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Case Report

Myocardial infarction and ischemic hepatitis complicated by postpartum hemorrhage

Meng-Han Chou [a], Ying-Chieh Chen [b], Kwei-Shuai Hwang [c], Mu-Hsien Yu [c], Her-Young Su [c, *]

[a] Department of General Medicine, National Defense Medical Center, Tri-Service General Hospital, Taipei, Taiwan
[b] Division of Pulmonary and Critical Care Medicine, Department of Internal Medicine, National Defense Medical Center, Tri-Service General Hospital, Taipei, Taiwan
[c] Department of Obstetrics and Gynecology, National Defense Medical Center, Tri-Service General Hospital, Taipei, Taiwan

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ABSTRACT

Objective: To present a rare case of life-threatening postpartum hemorrhage due to uterine atony complicated by acute myocardial infarction and ischemic hepatitis.
Case Report: A 37-year-old parturient, gravida 1 para 0, presented with symptoms and signs of shock due to postpartum hemorrhage after delivery. Ischemic hepatitis, pulmonary edema, and adult respiratory distress syndrome developed the following morning. On the 7th postpartum day, she developed chest pain and was subsequently diagnosed with acute inferior myocardial infarction based on serial changes on the electrocardiogram (ECG) and myocardial enzymes. The clinical condition improved after a series of resuscitative efforts and percutaneous transluminal coronary angioplasty.
Conclusion: The presented case demonstrated that when hypovolemic shock develops with complications of pulmonary edema or ischemic hepatitis, the possibility of cardiovascular disease should be immediately investigated and preventive measures initiated.
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Introduction

Postpartum hemorrhage (PPH) is one of the major causes of maternal morbidity and mortality worldwide. The definition of PPH varies. The postpartum loss of ≥500 mL of blood within 24 hours of delivery is commonly defined as PPH [1]. The prevalence of PPH is approximately 6% of deliveries, although a wide variation exists across different geographic regions of the world [2]. Multiple factors can cause PPH, including the leading cause (uterine atony) and others, such as genital tract lacerations, retained placenta, uterine inversion, and acquired or inherited coagulopathies.

Acute myocardial infarction (MI) in the postpartum period is very rare. Acute MI may occur as a result of coronary artery spasm or overt coronary artery disease. Pregnancy complicated by MI is associated with a high maternal mortality rate [3]. We report a case of life-threatening PPH due to uterine atony complicated by acute inferior MI and ischemic hepatitis. A series of aggressive treatments, including angiographic embolization and percutaneous transluminal coronary angioplasty (PTCA) with balloon dilatation and stent placement, were performed to successfully resolve the PPH and MI.

Case Report

A 37-year-old woman, gravida 1 para 0, was in labor at term. Her antenatal weight gain was 17.5 kg. There was no known history of cardiovascular disease and contributory factors, such as alcohol consumption, cigarette smoking, hypertension, diabetes mellitus, hyperlipidemia, coagulopathy, or a family history of MI. Her antenatal check-ups were uneventful and she had normal blood pressure. After a normal labor course, she delivered a healthy infant vaginally, weighing 3265 g and with Apgar scores of 8 and 9 at 1 minute and 5 minutes, respectively.

PPH developed soon after delivery. After excluding a genital tract laceration and retained placenta, the cause of PPH was diagnosed as an atonic uterus. The PPH precipitated extreme

* Corresponding author. Department of Obstetrics and Gynecology, National Defense Medical Center, Tri-Service General Hospital, 5F, 325, Section 2, Cheng-Gong Road, Nei-Hu District, 114 Taipei, Taiwan.
E-mail address: su108868@gmail.com (H.-Y. Su).

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hemorrhagic shock, with a massive blood loss of 1400 mL, a blood pressure of 68/39 mmHg, a pulse rate of 140 beats/min, and a respiratory rate of 22 breaths/min. Emergency treatment, consisting of bimanual massage, uterotonic administration, intrauterine packing, and blood transfusion failed to stabilize the PPH. The PPH persisted despite bilateral internal iliac artery embolization. The patient had an unstable hemogram, as follows: hemoglobin, 6.3 g/dL (normal range, 12.1–16.1 g/dL); hematocrit, 18.3% (normal range, 37–47%); and platelet count, 40×10^9/L (normal range, $150–450 \times 10^9$/L). The coagulation profile was as follows: prothrombin time (PT) international normalized ratio, 5.71 (normal range, 0–1.20); activated partial thromboplastin time, 143.4 seconds (normal range, 0–40 seconds); fibrinogen, 1.2 g/L (normal range, 1.5–4.5 g/L); and D-dimer > 0.2 μg/mL (normal range, <0.2 μg/mL). Surgical intervention was suspended due to disseminated intravascular coagulation (DIC). After correcting the coagulopathy by blood transfusion, the bleeding gradually stopped.

