·临床病理讨论·

Clinicopathological Conference

An 80-year-old woman with abdominal pain and vomiting

(The 15th case)

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Case Presentation

The patient, an 80-year-old female, retired worker, was admitted to the hospital because of vomiting and stomach discomfort for 5 days, abdominal pain for 1 day.

Five days before admission she had had a cold characterized by nausea, vomiting small amount of coffee-ground fluid, and stomach discomfort. On the day of admission (March 7), she complained of palpitation, shortness of breath, and epigastric pain without precordial discomfort, diarrhea and vomiting. On the way to the hospital, she got temporal abnormality of consciousness. In the emergency department, the patient's blood pressure was 60/50 mmHg and could not be measured gradually. After giving intravenous dopamine and other pressor agents, the BP could be maintained at above 80/60 mmHg. Initial electrocardiogram did not show any abnormality. The patient was then diagnosed to have shock for further observation and treatment. At 9 o'clock in the morning of March 8, a repeated EKG revealed high sharp R wave in the lead Ⅰ and Q waves in leads Ⅱ, Ⅲ, aVF. The levels of GOT and GPT were elevated to 1400 and 1014U/L respectively. Initial laboratory studies revealed a white-cell count of 13.6×10^9/L, the percentage of neutrophils was 80%, blood gas analysis showed PO_2 65.1 mmHg, PCO_2 30 mmHg, BE −6.9. The patient was immediately admitted to the ward for further treatment because of acute inferior myocardial infarction.

On physical examination, vital signs were BP 80/60, pulse 154/min, temperature 39.2℃, respirations 24/min. The patient was restless and the general condition was poor, her lip and finger tip showed cyanosis, the extremities were cool, pupils were equal in size, round and reactive to light. Neck veins were not prominent and distended, thyroids were not enlarged. Examination of the lungs revealed dry rales in the whole lung field and moist rales were not heard. The heart was not enlarged, heart rate was 154/min and regular, S_1 was diminished at the apex, $A_2 > P_2$, no murmur was heard. Examination of the abdomen was normal except for diffuse tenderness. There was no edema of both lower extremities. Signs of pathologic reflex were negative.

The patient had been diagnosed to have cor pulmonale 1 year ago because of recurrent attacks of asthma. She had no history of hypertension and diabetes mellitus. There was no personal history of cigarette smoking and alcohol drinking.

After admission, the patient received antishock therapy with pressor agents and fluid replacement (2500-3000ml/day), correcting acid-base imbalance therapy, and anti-infection therapy with 16×10^5U of penicillin. At 4 o'clock in the afternoon of March 8, the patient suddenly presented unconsciousness and speech disability. After neurologic examination, the consultant of the department of neurology concluded that the patient suffered from stroke. The neurologic signs included blurred consciousness, having reaction to stimulation, not being waken by calling, equal size of pupils, sluggish light reflex, edema of optic disc, neck rigidity, symmetric nasolabial grooves on two sides, decreased activities of the left lower extremity on scraping, decreased tendon reflexes of extremities, positive Babinski sign in the left, and negative Chaddock sign on both sides. Computerized tomographic scanning could not be done because of the patient's poor state. At night, her heart rate decreased to 45/min, BP fluctuated between 50/30—40/0mmHg after receiving pressor agents such as dopamine(220mg in 250ml fluid), aramine(40mg). At 8 o'clock, the patient started having apnea and did not

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show the sign of resuscitation after giving nikethamide(0.375g), external cardiac massage and artificial respiration.

Clinical discussion

Dr. GAO Lei: The clinical characteristics of this case were as follows: ①There were no history of coronary artery disease, cerebrovascular diseases, gastrointestinal disease; ② Nausea and vomiting were the primary symptoms; ③The patient presented refractory hypotension and temporal abnormality of consciousness on admission; ④EKG showed dynamic changes of QS waves in leads Ⅱ, Ⅲ, aVF and obvious elevation of serum myocardial enzymes; ⑤ The patient became unconscious again in spite of active anti-shock treatment. In short, the diagnosis of acute inferior myocardial infarction was established according to the changes of EKG and myocardial enzymes. However, following questions should be discussed: ① Were the gastrointestinal symptoms before admission associated with acute myocardial infarction (AMI)? ② What was the cause of refractory hypotension? ③It was still not clear what resulted in loss of consciousness of the patient just before dying; ④From the patient who died in 1990s, what can we learn according to current opinions about acute coronary syndrome?

