Case Report

Journal of INTERNATIONAL MEDICAL RESEARCH

Journal of International Medical Research
2019, Vol. 47(5) 2269–2273

DOI: 10.1177/0300060519837833
journals.sagepub.com/home/imr
SAGE

Acute myocardial infarction in pregnancy: spasm caused by hyperthyroidism?

LingQing Wang*, JianGuang Yang*, JiPing Zheng and XingJian Gu

Abstract

We present a 26-year-old woman with ST-segment elevation myocardial infarction in the 14th week of pregnancy. Coronary angiography revealed no abnormalities in the coronary arteries. She had no history of coronary risk factors such as smoking, diabetes mellitus, hypertension, or dyslipidemia. Although we do not have direct evidence of coronary spasm in this patient, several factors suggest that coronary spasm is the most likely cause of myocardial infarction. We suspect that hyperthyroidism may have played an important role in coronary spasm in this patient. Early use of coronary angiography is helpful to identify the types of coronary artery lesions.

Keywords

Pregnancy, coronary spasm, hyperthyroidism, acute myocardial infarction, coronary angiography, hyperemesis

Date received: 15 November 2018; accepted: 22 February 2019

Introduction

The incidence of acute myocardial infarction (AMI) in pregnancy ranges from 1 to 10 cases per 100,000 deliveries.[1–4] Physiological changes during pregnancy increase the risk of AMI. Only 2% of cases of myocardial infarction during pregnancy are caused by coronary spasms.[2] Patients with hyperemesis produce structural variants of human chorionic gonadotropin (hCG). High levels of hCG may induce gestational transient thyrotoxicosis (GTT) and increase the activity of thyroxine. Both GTT and increased thyroxine activity increase the sensitivity of vasoconstrictors to norepinephrine,[5,6] and the latter may

Department of Cardiology, Taizhou First People's Hospital, Wenzhou Medical University, Taizhou, Zhejiang Province, China

*These authors contributed equally to this work.

Corresponding author:
XingJian Gu, Wenzhou Medical University, Department of Cardiology, Taizhou First People's Hospital, Hejie Road 218#, Huangyan, Taizhou, Zhejiang Province, China.
Email: xingjian1967@sina.com

lead to coronary spasm. The mechanism of coronary spasm and the relationship between spasm and hyperthyroidism need further study.

Case report

A 26-year-old pregnant woman who presented with repeated nausea and vomiting at 14 weeks of gestation was admitted to our hospital after 2 days of symptoms. She did not have any chest pain but it was likely to be masked by severe vomiting. The electrocardiogram on admission showed ST elevation in leads II, III, and aVF (Figure 1). Cardiac troponin I (cTNI; 22.00 ng/mL) was significantly elevated, which is consistent with inferior myocardial infarction. We measured cTNI levels multiple times, with a maximum value of 22.00 ng/mL, suggesting a small area of myocardial damage, which may explain why echocardiography did not detect abnormal ventricular wall motion. The physical examination was normal. The patient was gravida 2, para 1, with no history of coronary risk factors. It is worth noting that the patient had hyperthyroidism with free thyroxine of 3.29 ng/dL and hypersensitive thyrotropin (hTSH) <0.0048 μIU/L.

Urgent coronary angiography (CAG) showed that the coronary artery was normal (Figure 2). We did not see evidence of atherosclerotic plaques, coronary dissection, coronary spasm, or thrombus. The electrocardiogram showed that the ST segment of the inferior wall leads (II, III, and aVF) returned to normal 30 minutes after CAG (Figure 3). Echocardiogram after the CAG showed no local asynergy of the inferior wall.

Treatment with heparin was initiated to prevent potential thrombus formation. Because we could not exclude coronary spasm, we used calcium channel blockers and nitrates to prevent spasm. The patient remained asymptomatic during follow-up with medical management.

This report was prepared in compliance with CARE guidelines (https://www.care-statement.org). The Ethics Committee of Taizhou First People's Hospital approved the study protocol. Written informed consent was obtained from the patient for publication of this report and accompanying images.

