# 临床病理讨论
# Clinicopathological Conference

## An elderly patient with chest pain, syncope and progressive multi-organ damage

**(the 39th case)**

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## Case presentation

A male patient, 65 years old, was admitted to Emergency Department of General Hospital of Beijing Military Command on Dec 29, 2009 because of syncope for 1 h. The patient suffered from chest distress and chest pain on left side with palpitation and subsequent syncope. He denied the history of hypertension, coronary artery disease and diabetes.

Physical examination Body temperature 36.1℃, pulse rate 110 beats/min, respiratory rate 18/min, blood pressure 60/30 mmHg. The patient was found in light coma and dysphoria. The patient was placed in a semi-reclining position. His left nasolabial fold was slightly shallow, and the angle of the mouth deflected toward the left side. The heart rate (HR) was 110 beats/min, and no pericardium friction sound was heard.

Accessory examination The routine blood test showed that WBC was $26.12\times10^9$/L and neutrophil 0.898. The blood biochemical test showed that glucose was 12.7 mmol/L and creatinine(CR) 220 μmol/L. Coagulation function examination showed that prothrombin time was 18.8 s, activated partial thromboplastin time 56 s. Creatine kinase (CK), myoglobin, Troponin I (TNI) and brain natriuretic peptide (BNP) levels in blood were normal. The electrocardiogram (ECG) showed sinus tachycardia and HR of 110 beats/min. Head CT scan showed no obvious infarction and hemorrhagic focus. Twelve hours later, CK, myoglobin and TNI all increased significantly (9.5, 500, 0.94 μg/L respectively). ECG showed extensive T-wave inversion. Abdominal ultrasound examination showed gallbladder wall edema, inferior vena cava dilation and right kidney stones. Echocardiography showed pericardial effusion.

Admission diagnosis (1) chest pain and syncope of unknown cause; (2) shock of unknown origin; (3) renal stones; (4) pericardial effusion.

Treatment procedures After admission, the patient received symptomatic treatment. His blood pressure was kept at 95-120/60-95 mmHg (with dopamine) and HR 85-115 beats/min. Chest radiograph showed widened mediastinum and cardiac enlargement (Fig 1). Abdomen CT scan showed splenomegaly, bilateral kidney stones and small amount of ascites. Echocardiography showed that (1) Visceral layer of pericardium slightly thickened (4 mm); (2) Ventricular wall motion was abnormal; (3) The ascending aorta was slightly dilated; (4) There was asymptomatic pericardial effusion. Abdominal ultrasound showed that there was irregular echo area when the patient was in horizontal position and the maximum depth was 6 cm. On Dec 30, the patient complained of asthenia without dyspnea; blood pressure was 115/70 mmHg; breath sounds were clear in the areas of both lungs, but there were diminished breath sounds in the right lower lung; HR was 95 beats/min. Head magnetic resonance imaging (MRI) showed acute left cerebellum and right occipital lobe infarction (Fig 2). On Dec 31, chest CT scan showed (1) possibility of aortic dissection; (2) bilateral pleural effusion; (3) dorsal atelectasis of right inferior lobe lung (Fig 3). After admission, the patient's heart, liver and kidney function was progressively injured.

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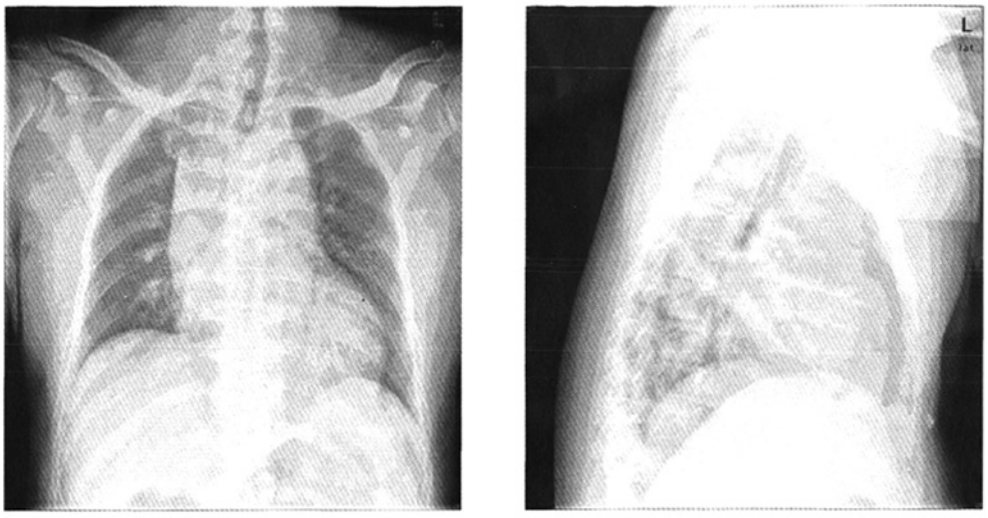

