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Menstrual Chest Pain in Women With History of Spontaneous Coronary Artery Dissection

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Spontaneous coronary artery dissection (SCAD) is an important etiology of myocardial infarction (MI) and sudden cardiac death in young women (1). Previously considered rare, substantial improvement in SCAD diagnosis and awareness has facilitated research of this disease. Many women with a history of SCAD have recurrent chest pain despite lack of demonstrable ischemia or coronary obstruction. Herein, we report the hypothesis-generating finding of women who experience chest pain that predictably occurs or worsens synchronous with menstruation. The objective was to describe the observation and treatment of cyclic chest pain associated with menstruation (catamenial chest pain) in SCAD.

The Mayo Clinic Institutional Review Board approved the study. We reviewed the records of patients in the Mayo Clinic SCAD Registry enrolled from 2012 to 2016 and identified those who voluntarily described chest pain associated with menstruation. These patients were contacted by telephone and participated in a focused survey.

A total of 11 women were identified (Table 1). Four had experienced post-partum SCAD, 2 had fibromuscular dysplasia, and 1 had Ehlers-Danlos syndrome type IV. Most (7 of 11, 64%) presented with ST-segment elevation MI at time of SCAD; 1 presented in cardiac arrest. The majority (7 of 11, 64%) had left anterior descending coronary artery SCAD.

All women described onset of chest pain just prior to menstruation, usually 1 to 2 days before onset of bleeding. Symptoms persisted into menses in 4 (36%). Two women (18%) had coronary artery obstruction on coronary angiography (CA) (1 with persistent dissection and another with instent stenosis); these patients' catamenial chest pain persisted despite revascularization and negative subsequent stress studies. Six women (55%) had negative stress tests, 2 of whom had patent coronaries upon imaging. Of these, patient #5 had chronic troponin I elevations in the absence of electrocardiogram changes or left ventricular wall motion abnormalities. Two (18%) underwent CA without a preceding stress test with the finding of patent coronaries; another woman (9%) had an unremarkable coronary computed tomography angiography.

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Most were treated with short acting nitrates. Another strategy included starting or up-titrating long-acting nitrate therapy just prior to menstruation and down-titrating the dose after symptom remittance. Other therapies included ranolazine, calcium-channel blockers, beta-blockers, and endometrial ablation.

To our knowledge, this is the first report of catamenial chest pain after SCAD. Chest pain most often occurred 1 to 2 days prior to menstruation, the time when ovarian hormones are lowest in the menstrual cycle. Similar observations have been noted in the context of other diseases. For example, Kawano et al. (2) studied 10 women with coronary vasospasm and found that most ischemia occurred in the days immediately preceding and at the beginning of menstruation, at which time these patients were found to have low estradiol levels and less flow-mediated brachial artery vasodilation. Likewise, another study found that women with menstrual migraines were 1.7× more likely to have a migraine 2 days before menstruation with greater symptom severity (3).

Estrogen vasodilates the vascular endothelium by increasing nitric oxide synthase activity and directly relaxing vascular smooth muscle (2). Its actions are likely sex-specific, as 1 study showed that women, but not men, had increased peripheral arterial vasodilation with supplemental estrogen (4).

One may hypothesize that certain women have vasculature that is negatively affected by the relative reduction in estrogen. Perhaps this predisposes to chest pains from undetectable ischemia, endothelial dysfunction, coronary vasospasm, or microvascular disease among those with SCAD. Additionally, SCAD MI is strongly associated with conditions affecting the hormonal milieu, such as female sex, pregnancy, and exogenous hormone therapy, and most pregnancy-associated SCAD events occur early postpartum when there is a rapid ovarian hormonal decline (1).

The novel observation of catamenial chest pain among SCAD patients emphasizes the potential vascular significance of ovarian hormones among these patients. If catamenial chest pain is recognized, and after excluding ongoing ischemia/infarction, anti-ischemic therapies can be titrated to achieve optimal symptom control. The role of pregnancy, endogenous/exogenous hormones, endometrial ablation, and menopause in SCAD needs to be further explored to better discern management of post-SCAD contraception, pregnancy considerations, and recurrent symptoms.

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TABLE 1

Patient Details Specific to History of SCAD and Subsequent Chest Pain Symptoms

Patient #	Age*, yrs	SCAD Risk Factors	Initial SCAD Management	Timing of Symptoms	% of Cycles Affected	Evaluation of Recurrent CP	Treatment
1	35	5 days PP	PCI	5 days pre-menstrual	100	PCI to residual dissection; persistent pain without ischemia on subsequent stress test	Short-acting nitrate prn
2	42	Prior SCAD	Unsuccessful PCI; CABG	1–2 days pre-menstrual	5–10	No ischemia on stress test	Short-acting nitrate prn, CCB
3	39	13 days PP, FMD	Unsuccessful conservative;† PCI	1–2 days pre-menstrual	90	Patent coronary arteries on CA	Short-acting nitrate prn, CCB, beta-blocker
4	46	None	Conservative	1–2 days pre-menstrual	100	Improved coronary artery caliber on CA	Long-acting nitrate, ranolazine, endometrial ablation
5	44	None	Conservative	1–2 days pre-menstrual through menses	100 for 2 yrs, now 5–10	No ischemia on stress test; stable CCTA	Short-acting nitrate prn
6	34	FMD	Unsuccessful conservative; PCI	1–2 days pre-menstrual through menses	50	Complex PCI for instent stenosis; intracoronary thrombus requiring emergent CABG; persistent pain without ischemia on stress test; patent stents/grafts on repeat CA	Ranolazine, CCB, beta-blockers, long-acting nitrate
7	45	EDS type IV	Unsuccessful conservative; unsuccessful PCI; interval CABG	1–2 days pre-menstrual during first 2 days of menses	100	Patent coronary arteries on CCTA	Short-acting nitrate prn, CCB, long-acting nitrate
8	43	Emotional stress	Conservative	3 days pre-menstrual during first 2 days of menses	100	No ischemia on stress test; patent coronaries on CA	Short-acting nitrate prn
9	37	10 weeks PP	PCI	1–2 days pre-menstrual	Only heavy cycles	No ischemia on stress test	Short-acting nitrate prn
10	37	15 days PP	CABG	1–2 days pre-menstrual	50	No ischemia on stress test	Short-acting nitrate prn, long- acting nitrate, ranolazine
11	41	None	Conservative	1–2 days pre-menstrual	20	No ischemia on stress test	Short-acting nitrates prn, CCB

*Age in years at the time of SCAD.

†Conservative = conservative management during which the patient received medications but no other invasive treatment strategies at time of SCAD.

CA = coronary angiography; CABG = coronary artery bypass grafting; CCB = calcium-channel blocker; CCTA = coronary computed tomography angiography; CP = chest pain; EDS = Ehlers-Danlos syndrome; FMD = fibromuscular dysplasia; PCI = percutaneous coronary intervention; PP = postpartum; pm = pro re nata (when necessary); SCAD = spontaneous coronary artery dissection.