Dr. LI Jindong: Possible causes of refractory hypotension after her admission were as follows: ①Acute inferior myocardial infarction might be accompanied by right ventricular infarction, leading to severe hypotension. Firstly, some patients with inferior infarction in whom the Bezold-Jarisch reflex is activated may transiently have systolic blood pressure below 90 mmHg; secondly, depending on the criteria used, approximately 50 percent of the patients with inferior infarction have some involvement of the right ventricle, these patients mostly present refractory hypotension because of limitation of right ventricular filling. ② Cardiogenic shock, by definition, has systolic pressure below 90 mmHg and evidence of end-organ hypoperfusion. ③Septic shock. It occurs most frequently in elderly or immunocompromised patients. Infections of the lung, abdomen, or urinary tract are most common cause. The patient was of advanced age and presented cold 5 days before admission, in addition, laboratory studies revealed a white-cell count of 13.6×10^9/L and the percentage of neutrophils was 80%. These all supported the possibility of septic shock. ④ Hypovolemic shock. According to the history of vomiting small amount of coffee-grounds fluid, we can not exclude loss of excessive blood into gastrointestinal tract. ⑤ Neurogenic shock. Strong neural stimulation caused release of vasoactive substances, leading to dilatation of peripheral blood vessels. The blood pooled in dilated microvessels, leading to sudden decrease in effective circulatory blood volume and shock. The patient had hemorrhage of digestive tract before admission, and complained of epigastric pain on admission, so gastric perforation could not be excluded, extreme epigastric pain might cause neurogenic shock.

Dr. ZHANG Wenli: The whole course of disease could be divided into 3 phases: ① Eraly stage, the patient suffered from gastrointestinal symptoms after cold. It might be acute episode of peptic ulcer; ② Period of onset, the patient developed acute inferior myocardial infarction and shock 5 days after afore-mentioned stress; ③ Terminal stage, ischemic stroke caused by cerebral hyoperfusion because of refractory hypotension. So, diagnosis of the patient included peptic ulcer, acute inferior myocardial infarction, shock, and ischemic stroke.

Dr. TIAN Jinwen: This patient was of advanced age, had been diagnosed to have cor pulmonale for 1 year and presented symptoms of cold at first. Physical examination on admission showed high temperature, bilateral dry rales in the whole lung fields. These findings all supported the existence of pulmonary infection. Dysfunction of multiple organs including heart, brain and digestive system appeared after admission. Pulmonary infection was an important initiative factor of multiple organ dysfunction syndrome (MODS) in old patient, the clinical course of the case was consistent with the hypothesis of lung initiating mechanism in multiple organ dysfunction syndrome in the elderly (MODSE).

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Dr. QIAN Xiaoshun: I did not agree with doctor TIAN. The patient had no obvious evidences of pulmonary infection. Although the patient had symptoms of cold early at the onset of disease and fever, high WBC count and dry rales in the whole lung field on admission, the dry rales could be caused by cor pulmonale. In addition, lack of moist rales typical of pulmonary infection and evidence in chest radiography also did not support the diagnosis of pulmonary infection.

Pathological discussion

Dr. YIN Tong: The major panthological findings were as follows: ①Extensive acute myocardial infarction located at left inferior ventricle and interventricular septum; ②Right coronary atherosclerosis (Stage Ⅳ) with old organized thrombosis; ③Coronary atherosclerotic heart disease and pneumocardial disease; ④ Aorta atherosclerosis (Stage Ⅲ - Ⅳ); ⑤ Brain basal artery atherosclerosis (Stage Ⅳ); ⑥Pulmonary emphysema with congestion at bilateral inferior pulmonary lobes; ⑦Liver atrophy complicated with chronic hepatic congestion; ⑧ Perforation of anterior wall of duodenal bulb (with area about 2cm × 1cm); Splenic and renal arteriolar sclerosis; ⑨Chronic enteritis and colonitis; ⑩Cerebella tonsillar hernia. From the aforementioned pathological changes, we could conclude that the death of the patient was caused by respiratory and circulatory failure as the result of extensive myocardial infarction and in turn the total cardiac failure, cerebral anoxia, oedema and finally cerebral hernia. Extensive myocardial infarction was most possibly induced by the perforation of duodenal bulb anterior wall.

