Case Report

Interventional Cardiology

Hepatic cyst rupture: A hidden complication of chest compression

Yusuke Takeda, Hisayoshi Murai*, Soichiro Usui, Kenji Sakata, Masaaki Kawashiri, Masayuki Takamura

Department of Cardiovascular Medicine, Kanazawa University Graduate School of Medical Sciences, Kanazawa, Japan

*Author for correspondence: Dr. Hisayoshi Murai, Department of Cardiovascular Medicine, Kanazawa University Graduate School of Medical Sciences, Kanazawa, Japan, E-mail: sakurasoma1209@yahoo.co.jp

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Abstract

Background: Chest compressions are absolutely essential to save the lives of those in cardiac arrest. However, chest compressions per se may induce certain complications. Here, we present a case of hepatic cyst rupture induced by chest compressions after ventricular fibrillation developed during percutaneous coronary intervention (PCI).

Case presentation: A 74-year-old male was admitted for PCI. Earlier computed tomography (CT) had incidentally revealed a large hepatic cyst. During PCI, ventricular fibrillation developed and the patient required cardiopulmonary resuscitation. Sinus rhythm recovery did not improve the hemodynamic stability. CT unexpectedly revealed an abdominal hematoma and extravasation of contrast medium from the ruptured hepatic cyst, causing hemorrhagic shock. We performed transcatheter arterial embolization, which stabilized the hemodynamics.

Conclusion: Chest compressions must be delivered carefully to patients at risk of hepatic cyst rupture. Careful consideration of adjacent organs around heart is required for prompt diagnosis and treatment to prevent serious shock.

Keywords: Hepatic cyst rupture ■ Complication of chest compression ■ Hemorrhagic shock Cardiopulmonary resuscitation ■ Triple therapy ■ Transcatheter arterial embolization

Abbreviations

PCI: Percutaneous Coronary Intervention; CT: Computed Tomography; CPR: Cardiopulmonary Resuscitation; LAD: Left Anterior Descending Artery

Background

Life-threatening cardiac arrhythmias are infrequently encountered in association with Percutaneous Coronary Intervention (PCI). During cardiopulmonary resuscitation (CPR), chest compressions are essential for survival. Although such compressions sometimes cause traumatic injuries, deeper compressions increase the success rate of spontaneous circulatory return [1,2]. Hepatic cysts are common benign lesions [3] that are usually asymptomatic, being discovered incidentally on ultrasonography and Computed Tomography (CT). Intraperitoneal cyst rupture is a rare complication. Most ruptures are caused by trauma, although spontaneous ruptures have been reported [4]. Here, we present a case of hepatic cyst rupture after chest compressions were delivered when ventricular fibrillation developed during PCI. We found no report on hepatic cyst rupture following chest compressions in the literature; our case is thus important.

Case Presentation

A 74-year-old male was admitted for PCI. His family history was unremarkable. Four months before admission he had undergone surgical thrombectomy to treat an acute embolic occlusion of the right brachial artery. At that time, an electrocardiogram and an echocardiogram revealed atrial fibrillation, mild enlargement of the left ventricle, reduced motion of the anterior septum, and an ejection fraction

of 40%. Anticoagulation therapy with 60 mg/day edoxaban, a β-blocker, and an angiotensin-converting enzyme inhibitor were prescribed. Incidentally, a large hepatic cyst was detected in the lateral segment of the liver that lay close to the heart (across the diaphragm) by computed tomography (CT) (Figure 1). One month before admission, routine coronary angiography was performed to explore the etiology of left ventricular dysfunction. Stenosis was detected in the mid-left anterior descending artery (LAD) that was attributable to the left ventricular dysfunction (Figure 2). Thus, staged PCI was scheduled; we planned to address the stenosis. Dual antiplatelet therapy (aspirin and clopidogrel in addition to the direct oral anticoagulant; so-called 'triple therapy') was commenced.

