· 临床病理讨论 ·

Clinicopathological Conference (the 50th case)

Acute anterior wall myocardial infarction complicated by cardiogenic shook in an elderly female patient

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【CLC number】 R542.2+2 【Document code】 A 【DOI】 10.3724/SP.J.1264.2012.00016

Case presentation

A 73-year-old female was admitted into the Institute of Geriatric Cardiology, Chinese PLA General Hospital because of sudden chest pain accompanied with nausea and vomiting for 15 hours. At 3:00 on August 14th, 2011, the patient suddenly suffered from severe chest pain accompanied with perspiration, nausea, vomiting, and cold extremities, but she was under normal conscious level. In the emergency room, electrocardiogram(ECG) at 18:00 showed ST segment elevation on the precordial leads. Cardiac biochemical markers increased proportionally. The patient had a history of hypertension for 10 years which was poorly controlled, chronic bronchitis for several years, cerebral ischemia attack one month ago, and diarrhea one day before admission. She denied any history of smoking, alcohol drinking, or illicit drugs use.

On admission examination, the blood pressure was 90/70mmHg and the respiratory rate 20 breaths per minute. Both lungs were clear with moist rales. Heart rate was 133 beats per minute. Percussion revealed a dull sound. A grade 2-3/6 diastolic murmur could be heard at the left second intercostal space(ICS). Dorsalis pedis artery pulse was weak. Transthoracic echocardiogram showed a left ventricular ejection fraction (LVEF) of 43%, a normal-size left ventricular cavity, moderate dysfunction involving the septal and anterior, lateral and apex segments of the midportion, the thinned left ventricle, and ventricular aneurysm at the apex. Chest radiogragh showed pulmonary infection.

Considering the diagnosis of acute ST-elevation myocardial infarction (STEMI) and cardiogenic shock, the patient received emergency intra-aortic balloon bumping(IABP) implantation and percutaneous coronary intervention(PCI). Cardioangiography (CAG) showed occlusion of the left anterior descending (LAD) artery right after the first diagonal opening. There was poor collateral flow to the distal LAD artery. The ostial diagonal branch presented a localized stenosis of about 90%. The left circumflex coronary artery and its branches appeared to be free of obstruction. Considering LAD as the culprit vessel, one stent was implanted in the middle LAD.

Medication therapy was administrated, including anti-platelet, anti-coagulation, nitrate, statin, liver and renal protection, anti-infection and nutritional support. Diuretics were given intermittently to control the input and output. Dopamine was given intravenously to maintain the blood pressure. Because of her poor heart function, high heart rate and frequent ventricular premature beats, cedilanid and amiodarone were also administrated. On August 16th, tracheal cannula placement and mechanical ventilator support were performed as a result of repeated acute left heart failure, pulmonary infection and liver and renal dysfunction. Sodium nitroprusside, imipenem and cilastatin were administrated, while amiodarone stopped. Intake and output were also controlled. After all these treatment, the liver and renal function improved, intermittent respirator suspension training went smooth and heart function improved. The platelet account presented a slight decrease, and IABP balloon induced platelet injury was highly suspected. Tracheal cannula and IABP were removed on the 22nd and 24th in sequence. Consequentially, respiratory rate increased later, accompanied with continuous low-blood-pressure state which even high dose of dopamine could barely correct. On the 26th, acute left heart failure attacked. ECG monitor suggested junctional rhythm and frequent ventricular premature. The patient lost consciousness. Digitalis intoxication was highly suspected. Laboratory examination showed serum level of digoxin ＞4ng/dl, confirming digitalis intoxication. Digitaloid medications were forbidden immediately. Tracheal intubation and IABP implantation were performed for the second time. Epinephrine, norepinephrine and high dose of dopamine, *etc*, were all given to keep the blood pressure stable. Respiratory acidosis was corrected after so-

收稿日期：2009-12-11；修回日期：2010-10-12
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dium bicarbonate intravenous administration. The patient regained consciousness. Between November the 6th and November the 16th, repeated intermittent ventilator weaning all failed and IABP counterpulsation failed to maintain the blood pressure. Heparin-induced thrombocytopenia (HIT) developed, so did continuous hyperpyrexia, pulmonary infection aggravation and frequent ventricular premature. The patient was under a severe condition persistently. At 19:00, November the 17th, 2011, blood oxygen saturation suddenly dropped to 50%-70%, heart rate 140-150 beats per minute, blood pressure 70/50mmHg. Pure oxygen was given, accompanied with epinephrine and bicarbonate injection. Her family members rejected the rescue treatment with cardiopulmonary resuscitation (CPR) and electric defibrillation, the patient was confirmed dead at 19:40.

