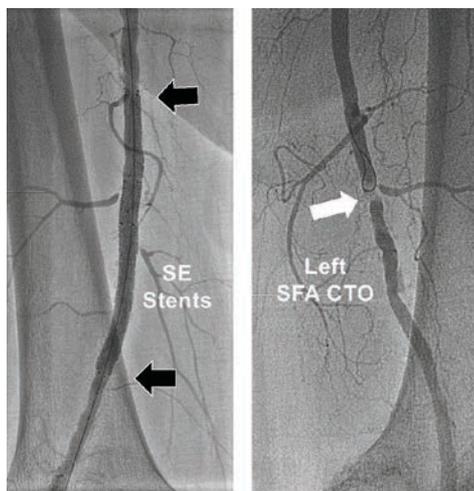


Cath Lab Digest

A product, news & clinical update for the cardiac catheterization laboratory specialist



PERIPHERAL INTERVENTION

Radial to Peripheral in Women With Small Radial Arteries: 5 French Access for Endovascular Therapy in Femoral, Iliac, Renal, Subclavian and Carotid Artery Disease

Robert L. Minor, Jr, MD

Transradial access (TRA) with same-day discharge for coronary interventions can save over \$3000 per case, as compared to femoral access with overnight hospital stay. In addition, it improves safety, reduces patient discomfort, and is strongly preferred by patients and nursing staff. TRA for endovascular interventions also allows same-day discharge and may provide similar benefits.¹

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Complete Stent Shaft Disruption During Complex CTO Angioplasty

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Distal Radial Sheath Extension Technique

Richard Casazza, MAS,
RT(R)(CI)

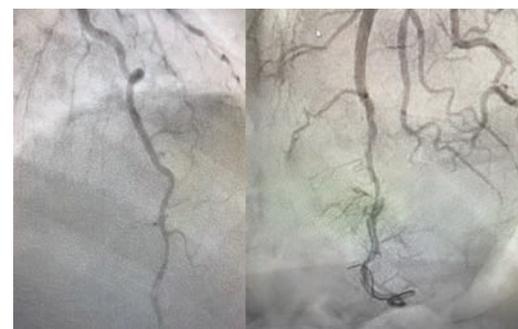
Online Exclusive

CARDIOLOGY CARE

Coronary Artery Disease in Women: A Review

Julie Billingsley, Richard J. Merschen, EdS, RT(R)(CV), RCIS

Cardiovascular disease (CVD) is the leading cause of death in women and men in the United States. Coronary artery disease (CAD), heart failure, and stroke cause around 500,000 deaths in U.S. women every year, with the most deaths caused by CAD.¹ While CAD is the leading cause of death



in both sexes, the etiology, recognition, management, and outcomes of CAD differ between men and women. Many of the common risk factors associated with CAD also have different clinical presentations and chronology in women. Additionally, women may have pregnancy-, ovarian-, and estrogen-related complications that increase their risk for developing CAD.²

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OUT-OF-HOSPITAL CARE

How One Healthcare System Creates and Partners Around Cardiovascular Ambulatory Surgical Centers

CLD talks with Kristi McShay, Associate Vice President, Cardiovascular Service Line, Banner Health.

Can you tell us about your role and how you are involved with cardiovascular ambulatory surgical centers (ASCs)?

I am the associate vice president for cardiovascular services at Banner Health, and in our organization, it is a corporate-level position. I report up through the vp of service lines to our chief strategy officer. Banner Health's service line executives are very firmly housed within the strategy side of the organization versus operations, meaning I am not "in the weeds" as an operational person.



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Continued from cover

Coronary Artery Disease in Women: A Review

Julie Billingsley, Richard J. Merschen, EdS, RT(R)(CV), RCIS

On average, CAD and other CVDs will present in women 10 years later than in men. This may be due to the potential benefits of estrogen in premenopausal women.³ While the incidence of CAD in women is initially lower than in men, it rises steadily after the fifth decade. Because CAD risk factors vary between men and women across age ranges, underestimating these differences is a reason why women with CAD are treated later in their events and have poorer outcomes than men.⁴ This is most evident during acute coronary syndrome (ACS) scenarios where women's clinical presentations are under-recognized, under-studied, under-diagnosed, and under-treated.⁵⁻⁷ While women's cardiac health screening and care has greatly improved, it is important to understand female-specific CAD risks, clinical presentations, treatment options, and outcomes.^{8,9} Despite updated CVD prevention guidelines that emphasize women's unique risk factors and clinical manifestations, treatment gaps in women's heart health continue to be a challenge.¹⁰

This review will discuss CVD risk factors and special considerations for women, including unique issues related to estrogen, menopause, and

pregnancy. CVD in women, such as microvascular disease (MVD), spontaneous coronary artery dissection (SCAD), and takotsubo cardiomyopathy (TCM) will also be discussed. Because of sex and gender differences, clinical management and recognition of these differences is essential in the diagnosis, management, and improved outcomes for women with CVD.⁵