Dr. ZHAO Yusheng: The main characteristics of acute coronary syndrome in the elderly are as follows: ① Insidious onset and atypical pain are the most important features; ②It is not uncommon that symptoms such as heart failure, shock, cerebral circulatory failure and gastrointestinal symptoms such as nausea and vomiting as a presentation of AMI are more likely to be seen in the period of onset; ③Symptoms of previous diseases such as cor pulmonale or asthma may be the outstanding symptoms. As for this case, symptoms of digestive tract played the central role in the onset of disease. However, pathological diagnosis included both gastrointestinal perforation and AMI. We need to pay more attention to elderly patients first presenting symptoms of digestive tract. We had to consider if the patient was suffered from gastrointestinal disease, acute coronary syndrome, or both. Perforation is the second most common ulcer-related complication being reported in as many as 6% to 7% of peptic ulcer patients. The main clinical characteristics include variation in the intensity or distribution of the abdominal pain as well as the associated symptoms such as nausea, vomiting, and symptoms of upper gastrointestinal bleeding. However, gastrointestinal symptoms which may occur in patients with AMI are nausea, vomiting, and abdominal pain. Nausea and vomiting occur more commonly in patients with inferior AMI. In this patient, gastrointestinal symptoms existed in the whole course of the disease and vomiting small amounts of coffee-grounds fluid occurred in the early stage, so we can suspect the diagnosis of upper gastrointestinal disorder. Abnormalities of myocardial enzymes and change in EKG after admission supported the diagnosis of AMI induced by gastrointestinal disorder. With regard to the cause of hypotension, I agree with the opinion of doctor Li. It was the comprehensive effect of decrease of myocardial contractility, infection, insufficiency of fluid, severe pain and so on.

Dr. WANG Shiwen: This patient had obvious neurological symptoms, even though the pathological examination did not find the evidence of distinct cerebrovascular accident and only find the presence of cerebellar tonsillar hernia, it must provoke the attention of geriatric cardiologists. Firstly, There may be coexistence of AMI and acute cerebrovascular accident in the elderly, constituting cardio-cerebral syndrome. Although the pathological diagnosis did not support the evidence of acute stroke in this case, more elderly AMI patients were companied with acute cerebral accident. Low cardiac output during AMI causes decrease in cerebral vascular blood flow. At the same time, regulation of nervous reflex results in arterial spasm, which aggravates deficiency of

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blood supply to brain. In addition, rupture of the arteriosclerotic blood vessel in brain or embolism from dislodging of heart thrombs can cause cerebrovascular disease. Secondly, refractory hypotension which resulted in cerebral hypoperfusion, cerebral ischemia and hypoxia, and at the same time, large amount of fluid infusion for treating shock were possibly the causes of cerebral edema and tonsillar hernia. I think that anitshock therapy for the patient needs the measures such as early revascularization including intra-aortic balloon pumping (IABP) in time, anti-infection treatment, appropriate fluid infusion and alleviating pain.

Translator GAO Lei

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1 病历摘要

患者女性，80 岁，退休工人，主因呕吐，胃部不适 5 d，腹痛 1 d 入院。患者 5 d 前患感冒，恶心、呕吐，胃部不适。吐少量咖啡色液体。3 月 7 日患者出现心慌，气短，上腹疼痛，但无明显心前区不适，无腹泻，呕吐，遂来解放军总医院就诊。途中曾一度出现意识反常，模糊。来院后血压 60/50 mmHg，后逐渐测不到。用多巴胺等升压药物后，可维持在 80/60 mmHg。心电图未见明确异常。按休克留观。3 月 8 日上午 9:00 复查心电图发现Ⅰ导联 R 波高尖，Ⅱ，Ⅲ，aVF 呈 QS 波，急查 GOT 1400%，GPT 1014%，WBC 13.6×10^9/L，中性 0.8，PO_2 65.1 mmHg，PCO_2 30 mmHg，BE −6.9。以急性下壁心肌梗死收入院。

