· 临床病理讨论 ·

Clinicopathological Conference

Left ventricular free wall rupture after acute myocardial infarction

(The 21th case)

Heart center, Beijing Chaoyang Hospital

Case presentation

A female patient, 69 years old, retired worker, was hospitalized for retrosternal distress for 16 hours. Her onset of symptom began from activities accompanied by gradual dyspnea. The patient denied history of coronary heart disease but had a history of hypertension. Physical examination on admission: body temperature 36.5℃, pulse rate 110 beats/min, respiratory rate 27 /min, blood pressure 90/60mmHg. The heart rhythm was regular with first heart sound dull and low-pitched. No cardiac murmur was found, while diastolic gallop at apex cordis and large amount of moist rales in both lungs could be detected. Cardiac enzymes were elevated and electrocardiography showed 0.1 — 0.3 mV ST elevation and Q-wave formation in anterior and high lateral leads. She was diagnosed of extensive anterior-wall acute myocardial infarction (AMI) with cardiac function Killip Ⅲ. She was not given early reperfusion treatment including emergent percutaneous coronary intervention(PCI) and thrombolysis because the time interval between onset of disease and admission had exceeded 12 hours and the symptoms had disappeared. She was treated with medication, including anticoagulation and antiplatelet drugs and nitroglycerin in coronary care unit(CCU). Platelet glucoprotein Ⅱb/Ⅲa receptor inhibitor was not administered. Intra-aortic balloon counterpulsation (IABP) was used immediately at bedside. Echocardiography indicated anteroapical motion weakened or disappeared and a ventricular aneurysm at apex cordis was detected. The patient's course through the next 5 days was uneventful. Counterchecked electrocardiography showed sustained ST elevation in anterior leads. Blood pressure and heart rate became stable and moist rales in both lungs reduced significantly after medication. She received staged coronary angiography(CAG) on the sixth day after onset. It showed a severe stenosis in the proximal left anterior descending coronary artery and the vessel was completely patent with forward blood flow grade TIMI 3. The rest of the epicardial coronary arteries was normal. Sudden respiratory arrest, loss of consciousness and subsequent hypotension happened when left coronary angiography was performed. Orotracheal intubation and artificial respiration was established immediately. Heart rate decreased soon. Sinus bradycardia was seen on monitor screen. Adrenalin 1mg was injected intravenously 5 times. Blood pressure and large artery pulsation disappeared. Dopamine 3mg was injected intravenously and then dopamine 120mg added in 250ml 5% glucose injection was infused. Sustained chest compression was performed . However, blood pressure did not recover and the heart rate decreased progressively. Electromechanical dissociation was then confirmed. Cardiac figure was found enlarged under cine images and the pulsation of the heart disappeared. An urgent bedside echo-

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作者单位:100020 北京市,首都医科大学附属北京朝阳医院心脏中心

作者简介:王乐丰,男,1962年3月生,江西省广丰县人,医学博士,主任医师,朝阳医院心脏中心副主任,导管室主任。Tel:010-85231170

cardiogram showed akinesia of heart wall, and a low echodense pericardial effusion suggested left ventricular free wall rupture. The patient soon died after unsuccess of cardiac-pulmonary resuscitation.

Clinicopathological discussion

Dr. ZHANG Yuan: The patient present here was an elderly woman of 69 years old. She was hospitalized for retrosternal distress for 16 hours. Diagnosis of extensive anterior-wall AMI was established by elevated cardiac enzymes and electrocardiography display, with cardiac function Killip Ⅲ. Her onset of symptom began from activities accompanied by gradual dyspnea. The patient had no history of coronary heart disease, diabetes mellitus or hyperlipidemia but a history of hypertension. Current myocardial infarction was her first cardiac event without preceding angina. Was her myocardial rupture induced by CAG performance?

Dr. YU Liping: The present patient was diagnosed of extensive anterior-wall AMI confirmed by ST elevation and Q-wave formation in anterior and high lateral leads and elevated cardiac enzymes on admission. The patient was in acute distress on admission with mild to numerous moist rales in both lungs; heart rhythm was regular; electrocardiography indicated sinus tachycardia; first heart sound was dull and low-pitched ; no heart murmur but diastolic gallop at apex cordis was detected. She was complicated with acute pulmonary edema. She was not given early reperfusion treatment including PCI and thrombolysis because of overtime and disappearance of chest pain. She was treated with medication including anticoagulation and antiplatelet drugs and nitroglycerin in coronary care unit(CCU). Platelet glucoprotein Ⅱb/Ⅲa receptor inhibitor was not administered. Intraaortic balloon counterpulsation (IABP) was used immediately at bedside. Counterchecked electrocardiography showed sustained ST elevation in anterior leads. Echocardiography indicated anteroapical motion weakened or disappeared and a ventricular aneurysm at apex cordis was detected. Might she avoid myocardial rupture if early reperfusion therapy such as emergent PCI had been adopted after admission?

