临床病例讨论
Clinicopathological Conference

A 51-year-old male patient with chest pain and dyspnea

(the 40th case)

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Case presentation

A 51-year-old man was admitted to hospital on Dec. 23, 2008 because of a 5-day history of chest and back pain, and dyspnea. On Dec. 18, 2008, the patient suffered from pain in right legs accompanied with movement disturbance when he defecated. Two hours later, the right leg pain and paralysis relieved, but chest and back pain appeared, and was exacerbated by deep inhalation, which was also accompanied with sweating and shortness of breath, but with no radiation pain, palpitations, nausea, or vomiting. Four days later, chest and back pain was relieved, but dyspnea still existed. The patient was then admitted to the Emergency Department of Affiliated Hospital, Chengde Medical College. The brain CT imaging was normal. The routine blood tests showed that white cell count was 23.23×10^9/L, and neutrophils 86.5%. The myocardial enzymes spectrum was normal. The ultrasonography of kidneys, bladder and ureters was also normal. The diagnosis of upper respiratory tract infection was given, and antibiotics were administrated. But after two-day treatment, the shortness of breath was still not relieved. For definite diagnosis and further treatment, the patient was admitted to Emergency Department, Chinese PLA General Hospital. The arterial blood gas analysis showed PO_2 51.9 mmHg (1 mmHg = 0.133kPa), PCO_2 26.5 mmHg, pH 7.404, HCO_3^- 16.2 mmol/L, BE −7.2 mmol/L. The routine blood test showed white cell count 14.04×10^9/L, neutrophils 77.8%. And D-dimer 8.95mg/L. Blood biochemical tests showed TB 53.6 μmol/L, IB 19.3 μmol/L, serum creatinine 159.9 μmol/L, urea nitrogen 11.24 mmol/L, and potassium 3.20 mmol/L. The chest X-ray examination reported bilateral pleural effusions. The transabdominal ultrasonography (TAS) showed widening of hepatic vein, bilateral pleural effusions and ascites. The diagnosis of pulmonary embolism was suggested. The patient was transferred to Institute of Geriatric Cardiology, Chinese PLA General Hospital with the supposed diagnosis of "chest pain with undefined cause".

The patient had a 3-year history of hyertension, and the blood pressure was not under satisfactory control. He denied history of angina and deep venous thrombosis. The body temperature was 36.7℃, pulse rate 105/min, respiration rate 18/min, blood pressure 171/100 mmHg. No moist rales were auscultated, but the respiratory sounds were weak over both lung fields and disappeared at the lower part of lungs. Cardiac rhythm was regular, the heart rate was 105/min, and the first heart sound was weak. No cardiac murmur or pericardial rub was detected on auscultation. The abdomen was flat, soft and no tenderness. The liver was not palpable. There was no shifting dullness and edema of legs. The nervous system had no abnormality. Electrocardiographic examination showed sinus tachycardia and $Q_{Ⅲ,avF}$. A transthoracic echocardiogram showed pericardial effusions, but no left ventricular hypertrophy or left ventricular segmental wall-motion abnormalities. Pulmonary embolism was the initial diagnosis, and low molecular weight heparin (LMWH) was given to the patient at a dose of 60mg every 12 hours. But lower limb venous

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ultrasound and perfusion lung scan didn't confirm the diagnosis of pulmonary embolism. LMWH administration was stopped. The ultrasound of renal artery, iliac artery and vein in legs were normal. Chest MRI showed aortic dissection. After consulting vascular surgeon, CT angiography (CTA) was performed and the descending aortic dissection, which originated in intimal tears just distal to the left subclavian artery, was identified.

