· 临床病理讨论 ·
Clinicopathological Conference

Multiple organ failure and death caused by lung infection in an elderly bedridden patient

(the 30th case)

Institute of Geriatric Cardiology of Chinese PLA General Hospital

Case report

An 83 years old man was admitted to the hospital because of the history of intermittent fever for two months and recurring for one day on December 22, 2007. The patient began to be febrile from October 2007, with the highest temperature to 40℃. He did not have the symptoms such as chilly, rhinocleisis, cough, expectoration, hemoptysis, night sweat, nausea and vomiting, palpitation, shortness of breath, chest pain, stomach-ache and diarrhea, thamuria, urgency and odynuria, etc. Each episode of fever lasted about one week. He had fever again because of accidental aspiration on December 21, 2007 with the highest temperature to 40℃. His temperature became nomal after indomethacin suppository 50mg was put in his anus. Then the man was admited to this hospital for "acute upper respiratory infection".

The patient had the history of cerebral infarction and Parkinson's syndrome, but he denied history of hypertension, diabetes mellitus, and coronary heart disease. He also denied history of hepatitis, typhoid fever, tuberculosis, or trauma. He had no history of drug and food allergy. He did not smoke or use illicit drugs, drank little. He had no recent history of travel to epidemic area or exposure to sick persons, pets, or other animals.

On examination, the temperature was 36.7℃, the pulse 76 beats per minute, the respiratory rate 20 times per minute, the blood pressure 170/90mmHg. He was in somnolent condition. There was no cyanosis. He had pharyngeal hyperaemia, but without paristhmion enlargement. The neck was soft, bilateral jugular veins did not engorge. The breath sounds of the lungs were coarse and low. Moderate and fine moist rales were heard at the bottoms of the two lungs. No cardiac murmur or pericardial rub was detected on auscultation. The abdomen was flat and soft, no tenderness and rebounding pain were found, liver and spleen were not enlarged. Other abdominal examinations showed no abnormality. The arms and legs were warm, with symmetric pulses and without edema. Muscle force of both lower extremities was grade 3 on a scale of 0 to 5 (higher numbers indicate better force). Arterial pulse of dorsa of two feet was good. Babinski's sign was negative.

Laboratory tests were performed and the results are shown in tables 1-4.

Two chest radiographs (12, 22 and 27, 2007) disclosed enlarged bilateral hili of lung.

After admission, the patient was given meropenem and ornidazole for combating infection, pyretolysis, atomization inhalation for diluting sputum and parenteral nutrition, but the fever still persisted. The patient presented tachypnea at 12:00, December 27, 2007. Oxygen saturation measured by pulse oxymeter progressively decreased to 76%. Oxygen saturation could not rise to normal even though he breathed 2-4 liters of oxygen per minute by nasal cannula. Moist rales and

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phlegm wheezing were heard in the lungs. The trachea was intubated by nose under the guidance of bronchofiberscope at 12:25 and continuous mechanical ventilation by respirator was given to supply pure oxygen, but pulse oxymeter only showed 77%—85% oxygen saturation. PEEP was 8 cmH_2O. Suction of sputum, turnover and percussion of back were strengthened, but the conditon of the patient was not ameliorated after these managements. The blood pressure was maintained at 83/47 mmHg under continuous infusion of dopamine hydrochloride by a microamount pump. Blood gas analysis results showed that pH was 7.140, PCO_2 38.0 mmHg, PO_2 64.4 mmHg, HCO_3^- 12.6mmol/L and BE −15.5 mmol/L. Sodium bicarbonate 150ml was infused intravenously. At 13:45, a right subclavian vein central venous line was placed. The central venous pressure was 4 cm H_2O and normal saline with glucose was infused. The temperature was 40.2℃. Ice blanket was spread under the patient to cool him. The temperature reduced to 38.6℃ at 4:00, December 28, 2007. The patient's heart rate fluctuated at about 140 beats per minute with sinus tachycardia. The blood pressure was maintained at 80/40 mmHg under the infusion of 2 liters of saline and continuous infusion of dopamine hydrochloride through central venous line. Pulse oxygen saturation was about 80%. The vital signs remained unstable and he was very critical during the night. Blood gas analyses at 19:00 showed pH 7.299, PCO_2 30.7mmHg, PO_2 48.0mmHg, SO_2 80.2% and BE −10.5mmol/L. Sodium bicarbonate was intermittently infused intravenously.