Dr. LIU Yu: Myocardial rupture occurs in 10% of AMI. LVFWR is rare but lethal form of myocardial rupture and is associated with rapid hemodynamic collapse. LVFWR occurs always during the first 24 hours to 3 weeks after an AMI. In the first week of AMI, myocardium is in the stage of pathological malacia. The heart is prone to rupture in this stage because fibrosis and cicatrization process of infarct area are insufficient. The heart rupture usually lies at the juncture between normal tissue and infarct area. It was reported that older age, female sex, previous hypertension, and an anterior-wall AMI for the first time were the risk factors for LVFWR. Physical exercise and hypertension are common inducing factors. The present case was at high risk for myocardial rupture for her clinical characteristics.

Dr. XU Li: Transmural infarction of left ventricular free wall is prone to rupture in anatomy, especially infarction of anterior and lateral wall and apex cordis. Blood supply for these areas comes from the terminal branches of left descending coronary artery(LAD) with few collaterals. The present patient was an elderly woman of 69 years old with a history of hypertension. Her symptom of retrosternal distress had lasted 16 hours before admission. She had missed optimal reperfusion time. Coronary angiography (CAG) on the sixth day showed single vessel disease and the criminal artery was patent. Myocardial rupture is rare but lethal complication of AMI. Such patients often die of refractory cardiac failure, cardiogenic shock or malignant arrhythmia in a very short time.

Dr. CHEN Mulei: The present patient showed typical signs of cardiac rupture during CAG performance indicated by sudden respiratory arrest, loss of consciousness and continuous bradycardia. As far as the present case was concerned, LVFWR was confirmed by echocardiography. Echocardiography is considered as the best method for identifying LVFWR. Echocardiography has a diagnostic sensitivity of 100% and a specificity of 93%.

The main echocardiographic evidence is pericardial effusion and intrapericardial low echodense. The actual tear itself can seldom be seen.

Dr. GE Yonggui: Orotracheal intubation and artificial respiration was established immediately when this patient was found to have sudden respiratory arrest and subsequent hypotension. Heart rate decreased soon. Adrenalin 1mg was injected intravenously 5 times. Blood pressure and large artery pulsation disappeared. Dopamine 3mg was injected intravenously and then dopamine 120mg added in 250ml of 5% glucose injection was infused. Sustained chest compression was performed. Blood pressure still could not be measured, heart rate decreased continually, electromechanical dissociation was then confirmed. We were aware of cardiac rupture immediately. LVFWR was confirmed by echocardiography with finding of profuse pericardial effusion. Such patients have considerable tear of heart and are always too late to be rescued. It was reported that early percutaneous intervention can not reduce incidence of myocardial rupture after AMI.

Dr. WANG Lefeng: Myocardial rupture was considered rapidly when sudden respiratory arrest and electromechanical dissociation were found, still accompanied by loss of consciousness and large artery pulsation. Rupture was later confirmed by echocardiogram. Rapid fluid infusion, administration of positive inotropic agents, and pericardiocentesis may be useful but are temporary measures after prompt diagnosis of myocardial rupture. The definitive treatment for LVFWR is emergent surgical repair. Either biological glue or epicardial sutures can be used. Other surgical techniques include infarctectomy with patch placement and ventricular wall reconstruction. However, only a few patients can undergo operation before death. The role of IABP support in patients with LVFWR is not clear. In the present case, IABP was implanted without delay from lack of emergent PCI. The pump did improve her early hemodynamic status by reducing both afterload and wall tension, but the patient still underwent lethal LVFWR. Primary percutaneous intervention might not prevent myocardial rupture after AMI. Patency of her criminal artery was speculated on admission. Myocardial rupture might be coincident with CAG performance.

Dr. YANG Xinchun: As far as the present case was concerned, LVFWR was coincident with CAG performance. For the patients characterised by female, over 60-year old, and infarction for the first time, especially extensive anterior wall infarction, we should be on the alert for the danger of cardiac rupture. In our opinion, IABP support is helpful for hemodynamic stabilization but might not prevent cardiac rupture after AMI. Early opening of the criminal vessel seems not crucial to prevent LVFWR after AMI. Cautious medical therapy with absolute bed rest, adequate control of blood pressure, sedation and analgesia are likely to contribute to patient's survival through early weeks to safe phase. Optimal intervention of coronary artery should not be decided until fibrosis and cicatrization process of infarct area are sufficient.