A B

**Fig 1 Chest X-ray examination**

A: widened mediastinum, suggesting further chest CT scan; B: cardiac enlargement

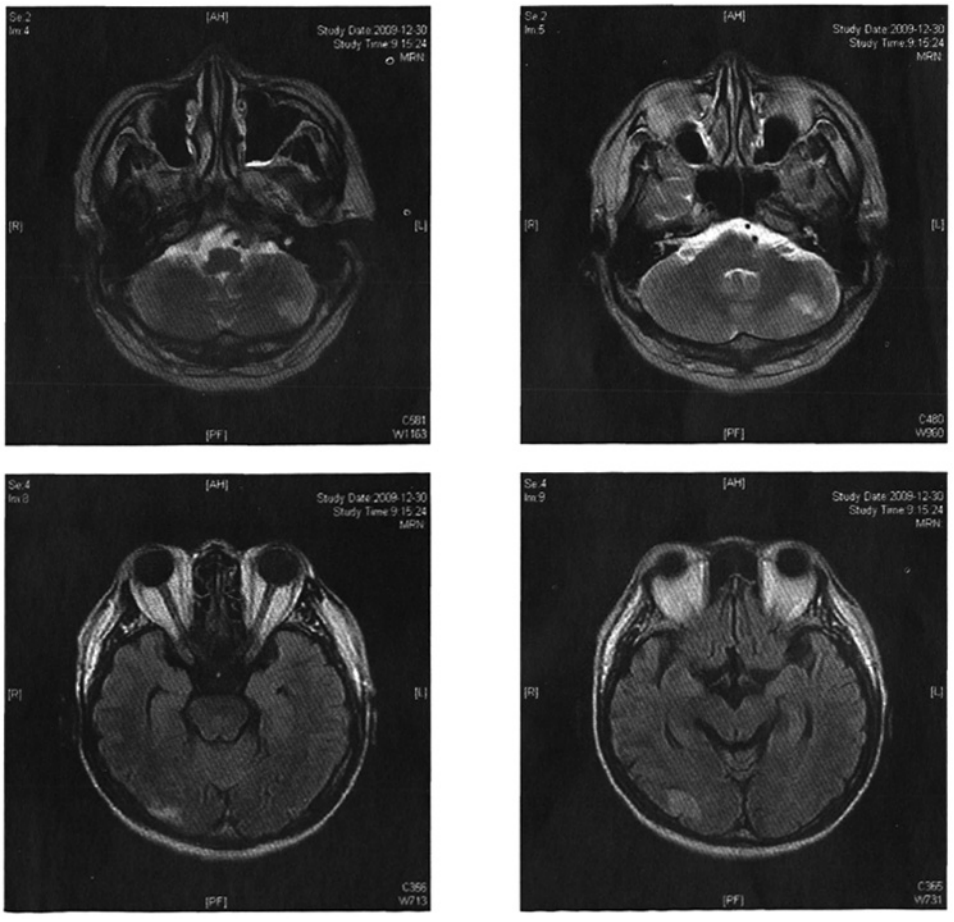

**Fig 2 Head magnetic resonance imaging shows acute infarction of left cerebellum and right occipital lobe**

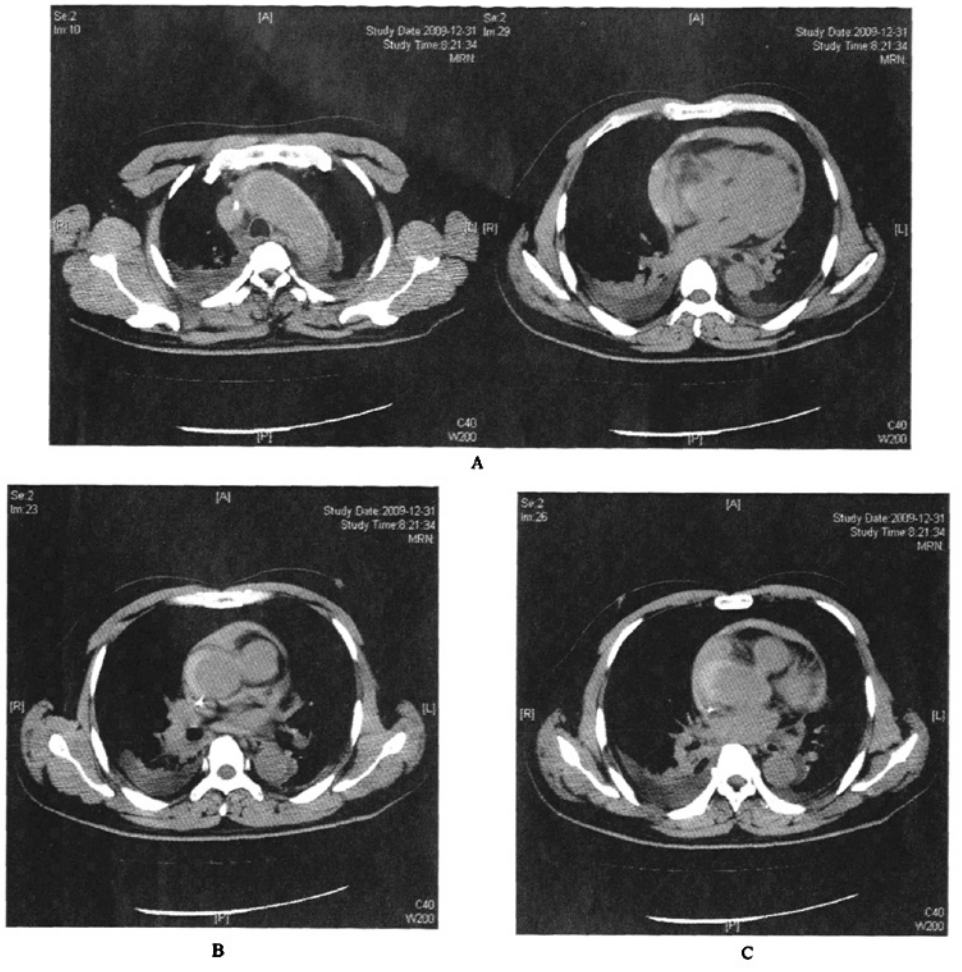


**Fig 3 Chest CT scan**

A: aortic dissection; B: bilateral pleural effusion; C: dorsal atelectasis of inferior lobe of the right lung

The CK reached 808 U/L, creatine kinase MB 128 U/L, serum myoglobin 637 μg/L, serum cardiac troponin T 2.5 μg/L, lactic dehydrogenase 2357 U/L, alanine aminotransferase(ALT) 2445 U/L, aspartate aminotransferase (AST) 2429 U/L. Uric acid was 824 mmol/L, CR 357 μmol/L, blood urea nitrogen 18.2 μmol/L. Coagulation function examination revealed that prothrombin activity (PTA) declined, and fibrinogen increased (15 g/L). Urinalysis showed that urine protein was 1.5 g/L and there were a large number of red blood cells. On Jan 2, the patient's mental status was obviously improved. The routine blood test results, liver function, kidney function, myocardial enzymes and coagulation function all became better than before; but PTA still declined. At 19: 42, the patient presented with sudden limbs twitching, with severe lips and facial cyanosis, unconsciousness, breathlessness and urinary incontinence. On physical examination, blood pressure was 150/70 mmHg; light reflex disappeared with anisocoria (right > left); breath sounds were not heard; HR was 83 beats/min in sinus rhythm; cardiac rhythm was regular; limbs had no response to strong pain stimulus and pathological sign was not elicited. Although immergency treatment was performed immediately, clinical death was declared for the patient at 21:35.