The Role of Estrogen in CVD Prevention

Estrogen provides many health benefits to women, including higher high-density lipoprotein (HDL) cholesterol levels, promotion of normal blood clot formation, the dilation and relaxation of blood vessels to improve blood flow, blood pressure control, and reduced rates of CVD in comparison to men. Estrogen is largely responsible for the average 10-year gap in the onset of CAD between men and women. When women go through menopause, estradiol levels decrease by 75%.¹⁰⁻¹² The reduction in estrogen is associated with increased rates of CVD and cardiovascular risk factors. In women who use tobacco or have diabetes, the benefits of estrogen are significantly reduced, to the point where no discernable benefits from estrogen are evident.³⁻⁵

Risk Factors

While CVD risk factors are often similar for men and women, there are important considerations that are specific to women. Smoking, for instance is a major CVD risk factor for both sexes. However, smoking is more detrimental to women, with the risk of CAD and myocardial infarction (MI) for women twice as high as for men.^{3,11} There is also a compounded CAD risk between smoking and the use of hormonal treatment, which may be due to prothrombotic effects.¹² Smoking more than a pack a day of cigarettes may increase a women's risk of myocardial infarction by 12-fold, and this risk is more than doubled in women using oral contraception.¹² Furthermore, smoking is known to reduce the concentration of HDL in both men and women, but is more significant in women.³ Smoking also has anti-estrogenic effects in women and has been linked to premature menopause.³ Due to these effects, smoking quadruples a premenopausal woman's risk for CAD.⁴

Dyslipidemia is common in women and changes in their lipid profiles are associated with menopausal changes.¹³ The Study of Women's Health Across the Nation (SWAN) study found that during menopause, women have substantial increases in total cholesterol, triglycerides, and low-density lipoprotein (LDL) cholesterol. On average, women over the age of 50 have higher total cholesterol levels than men over 50.^{14,15} An increase in HDL levels, which has major health benefits such as atheroprotective properties in the premenopausal phase, has been found to be paradoxically associated with increased atherosclerosis progression after menopause.¹⁴ This may be due to changes in HDL function resulting from hormonal changes.¹⁵ Elevated triglyceride levels also increase CAD risks in postmenopausal women. During menopause, the concentration of LDL cholesterol in women can also significantly increase.

Hypertension (HTN) is the most common modifiable risk factor for CVD, and occurs in over 80 million adults in the United States.¹⁶ HTN is associated with shorter life expectancy, a shorter life expectancy free of CVD, and more years lived with CVD.¹⁶⁻¹⁸ Women tend to have lower rates of hypertension than men under the age of 65, but women over 65 are more likely to have hypertension than men over 65 (Figure 1).

Systolic blood pressure (SBP) increases in women as they age. After menopause, there is an increase in SBP that has been theorized to be secondary to factors such as the withdrawal of vasodilator effects of endogenous estrogen, increased arterial stiffness, reduced vascular compliance, and decreased endothelial nitric oxide production.¹⁹ The increase in both SBP and pulse pressure in postmenopausal women is greater than in men in the same age group, while diastolic blood pressure shows little difference between the sexes.¹⁷⁻¹⁹ Isolated systolic hypertension (ISH) significantly increases the risk for CVD, such as stroke, left ventricular