Discussion

The incidence of AMI in pregnancy ranges from 1 to 10 cases per 100,000 deliveries. The physiological changes during pregnancy,

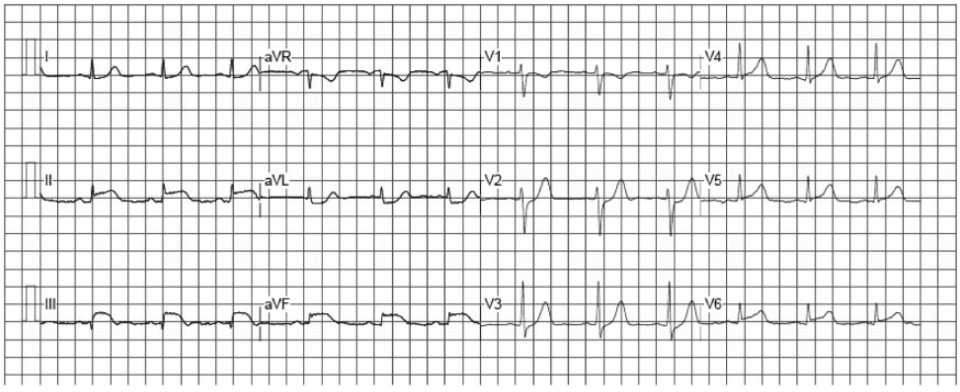

Figure 1. The electrocardiogram at admission indicated that the II, III, and aVF lead ST segments were elevated.

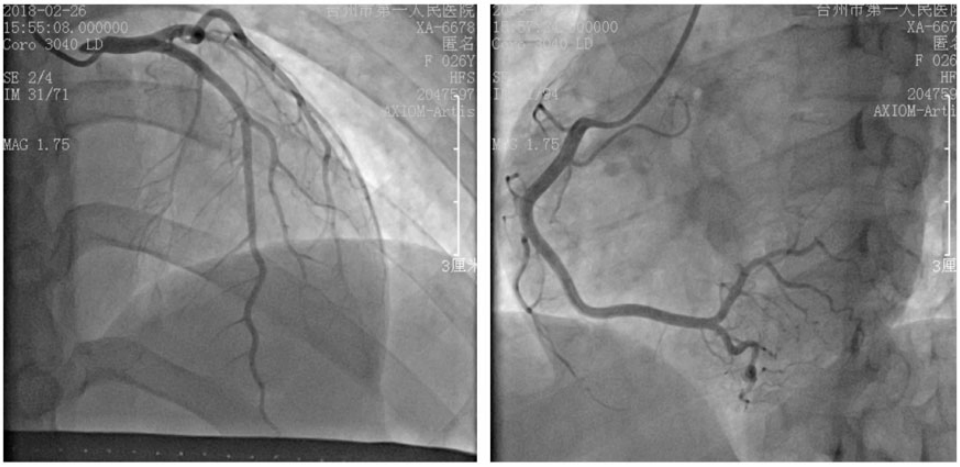

Figure 2. No lesions of the coronary artery were found by coronary angiography.

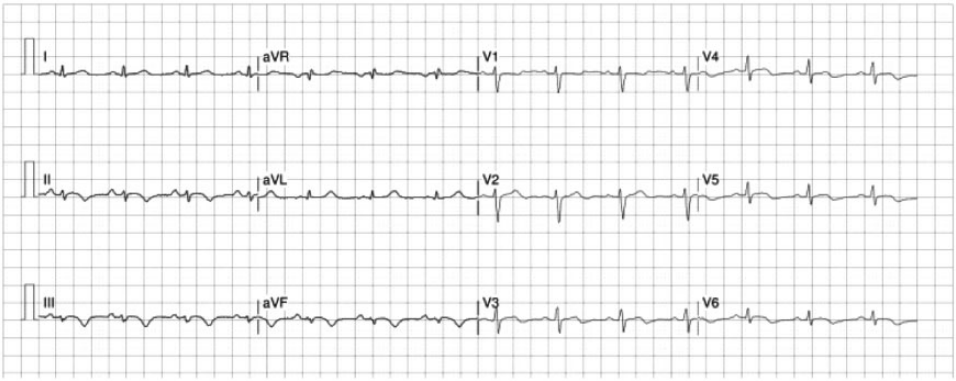

Figure 3. The electrocardiogram indicated that the ST segment of the inferior wall leads (II, III, and aVF) returned to normal 30 minutes after coronary angiography.

such as a procoagulant state and increased stress on the cardiovascular system, can increase the risks of AMI. Under such physiological conditions, coronary thrombosis or spontaneous coronary dissection is more likely to occur.[2,3]