## Clinical discussion

*Dr. SUN Ming*: This patient was an old male, who was admitted to the emergency department because of syncope and unconsciousness. As

the disease progressed, systemic multi-organ dysfunction developed, with significant deterioration of lung, heart, liver, kidney and brain function. After symptomatic treatment, the patient's condition gradually improved, but during which, he died suddenly. Before admission, the patient experienced chest pain and palpitation with no obvious incentive, followed by syncope, which was initially considered as cardiac syncope induced by acute myocardial infarction (AMI). But CK, myoglobin, TNI and BNP at admission were normal, which excluded AMI. Twelve hours after onset, CK, myoglobin, and TNI all increased significantly and ECG showed extensive T-wave inversion in precordial leads, which suggested that coronary arteries were involved and myocardial ischemia happened. However, changes of myocardial enzyme did not support the diagnosis of AMI. The patient was in light coma and dysphoria. The left nasolabial fold was shallow and the angle of the mouth deflected toward the left side. It was indicative of acute cerebrovascular disease. Head CT scan showed no abnormality, but the brain stem lesion could not be excluded. Head MRI showed acute cerebral infarction in the left cerebellum, right occipital leaves. Reexamination of blood, liver and kidney function, and myocardial enzymes showed a significant increase in all indices, suggesting that systemic diseases couldn't be excluded.

*Dr. WU Hangyu*: The patient presented mainly with symptom of shock at admission. He experienced no excessive bleeding and body fluid loss, which did not support the diagnosis of hemorrhagic shock. The patient displayed no allergy-related symptom, so the anaphylactic shock was excluded. He did not take any antihypertensive drugs and experienced no exposure to any chemicals, so toxic shock was also excluded. The patient complained of dyspnea and chest pain, therefore the neurogenic shock might be taken into consideration. Besides, the routine blood test showed abnormally high levels of the indices, so the septic shock could not be excluded. Before admission, the patient experienced chest pain and palpitation, and ECG and myocardial enzymes was normal, but echocardiography showed small amount of pericardial effusion. Comprehensively, the acute pericardial tamponade induced cardiac shock could not be excluded.

*Dr. WANG Yuhong*: I totally agree with the above analysis. On Dec 31, chest CT scan suggested possibility of aortic dissection. On Jan 2, the patient's mental status was good and his blood pressure was kept at 140-170/70-90 mmHg. He should have a history of hypertension. Atherosclerosis is an essential enhancing factor of aorta dissection. About 3/4 patients with aorta dissection also suffered from hypertension, especially in elderly male individuals. The aortic aneurysm might be asymptomatic if it does not oppress adjacent tissues. The aortic aneurysm can extend to the distal aorta, involving the full-length of thoracic aorta and the branches of abdominal aorta. It can also extend to the proximal aorta, involving coronary artery and aortic valve, which will result in coronary blood flow occlusion and aortic insufficiency. If it involved the carotid artery, the cerebral ischemia would be caused, and the aggravation of ischemia could lead to acute cerebral infarction. If the aortic aneurysm perforated into aorta cavity, aortic wall dissection would no longer develop, and the condition may be relieved. From Dec 31 to Jan 1, the patient's condition was relatively stable, this situation should be taken into consideration. Immediately before death, the patient had the symptom of decerebrate rigidity, which was the manifestation of midbrain injury. So, in this case, we considered that aortic dissection extended to the vertebral basilar system and caused acute brain stem injury. This may be the cause of sudden death in the patient.

*Dr. DONG Lan*: In this case, liver and kidney function was abnormal. Possibly, aortic dissection extended to the branches of abdominal cavity organs and caused insufficient blood supply of liver or kidney, which resulted in liver and kidney function injuries. Chest CT scan showed dorsal atelec-

tasis of right inferior lobe lung. Ascending aortic aneurysm oppressed right bronchus and pulmonary artery, resulting in pulmonary atelectasis. As the patient's symptoms were not typical, and the etiological factors were not defined, what we can do was only to give symptomatic treatment targeting to the shock, such as restoration of blood volume with fluid infusion. But that is exactly the contraindicative for aortic dissection. This intervention may induce or accelerate the rupture of aortic dissection, leading to death. Aortic rupture led to acute pericardial effusion and acute pericardial tamponade, which caused sudden cardiac arrest. This may be the cause of death in the patient.