Clinical discussion

Dr. Chen Yanming: The results of cardiac biomarkers and ECG excluded the possibility of acute myocardial infarction and acute pericarditis. The key point was the differentiation diagnosis of aortic dissection from pulmonary embolism. (1) Chest pain, dyspnea, sinus tarchycardia, hypoxia, the elevation of D-dimer, widening of liver veins and ascites provided clues to the diagnosis of pulmonary embolism. So on the first admission day, LMWH was administered subcutaneously. But no deep venous thrombosis was found in the lower extremities, and the perfusion lung scan excluded the initial diagnosis on the second admission day. (2) The patient had a 3-year history of hypertension, which was not controlled well. The chest pain was accompanied with right leg paralysis. MRI of ascending aortic dissection and CTA confirmed the diagnosis of aortic dissection. But there were a few questions about the diagnosis. (1) whether the aortic dissection could lead to dyspnea and pleural effusions. (2) Vascular CTA and angiography indicated that right renal artery and bilateral iliac artery were involved, but the ultrasonography was normal. Then what is the significance of vascular ultrasound in aortic dissection? (3) The patient reported renal dysfunction, bad-controlled blood pressure, hypopotassemia, and right leg paralysis, so secondary hypertension was considered. Then what is the reason for the secondary hypertension? (4) What is the reason for white blood cell increase and red blood cell decrease? (5) What are the other disorders which need differentiation from aortic dissection in this patient?

Dr. Gao Lei: (1) Aortic dissection may cause blood exudation and lead to pleural, abdominal, and cardiac effusions. But dropsy of multiple serous cavities was seldom reported. Pleural effusions could cause dyspnea and hypoxia. (2) Transoesphageal echocardiography is sensitive and specific for ascending aortic dissection, which can show the site of intimal tears, blood flow of real and false cavity, aortic insufficiency, pleural effusion and blood flow of branches of aortic artery. But it is not sensitive and specific for descending aortic dissection. In this case, the renal and iliac arteries were involved, but the TAS didn't reveal abnormality, which showed that TAS had limited sensitivity for the diagnosis of descending aortic dissection. (3) If the artery which supplies the blood for nerve was involved, the feeling and movement of legs could disappear. This can explain the right leg paralysis of the patient. (4) According to renal ultrasound, we can speculate that the ischemia of the kidneys caused the elevation of serum creatinine, hypokalemia and activation of RAS system, and that is the reason for the hard-controlled blood pressure. (5) White blood cell counts are usually elevated and hemoglobin reduced in patients with aortic dissection. It has been reported that they are related to the prognosis of aortic dissection. (6) An elevated D-dimer is a sensitive marker for pulmanory embolism. Unfortunately, it is not very specific. Its elevation is also observed in patients with acute myocardial infarction and aortic dissection.

Dr. Xueqiao: After admission, the key point for the patient was the differential diagnosis between aortic dissection and pulmonary embolism. The electrocardiographic findings in patients with aortic dissection are usually nonspecific. The chest X-ray film often suggests the diagnosis of dissection, but cannot identify it is on earth ascending aorta or descending aorta that is involved, and the sensitivity is low. CT and MRI both have high accuracy for identifying aortic dissection. Especially, CTA has important significance for differential diagnosis between pulmonary embolism and aortic dissection. Moreover, the level of serum creatinine (159.9 μmol/L) was not a contraindication for CT

angiography. If the renal function was impaired due to aortic dissection, it can be reversed by early diagnosis and treatment. What's more, CTA is very important for guiding stent selection. So, timely CTA can significantly shorten the time to get final diagnosis.

Dr. Zhao Yusheng: Aortic dissection had variable manifestations, including the severe pain in chest, back or abdomen, hypertension, the ischemia of organs (spinal cord, limbs, and kidneys), the rupture of dissection (pericardial effusions, ascites, hemoptysis and hematemesis), aortic insufficiency, and spasmophonia, *etc*. Even in some high level hospitals, miss diagnosis was reported in about 38% of the patients with aortic dissection, and the diagnosis was confirmed by autopsy in 28% patients. So it is not easy to diagnose this illness early. In this case, besides pulmonary embolism and aortic dissection, other diseases should also be considered. (1) Connective tissue disease also can manifest hypertension, renal dysfunction, fever, hypoxia and pleural effusions. For example, systemic lupus erythematosus also can cause aortic dissection. Patient's medical history and laboratory tests are quite useful for the differential diagnosis. (2) When hypertension was combined with right limb paralysis, stroke should be considered. When the aorta branches, such as carotid artery, vertebral artery, were involved in aortic dissection, stroke often happened. (3) The white blood cells always increase as a stress reaction for aortic dissection. In aortic dissection, the pseudocoel releases pyrogen, which causes fever. Simultaneously, bloody pleural effusions often appear, so aortic dissection should be carefully differentiated from pulmonary infection and tuberculous pleurisy.