入院时查体：体温：39.2℃，心率 154 次/min，呼吸 24 次/min，血压 80/60 mmHg，一般状况差，意识清，烦躁，口唇及指端紫绀，四肢凉，两瞳孔等大同圆，对光反射存在，结膜无充血，巩膜无黄染，口唇无紫绀，耳鼻无异常。咽红，扁桃体不大，无颈静脉怒张，气管居中，甲状腺不大，两肺密布干鸣音，无湿啰音，心脏不大，心率 154 次/min，律齐，心尖部第一心音弱，无杂音，A2 > P2，腹部略膨隆，移动性浊音阴性，腹软，肝脾不大，全腹弥散性轻度压痛，无反跳痛，无肌紧张。下肢不肿，双膝反射弱，无病理反射。1 年前因哮喘反复发作诊断为“肺心病”。无高血压、糖尿病史。

入院后给予升压药物，全天补液 2500 ~ 3000 ml。积极抗休克治疗，并纠正酸碱失衡，青霉素 160 万单位抗感染。考虑到入院时Ⅱ导联有 R 波但 1 d 后衍变为 QS 波，患者病情呈动态变化，认为患者预后较差，并向家属交待病情。肘静脉压 13cm H_2O，患者于 16:00 突然出现意识不清，言语不能，请神经内科会诊，神经科查体：意识混沌，呼之不应，对刺激有反应，双侧瞳孔等大 4 mm，对光反射迟钝，视乳头水肿，颈项强直，双侧鼻唇沟对称，伸舌不能，搔刮刺激左侧下肢活动较对侧少，四肢腱反射减低，Babinski征左侧阳性，Chaddock 征双侧阴性，Kernig 征双侧阳性。考虑为脑卒中，因病人一般状况差，无法做进一步 CT 等检查。入夜，患者心率 45 次/min，血压靠升压药物维持，浓度最高为 250 ml 液体内加入多巴胺 220 mg，阿拉明 40 mg，血压波动在 50 ~ 30/40 ~ 0 mmHg，20:15 患者出现呼吸暂停，液体中加入尼可刹米 0.375 g，效果不佳，心率开始逐步减慢至 20 次/min，血压测不出，22:35 呼吸心跳停止，立即予以心内注射“三联针”2 支，并给予体外心脏按摩及人工呼吸，抢救无效，患者死亡。病故前未见腹部症状加重。

2 临床与病理讨论

高磊医师：该病历特点包括：①既往无冠心病、脑血管疾病、消化系统疾病病史；②此次以恶心、呕吐，吐少量咖啡色液体为首发症状；③入院时表现为顽固低血压，一过性神志异常；④入院后 1 d 心电图呈动态演变，Ⅱ，Ⅲ，aVF 呈 QS 波，心肌酶谱明显升高；⑤虽经积极抗休克治疗却突然出现神志障碍；综上，根据心电图及心肌酶学改变，该患者急性下壁心肌梗死诊断成立，但仍有以下疑问：①入院前消化系统症状与本次急性心肌梗死有无联系；②患者顽固性低血压的原因如何；③患者临终前出现神志障碍的原因和诊断目前不清楚；④结合目前对急性冠脉综合征的诊断和治疗，该病例有何借鉴之处。请各位医师就上述或其他问题展开广泛讨论。

李进东医师：从入院后心电图的动态演变及心肌酶学改变，分析患者急性心肌梗死诊断是成立的，但患者入院后出现顽固的低血压状态，可能有以下原因：① 急性下壁心梗合并右室心梗导致严重低血

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压;② 心源性休克,大面积心梗导致泵功能衰竭,出现严重低血压;③ 感染性休克,该患者高龄,入院前有感冒病史,同时合并消化道症状,入院时体温39.2℃,WBC 13.6×10^9/L,中性0.8,故感染性休克也不能排除;④ 低血容量性休克,该患者入院前有恶心、呕吐、吐咖啡色液体病史,不能排除体内仍有出血导致低血容量性休克的可能;⑤ 神经源性休克,多为强烈的神经刺激,引起血管活性物质释放,导致周围血管扩张,大量血液淤滞于扩张的微循环中,导致有效循环血量突然减少而引起休克。该患者入院前有消化道出血病史,入院当天出现上腹部疼痛,不能除外因消化道穿孔、出血等急腹症引起的神经源性休克。

张文莉医师:综合该患者整个发病过程可以分为以下几个阶段,发病早期:感冒后出现消化道症状,考虑为消化道溃疡急性发作;发病期:由于上述应激导致5 d后出现急性下壁心肌梗死并合并休克;临终期:脑卒中,考虑顽固低血压导致脑灌注不足引起缺血性脑卒中的可能性大。