Clinical discussions

Dr. HOU Yuntian: This elderly female was diagnosed with acute STEMI in the anterior wall and cardiogenic shock. Considering the hospital admission delay, together with liver and kidney dysfunction, as well as pulmonary infection, the patient was in severe condition and had a great risk of death. As for treatment, the following points should be considered. (1) Digitalis intoxication. Regarding the cardiac dysfunction, higher heart rate and frequent ventricular premature, digoxin and cedilanid were administrated. Because digitalis was in high dose, the plasma concentration should be monitored frequently. In this case, ECG revealed junctional rhythm, which was highly due to digitalis intoxication. (2) Infection. This patient presented with cardiogenic shock, a long history of bed rest, poor nutritional status, weak cough, tracheal intubation, previous history of chronic bronchitis. In addition of IABP implantation, deep-vein catheterization and urinary catheter placement, she was in a great risk for catheter associated infection. The patient′s body temperature, blood routine test, chest radiogragh, clinical signs, especially the result of sputum culture should all be taken into consideration in antibiotics application.

Dr. XUE Qiao: Multi-organ dysfunction is a common problem in AMI patients after PCI, mostly according to illness progression and iatrogenic reasons. In this case, cardiogenic shock had been present before admission. With a low-blood-pressure state for several hours, hypoperfusion developed, which manifested as paleness, cold extremities, weak dorsal pedis artery pulse, hepatic congestion, pulmonary venous congestion, and renal dysfunction. All these suggested severe multi-organ dysfunction which led to a poor prognosis. Clinical doctors should pay much more attention to iatrogenic liver injury, acute kidney injury (AKI), digitalis intoxication and HIT. Liver injury was usually associated with liver disease history, cardiac dysfunction, congestive & ischemic hepatopathy and iatrogenic liver injury(intravenous administration of amiodarone). Acute kidney injury can be induced not only by hypoperfusion, but also by radiographic contrast application. Patient with repeated left heart failure usually received high dose of digitaloid drugs orally and intravenously, so digitalis intoxication should be carefully monitored by close observation of clinical symptoms, electrocardiogram and plasma digoxin concentration. For a post-PCI patient with cardiac dysfunction and IABP implantation, the platelet count decreased, which may be related to either HIT or IABP balloon induced mechanical platelet injury. In this case, platelet count displayed no change under IABP support and after IABP removal, while it increased greatly after heparin was replaced by argatroban, so HIT was strongly suggested.

Dr. ZHAO Yusheng: For AMI patients with cardiac dysfunction, some factors predicted poor prognosis, including advanced age, pulmonary infection, liver and renal dysfunction, large infarction area, less survival myocardium, low LVEF, multiple post-infarction complications, history of adverse cerebral events, *etc*. Considering these factors, in addition to the impatience aspects in her characteristics and delayed revascularization time, this patient was more likely to have poor prognosis and in-hospital death. Several dilemma made the treatment even more complicated. Firstly, failure in removing IABP suggested severe myocardial stunning which necessiates prolongation of mechanical support, while considering pulmonary infection and potential catheter-related infection, an earlier removal of IABP was undoubtedly preferable. Maintenance of blood pressure was another dilemma. Vasoconstriction agents may increase afterload and labor the myocardium, while coronary hypoperfusion may occur if vasoconstriction agents were not applied. Additionally, high dose of dopamine improved the blood pressure but constricted peripheral vessels simultaneously which induced multi-organ hypoperfusion consequently, especially liver and kidney. Meanwhile, small dose of sodium nitroprusside dilated peripheral vessels to improve multi-organ hypoperfusion, but left blood pressure maintenance a great problem. All these made it difficult to balance between the advantage and disadvantage of every treatment, which increased the risk of poor prognosis.

In brief, cardiac dysfunction complicated AMI patients are of high risk and poor prognosis. Iatrogenic multi-organ injuries should be avoided by every means possible. More attention should be paid to drug use, including indications, contraindications, therapeutic effects, and adverse reaction. Based on clinical symptoms, signs and laboratory tests, treatment strategy should be adjusted by comprehensively balancing between

the advantages and disadvantages. Prognosis evaluation plays important roles in keeping abreast of the disease progress, performing the treatment actively and informing the patient's family members effectively. It is strongly recommended that clinical doctors give intensive attention to this kind of patients in hope of accumulating experience, increasing survival rate, and improving the patient's long-term outcome.