(Translator: Chen Yanming)

胸痛、呼吸困难男性1例

患者男性,51岁,主因"发作性胸背部疼痛伴呼吸困难5 d"于2008年12月23日急诊入院。2008年12月18日解大便时出现右下肢剧烈疼痛并伴有活动障碍,2 h后右下肢疼痛和无力缓解,但出现胸背部剧痛,吸气时加重,并伴呼吸困难,出汗,无反射痛,无心悸,无恶心呕吐。4 d后胸背部疼痛逐渐消失,但仍有呼吸困难,遂入承德医学院附属医院,急查头颅CT未见出血灶,血常规提示白细胞总数23.23×10^9/L,中性粒细胞86.5%,心肌酶未见明显异常,腹部超声提示双肾、膀胱、输尿管未见异常,诊断为上呼吸道感染,治疗2 d后背部疼痛减轻,但仍间断有呼吸困难,为进一步明确诊断入解放军总医院急诊,血气分析:PO_2 51.9 mmHg (1 mmHg=0.133 kPa),PCO_2 26.5 mmHg,pH 7.404,HCO_3^- 16.2 mmol/L,BE−7.2 mmol/L,血常规:白细胞总数14.04×10^9/L,中性粒细胞77.8%,D-二聚体8.95 mg/L,生化:总胆红素53.6 μmol/L,直接胆红素19.3 μmol/L,肌酐159.9 μmol/L,尿素氮11.24 mmol/L,血钾3.20 mmol/L。胸片提示:胸腔积液。腹部超声提示:(1)肝静脉增宽;(2)双侧胸腔积液;(3)腹水。考虑肺栓塞可能性大,遂拟诊"胸痛原因待查?"收入解放军总医院老年心血管病研究所。既往有高血压病史3年,血压控制欠佳。否认心绞痛病史。无下肢深静脉血栓病史。体温:36.7℃,脉搏:105次/min,呼吸:18次/min,血压:171/100 mmHg。双肺呼吸音弱,双下肺未闻及呼吸音,未闻及干湿性啰音。心率105次/min,律齐,第一心音弱,各瓣膜听诊区未闻及病理性杂音。腹软,无压痛和肌紧张,肝肋下未触及,移动性浊音阴性。双下肢无水肿。神经系统查体正常。心电图(2008-12-23):窦性心动过速,$Q_{III,avF}$。超声心动图:心脏功能正常,心包积液。入院后首先考虑肺栓塞,控制血压、心率情况下,同时给予低分子肝素钠60 mg 1次/12 h抗凝。入院后第2天行下肢静脉超声和肺灌注扫描,未见异常后停用低分子肝素。后考虑主动脉夹层可能性大,鉴于患者存在肾功能不全,申请了胸部血管磁共振检查,在等待检查的过程中(共8 d),患者行双侧肾动脉超声、双侧髂动脉超声检查未见异常。胸部血管磁共振提示为主动脉夹层,请血管外科会诊后,行胸腹主动脉CT血管造影(CT angiography,CTA),进一步证实主动脉夹层(Stanford B,内膜破口起源于左锁骨下动脉

远端)。

陈艳明住院医师:患者入院后,根据心肌酶、心电图的变化,首先排除了急性心肌梗死和急性心包炎。诊疗过程中主要进行肺栓塞和主动脉夹层的鉴别诊疗。(1)肺栓塞:患者胸痛、呼吸困难明显,窦性心动过速,血气(PO_2 51.9 mmHg, PCO_2 26.5 mmHg)、D-二聚体升高,肝静脉增宽、腹腔积液,首先考虑肺栓塞,入院后第一天给予抗凝治疗,但该患者无双下肢静脉血栓,并且行肺灌注扫描未见明显异常,不支持该诊断;(2)主动脉夹层:患者既往有高血压病史,而且控制不佳,胸痛伴右下肢无力,查体腘动脉、足背动脉、胫后动脉搏动弱。主动脉的磁共振和CTA支持该诊断。①但主动脉夹层是否会引起呼吸困难,胸腔、腹腔、心包积液?②血管CTA及血管造影提示右肾动脉受累和双侧髂动脉受累,但肾动脉和髂动脉超声检查未见异常,血管超声在诊断主动脉夹层中的意义?③诊疗过程中患者肾功能不全(肌酐159.9 μmol/L),血压难控制、顽固的低钾血症,发病时有一侧肢体活动障碍,考虑存在继发性高血压,继发原因是什么?④患者白细胞升高、血红蛋白下降的原因是什么?⑤需与主动脉夹层鉴别的其他疾病有哪些?