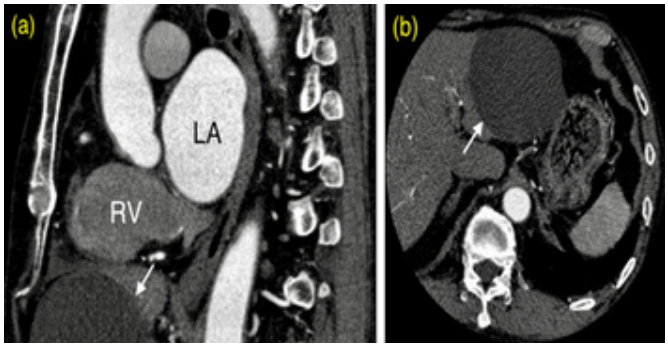

Figure 1: Computed tomography revealed a hepatic cyst (7 cm in diameter) in the lateral segment of the liver (arrow) 1a: Sagittal plane; 1b: Axial plane. (LA: Left Atrium, RV: Right Ventricle).

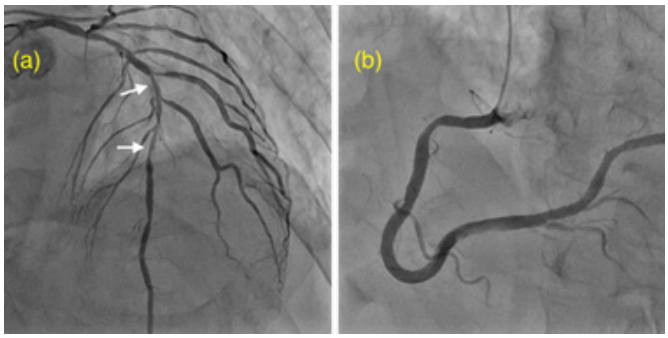

Figure 2: 2a: Coronary angiography revealed a 90% stenotic lesion in the left anterior descending artery (arrows); 2b: A mild stenotic lesion in the right coronary artery.

On admission, the patient's blood pressure was 126/76 mmHg, his heart rate was 62 beats/min, and his SpO2 was 98% on room air. The conjunctiva showed no sign of anemia or jaundice. The first and second heart sounds were normal. A pan-systolic murmur was evident at the cardiac apex (Levine 2/6). No lung rales were audible. The abdomen was flat and soft; the liver was not palpated. We noted no edema. As shown in the Table 1, laboratory tests revealed mild renal dysfunction and an elevated level of brain natriuretic peptide. There was no sign of an inflammatory response, anemia, or liver dysfunction. A chest X-ray revealed a cardiothoracic ratio of 56.9% and no evidence of pleural effusion. The electrocardiogram revealed atrial fibrillation, poor R-wave progression in V1–V3, and ST depression in V4–V6 (Figure 3).

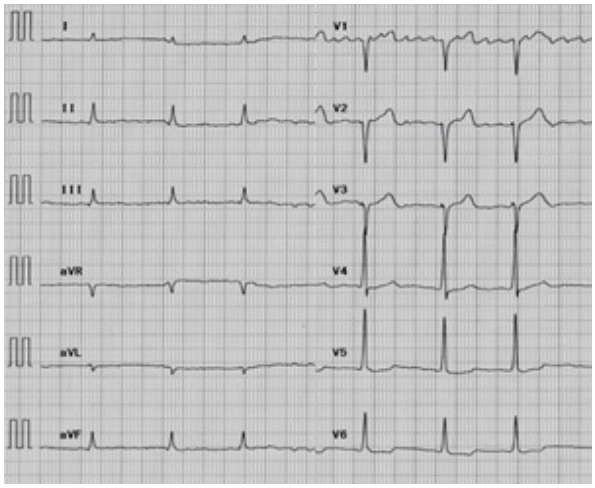

Figure 3: Electrocardiogram performed on admission revealed atrial fibrillation, poor R-wave progression in V1–V3 and ST depression in V4–V6.

Table 1: Laboratory data on admission.