The next morning, the patient developed pulmonary edema, ischemic hepatitis (alanine aminotransferase [ALT], 1990 U/L; and aspartate aminotransferase [AST], 1600 U/L), and adult respiratory distress syndrome, which was treated with high-pressure ventilation. Table 1 shows the clinical course of the patient and the treatment received.

The patient's clinical condition stabilized 7 days later, until the sudden onset of chest pain with radiation to the left arm and bradycardia developed. An electrocardiogram (ECG) obtained at that time showed ST segment elevation in leads III and avF, which was compatible with an acute inferior wall myocardial infarction. Echocardiography showed mild hypokinesia of the inferior and apical walls of the left ventricle, with an ejection fraction of 40–45%. Acute inferior wall MI was confirmed by serial changes in the ECG and myocardial enzymes, as follows: creatine kinase (CK), 544 U/L (normal range, 30–135 U/L); creatine kinase–MB isoenzyme (CK–MB), 53 U/L (normal range, 0–16 U/L); and troponin-I, 16.11 ng/mL (normal range, <0.5 ng/mL). Emergency coronary angiography (CAG) revealed discrete eccentric stenosis (75%) of the middle portion of the left descending coronary artery and segmental eccentric stenosis (80%) of the distal portion of the right coronary artery. PTCA was performed using a 2.0 mm × 20-mm and a 3.5 mm × 20-mm balloon to dilate the middle portion of the left descending coronary artery and the distal part of the right coronary artery, respectively. The residual stenosis was 25% in the left descending coronary artery and 55% in the right coronary artery. Additionally, a 3.5 mm × 20-mm stent was successfully deployed in the distal portion of the right coronary artery without any residual stenosis. A pre- and post-treatment comparison of these two coronary arteries is shown in Figures 1–4.

The patient's clinical condition improved 3 days after PTCA treatment. Echocardiography showed that the previous regional wall motion abnormalities disappeared and the left ventricular ejection fraction increased to 68%. Eleven days after delivery, the patient was discharged in stable clinical condition. Three months after delivery the patient had no symptom of chest discomfort or dyspnea during her first postpartum menstrual period, and the hepatic and myocardial enzymes were normal.

Discussion

The management of PPH remains one of the significant challenges to obstetricians because PPH causes major maternal morbidity and mortality worldwide. Acute management of PPH includes bimanual compression, medical therapy (uterotonic drugs, such as oxytocin, methylergonovine, misoprostol, and others), conservative surgical procedures (suturing of lacerations and bilateral or unilateral internal iliac artery or uterine artery ligation

Table 1
Summary of clinical course, laboratory results, and treatments.

Postpartum (d)	1	2	3	4	5	6	7	8	9
Blood transfusion (U)									
RBCs	12	8	—	2	—	—	—	—	2
FFP	12	8	4	8	4	4	—	—	—
Platelet	3	2	—	2	—	—	—	—	—
Cryoprecipitate	20	—	10	—	—	—	—	—	—
Clinical manifestation	PPH atarted 1 h after delivery, hemorrhagic shock	Pulmonary edema, ischemic hepatitis, DIC, ARDS	Ischemic hepatitis	Ischemic hepatitis	Improvement of ischemic hepatitis	—	Chest pain, bradycardia	Acute myocardial infarction	—
Treatments	Oxytocin, ergonovine maleate, gauze packing, bilateral internal iliac artery embolization	Blood transfusion with coagulative factor, high pressure ventilation	Blood transfusion with coagulative factor	Blood transfusion with coagulative factor	Blood transfusion with coagulative factor	Blood transfusion with coagulative factor	—	PTCA with balloon dilatation and stent implantation	—
PT (INR)/APTT (s)	5.71/143.4	2.96/49.6	3.14/38.2	3.29/37.5	2.22/35.7	1.81/31.1	1.46/31.4	1.38/28.4	1.32/39
Hct (%)/ Hb (g/dL)	18.3/6.3	22.7/7.7	31.7/11.0	27.4/9.5	26.5/9.0	30.6/10.4	29.5/9.9	29.3/9.9	26.7/8.9
Platelet count ($\times 10^9$/L)	40	63	105	66	117	113	88	77	89
AST/ALT (U/L)	1990/1600	3640/2170	5890/3850	6280/4320	1840/2000	525/1234	163/680	137/482	63/230
CK/CK–MB (U/L)	—/—	—/—	—/—	—/—	—/—	—/—	54/10	544/53	171/27
Troponin-I (ng/mL)	—	—	—	—	—	—	0.14	16.11	7.75