(Translator: GAO Lei)

高龄女性急性广泛前壁心肌梗死合并心源性休克死亡 1 例

1 病例摘要

患者因"发作性胸痛伴恶心呕吐 15h "收入解放军总医院老年心血管病研究所病房。2011 年 8 月 14 日 3:00 患者无明显诱因出现明显胸痛，伴恶心、呕吐、大汗、四肢湿冷，无意识丧失，18:00 就诊于我院急诊，心电图提示胸前导联 ST 段抬高，心肌损伤标志物明显成比例升高。患者既往高血压病史 10 年，目前控制不佳；慢性支气管炎病史多年；1 个月前发生脑梗死；入院前一天腹泻。既往无吸烟、饮酒、违禁药物应用史。

入院查体：体温 36.5℃，血压 90/70 mmHg，双肺呼吸音清，双肺底可闻及少量湿性啰音。心率 133 次/min，心音低钝，胸骨左缘第二肋间可闻及 2-3/6 级舒张期杂音，双侧足背动脉搏动减弱。超声心动图提示：左室射血分数（left ventricular ejection fraction，LVEF）43%，室间隔中段至心尖段、左室前壁、侧壁运动减弱，室壁变薄，心尖部室壁瘤形成，矛盾运动，未见明显心包积液。胸片提示双肺炎症。

考虑急性前壁 ST 段抬高型心肌梗死、心源性休克诊断明确，急诊行主动脉内气囊泵疗法（intra-aortic balloon bumping, IABP）和经皮冠脉介入术（percutaneous coronary intervention, PCI）。造影提示前降支发出第一对角支后完全闭塞，未见侧枝供应，第一对角支开口受累，局限性狭窄 90%，回旋支未见明显狭窄，右冠弥漫性病变。考虑前降支为罪犯血管，于前降支近中段狭窄处植入支架 1 枚。

术后予抗血小板、抗凝、扩冠、调脂、改善肝、肾功能、抗感染、解痉平喘、营养支持等治疗，间断利尿，多巴胺持续泵入维持血压。考虑患者心功能差，心率快，频发室性早搏，予间断西地兰静脉推注、胺碘酮静脉滴注。2011 年 8 月 16 日因反复急性左心衰发作，双肺感染、肝功能不全、肾功能不全，行气管插管、呼吸机辅助呼吸，增加硝普钠泵入改善外周循环及降低后负荷，停用胺碘酮，升级抗生素，并严格控制出入量，肝肾功能有所恢复，呼吸机脱机训练顺利，心功能好转。查血小板计数出现一过性下降，考虑不排除 IABP 球囊对血小板的机械性损伤。综上所述，8 月 22 日、8 月 24 日先后拔除气管插管、IABP，患者呼吸频率快，持续低血压状态，大剂量多巴胺无法维持，26 日凌晨发生急性左心衰并出现交界区心律伴频发室早，进入深昏迷，考虑患者存在洋地黄中毒，查血浆地高辛药物浓度＞4ng/dl，予立即停用洋地黄类药物，二次行气管插管及 IABP，同时给予肾上腺素、大剂量多巴胺、去甲肾上腺素等血管活性药物治疗，并碳酸氢钠纠正酸中毒后病情逐渐平稳，神志恢复。9 月 6 日至 16 日间反复尝试呼吸机脱机失败，降低 IABP 反搏比例后患者血压不能维持，且发生肝素诱导性血小板减少，稽留高热，肺部感染加重，频发室早，病情持续危重状态。2011 年 9 月 17 日 19:00 患者突然出现血氧饱和度下降至 50%～70%，心率 140～150 次/min，血压 70/50mmHg，给予 100%纯氧吸入，肾上腺素、碳酸氢钠注射液等药物救治。家属拒绝行心肺复苏、电除颤等有创抢救措施，19: 40 呼吸心跳停止，医治无效死亡。