*Dr. ZHOU Rongbin*: This case is a 65-year-old male. He should have a history of hypertension. He was admitted to the hospital because of chest pain and syncope with progressive systemic multi-organ damage. We should consider acute cardiovascular disease, cerebrovascular disease and systemic diseases, and perform related accessory examination. (1) ECG showed left ventricular hypertrophy and non-specific ST-T changes. If the coronary artery was involved, ECG would be indicative of acute myocardial ischemia, and even acute myocardial infarction. If there was hemopericardium, ECG would be indicative of acute pericarditis. (2) Chest X-ray examination revealed widened aorta. Although there is no diagnostic value, it may be suggestive of further examination. (3) CT scan and MRI both have decisive diagnostic value for aortic dissection. Especially, CT scan can easily reveal the intraluminal thrombosis and blood vessel wall calcification. It can also display the spacial relationship between aortic aneurysm and adjacent structures, such as renal artery, retroperitoneal cavity, and spine. (4) Echocardiography plays important role in the diagnosis of aortic dissection and is easy to identify the complications such as hemopericardium, aortic valve insufficiency and hemothorax, and so on. (5) Aortic angiography is helpful to the localization of aortic aneurysm, but needs to be operated cautiously.

Final diagnosis included (1) aortic dissection with pericardial effusion and ascites; (2) acute brain infarction; (3) acute kidney insufficiency; (4) liver injury; (5) bilateral kidney stones.

Aortic dissection is a clinical presentation in which a tear on the wall of the aorta causes blood to flow between the layers of the aorta wall and forces the layers apart and forms a hematoma finally. It mainly attacks male individuals. With the progression of the disease, it can cause insufficient blood supply to the relevant organs; if the aortic aneurysm oppresses neighboring soft tissues or involves the main branches of aorta, the damage of the corresponding systems could be caused. In this case, aortic dissection involved the carotid artery, which led to cerebral hypoperfusion, manifested with syncope, shallow left nasolabial fold and deflect of mouth angle to the left. Dissection involved coronary artery, causing severe chest pain and palpitations; ECG showed sinus tachycardia; CK, myoglobin and TNI all increased significantly. Dissection ruptured into the pericardium, then pericardial effusion was caused. Once ascending aortic aneurysm oppressed right bronchus and pulmonary artery, dyspnea and right pulmonary atelectasis were caused. Rupture of dissection into peritoneal cavity resulted in ascites. Once the aortic dissection involved the abdominal aorta and its branches, liver injury was induced. It was shown that AST and ALT increased significantly. If the renal artery was involved, hematuria may be caused. The cause of death was considered as the following: (1) Aortic dissection extended to the vertebral basilar system, caused complete occlusion of arteries and acute brain stem injury, inhibited the respiratory center, and then led to respiratory failure. (2) Dissection rupture into the pericardium cavity led to acute pericardial tamponade and sudden cardiac arrest.

(Translator: WANG Limin, ZHOU Rongbin)

# 胸痛、晕厥合并进行性多器官损害1例

## 1 病历摘要

患者男性，65岁，主因"晕厥1小时"于2009年12月29日入院。患者入院前无明显诱因出现左侧胸闷、胸痛（呈撕裂样痛）、憋气，伴心悸，随即晕厥，遂来北京军区总医院急诊。既往体健。

查体：体温36.1℃，脉搏110次/min，呼吸18次/min，血压60/30 mmHg。浅昏迷，烦躁状态，半卧位，左侧鼻唇沟变浅，口角向左偏斜。心率110次/min，律齐，各瓣膜区未闻及病理性杂音。

辅助检查：血常规：白细胞26.12×10⁹/L，中性粒细胞0.898。血生化：血糖12.7 mmol/L，肌酐220 μmol/L。凝血功能：血浆凝血酶原时间18.8 s，活化部分凝血活酶时间56 s。血肌酸激酶（creatine kinase，CK）、肌红蛋白（myoglobin，MYO）、肌钙蛋白I（Troponin I，TNI）及脑钠肽（brain natriuretic peptide，BNP）水平均正常。心电图示窦性心动过速，心率110次/min。头颅CT未见异常。发病12 h后复查血CK、MYO、TNI分别为9.5，500，0.94 μg/L，提示3项均升高。心电图示广泛导联T波倒置。腹部超声提示胆囊壁水肿，下腔静脉扩张，右肾多发结石。心脏超声示心包积液。