At 7:50, December 28, 2007, the cardiac monitor showed asystole, and the patient was found pulseless and unresponsive, blood pressure and pulse oxygen saturation could not be detected. Transthoracic cardiac massage was practiced. Adrenalin at the dose of 1mg and atropine at the dose of 1mg were given intravenously every 5 min. About 15 min later, autonomous sinus rhythm recovered with palpable distal pulse and systolic blood pressure of 100 mmHg. However, he did not awake and was not alert. Adrenalin and dopamine by intravenous pumping were continued. At 8:25 of the morning, large amount of bloody gastric fluid was drawn from gastric tube. The patient was diagnosed as upper gastrointestinal bleeding. Haemostasis therapy was initiated with reptilase 1kU and omeprazole 40mg intravenous injection. One hundred milliliter of icy saline was repeatedly injected into and pumped out his stomach for gastric lavage until the color of gastric juice fainted. Thrombase 2000U was injected into his stomach by the gastric tube. At the same time, 200ml of RBC suspension was emergently given. Shortly thereafter, the cardiac monitor showed asystole again and his blood pressure fell to 0 mmHg at 11:00. Large amount of fresh blood effused from his mouth and nose. About 30 min later, vital signs disappeared and he was declared to be dead.

Sputum culture (6:30, Dec. 26, 2007): *Streptococcus viridans* and *Monilia albicans*.

Blood culture (00:14, Dec. 27, 2007): both aerobe and anaerobe were negative.

Clinicopathological discussion

Dr. ZHU Bingpo: The patient was an elderly man and presented hyperpyrexia up to over 40℃ which unresponded to antipyretics. The lung infection caused these conditions and led to his death because of respiratory and circulatory collapse.

Dr. GAO Lei: The patient suffered from severe infection after admission. The laboratory tests showed that WBC count was not elevated, but decreased. Furthermore, his body temperature could not be controled well. In addition to the common bacterial infection, the possibility of infections caused by rare bacteria and virus and malignant tumor can not be ruled out.

Dr. GAO Wei: This 83-year-old patient was admitted mainly because of fever. He was diagnosed as aspiration pneumonia. The infection was severe and progressed rapidly. After admission, the fever rose up to 40.2℃. However, the white blood cell count was not high and decreased in the course of progress of the disease. This may be

caused by severe pulmonary infection and low resistance of the patient. The pulse oxygen saturation could not rise to over 90% after continuous assisted respiration by respirator, and inhalation of 100% oxygen was ineffective. Thus, adult respiratory distress syndrome (ARDS) is the most likely explanation of this patient's hyoxemia. Severe infection induced septic shock. On the basis of hypotensive shock, disseminated intravascular coagulation (DIC) appeared, which caused digestive tract and respiratory tract massive hemorrhage. The pathophysiologic process was consistent with the hypothesis of "lung initiation" of multiple organ dysfunction syndrome in the elderly (MODSE) proposed by Professor Wang Shiwen. The direct death cause was severe pulmonary infection and septic shock.

Dr. ZHAO Yusheng: The patient was an elderly man who had the history of cerebral infarction. He was in bedridden condition for a long time, and had the possibility of accidental aspiration. He had had high fever twice before this admission and the disease was very severe this time. Possibility of avian influenza had been ruled out in fever OPD. According to his medical history, there was pulmonary infection. Because the patient was an elderly man, and chest radiograph showed dilated hili of lung, the possibility of tuberculosis or tumor which could induce obstructive pneumonia could not be ruled out. After the admission, the patient manifested apathy, mental state deteriorated progressively, high fever persisted. The breath sound of the lungs was low and little dry and moist rales were heard in the two lungs. Respiratory embarrassment appeared at about 12 o'clock of December 27, 2007. The respiratory rate was 30-40 times per minute, heart rate 110-120 beats per minute, pulse oxygen saturation fell to 85%, piebaldism appeared all over his body. Trachea was intubated by nose under guidance of bronchofiberscope and respirator was used to help his breath with pure oxygen inhalation, but his pulse oxygen saturation fluctuated only between 77%-85%. The cause of his respiratory embarrassment may be ARDS. However, other diseases, such as pulmonary embolism, sputum embolism and acute left heart failure could not be excluded. By referring to his chest X-ray findings and past medical history, we could find that these diseases were less possible.

The efficacy of strengthening suction of sputum, turnover and percussion of back were unsatisfactory. Blood pressure and pulse oxygen saturation did not rise. The patient experienced ARDS because of the septic shock. The clinical manifestaions, such as severe infection, low blood pressure, rise of D-dimer from 0.61mg/L to 7.81 mg/L, prolongation of APTT to >180s and decrease in platelet to 41×10^9/L, indicated appearance of DIC, which induced digestive tract and respiratory tract hemorrhage. It is considered that DIC is the reason of his death. Some experiences and lessons can be learned from the management of the patient. The bedridden elderly person frequently develops lung infection because of accidental aspiration. It is difficult to cure this kind of infection. Pulmonary infection in elderly patients is apt to develop MODSE. It was suggested that when infection and sepsis occur, continuous renal replacement therapy (CRRT) at bedside may be helpful to filtering out some inflammatory cytokines and to controlling the patient's sepsis and unbalanced inflammatory reaction.