2 临床病理讨论

侯允天副主任医师：患者主要诊断明确为急性前壁 ST 段抬高型心肌梗死，心源性休克。就医时间晚，高龄，合并肝、肾功能不全及肺部感染等多脏器功能不全，病情危重，死亡风险极高。治疗过程中存在以下问题：（1）洋地黄中毒。患者心功能低下，心率快，频发室性期前收缩，予口服地高辛及间断西地兰静脉推注，洋地黄类药物剂量大，应监测血药浓度。心电监护提示交界区心律，应高度怀疑洋地黄中毒可能，抢救中判断准确，处理果断。（2）感染。患者心源性休克、长期卧床、营养状况差、咳痰无力、气管插管、既往慢性支气管炎病史等，均增加肺部感染的可能性，同时长期 IABP、大静脉置管、留置尿管又存在导管相关性感染风险。抗生素选用时应注意结合体温、血象、胸片、体征，特别是痰培养结果来判断，同时注意同种抗生素应用时间。

薛桥副主任医师：多脏器功能衰竭是心肌梗死

伴心源性休克患者治疗中的常见问题，主要分为病程进展引起的脏器功能损伤和医源性脏器功能损伤。本病例中，患者入院前已发生心源性休克，持续低血压状态数小时，多器官灌注严重不足，表现为面色苍白、四肢湿冷、末梢动脉搏动弱、肝淤血、肺淤血、肾功能不全，均提示患者基础脏器功能差，预后不良。医源性肝功能损害、急性肾损伤、洋地黄中毒、肝素诱导血小板减少更应引起临床医生高度重视。造成肝功能损害的原因包括既往基础肝病、心功能不全、充血性/缺血性肝病以及药源性肝功能损害，如静脉应用胺碘酮。急性肾功能损害除了低灌注造成的肾前性急性肾功能损害外，还应考虑冠脉造影及支架植入术中造影剂对肾脏功能的影响。反复发作急性左心衰的患者口服及静脉应用大量洋地黄类药物，应警惕洋地黄中毒发生，及时观察临床症状、心电图变化及监测地高辛浓度。PCI 术后心功能不全且应用 IABP 的患者血小板减少，可能与肝素大量应用致肝素诱导血小板减少或 IABP 球囊对血小板的机械性破坏有关。本例患者 IABP 拔除期间未见明显血小板计数变化，而用阿加曲班代替肝素后血小板计数明显回升，考虑应为肝素诱导血小板减少。

赵玉生主任医师：急性心肌梗死合并心功能不全患者提示预后不良的的危险因素包括有高龄、肺部感染、肝肾功能不全、心肌损伤面积大、存活心肌数目少、射血分数低下、多种心肌梗死机械并发症、既往脑血管意外病史等。该患者高龄，性格急躁，急性心肌梗死面积大，心肌损伤严重，心功能差，加之血管再通延迟时间较长，双肺感染、肝肾功能不全，使该患者危险评分极高，院内死亡可能极高，预后差，且患者在治疗中存在多个矛盾，为临床治疗带来困难。在 IABP 拔除问题上，一方面始终不能耐受 IABP 拔除，提示心肌顿抑严重，心功能恢复缓慢，另一方面双肺炎症持续存在，随时可能发生导管相关性感染，使拔除时间难以确定。在持续心源性休克状态血压的调整问题上，如扩容升压则加重心衰，反之，则血容量不足心功能减退。在外周灌注的问题上，大剂量多巴胺的强烈缩血管作用可升压，但导致肝、肾等脏器及四肢供血不足，增加应用硝普钠可改善周围组织器官灌注，减轻后负荷，但其强大的降压作用使维持血压成为困难。以上治疗矛盾增加了治疗难度，提高了患者不良预后风险。

总之，急性心肌梗死合并心源性休克的患者预后差，风险高。治疗过程中应针对多脏器功能衰竭进行一体化治疗，尽量避免医源性脏器功能损伤。严格掌握用药适应证及禁忌证，观察药物疗效及不良反应。面对治疗矛盾，多角度权衡利弊，根据症状、体征及辅助检查结果精细调整治疗方案。而重视患者预后风险评估，有助于掌握病情进展、积极治疗及更好地与患者家属沟通。临床医生应高度重视此类患者的诊治，不断积累经验，提高生存率并改善远期预后。

（参加讨论医师：候允天，薛　桥，赵玉生）

（高　磊　整理）

（编辑：王雪萍）

· 启　事 ·

《中华老年多器官疾病杂志》已由双月刊改为月刊出版，80 页，刊物载文量大幅增加，稿件处理周期亦有所缩短，欢迎广大专家学者踊跃投稿!

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