Laboratory data			
White blood cell(/μL)	5520	CRP(mg/dL)	0.11
Hemoglobin(g/dL)	15.3	CK(IU/L)	168
Platelet(× 10^3/μL)	249	ALP(IU/L)	167
PT(sec)	17.2	γ-GTP(IU/L)	41
APTT(sec)	35.5	AST(IU/L)	20
Fibrinogen(mg/dL)	358	ALT(IU/L)	22
Glucose (mg/dL)	118	LDH(IU/L)	162
Hemoglobin A1c(%)	6.7	T-Bil(mg/dL)	0.5
Sodium(mEq/L)	140	TP(g/dL)	7
Potassium (mEq/L)	5	T-Cho(mg/dL)	156
Chlorine(mEq/L)	104	LDL-Cho(mg/dL)	96
BUN(mg/dL)	17	HDL-Cho(mg/dL)	39
Creatinine(mg/dL)	0.99	Triglyceride(mg/dL)	107
		BNP (pg/mL)	191.9

*PT: Prothrombin time; APTT: Activated Partial Thromboplastin Time; BUN: Blood Urea Nitrogen; CRP: C-Reactive Protein; CK: Creatine Kinase; ALP: Alkaline Phosphatase; γ-GTP: γ-Glutamyl Transpeptidase; AST: Aspartate Transaminase; ALT: Alanine Transaminase; LDH: Lactate Dehydrogenase; T-Bil: Total Bilirubin; T-Cho: Total Cholesterol; LDL-Cho: LDL- Cholesterol; HDL-Cho: HDL- Cholesterol; BNP: Brain Natriuretic Peptide

On hospital day 3, we performed PCI. A 7-Fr sheath was inserted into the right femoral artery and 8,000 U of heparin sodium were intravenously injected. After predilation with a 2.0 mm balloon at nominal pressure, a 2.5 mm zotarolimus-eluting stent was implanted at nominal pressure. Immediately after implantation, the patient complained of chest pain. Although isosorbide dinitrate

was injected into the left coronary artery, coronary angiography revealed severe stenosis at the distal end of the stent, and coronary vasospasm (Figure 4). Suddenly, the electrocardiogram reported ventricular fibrillation. First, we performed CPR while we prepared for direct current cardioversion; 150-J direct-current cardioversion converted the ventricular fibrillation into a normal sinus rhythm. The angiographic stenosis in the LAD resolved, but the patient's blood pressure indicated shock. An intra-aortic balloon pump was immediately inserted, but the blood pressure did not recover. We suspected occult bleeding; abdominal CT revealed a very small intra-abdominal hematoma and hepatic cyst rupture (Figure 5a). We felt that this explained the fall in blood pressure. We first employed conservative treatment; we discontinued the heparin infusion. However, the patient became gradually hypotensive and required large volumes of intravenous fluids and catecholamine. Enhanced CT of the abdomen revealed that the hematoma had grown; contrast medium was extravasated from the ruptured hepatic cyst (Figure 5b). We performed emergency angiography and transcatheter arterial embolization. CT angiography demonstrated extravasation from small arterial branches suppling the hepatic cyst (Figure 6). We embolized these arteries using gelatin sponges; the hemodynamics became stable. The hematoma gradually reduced and was barely visible after 1 week. Three weeks after the event, the patient was discharged in good health.

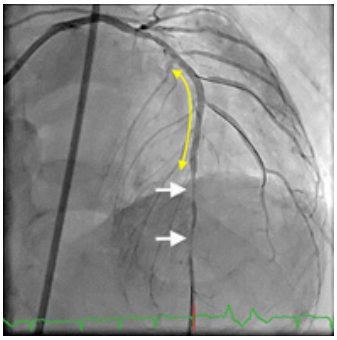

Figure 4: Coronary angiography revealed that stenosis (white arrows) developed after implantation of a drug-eluting stent (yellow arrow).