Coronary spasm is also one of the reasons for AMI in pregnancy. Although there have been reports of coronary artery spasm in pregnancy, there is no direct evidence to suggest any relationships between them.[7,8] The mechanism of spasm is unknown but has been linked with hyperthyroidism in some cases.[9] GTT is a transient, mild hyperthyroidism that occurs early in pregnancy and is caused by hCG. GTT occurs in about 1.4% of pregnant women and is frequently associated with emesis.[10] Although we did not see spasms on coronary angiography, we speculated that our patient's AMI was caused by coronary spasm for the following reasons. First, no other lesions, such as dissection, atherosclerotic lesions, or thrombus, were seen on coronary angiography. Unfortunately, we did not perform intravascular ultrasound, and

some lesions, such as intramural hematoma, can be missed without intravascular ultrasound. However, we believe that intramural hematomas are unlikely to cause coronary blockage under stable conditions. If the myocardial infarction were caused by spontaneous coronary dissection, CAG would show a significant interruption of blood flow, continuous elevation of the ST segment of ECG, and further elevation of troponin I levels. Second, electrocardiography and myocardial markers suggested severe myocardial damage at the time of onset, which cannot be explained by other diseases. Third, Napoli et al.[6] found that the vasoconstrictor response to norepinephrine was significantly increased in patients with untreated hyperthyroidism compared with the response in control subjects and in those effectively treated. In patients with hyperthyroidism, noradrenaline-induced coronary spasm is more likely to occur. Furthermore, Kimura et al.[5] hypothesized that patients with hyperemesis produce molecular variants of hCG with increased thyroid-stimulating activity. Our patient with GTT was hospitalized because of hyperemesis, suggesting that her thyroid-stimulating activity was augmented. Finally, treatment with nitrates and calcium antagonists was effective in preventing recurrence.

AMI caused by coronary spasm is extremely rare in pregnant women. Roth et al.[2] retrospectively analyzed 93 pregnant women with acute coronary syndrome and only 2% were found to have a coronary spasm. Ladner et al.[4] noted that hypertension, diabetes, advanced maternal age, preeclampsia, and eclampsia were independent risk factors for pregnancy-associated AMI. James et al.[3] found that thrombophilia, transfusion, and postpartum infections were also significant risk predictors for AMI. Our patient was young and did not have any risk factors.

In general, the prognosis of hyperthyroidism-related coronary spasm is good, and nitrates and calcium channel blockers can be used to prevent recurrence. Radioactive iodine and surgical treatment may be considered for refractory patients.

In conclusion, AMI caused by coronary spasm is extremely rare in pregnant women. Our patient had hyperthyroidism with hyperemesis during the course of the disease, suggesting the possibility of hyperthyroidism leading to coronary spasm; she had no associated risk factors for AMI. The mechanism of spasm and the relationship between spasm and hyperthyroidism need further study.

Declaration of conflicting interest

The authors declare that there is no conflict of interest.

Funding

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

ORCID iD

XingJian Gu http://orcid.org/0000-0003-3355-4571

References

1. Dwyer BK, Taylor L, Fuller A, et al. Percutaneous transluminal coronary angioplasty and stent placement in pregnancy. *Obstet Gynecol* 2005; 106: 1162–1164.
2. Roth A and Elkayam U. Acute myocardial infarction associated with pregnancy. *J Am Coll Cardiol* 2008; 52: 171–180.
3. James AH, Jamison MG, Biswas MS, et al. Acute myocardial infarction in pregnancy: a United States population-based study. *Circulation* 2006; 113: 1564–1571.
4. Ladner HE, Danielsen B and Gilbert WM. Acute myocardial infarction in pregnancy and the puerperium: a population-based study. *Obstet Gynecol* 2005; 105: 480–484.
5. Kimura M, Amino N, Tamaki H, et al. Gestational thyrotoxicosis and hyperemesis gravidarum: possible role of hCG with

higher stimulating activity. *Clin Endocrinol (Oxf)* 1993; 38: 345–350.
6. Napoli R, Biondi B, Guardasole V, et al. Impact of hyperthyroidism and its correction on vascular reactivity in humans. *Circulation* 2001; 104: 3076–3080.
7. Başkurt M, Ozkan T, Arat Ozkan A, et al. Acute myocardial infarction in a young pregnant woman. *Anadolu Kardiyol Derg* 2010; 10: 285–286.
8. Iadanza A, Del Pasqua A, Barbati R, et al. Acute ST elevation myocardial infarction in pregnancy due to coronary vasospasm: a case report and review of literature. *Int J Cardiol* 2007; 115: 81–85.
9. Nannaka VB and Lvovsky D. A rare case of gestational thyrotoxicosis as a cause of acute myocardial infarction. *Endocrinol Diabetes Metab Case Rep* 2016; 2016: pii: 16-0063.
10. Glinoer D. Thyroid hyperfunction during pregnancy. *Thyroid* 1998; 8: 859–864.