田进文医师:该患者为高龄患者,既往诊断"肺心病"1年,此次发病前曾出现感冒症状,入院时查体提示体温、血象显著增高,双肺可闻及干鸣音,入院后出现心、脑、消化系统等多器官功能障碍,认为符合王士雯院士提出"老年多器官功能不全肺启动假说",以肺为首发,随后依次出现消化、心、脑等器官功能障碍。

钱小顺医师:不同意田进文医师的观点,该患者没有明显的肺启动过程。虽然本例患者在发病早期有感冒症状,入院时有体温、血象显著增高,双肺可闻及干鸣音,但该患者入院时腹部症状更加明显,不能排除消化系统感染的可能。同时患者既往有肺心病病史,双肺可长期存在干鸣音,另外缺乏典型肺感染湿性啰音、缺乏胸片等影像学依据,也不支持肺感染诊断。

尹彤医师报告病理诊断:①左心室下壁,室间隔广泛性心肌梗死,伴心源性休克及全心衰竭;②右冠状动脉粥样硬化(Ⅳ级),伴陈旧性机化的血栓形成;③冠状动脉粥样硬化性及肺源性心脏病;④主动脉粥样硬化 Ⅲ~Ⅳ 级;⑤基底动脉粥样硬化Ⅳ级;⑥肺气肿,两肺下叶淤血;⑦肝萎缩合并慢性肝淤血;⑧十二指肠球部前壁穿孔(面积2 cm×1 cm);⑨慢性小肠炎及结肠炎;⑩小脑扁桃体疝。患者的死亡原因是十二指肠球部前壁穿孔,诱发广泛性心肌梗死,造成全心衰竭,缺氧水肿而至脑疝,引起循环和呼吸衰竭而死亡。

赵玉生医师:老年人急性冠脉综合征的临床特点是①发病隐匿,疼痛症状不典型;②以心力衰竭、休克、脑循环衰竭和胃肠道症状为首发症状者不少见;③原有的基础疾病症状多突出,如肺心病、哮喘等以咳嗽、心悸症状为主;结合此患者,本次发病以消化系统症状为主,最终病理诊断包括消化道穿孔和急性心肌梗死两个诊断,这需要引起老年心脏科医师的高度重视,对于以消化道症状首诊的老年患者既要考虑消化系统疾病或急性冠脉综合征的存在,也要考虑两种疾病并存的可能。消化道穿孔是消化道溃疡第二常见的并发症,在消化道人群中发病率为6%~7%。其主要特点包括,腹部疼痛程度、部位的变化,同时伴有恶心、呕吐、消化道出血症状。而急性心肌梗死时消化道症状常表现为恶心、呕吐、腹部疼痛等,其中恶心、呕吐多见于下壁心肌梗死患者。该患者整个发病过程中消化道症状始终存在,同时发病早期有上消化道出血表现,因此应高度怀疑上消化道疾病的可能,入院后患者心肌酶、心电图等改变支持由于消化道疾病引发急性心肌梗死诊断。对于该患者顽固性低血压,我同意李进东医师的意见,考虑是多种因素的综合效应,包括心肌收缩力下降、感染、液体不足,剧烈疼痛等。

王士雯医师:本病例虽然有明显神经系统症状,但最终尸检结果没有发现明确的脑血管意外依据,而是存在小脑扁桃体疝,应引起老年心脏病专科医生的重视。一方面不少老年急性心梗患者与急性脑血管意外并存,这在临床上称为心脑综合征。这是因为急性心肌梗死时的低排心血量可使脑血管血流减少,神经反射的调节可使动脉痉挛,加重脑供血不足或使老年人本来已经硬化的脑血管破裂,或因心脏的附壁血栓脱落致脑血管栓塞。另一方面,由于该患者入院后血压始终偏低,脑灌注不足,缺血、缺氧;同时为纠正休克补液量较大均是最终患者脑水肿,脑疝的可能原因,针对该患者休克表现,纠正低血压应包括尽早采用血运重建治疗,必要时选择主动脉球囊反搏改善血流动力学状态,以及抗感染,适当补液,止痛等综合治疗。

(高磊 整理)

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