ALT = aspartate aminotransferase; APTT = activated partial thromboplastin time; ARDS = adult respiratory distress syndrome; AST = alanine aminotransferase; CK = creatine kinase; CKMB = creatine kinase-MB isoenzyme; DIC = disseminated intravascular coagulation; FFP = fresh frozen plasma; Hb = hemoglobin; Hct = hematocrit; INR = international normalized ratio; PPH = postpartum hemorrhage; PT = prothrombin time; PTCA = percutaneous transluminal coronary angioplasty; RBCs = red blood cells; U = units.

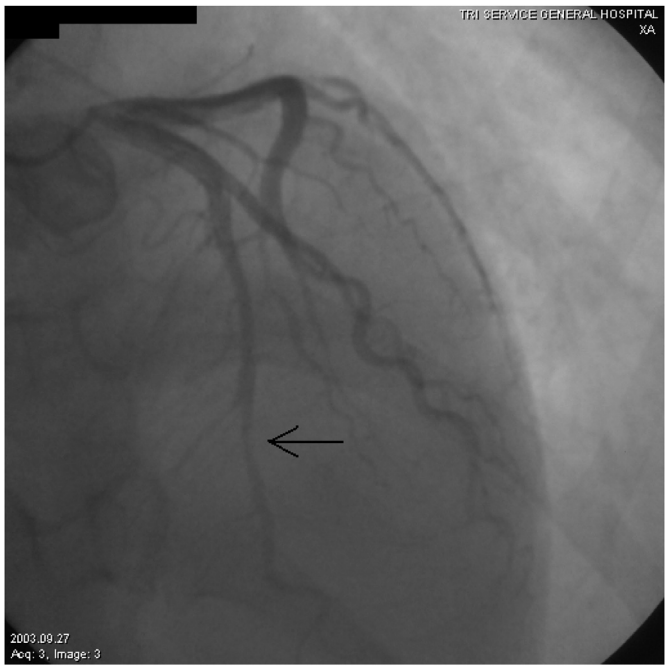

Figure 1. Preoperative condition of left descending coronary artery. A discrete 75% eccentric stenosis was noted over the middle portion of the left descending coronary artery.

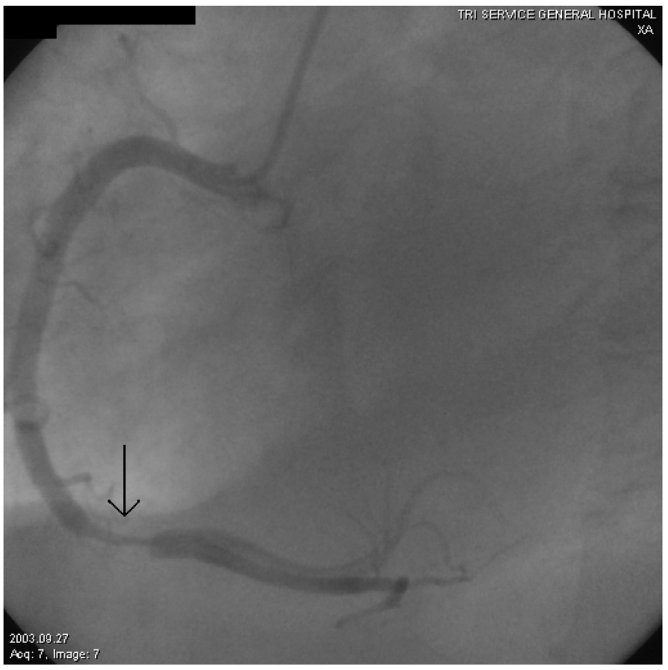

Figure 3. Preoperative condition of right coronary artery. A segmental 80% eccentric stenosis was noted over the distal portion of the right coronary artery.