HYPERTENSION RATES ACROSS AGE GROUPS

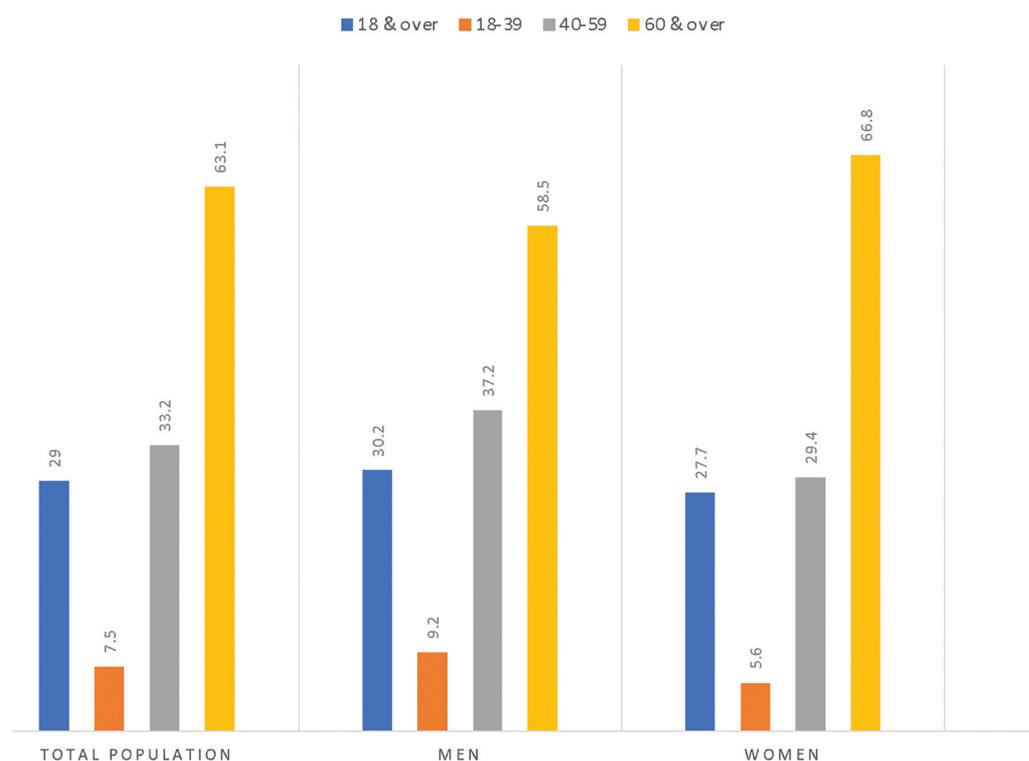


Figure 1. Prevalence of hypertension in men and women as they age.

Data obtained from <https://www.cdc.gov/nchs/data/databriefs/db289.pdf>

TABLE 1. Cardiac complications following preeclampsia.

Complication	Increase in risk	Average time span (years)
Hypertension	3.7	14.1
Ischemic heart disease	2.16	11.7
Stroke	1.81	10.4
Venous thrombosis	1.78	4.7

The increased risk of cardiovascular disease in women diagnosed with preeclampsia and the average time in years for these complications to develop.

Source: <https://www.bmj.com/content/335/7627/974> ref 28

hypertrophy and CAD. ISH continues to increase disproportionately in women until the age of 80, is closely linked to obesity, and is 6 times higher in women with a body mass index (BMI) >30.^{17,18}

Diabetes mellitus (DM) and obesity are also common risk factors contributing to CAD in both men and women. In women, type 2 DM is associated with a higher CAD risk than in men, and a higher mortality rate from MI.^{3,19} A BMI >30 increases CAD risks in women by 64% and men by 46%.³ Compared with men with the same condition, women with diabetes have higher rates of cardiovascular death and overall mortality than men.²⁰ While men typically develop CVD about a decade before women, in diabetic women, CVD risk occurs around the same age as men. It is theorized that diabetes eliminates much of the protection premenopausal women would normally get from estrogen. DM increases the risk of cardiovascular disease by three to four times in women and two

to three times in men, after adjusting for other risk factors.²¹ Diabetes and obesity are also linked to pregnancy-related hypertension risks such as preeclampsia and gestational hypertension.

For women, a family history of heart disease or sudden death from MI is a significant risk factor for the development of CAD or an MI. The risk increases based on which family member is involved. For example, if a parent has history of MI or sudden death, the risk increases 3-fold. If the family member is a brother, the risk increases 6-fold, and in a sister, the risk increases 12-fold.⁴

Risks Specific to Women

There are also multiple risk factors for CVD that are specific to women, including menopausal and pregnancy-related issues. The largest increase of CVD-related deaths in women occurs in the menopausal and postmenopausal periods.^{22,23} A reduction in estrogen is related to a higher incidence of hypertension, diabetes, dyslipidemia, and obesity.²⁴⁻²⁶ In the peripartum setting, CVD presentations include:

- Chronic hypertension (CHTN);
- Peripartum cardiomyopathy (PPCM);
- Gestational hypertension (GHTN);
- Preeclampsia.

These pregnancy-related complications have been shown to substantially increase the risk for CVD in the short and long term.^{22,23} Additionally, PPCM, GHTN, and preeclampsia are also causes for preterm delivery. HTN and PPCM are associated with vascular dysfunction, and pregnancy may cause the manifestation of a vascular dysfunction or a preexisting medical condition.^{23,24} PPCM can develop in late pregnancy or shortly postpartum, and is characterized by significant left ventricular dysfunction with a low ejection fraction and heart failure. PPCM can also cause the development of thromboembolism, arrhythmias, and progressive heart failure.²⁶

Preeclampsia is a pregnancy-related hypertensive disorder, defined by a new onset of HTN after 20 weeks of gestation, and accompanied by