Dr. CHEN Mingzhi: The patient's pathological slides showed that alveolar cavities of the inferior lobes of the two lungs were filled with neutrophils, phagocytes and edematous fluid. Individual bronchiolar lumina were filled with neutrophils. Small vessels of lung interstitium dilated and congested. There were fibrinous exudate in alveolar cavities, alveolar dilatation, alveolar septum breakage and alveolar fusiong in upper lobes of both lungs and the middle lobe of right lung. Heart slides showed that intima of coronary artery had fibrous eccentric thickening and calcification of various degrees. Tunica media was atrophic. Tunica externa had no significant change. The cause of death is consistent with clinical diagnosis. The lower lobes of both lungs presented diffuse infiltra-

tion of neutrophils, and hyaline membrane formation was found in the middle lobe of right lung, the lung function was severely impaired, leading to respiratory failure. According to his medical history, the septic shock caused by lobar pneumonia led to circulation failure and ultimately to death.

Main pathological diagnosis: (1) lobar pneumonia of lower lobes of the two lungs; (2) emphysema of upper lobes of the two lungs and the middle lobe of right lung; (3) diffuse alveolar damage of the middle lobe of right lung accompanied with hyaline membrane formation; (4) hypertension (stage 3), hypertensive heart disease with 2.5cm thickness of left ventricle wall, arteriolosclerosis of arteriales of renal, pancreas and adrenal gland capsule; (5) coronary arteriosclerotic heart disease: coronary atherosclerosis, the degree of stenosis of LAD was grade Ⅰ-Ⅲ and that of RCA was grade Ⅰ-Ⅱ.

Dr. ZHAO Yusheng: Despite great advancement in the understanding of the pathophysiology of sepsis and in the development of novel therapeutic methods, mortality of sepsis still remains unacceptably high. Adequate laboratory diagnosis is indispensable for the improvement of this situation. It was for a long time that the measurement of C-reactive protein as a major acute phase protein and differential WBC count were used as the basis for diagnosing inflammatory and infectious disease. Because of better understanding of the role of immunological disorder in this disease, several new markers are now available for routine clinical laboratory diagnosis. They include the cytokines interleukin (IL) -6, IL-8, procalcitonin and the LPS-binding protein. These novel markers used in clinical diagnosis may be helpful to the early discovery and diagnosis of sepsis and systemic inflammatary reaction syndrome and elevating the cure rate and survival rate.

(Translator: GAO Wei)

老年卧床患者肺炎致多脏器衰竭及死亡

1 病例摘要

患者，男性，83岁，主因“间断发热2个月余再发1d余”于2007年12月22日入院。患者曾于2007年10月始无诱因出现发热，体温最高达40℃。无畏寒、寒战、鼻塞、流涕，无咳嗽、咳痰、咯血、盗汗，无头痛、头晕、恶心、呕吐，无心悸、胸闷、胸痛、呼吸困难，亦无腹痛、腹泻、腰痛，无尿急、尿频、尿痛等，每次发热时间一般持续1周。2007年12月21日因误吸再次出现发热，体温最高达40℃，给予消炎痛栓剂50mg纳肛后体温降至正常。门诊以“急性上呼吸道感染”收入院。既往有脑梗死及帕金森综合征病史，否认高血压、糖尿病、冠心病病史。否认肝炎、伤寒、结核等传染病病史，否认外伤史。否认药物及食物过敏史，预防接种史不详。无疫区、疫水接触史，否认吸烟饮酒史，否认吸毒史。

体温36.7℃，脉搏76次/min，呼吸20次/min，血压170/90 mmHg，嗜睡状态，语言差，平卧位。口唇无发绀，咽部充血，扁桃体不大。颈软，双侧颈静脉无怒张，双肺呼吸音减弱、粗糙，两肺底可闻及中细湿啰音。心前区无隆起，未扪及心前区震颤，心率76次/min，律齐，各瓣膜听诊区未闻及病理性杂音。腹平软，全腹无压痛及反跳痛，肝脾不大，腹部移动性浊音阴性，肠鸣音无增强或减弱，腹主动脉、肾区及股动脉未闻及明显血管杂音。双下肢不浮肿，双下肢肌力Ⅲ级。双侧足背动脉搏动好，巴氏征未引出。

实验室及辅助检查：实验室检查结果见表1～4。X线检查(2007年12月22,27)：双肺门增大。

入院后选用美罗培南、奥硝唑抗感染，并退热、

表1 血浆凝血指标标化

时间	D-Dimer (mg/L)	APTT(s)	血浆纤维蛋白原(g/L)
2007-12-25	0.61	46.8	5.03
2007-12-28	7.81	>180	3.68