高磊主管医师:(1)主动脉夹层在急性期由于夹层外壁薄,表面可有血液渗出,并且引起胸腔、腹腔、心包积液,但同时出现多浆膜腔积液少见。胸腔积液量大时可有低氧血症、呼吸困难;(2)经食管超声心动图对升主动脉夹层敏感性和特异性高,可以确定内膜裂口的位置,显示真、假腔的血流状态,是否伴发主动脉关闭不全、心包积液及主动脉弓分支血管的阻塞。但经食管超声对腹主动脉受累情况的观察效果不佳。本例当夹层累积肾动脉、髂动脉时,血管超声检查未见异常,说明经腹血管超声检查对腹主动脉夹层的敏感性低;(3)患者出现右下肢感觉运动丧失的原因:夹层累及患侧动脉时,引起缺血性外周神经疾病,可引起感觉、运动丧失;(4)患者有入院后高血压病史,血压控制不理想,行肾脏超声检查提示为慢性改变,表明在慢性基础上,肾脏急性缺血后,肾素-血管紧张素-醛固酮系统激活,引起肌酐升高、血压难控制、低血钾;(5)主动脉夹层患者常有白细胞升高、血红蛋白下降,有资料表明与患者预后相关;(6)正常水平的D-二聚体对于除外肺栓塞具有意义。但是该指标特异性差,在急性心肌梗死和主动脉夹层时也可升高。

薛桥主诊医师:入院后患者的鉴别诊断主要围绕肺栓塞和主动脉夹层。主动脉夹层时心电图的改变不特异:如本例为窦性心动过速。胸片只能提示主动脉影增宽,但不能提示为升主动脉夹层还是降主动脉夹层,而且该提示作用敏感性差,如本例患者虽行胸片检查但未见主动脉增宽。CT和MRI诊断主动脉夹层的准确率高。而且血管CTA对肺栓塞、主动脉夹层鉴别具有重要意义。CT检测速度快,作一次检查即可鉴别两者,并且肌酐159.9 μmol/L并不是血管CT的检查禁忌。如果肾功能的恶化因主动脉夹层引起,及早诊断治疗可使肾功能及早恢复,并且血管CT对血管外科支架的选择具有指导意义。患者入院后如果及时行血管CTA的检查可以缩短患者确诊的时间。

赵玉生主任:主动脉夹层的临床表现复杂多样。临床表现有前胸、后背、腹部的剧烈疼痛,高血压,脏器缺血表现(脊髓、四肢、肾脏),夹层破裂(心包积液、腹腔积液、咯血、呕血),主动脉瓣关闭不全、声嘶等。国外较大的医疗机构也有38%患者首诊漏诊,28%的患者在尸检后确诊,提示早期诊断主动脉夹层患者并不容易。从该患者的诊治过程,除了急性心肌梗死、肺栓塞,主动脉夹层还需要考虑的疾病:(1)患者高血压病、肾功能不全、发热、低氧血症、胸腹腔心包积液,应与结缔组织病相鉴别,并且某些结缔组织疾病如系统性红斑狼疮也可引起主动脉夹层,病史和实验室检查有助于鉴别诊断;(2)高血压,右侧肢体活动障碍应与脑卒中相鉴别。主动脉夹层累及主动脉主要分支如颈动脉、椎动脉时,可同时有脑卒中,二者可并存;(3)主动脉夹层作为应激反应,白细胞总数可升高,假腔释放致热原体温升高,同时由于胸腔积液存在,应与肺部感染、结核性胸膜炎相鉴别。

(参加讨论的医师:陈艳明、高 磊、薛 桥、赵玉生)

(陈艳明 整理)

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