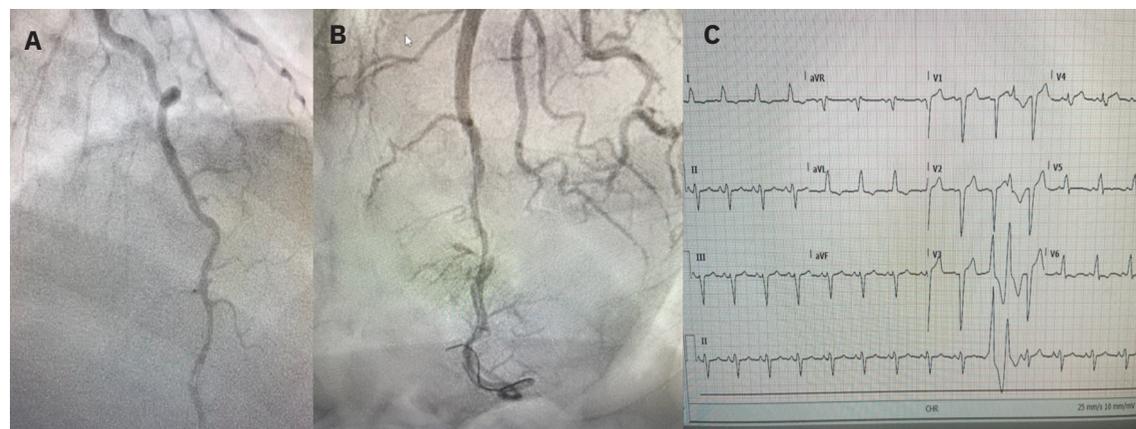


Figure 2. Spontaneous coronary artery dissection in women. The first patient was treated with medical therapy (A) and the second patient (B) was treated with percutaneous coronary intervention. The electrocardiogram (ECG) (at right) represents precordial ST-elevation myocardial infarction (MI) changes seen on these patients, both of whom presented through the emergency department.

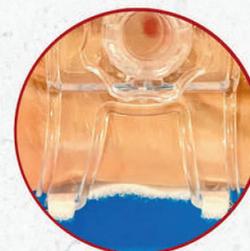
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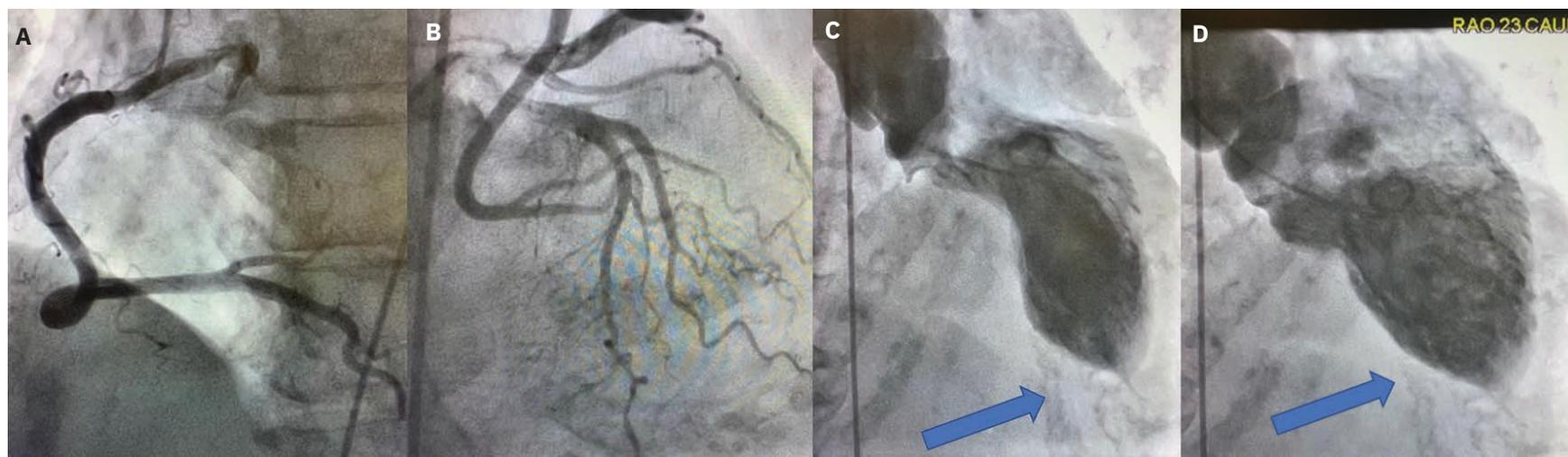


Figure 3. Takotsubo syndrome. This patient ruled in for acute myocardial infarction. Angiography of the coronary arteries was normal (A-B), but a classic Takotsubo pattern of left ventricular apical stunning is present (C-D).

TABLE 2. Symptoms of coronary artery disease in women vs men.

Women	Men
Chest pain	Chest pain
Low chest or abdominal pain/pressure	Jaw, neck, and back pain
Jaw, neck, and upper back pain	Nausea and vomiting
Nausea and vomiting	Shortness of breath
Extreme fatigue	
Indigestion	
Syncope	
Shortness of breath	

Some of the differences between men and women with acute coronary syndrome symptoms. Women are more likely to experience multiple symptoms at once.

proteinuria or systemic disease.²⁷ Evidence shows the effects and damage of eclampsia may continue postpartum, and in some cases, preeclampsia may develop postpartum. It is believed that the underlying cause of preeclampsia is endothelial dysfunction that corresponds to higher levels of coronary calcium. The damage from preeclampsia may not resolve postpartum, doubling women's long-term risk for CVD, particularly stroke, cardiac ischemia, and venous thrombosis (Table 1).^{2,27,28} Preeclampsia may also progress and cause eclampsia or hemolysis, elevated liver enzymes, and low platelet count, in a syndrome known as (HELLP).²⁷⁻²⁹