(Translator: WANG Lefeng)

69岁老年女性急性心肌梗死后心脏破裂一例报道

1 病例摘要

患者 女性，69岁，退休，以胸闷 16h 入院。患者活动后发病，逐渐出现呼吸困难。否认既往冠心病病史，合并高血压。入院时查体体温 36.5℃，心率 110 次/min，呼吸 27 次/min，血压 90/60mmHg。精神差，心律齐，心音低钝，各瓣膜区未闻及杂音。心尖部可闻及舒张期奔马律，双肺可闻及中到大量湿啰音。急诊心电图：窦性心动过速，前壁及高侧壁导联 ST 段抬高 0.1～0.3mV，Q 波形成。心肌酶升高。诊断急性广泛前壁心肌梗死。心功能 KillipⅢ级。因为发病距入院就诊已超过 12h 且症状消失，

所以未行包括急诊经皮冠状动脉介入(percutaneous coronary intervention，PCI)和溶栓在内的紧急再灌注治疗策略。入住心脏病监护病房(cardic care unit，CCU)后立即开始应用药物扩张冠状动脉、抗凝、抗血小板聚集，纠正左心功能不全等治疗。未应用Ⅱb/Ⅲa受体拮抗剂。于床旁植入主动脉内球囊反搏(intra-aortic balloon pump，IABP)。超声心动图：心室前壁节段性运动障碍，心尖部室壁瘤形成。随后5d患者病情平稳，复查心电图显示胸前导联ST段持续抬高。经过药物治疗后心率、血压平稳，双肺湿啰音明显减少。第6天行选择性冠状动脉造影，结果显示单支病变，左前降支近端90%狭窄，未见血栓，血管完全通畅，前向血流TIMI 3级。术中突然呼吸骤停，意识丧失，血压测不出，给予气管插管，人工辅助呼吸，心率变慢，心电监护显示窦性心动过缓。给予肾上腺素5mg分次静脉注射，血压测不出，大动脉搏动消失。给予多巴胺3mg静脉注射，并予5%葡萄糖250ml内加入120mg多巴胺静点维持。持续胸外按压。血压仍然测不出，心率进行性减慢，电机械分离。X线透视下可见心影增大，心脏搏动消失。即刻行心脏超声检查显示室壁运动消失，心包腔大量积液，心脏游离壁破裂。抢救无效而死亡。

2 临床与病理讨论

张媛医师：本例特点为老年女性，以突发胸闷16h入院，患者活动后发病，逐渐出现呼吸困难。经查心电图和心肌酶明确诊断为冠心病急性广泛前壁心肌梗死，心功能KillipⅢ级。既往有高血压病史，而无糖尿病、吸烟、高脂血症等危险因素。否认既往冠心病病史。首次发生心肌梗死，无梗死前心绞痛。患者心脏破裂是否与冠状动脉造影有关？

于丽萍主治医师：本患者入院诊断明确，前壁及高侧壁导联ST段抬高0.1～0.3mV伴Q波形成，心肌酶升高，诊断急性广泛前壁心肌梗死。患者入院时精神差，双肺可闻及中到大量湿啰音，心律齐，心电图显示窦性心动过速。心音低钝，各瓣膜区未闻及杂音。心尖部可闻及舒张期奔马律。急性广泛前壁心肌梗死同时合并急性肺水肿。心功能不全。因发病距入院就诊已经超过最佳干预时间，且胸痛症状消失所以未行包括急诊PCI和溶栓在内的紧急再灌注治疗策略。入住CCU后立即开始应用药物扩张冠状动脉、抗凝、抗血小板聚集，以及纠正左心功能不全等治疗。未应用Ⅱb/Ⅲa受体拮抗剂。于床旁植入IABP。多次复查心电图显示胸前导联ST段持续抬高。超声心动图：心室前壁节段性运动障碍，心尖部室壁瘤形成。如果入院后尽快行急诊PCI治疗，是否可以避免心脏破裂的发生？

刘宇主治医师：文献报道，心脏破裂占急性心肌梗死死亡原因的10%。左心室游离壁破裂在心脏破裂中较为少见，但非常致命，常常迅速引起循环衰竭。破裂可发生在心肌梗死起病后的1d至3周。急性心肌梗死第1周为病理软化期，梗死区的纤维化和瘢痕修复不完善，最易产生心脏破裂。破裂通常发生在正常心肌与梗死组织交界处，左室游离壁破裂常导致心包积血，病人死于心脏压塞。60岁以上老年患者、女性、首次发生急性透壁性心肌梗死的患者及前壁心肌梗死者发生率高。体力活动和高血压是心脏破裂的常见诱发因素。本例患者为69岁老年女性、首次发生急性透壁性心肌梗死，梗死部位为前壁，是发生心脏破裂的高危患者。