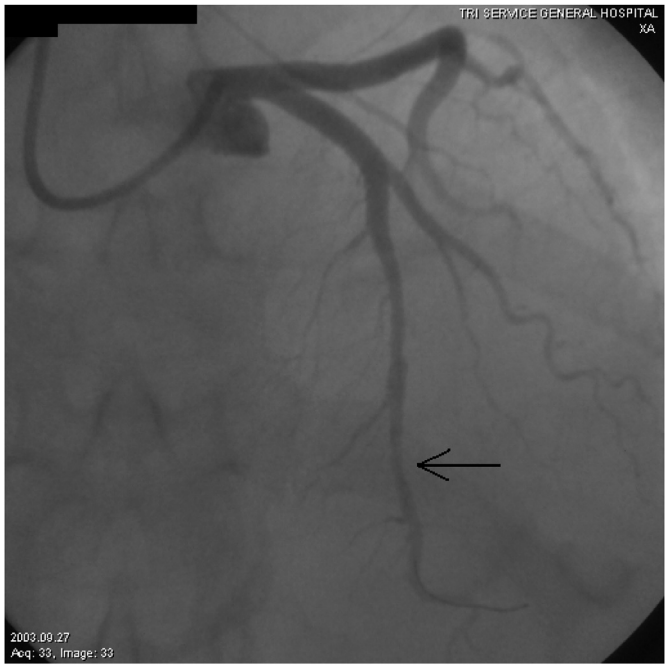

Figure 2. Postoperative condition of the left descending coronary artery. A 2.0 mm × 20-mm balloon was applied to dilate the middle portion of the left descending coronary artery. The residual stenosis is 25%.

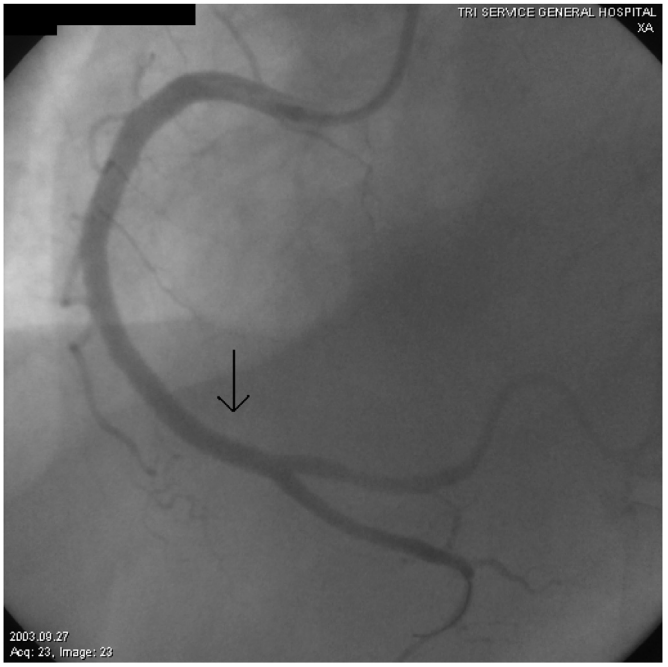

Figure 4. Postoperative condition of right coronary artery. A 3.5 mm × 20-mm stent was deployed at the distal portion of the right coronary artery. There was no residual stenosis.

or embolization), and emergency hysterectomy. An emergency hysterectomy can result in additional significant blood loss and postoperative complications, and have a major psychological impact on the mother [4,5].

There are few reported cases of postpartum MI in the literature. Taylor et al [6] reported the first case of postpartum MI, which was probably induced by ergonovine maleate, a drug known to provoke coronary artery spasm in susceptible patients. Nonetheless, Bateman et al [7] suggested that the increase in the risk of MI after methylergonovine exposure, if present at all, is extremely small. The effect of ergonovine is still controversial. Other possible etiologies of postpartum MI include spontaneous coronary artery dissection, bromocriptine use, antiphospholipid syndrome, and other systemic medical diseases [8–10].