Women and Coronary Artery Disease

While CAD is the most common type of heart disease and the leading cause of death in both sexes, there are other coronary and cardiac syndromes that affect women at much higher rates than men. This includes coronary microvascular disease (CMD), spontaneous coronary artery dissection (SCAD), and takotsubo syndrome. CMD is characterized by ischemia and/or angina with no obstructive CAD. CMD is an increasingly recognized cause of angina and is much more common in women. CMD is associated with abnormal dilation and constriction of the small vessels of the heart, and is a major cause of ischemia and nonobstructive coronary artery disease (INOCA) in as many as 50% of women with ischemia and angina symptoms.^{30,31} Low levels of estrogen before menopause raise a woman's risk for developing CMD and it is a major source of angina in younger women.²⁹⁻³¹

In the January 2023 edition of *Cath Lab Digest*, Dr. Jennifer Tremmel explains that while CMD, either structural or functional, may be independently responsible for the development of angina with nonobstructive coronary arteries (ANOCA), there are several other possible causes.³² These etiologies include endothelial dysfunction, epicardial vasospasm, and myocardial bridging.³² If a patient presents with ANOCA, it is important to determine the underlying cause. Typical stress testing may not find any abnormality in these patients. Instead, patients presenting with ANOCA may need further invasive testing in the cardiac catheterization lab that includes the use of acetylcholine, fractional flow reserve, and coronary flow reserve.³²

Spontaneous coronary artery dissection (SCAD) is a nonatherosclerotic etiology of ACS, including sudden cardiac death, which frequently affects younger women.⁸⁻¹⁰ Among women, SCAD is the cause of ACS in as many as 35% of cases^{8,10} and the most common etiology of MI associated with pregnancy.^{31,33} SCAD can occur in men and at any age, but is most common in women in their fourth and fifth decades.³³⁻³⁵ Most people with SCAD do not have other risk factors like hypertension or dyslipidemia. However, women who

have experienced childbirth, fibromuscular dysplasia, and hormone use are at increased risk of SCAD.^{34,35} While SCAD has been linked to childbirth, most often occurring in the first few weeks postpartum, it can also occur during pregnancy.^{34,35} Oral contraceptives, hormone replacement therapy, and infertility treatments have also been linked to SCAD.^{35,36}

Figure 2 illustrates two cases of SCAD diagnosed in women with ST-elevation MI symptoms. In the first case, the patient was treated with medical therapy because the dissection was located too distal in the left anterior descending coronary artery to deploy a stent. The second patient was treated with a drug-eluting stent. Both patients had successful outcomes, and are also being managed with medications and regular cardiology follow-up care.

Takotsubo cardiomyopathy (TCM), also known as takotsubo syndrome, stress-induced cardiomyopathy, transient apical ballooning, or broken heart syndrome, is a condition that is generally triggered by emotional or physical stress, and is characterized by transient regional left ventricular systolic dysfunction.^{36,37} Symptoms of TCM are often similar to ACS prior to clinical assessment and imaging, and may present as an apical, anterior STEMI or non-STEMI. The onset of symptoms in most TCM cases is usually preceded by an identifiable acute emotional or physical stressor, or a combination of both.²⁹ In the International Takotsubo Registry study, 36% of patients had experienced a physical trigger, 28% reported an emotional trigger, and 8% experienced both.^{35,36} It is estimated that >90% of TCM occurs in women.^{37,38} Fortunately, TCM will usually resolve within several months without complications and rarely causes other cardiovascular events.

In Figure 3, a 70-year-old woman, whose spouse had recently died presented with signs and electrocardiographic presentation of an anterior wall MI. Angiography revealed no obstructive coronary disease. Left ventriculography revealed a classic apical ballooning pattern associated with TCM. The patient was treated with medications and followed up with serial echocardiography. In less than 2 months, her left ventricular function had returned to normal.

