementioned vascular remodeling can alter the framework of vascular layers, hence enhancing the risk for intimal layer rupture, a phenomenon referred to as inside-out hypothesis [1, 23, 37].

Alternatively, blood vessels supplying the coronary arteries namely vasa-vasorum are prone to rupture and unleash their luminal blood contents into the tunica media, an eventuation referred to as outside-in hypothesis [23, 38, 39]. Both these aforementioned turn of events converge to spark off generation of false lumen and enkindle the amassment of blood within the tunica media[40]. These turns of events will divert the blood from the true lumen of the coronary arteries, thus giving rise to myocardial ischemia.

Some speculated an autoimmune component to pathogenesis of this clinical disorder. In autoimmune diseases, one can contemplate that unfolding of vasculitis with associated stockpiling of inflammatory infiltrate such as histiocytes and eosinophils adjoining the microvasculature supplying the coronary arteries is a common state of affairs[9]. These histiocytes and eosinophils exude pro-inflammatory cytokines including collagenase, and myelin basic protein which are formidable for bringing about crumbling of collagen and elastic fibers of the coronary arteries[4, 9, 16].

It is easy to wrap one's mind around the fact these destabilized and shriveled coronary arteries are easily amenable for coronary artery dissection. Given the friability of the arterial walls, a guarded and cautious approach seems to be the priority during coronary angiography for avoiding the progression of dissection. Specifically, focus should be directed towards avoiding deep intubation, utilization of less toxic & non-selective contrast agents, as well as emphasis on pressure tracing[3]. It is not unconventional to rely on intravascular ultrasound to further delineate and demarcate the intimal tears, hematoma and degree of vessel narrowing[3].

In majority of the cases (80%), postpartum SCAD can be managed conservatively with medical management[32]. ACS optimized therapy including dual antiplatelet therapy, heparin and beta-blockers is usually reserved for conservative management of uncomplicated cases [41].[42]

However, in the event of cardiogenic shock, ventricular arrhythmias, or ongoing ischemia, left main vessel disease, extensive proximal multi-vessel disease and hemodynamic instability, it would be prudent to move forward with percutaneous coronary intervention (PCI) [43]. PCI in these patients is not always successful as long-term vessel patency is attained in only meagre 30% cases [44, 45]. It was reported that in a few cases, there are instances where dissection has progressed

after the PCI because it is possible that guidewire catheter has mistakenly entered the false lumen, thereby proving an impetus for the progression of hematoma[3]. Reports indicate that the technical failure rate of PCI as high as 35% due there is diversion of blood through the false lumen and stent induced extension of the hematoma [45, 46].

To circumvent this predicament, cardiologists should confirm as well as differentiate between false and true lumen within coronary arteries with the help of intravascular ultrasound[3]. In addition, they have used drug eluding stents proximal and distal to the dissection [44]. Even with PCI, some patients fail to recuperate their baseline cardiac function, thus presenting with recurrent congestive cardiac failure. This calls for enforcement of guideline directed medical therapy (GDMT) for bolstering the cardiac pump function including beta-blockers, mineralocorticoid receptor inhibitors (MRA), sodium-glucose cotransport inhibitors, ACEI/ARB/angiotensin-nephrolysin inhibitors[41, 47].These should supplemented with cardiac rehabilitation and stress avoidance[29].

It is not uncommon to encounter progression of coronary dissection despite performance of PCI. In such cases, coronary artery by-pass graft (CABG) should be warranted in multi-vessel SCAD or failure of PCI[32]. Some of the challenges encountered with CABG can range from finding patent grafted vessels and securing and demarcating patent non-dissected coronary vessel areas for graft fixation[3]. If CABG is unsuccessful or hemodynamic compromise still persists, as a last resort intra-aortic ballon pump, extracorporeal membrane oxygen (ECMO), left ventricular assist device or heart transplantation can be considered in case by case basis[48]. In a Canadian study, among the patients presenting with postpartum SCAD, majority of cases were managed with conservative therapy, while small percentage required PCI (2%) and CABG (0.4%)[49].

As compared to non-postpartum SCAD, postpartum SCAD is linked to high troponins, weaker LV pump function, severe congestive heart failure and extensive cardiogenic shock, thus setting in motion higher mortality rate [3, 50, 51]

Following first episode, a second recurrence can be foreseeable event within first 30-60 days in 17-50% cases [52]. With that being said, postpartum women who has first episode of SCAD are more likely to have increased propensity to develop after second pregnancy. This warrants prompt screening for pertinent risk factors and appropriate counselling to avoid future pregnancies would be prudent, given the complications, and increased mortality rate associated with it[25].

Patients who experienced postpartum SCAD are more liable to have an extra-coronary fibromuscular dysplasia and major cardiovascular event [49]. In multi-centered observational study, complications ensuing after 3-yr median follow up after postpartum SCAD include extension of previous SCAD (3.5%), de-novo recurrent SCAD (2.4%), iatrogenic SCAD (1.9%) and major adverse cardiovascular event (14%)[49]. Depending upon the severity of the disease, mortality can be as high as 50% and with those presenting with myocardial infarction it is projected to be as high as 70%[11]. Albeit, patients who would persevere through the initial clinical course are prone to have modest survival and prognosis[11].

Conclusion

Postpartum SCAD presents with myocardial infarction following pregnancy. It can occur early [6 weeks], late [6 weeks-12 months] or very late postpartum [12-24 months]. Most common clinical presentation usually befalls within 6 weeks of delivery. By definition, SCAD is liable to unfold in physiologically normal coronary arteries with no evidence of atherosclerosis. Thereupon, in the pregnant women with relevant risk factors, intimal rupture and rupture of vaso-vasorum are the primordial events that are usually followed by fabrication of false lumen, and inception of intramural hematoma within the coronary vessels. Over and above this, ancillary histological changes ranging from peri-advential inflammation, medial degeneration, cystis medial necrosis and fibrimuscular dysplasia are not unusual. These aforementioned events become the springboard for narrowing of the coronary vessel lumen, thus ultimately crystalizing as ischemic cardiomyopathy. Consequently, postpartum women present with chest pain, shortness of breath and palpitations. Given the severity and disease progression, these women warrant prompt evaluation with coronary angiography, and intracoronary ultrasound. Precise localization of site and extent of coronary dissection should be the prerequisite before moving forward with management strategies. Most of the patients the can be adequately managed with conservative therapy. In rest of them, the therapeutic options can range from PCI, CABG, intra-aortic ballon pump, LV assist devices to heart transplantation, which needs to tailored according to clinical scenario. Postpartum women with first episode of SCAD needs appropriate counselling to avoid future pregnancies due to increased recurrence and mortality associated with second SCAD episodes.

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