入院诊断：（1）胸痛与晕厥原因待查；（2）休克原因待查；（3）肾结石；（4）心包积液。

诊疗经过：入院后给予对症支持治疗，血压95～120/60～95 mmHg（多巴胺维持），心率85～115次/min。胸片报告纵隔增宽，心脏增大（图1）。腹部CT提示，（1）脾大；（2）双侧肾结石；（3）少量腹水。心脏超声提示，（1）心包脏层稍增厚，厚约4 mm；（2）室壁运动不协调；（3）升主动脉内径轻度增宽；（4）少量心包积液。腹腔超声示腹腔平卧位可见不规则回声，最大深度6 cm。12月30日患者自诉乏力，血压115/70 mmHg，双肺呼吸音清，右下肺呼吸音减弱，心率95次/min。头颅MRI示左小脑、右枕叶急性脑梗死（图2）。12月31日胸部CT示（1）主动脉夹层？（2）双侧胸腔积液；（3）右肺下叶背侧膨胀不全（图3）。入院后心、肝、肾功能进行性损害，最高值分别为CK 808 U/L，肌酸激酶同工酶128 U/L，血清MYO 637 μg/L，血清肌钙蛋白T 2.5 μg/L，乳酸脱氢酶2357 U/L；谷丙转氨酶2445 U/L、谷草转氨酶2429 U/L。尿酸824 mmol/L，肌酐357 μmol/L，尿素氮18.2 μmol/L；凝血功能：凝血酶原活动度（prothrombin activity，PTA）降低，纤维蛋白原定量增高（15 g/L）。尿常规：尿蛋白1.5 g/L，大量红细胞。2010年1月2日患者精神状态明显好转，复查血常规、肝肾功能、心肌酶及凝血功能，各项指标均较前有所改善；PTA继续降低。1月2日19：42患者突发四肢抽搐，口唇、颜面重度紫绀，意识丧失，呼吸停止，尿失禁。查体：血压150/70 mmHg，双侧瞳孔不等大（右侧＞左侧），对光反射消失，未闻及呼吸音，心率83次/min，窦性心率，律齐，四肢强痛刺激无回缩，病理征未引出。经抢救无效，21：35宣布临床死亡。

## 2 临床病例讨论

孙明医师：患者老年男性，因晕厥入院。随着病情的发展，出现全身多器官损害：肺、心、肝、肾、脑功能急剧恶化，对症处理后病情逐渐好转过程中突然死亡。患者入院前突然出现胸闷、胸痛、憋气、心悸，随即晕厥，考虑急性心肌梗死（acute myocardial infarction，AMI）引起的心源性晕厥，入院当天血CK、MYO、TNI及BNP水平均正常，不支持AMI；发病12 h后复查CK、MYO、TNI均升高，心电图示广泛导联T波倒置，提示病变累及冠状动脉引起心肌供血不足，但心肌酶谱变化规律不支持AMI。入院后浅昏迷，烦躁状态，左侧鼻唇沟变浅，口角向左偏斜，考虑急性脑血管病，头颅CT未见异常，不除外脑干病变，进一步行头颅MRI示左小脑、右枕叶急性脑梗死。复查血常规、肝肾功能、心肌酶显示各项指标均显著增加，综合考虑不除外全身系统性疾病。

吴航宇主治医师：患者入院主要表现为休克，复习病史其入院前无大出血及大量体液丢失，不支持低血容量性休克；无过敏表现，可排除过敏性休克；未服用降压药及接触化学物品，亦可排除药物或化学品中毒所引起的休克。患者胸闷、胸痛，不排除因疼痛引起神经源性休克的可能；入院后血白细胞成倍升高，患者虽无发热，不能排除感染性休克；该患者入院前表现为胸闷、胸痛、憋气、心悸，查心电图、心肌酶均无明显异常，但超声心动图提示心包腔内