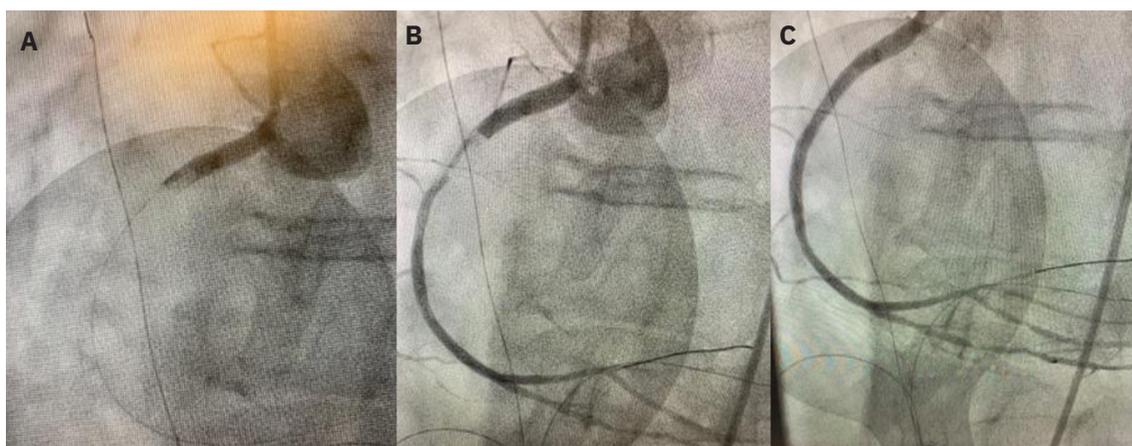


Figure 4. This patient presented with nausea, vomiting, and fatigue for 24 hours. She then developed atypical chest pain and was seen in the emergency department. A 12-lead ECG showed an inferior wall MI pattern and she was sent to the cath lab. Angiography revealed an inferior wall MI that was successfully treated with thrombectomy and a drug-eluting stent.

ACS Presentation and Treatment

ACS symptoms may also differ between men and women. Women can present with different symptoms such as shortness of breath. Angina in women has a higher likelihood of being present during rest and mental stress, while in men, angina is most often caused by physical exertion.^{4,39} Women may also present with symptoms such as back pain, abdominal discomfort, fatigue, lightheadedness that have been traditionally classified as noncardiac, or atypical symptoms (Table 2). The Women's Ischemia Syndrome Evaluation (WISE) study concluded 65% of women who had CAD were misdiagnosed because of disparity in symptoms compared to men.⁴

Studies also show that women present to the hospital later in the course of an MI than men. Some of the reasons for this may be because women's symptoms develop more gradually, have longer ischemic times than men, and may be described as "atypical".³⁹⁻⁴¹ Women generally have a poorer prognosis and more severe outcomes than men after MI, PCI, and coronary artery bypass grafting (CABG) that include heart failure, re-infarction, recurrent ischemia, bleeding and stroke.³⁸⁻⁴⁰

Women are also more likely than men to die after a first MI, and for survivors, there is higher risk of recurrent MI, heart failure, or death.⁸ In the Framingham Heart Study, the one-year mortality rate following an MI was 44% in women versus 27% in men.^{41,42} Research has shown that women with ACS

are less likely to be treated with guideline-directed medical therapy, less likely to undergo PCI, and more likely to have a time delay to reperfusion.^{38,39}

In Figure 4, a 75-year-old female initially developed nausea and vomiting, and self-treated at home for gastrointestinal distress. The next day, she developed atypical chest pain. She went to her family physician and had 12-lead ECG performed that suggested an inferior wall STEMI. She was transported to an acute care facility for STEMI and had a right coronary artery infarct that was successfully treated with PCI. Patient follow-up will help determine the possible long-term effects of the delayed time to PCI.

Conclusion

The goal of this literature review is to promote awareness of the complexity of women's cardiovascular health. While significant advancements have been made in women's cardiovascular health due to increased awareness, there are still many disparities between men and women in terms of timely care received and treatment outcomes. These disparities are most apparent when treating women with suspected ACS. Risk factors are strongly influenced by menopause and estrogen levels in women, requiring skilled care providers to address the unique clinical challenges in women's cardiovascular health. There are also unique presentations such as PPCM, GHTN, preeclampsia, MVD, SCAD, and TCM, further complicating diagnoses and treatment for women with cardiac disease. ■