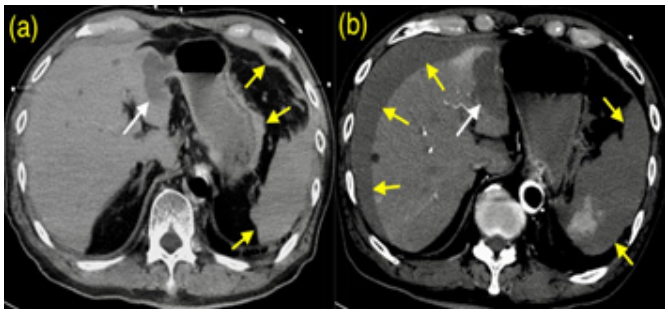

Figure 5: 5a: Plain abdominal computed tomography (CT) performed after percutaneous coronary intervention (PCI); 5b: Enhanced abdominal CT performed 4 hours after PCI. The hepatic cyst had ruptured (white arrows) and a hematoma developed rapidly (yellow arrows).

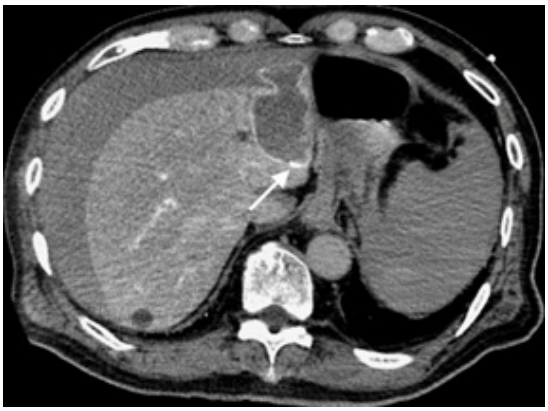

Figure 6: Computed tomography angiography revealed extravasation from the subcapsular artery of the hepatic cyst (arrow).

Discussion

Simple hepatic cysts are common benign lesions; the population frequency is about 2.5% [3]. The cysts are usually asymptomatic, being incidentally discovered during abdominal ultrasonography or CT. Rarely, cysts cause abdominal symptoms via the compression of adjacent organs. Asymptomatic hepatic cysts do not require treatment. If a cyst is symptomatic, percutaneous aspiration, sclerosis with alcohol or minocycline, or laparoscopic deroofing, is performed [5-7].

Hepatic cyst rupture is a rare complication, triggering sudden abdominal pain. Rupture may be caused by trauma or infection, but it is sometimes spontaneous [4]. No definitive treatment is available. Earlier case reports employed conservative strategies, including abdominal drainage, surgery, and transcatheter arterial embolization [8,9]. In our present case, the abdominal hematoma grew rapidly and the hemodynamic status became unstable; intervention was thus required. As CT revealed extravasation of the contrast medium, and as the effects of triple antiplatelet therapy persisted, we chose transcatheter arterial embolization, not surgical intervention. In our case, triple heparin antiplatelet therapy may have contributed to the hemorrhagic shock; hepatic cyst rupture rarely induces such shock. On the other hand, there are some cases that hepatic cysts induced cardiac compression and caused hemodynamic instability [10,11]. From these cases and our case, the heart and the liver are in proximity and an abnormality in one may affect the other.

Patients in cardiopulmonary arrest require CPR. The European Resuscitation Council and the American Heart Association have published relevant guidelines. Pressure should be applied to the lower half of the breastbone; it is important to avoid compressing the upper abdomen or the bottom of the bony sternum [1,2]. However, CPR-related complications have been described [12,13].

Abdominal complications develop in about 30% of those who undergo CPR [14]. In our case, it is likely that the hepatic cyst was injured via chest compression slightly caudal to the sternal bone. As a 7-cm-diameter hepatic cyst was noted prior to PCI, we should have performed chest compression in a more superior position. However, the placement of the X-ray tube and plate detector during PCI disturbed the number of sites available for chest compression.

Conclusion

We experienced a case of hepatic cyst rupture caused by chest compressions during PCI. Such compressions increase the risk of hepatic cyst rupture. Careful consideration of adjacent organs around heart is required for prompt diagnosis and treatment to prevent serious cardiogenic shock and secondary hemorrhage.

Conflicts of Interest

The authors declare that they have no conflict of interest.

Acknowledgements

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