徐立主治医师：解剖学上，心脏破裂多发生在左室透壁梗死，尤其是前壁、侧壁、心尖部，该部位接受左冠状动脉前降支终末分支供血，侧支循环少。本例为一首次发生心肌梗死的69岁女性患者，既往有高血压病史。入院时距发病已16h，症状消失，因此未接受再灌注治疗。发病后第6天行冠脉造影检查：单支病变，罪犯血管为前降支，已开通。心脏破裂是急性心肌梗死的一种严重并发症，临床发生率较低。一旦出现，短期内迅速出现顽固性心衰、心源性休克及恶性心律失常而死亡。

陈牧雷副主任医师：本例患者在术中突然发生呼吸骤停，意识丧失，心率尚能维持，但逐渐变慢，这是急性心脏破裂较典型的临床过程。即刻行心脏超声检查显示心包腔大量积液，证实心脏游离壁破裂。心脏超声是目前确诊心脏游离壁破裂的最佳最便捷的检查方法，敏感度100%，特异度93%。超声影像学的主要依据是心包腔内发现低回声积液，偶尔可以见到游离壁破口。

葛永贵副主任医师：患者术中突发呼吸骤停，血压下降和心率变慢，立即给予气管插管，人工辅助呼吸，给予肾上腺素5mg分次静脉注射，血压测不出，大动脉搏动消失。给予多巴胺3mg静脉注射，并予5%葡萄糖250ml内加入120mg多巴胺静点维持。持续胸外按压。血压仍然测不出，心率进行性减慢，电机械分离。当时就考虑到可能发生心脏破裂。即刻行心脏超声检查显示心包腔内大量积液，证实心脏游离壁破裂。此类患者心脏破裂口较大，往往来不及抢

救。文献报道显示，急性心急梗死后心脏破裂的高危患者尽早开通冠状动脉并不能避免心脏破裂的发生。

王乐丰主任医师：患者术中突发呼吸骤停，意识丧失，大动脉搏动消失，电机械分离。考虑到可能发生心脏破裂。而且经心脏超声确诊。一旦确诊，可立即心包穿刺抽液，静脉快速输液。但均为临时措施，最有效的治疗方法为紧急外科修补术。生物胶和心脏外膜缝合的方法均可采用，其他有效的外科技术手段还包括切除梗死区、放置补片，心室壁重建等。然而，仅有少数患者能够生存到接受外科手术的那个时刻。本例患者未行急诊 PCI，但是入院后立即置入 IABP。IABP 的应用通过减轻后负荷和室壁张力改善了患者的早期血流动力学状态。但是患者仍然发生了致命性的心脏破裂。对于早期行急诊 PCI 开通罪犯血管能否防止或降低心肌梗死后的心脏破裂目前存在争议。实际上，本例入院后症状消失，可能罪犯血管在那时已经开通。但是行冠脉造影时突然发生心脏破裂，可能与搬动，患者情绪紧张有关。

杨新春教授：从本例来看，为心脏破裂时机与冠状动脉造影操作巧合。对于年龄≥60 岁、女性、首次发生、特别是广泛前壁梗死的患者，应该高度警惕心脏破裂。应用 IABP 有助于血流动力学参数的稳定。然而 IABP 和早期开通罪犯血管可能不能防止心肌梗死后的心脏破裂。应绝对卧床休息、镇静止痛、控制高血压、防止大小便用力，平稳度过心脏破裂危险期，等待心肌瘢痕修复完善再选择最佳冠脉干预策略。

（参加讨论医师：张媛、于丽萍、刘宇、徐立、陈牧雷、葛永贵、王乐丰、杨新春）

（王乐丰、刘宇、杨新春整理）

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作为指标来评估诊断中心性肥胖的腰围切点，结果显示男性腰围切点为 90cm，女性为 80cm，与 IDF 的建议相符合。该研究是一项横断面人群研究，可以作为城市中老年人的代表人群，因此统计结果适用于中国城市中老年人群。目前诊断中心性肥胖的腰围切点还存在不同意见，尚需进一步的前瞻性研究来分析腰围与心血管疾病发生率和死亡率的关系等，从而制定适用于我国人群的与 MS 密切相关的腰围切点。

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