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References

1. Bybee KA, Stevens TL. Matters of the heart: cardiovascular disease in U.S. women. *Mo Med*. 2013; 110(1): 65-70.
2. Gongora MC, Wenger NK. Cardiovascular complications of pregnancy. *Int J Mol Sci*. 2015; 16(10): 23905-23928. doi:10.3390/ijms161023905
3. Duda-Pyszny D, Trzeciak P, Gąsior M. Coronary artery disease in women. *Kardiolog Torakochirurgia Pol*. 2018; 15(1): 44-48. doi:10.5114/kitp.2018.74675
4. Pathak LA, Shirodkar S, Ruparelia R, Rajebahadur J. Coronary artery disease in women. *Indian Heart J*. 2017; 69(4): 532-538. doi:10.1016/j.ihj.2017.05.023
5. Mehta LS, Beckie TM, DeVon HA, et al. Acute myocardial infarction in women: A scientific statement from the American Heart Association. *Circulation*. 2016; 133(9): 916-947. doi:10.1161/CIR.0000000000000351
6. Norris CM, Yip CYY, Nerenberg KA, et al. State of the science in women's cardiovascular disease: A Canadian perspective on the influence of sex and gender. *J Am Heart Assoc*. 2020; 9(4): e015634. doi:10.1161/JAHA.119.015634
7. Pinn VW. Sex and gender factors in medical studies: implications for health and clinical practice. *JAMA*. 2003; 289(4): 397-400. doi:10.1001/jama.289.4.397
8. Pepine CJ, Kerensky RA, Lambert CR, et al. Some thoughts on the vasculopathy of women with ischemic heart disease. *J Am Coll Cardiol*. 2006; 47(3 Suppl): S30-S35. doi:10.1016/j.jacc.2005.09.023
9. Vogel B, Acevedo M, Appelman Y, et al. The Lancet women and cardiovascular disease Commission: reducing the global burden by 2030. *Lancet*. 2021; 397(10292): 2385-2438. doi:10.1016/S0140-6736(21)00684-X
10. Bairey Merz CN, Andersen H, Sprague E, et al. Knowledge, attitudes, and beliefs regarding cardiovascular disease in women: The Women's Heart Alliance [published correction appears in *J Am Coll Cardiol*. 2017; 70(8): 1106-1107]. *J Am Coll Cardiol*. 2017; 70(2): 123-132. doi:10.1016/j.jacc.2017.05.024
11. Njølstad I, Arnesen E, Lund-Larsen PG. Smoking, serum lipids, blood pressure, and sex differences in myocardial infarction. A 12-year follow-up of the Finnmark Study. *Circulation*. 1996; 93(3): 450-456. doi:10.1161/01.cir.93.3.450
12. Rosenberg L, Palmer JR, Rao RS, Shapiro S. Low-dose oral contraceptive use and the risk of myocardial infarction. *Arch Intern Med*. 2001; 161(8): 1065-1070. doi:10.1001/archinte.161.8.1065
13. El Khoudary SR, Aggarwal B, Beckie TM, et al. Menopause transition and cardiovascular disease risk: Implications for timing of early prevention: A scientific statement from the American Heart Association. *Circulation*. 2020; 142(25): e506-e532. doi:10.1161/CIR.0000000000000912
14. Matthews KA, Crawford SL, Chae CU, et al. Are changes in cardiovascular disease risk factors in midlife women due to chronological aging or to the menopausal transition? *J Am Coll Cardiol*. 2009; 54(25): 2366-2373. doi:10.1016/j.jacc.2009.10.009
15. El Khoudary SR, Wang L, Brooks MM, Thurston RC, Derby CA, Matthews KA. Increase HDL-C level over the menopausal transition is associated with greater atherosclerotic progression. *J Clin Lipidol*. 2016; 10(4): 962-969. doi:10.1016/j.jacl.2016.04.008
16. Benjamin EJ, Blaha MJ, Chiuve SE, et al. Heart Disease and Stroke Statistics-2017 Update: A report from the American Heart Association [published correction appears in *Circulation*. 2017; 135(10): e646] [published correction appears in *Circulation*. 2017; 136(10): e196]. *Circulation*. 2017; 135(10): e146-e603. doi:10.1161/CIR.0000000000000485
17. Harsha DW, Bray GA. Weight loss and blood pressure control (Pro). *Hypertension*. 2008; 51(6): 1420-1425. doi:10.1161/HYPERTENSIONA-HA.107.094011
18. Fields LE, Burt VL, Cutler JA, Hughes J, Roccella EJ, Sorlie P. The burden of adult hypertension in the United States 1999 to 2000: a rising tide. *Hypertension*. 2004; 44(4): 398-404. doi:10.1161/01.HYP.0000142248.54761.56
19. Izumi Y, Matsumoto K, Ozawa Y, et al. Effect of age at menopause on blood pressure in postmenopausal women. *Am J Hypertens*. 2007; 20(10): 1045-1050. doi:10.1016/j.amjhyper.2007.04.019
20. Norhammar A, Schenck-Gustafsson K. Type 2 diabetes and cardiovascular disease in women. *Diabetologia*. 2013; 56(1): 1-9. doi:10.1007/s00125-012-2694-y
21. Martín-Timón I, Sevillano-Collantes C, Segura-Galindo A, Del Cañizo-Gómez FJ. Type 2 diabetes and cardiovascular disease: Have all risk factors the same strength? *World J Diabetes*. 2014; 5(4): 444-470. doi:10.4239/wjd.v5.i4.444