The diagnosis of MI in our patient was based on finding increased levels of myocardial enzymes (CK, CK–MB, and troponin-I), acute ischemic changes in the ECG, typical angina pectoris, and echocardiography showing regional wall motion abnormalities. Our patient had occasional chest tightness, but she had no history of exercise intolerance, dyspnea, radiating chest pain, or previous cardiovascular disease. Furthermore, antenatal checkups showed an otherwise normal pregnancy course. The CAG showed discrete eccentric stenosis (75%) of the middle portion of the left descending coronary artery and segmental eccentric stenosis (80%) of the distal portion of the right coronary artery. However, our patient had no coronary artery disease symptoms. Interestingly, she tolerated the changes in blood volume and cardiac output secondary to the physiologic changes of pregnancy. Administration of ergonovine maleate is contraindicated in hypertensive patients due to the vasoconstrictive effects; however, our patient had no antenatal history of hypertension to indicate withholding the clinical use of ergonovine maleate. In spite of a confounding effect with coronary artery spasm, ergonovine maleate may have been a factor causing the MI in the patient reported here. Another cause of the MI may have been PPH-related hypoperfusion.

The physiology of pregnancy is such that the normal blood volume in a singleton pregnancy increases by up to 60% at term. Thus, once hypotension is present, approximately 1.5 L of blood may have been lost [11]. If hypovolemia is sufficient to result in shock, significant fluid deficiencies must have already existed. Hypovolemia not only leads to decreased cardiac output, but also decreased blood flow in the coronary arteries. The compromised coronary blood flow contributes to the ischemia of the myocardium, which is supplied by the stenotic coronary artery, resulting in myocardial stunning and infarction.

The relevance between PPH and acute myocardial infarction at a 7-day interval is interesting. It is plausible to speculate that vulnerable arteriosclerotic plaques are prone to rupture during stress, dramatic hemodynamic changes, and tachycardia. The narrowing lumen of the distal portion of the right coronary artery may be even more compromised to be obstructed by the wandering plagues.

The liver is a highly vascular organ, receiving approximately 20% of the cardiac output. The rapid and marked elevation of transaminase levels due to an acute fall in cardiac output is referred to a cardiogenic ischemic hepatitis [12]. Ischemic hepatitis may occur in the setting of overt cardiogenic shock in patients without preexisting heart failure, an acute decline in cardiac output in the absence of hypotension if the patient has underlying severe heart failure, or during the course of MI complicated by cardiogenic shock [13]. An important finding of recent studies is that all patients with a clinical diagnosis of ischemic hepatitis have evidence of cardiac disease; most of the patients had evidence of right-sided heart failure and induced hepatic venous congestion. Passive congestion may predispose hepatocytes to hypoxic injury resulting from hypotension [14]. The diagnosis of ischemic hepatitis includes sharp elevation of ALT and AST to at least 20 times the upper limit of the normal range, elevation of lactate dehydrogenase between 10 times and 20 times the upper limit of the normal range, elevation of serum bilirubin, and prolongation of PT. Furthermore, ischemic hepatitis is followed by a >50% decrease in these analytes within 72 hours if the causative disturbance is eliminated. Therapy for ischemic hepatitis is administered to increase or improve cardiac output, blood perfusion of the liver and kidneys, and tissue oxygenation. Ischemic hepatitis is usually self-limiting when it affects the normal liver, but more serious superimposed changes may occur when the liver has been previously damaged. The AST and ALT levels in our patient were elevated to 1990 U/L and 1600 U/L within 8 hours after PPH, respectively. The maximum levels of AST (6280 U/L) and ALT (4320 U/L) occurred on the 3rd day after PPH, then decreased dramatically without hepatic treatment.

Our patient had asymptomatic coronary artery disease antenatally. Nonetheless, severe PPH caused hypovolemic shock. Then, severe pulmonary edema and ischemic hepatitis developed during fluid resuscitation. This might have been a clue that the patient's complications were caused more by heart disease than hypovolemic shock and DIC. Secondly, acute MI developed on the 7th postpartum day. As the patient was in the intensive care unit, there was no possibility of a delayed MI diagnosis. Whether or not acute MI was a coincident occurrence with PPH or was a sequela of PPH remains unclear. When the symptoms of acute MI appeared, PTCA and stent deployment were immediately performed. After PTCA and stent deployment, the patient was transferred to the postpartum ward and discharged in stable condition.

In conclusion, this was a rare case of severe PPH complicated by acute MI and ischemic hepatitis, which were successfully treated. If a patient with hypovolemic shock develops complications of pulmonary edema or ischemic hepatitis, the possibility of cardiovascular disease should be immediately investigated and preventive measures initiated.

Conflicts of interest

The authors have no conflicts of interest relevant to this article.

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