少量积液，故不除外急性心脏压塞引起心源性休克可能。

王玉红副主任医师：同意上述分析。12月31日胸部CT提示可能为主动脉夹层。患者在1月2日精神状态较好的情况下血压140～170/70～90 mmHg，考虑其既往应有高血压病史。动脉粥样硬化是主动脉夹层的重要促发因素，约3/4的主动脉夹层患者有高血压，老年男性发病率高。主动脉瘤若不压迫邻近组织，并无症状。夹层动脉瘤形成后，可向远段主动脉延伸，累及胸主动脉全长和腹主动脉及其分支；向近段主动脉延伸则累及冠状动脉和主动脉瓣，引致冠脉循环血流阻断或主动脉瓣关闭不全。夹层病变累及颈总动脉则产生脑缺血症状，缺血加重，可导致急性脑梗死。有的患者动脉瘤内层穿破入主动脉腔，则主动脉形成两个血流通道，主动脉壁剥离过程就不再发展，病情得到缓解。该患者12月31日至1月1日病情相对平稳，考虑该情况。患者死亡前表现去大脑强直状态，系中脑损伤的重要表现，考虑为主动脉夹层延展至椎基底动脉系统引起急性脑干损伤，这可能是导致患者死亡的原因。

冬兰教授：根据检验结果，肝、肾功能异常，考虑夹层扩展到腹腔脏器分支引起肝、肾供血不足，造成肝、肾功能损害；胸部CT示右肺下叶背侧膨胀不全，此为夹层压迫右侧支气管和右侧肺动脉产生肺不张。因本病例病因未明，故只能对症处理，予大量输液补充血容量及升压等抗休克治疗。但这正是主动脉夹层的禁忌。此措施可诱发或加速主动脉夹层的破裂出血。主动脉夹层血肿破裂至心包腔导致急性心包积液、心脏压塞，心脏骤停，这可能是导致患者死亡的原因。

周荣斌教授：该患者为65岁老年男性患者，既往存在隐匿高血压病史，以胸痛、晕厥伴有全身多器官损害为主要临床表现，需考虑急性心、脑血管疾病和全身系统性疾病，行相关辅助检查。(1)心电图可示左心室肥大，非特异性ST-T改变。病变累及冠状动脉时，可出现心肌急性缺血甚至急性心肌梗死改变。心包积血时可出现急性心包炎的心电图改变。(2)胸部X线检查示主动脉增宽，虽无诊断价值但可提示进一步检查。(3) CT及MRI检查均有很高的决定性诊断价值：CT扫描尤其更易发现腔内血栓及壁的钙化，并能显示动脉瘤与邻近结构如肾动脉、腹膜后腔和脊柱等的关系。(4)超声心动图对诊断升主动脉夹层分离具有重要意义，且易识别并发症(如心包积血、主动脉瓣关闭不全和胸腔积血等)。(5)主动脉造影对主动脉夹层定位诊断也有帮助，但具有潜在危险，需谨慎操作。

最后诊断：(1)主动脉夹层合并心包积液、腹腔积液；(2)急性脑梗死；(3)急性肾功能不全；(4)肝损伤；(5)肾结石(双侧)。

主动脉夹层指主动脉腔内的血液通过内膜的破口进入主动脉壁中层而形成的血肿。本病男性多见。在发病和扩展过程中，可引起相关脏器供血不足，夹层血肿压迫周围软组织或波及主动脉各大分支，可引起相应器官系统功能受损。对本患者而言，主动脉夹层血肿累及颈动脉导致脑灌注不足出现晕厥、左侧鼻唇沟变浅，口角向左偏斜。随着疾病的进展，夹层波及冠状动脉，出现左胸部疼痛、心悸，心电图示窦性心动过速，CK、MYO、TNI成倍增加；血肿破裂入心包引起心包积液。压迫右侧支气管及肺动脉引起呼吸困难、右侧肺不张。夹层破裂入腹膜腔引起腹腔积液。累及腹主动脉及其分支引起肝功能损伤：谷丙转氨酶和谷草转氨酶显著增加。累及肾动脉可有血尿。死亡原因考虑为：(1)主动脉夹层累及后循环系统，导致动脉完全闭塞，从而引发后循环缺血，并导致急性脑干梗死，抑制呼吸中枢，呼吸衰竭；(2)夹层血肿破裂至心包腔导致急性心脏压塞，心脏骤停。

(参加讨论医师：孙 明，吴航宇，
周荣斌，王玉红，冬 兰)
(孙 明，周荣斌 整理)