22. Johnson RR, Sweeney ME. Debate: The potential role of estrogen in the prevention of heart disease in women after menopause. *Curr Control Trials Cardiovasc Med*. 2000; 1(3): 139-142. doi:10.1186/cvm-1-3-139
23. Arnaout R, Nah G, Marcus G, et al. Pregnancy complications and premature cardiovascular events among 1.6 million California pregnancies. *Open Heart*. 2019; 6(1): e000927. doi:10.1136/openhrt-2018-000927
23. Vaccarino V, Krumholz HM, Berkman LF, Horwitz RI. Sex differences in mortality after myocardial infarction. Is there evidence for an increased risk for women? *Circulation*. 1995; 91(6): 1861-1871. doi:10.1161/01.cir.91.6.1861
24. Luger RK, Kight BP. Hypertension In Pregnancy. [Updated 2022 Oct 3]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2023. www.ncbi.nlm.nih.gov/books/NBK430839/
25. Stangl V, Baumann G, Stangl K. Coronary atherogenic risk factors in women. *Eur Heart J*. 2002; 23(22): 1738-1752. doi:10.1053/euhj.2002.3329
26. Rodriguez Ziccardi M, Siddique MS. Peripartum Cardiomyopathy. [Updated 2022 Jul 19]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2023. www.ncbi.nlm.nih.gov/books/NBK482185/
27. Chaiworapongsa T, Chaemsaitong P, Yeo L, Romero R. Pre-eclampsia part 1: current understanding of its pathophysiology. *Nat Rev Nephrol*. 2014; 10(8): 466-480. doi:10.1038/nrneph.2014.102
28. Khalid F, Mahendrakar N, Tonismae T. HELLP Syndrome. [Updated 2022 Jun 16]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2023. www.ncbi.nlm.nih.gov/books/NBK560615/
29. Bellamy L, Casas JP, Hingorani AD, Williams DJ. Pre-eclampsia and risk of cardiovascular disease and cancer in later life: systematic review and meta-analysis. *BMJ*. 2007; 335(7627): 974. doi:10.1136/bmj.39335.385301.BE
30. Tjoe B, Barsky L, Wei J, et al. Coronary microvascular dysfunction: Considerations for diagnosis and treatment. *Cleve Clin J Med*. 2021; 88(10): 561-571. doi:10.3949/ccjm.88a.20140
31. Kothawade K, Bairey Merz CN. Microvascular coronary dysfunction in women: pathophysiology, diagnosis, and management. *Curr Probl Cardiol*. 2011; 36(8): 291-318. doi:10.1016/j.cpcardi.2011.05.002
32. Tremmell JA. Evaluating patients with ANOCA (Angina With Nonobstructive Coronary Arteries). *Cath Lab Digest*. 2023; 31(1): 18-20. https://www.hmpgloballearningnetwork.com/site/cathlab/clinical-update/evaluating-patients-anoca-angina-non-obstructive-coronary-arteries#:~:text=In%20evaluating%20these%20patients%2C%20the,-cath%20lab%20for%20ANOCA%20testing.
33. Elkayam U, Jalnapurkar S, Barakkat MN, et al. Pregnancy-associated acute myocardial infarction: a review of contemporary experience in 150 cases between 2006 and 2011. *Circulation*. 2014; 129(16): 1695-1702. doi:10.1161/CIRCULATIONAHA.113.002054
34. Tweet MS, Hayes SN, Codsí E, Gulati R, Rose CH, Best PJM. Spontaneous coronary artery dissection associated with pregnancy. *J Am Coll Cardiol*. 2017; 70(4): 426-435. doi:10.1016/j.jacc.2017.05.055
35. Lettieri C, Zavalloni D, Rossini R, et al. Management and long-term prognosis of spontaneous coronary artery dissection. *Am J Cardiol*. 2015; 116(1): 66-73. doi:10.1016/j.amjcard.2015.03.039
36. Assad J, Femia G, Pender P, Badie T, Rajaratnam R. Takotsubo Syndrome: A review of presentation, diagnosis and management. *Clin Med Insights Cardiol*. 2022; 16:11795468211065782. doi:10.1177/11795468211065782
37. Ghadri JR, Cammann VL, Jurisic S, et al. A novel clinical score (InterTAK Diagnostic Score) to differentiate takotsubo syndrome from acute coronary syndrome: results from the International Takotsubo Registry. *Eur J Heart Fail*. 2017; 19(8): 1036-1042. doi:10.1002/ejhf.683
38. Templin C, Ghadri J, Diekmann J, et al. Takotsubo (stress) cardiomyopathy. *N Engl J Med*. 2015; 373: 2688-2691. doi:10.1056/NEJMc1512595
39. Mehta LS, Beckie TM, DeVon HA, et al. Acute myocardial infarction in women: A scientific statement from the American Heart Association. *Circulation*. 2016; 133(9): 916-947. doi:10.1161/CIR.0000000000000351
40. DeFilippis EM, Collins BL, Singh A, et al. Women who experience a myocardial infarction at a young age have worse outcomes compared with men: the Mass General Brigham YOUNG-MI registry. *Eur Heart J*. 2020; 41(42): 4127-4137. doi:10.1093/eurheartj/ehaa662
41. Perdoncin E, Duvernoy C. Treatment of coronary artery disease in women. *Methodist Debaque Cardiovasc J*. 2017; 13(4): 201-208. doi:10.14797/mdcj-13-4-201
42. Mahmood SS, Levy D, Vasan RS, Wang TJ. The Framingham Heart Study and the epidemiology of cardiovascular disease: a historical perspective. *Lancet*. 2014; 383(9921): 999-1008. doi:10.1016/S0140-6736(13)61752-3