表2 血常规化验项目变化

时间	血红蛋白(g/L)	白细胞($\times10^9$/L)	中性粒细胞(%)	淋巴细胞(%)	单核细胞(%)	血小板($\times10^9$/L)
2007-12-22	163	7.16	0.706	0.232	0.061	203
2007-12-23	164	4.78	0.549	0.372	0.079	192
2007-12-25	167	6.36	0.44	0.498	0.06	168
2007-12-27	137	1.16	0.25	0.595	0.069	131
2007-12-28	98	3.83	0.36	0.6	0.02	41

表3 血生化指标变化

时间	尿素(mmol/L)	肌酐(mmol/L)	TnT(ng/L)	血清钾离子(mmol/L)
2007-12-22	7.23	76	<0.01	3.33
2007-12-25	9.75	78	0.014	3.62
2007-12-27	22.47	177	0.046	4.82

表4 血气分析指标变化

时间	pH	$Pa(O_2)$(mmHg)	$PaCO_2$(mmHg)	SaO_2(%)	HCO_3(mmol/L)
2007-12-23-23:01	7.423	68	30	94.6	19.1
2007-12-25-11:51	7.433	69	29	95	1931
2007-12-27-10:37	7.433	74	27	95.4	17.4
2007-12-27-14:12	7.14	64	38	82.8	12.6
2007-12-27-19:17	7.299	48	31	80.2	14.7
2007-12-27-21:15	7.431	54	33	88.9	21.1
2007-12-27-23:44	7.412	44	33	78	20.6
2007-12-28-6:12	7.402	41	30	80	18.5
2007-12-28-8:09	7.221	68	63	86.2	25.3
2007-12-28-10:35	7.099	19	73	23.5	22.2

雾化吸入稀释痰液、加强营养等治疗，仍持续高热。于2007年12月27日12:00呼吸急促，氧饱和度进行性下降，最低至76%，加大吸氧浓度无明显上升，听诊满肺湿啰音及痰鸣音。12:25在纤维支气管镜引导下给予经鼻气管插管，持续呼吸机辅助呼吸，调整吸氧浓度为100%，脉氧饱和度波动在77%～85%，呼气末正压8cmH_2O，加强吸痰、翻身、叩背，效果均不理想。血压在持续多巴胺泵入下维持在83/47mmHg左右。血气分析提示：pH 7.140，PCO_2 38.0 mmHg，PO_2 64.4 mmHg，HCO_3^- 12.6mmol/L，BE－15.5mmol/L，补充碳酸氢钠150ml。13:45行右侧锁骨下大静脉置管，测中心静脉压为4cmH_2O，快速扩容。测体温最高40.2℃，间断启用电冰毯降温。2007年12月28日凌晨4:00体温降至38.6℃。患者心率一直波动在140次/min左右，血压在多巴胺持续泵入下维持在80/40mmHg左右，脉氧饱和度约80%。夜间病情危重，患者一直处于低氧、低血压状态，给予多巴胺、654-2持续泵入。19:00复查血气分析，pH 7.299，PCO_2 30.7mmHg，PO_2 48.0mmHg，SO_2 80.2%，BE－10.5mmol/L，间断给予碳酸氢钠静滴。2007年12月28日7:50心率降为0，血压、脉氧饱和度测不到，给予胸外按压，反复肾上腺素、阿托品静推，8:00恢复为自主心律，多巴胺持续泵入，间断静脉注射肾上腺素。8:25从胃内抽出大量血性液体，考虑急性上消化道出血。给予止血治疗，立止血1kU，静脉注射；奥美拉唑40mg，静脉注射。100ml冰盐水反复灌洗胃部直至胃液颜色变淡，给与凝血酶粉2000U胃管注入。急申请悬浮红细胞200ml。11:00心率再次降为0，血压、脉氧饱和度测不到，口腔涌出大量鲜血，11:30经抢救无效，患者临床死亡。

2 临床病理讨论

朱冰坡医师：患者84岁高龄，持续高热，最高体温达40℃，用退热药物疗效不佳，感染最终导致呼吸循环衰竭死亡。

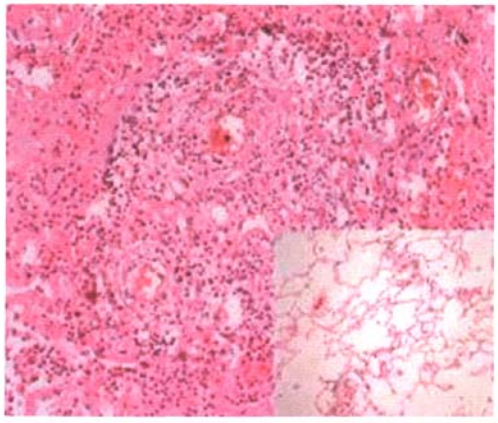
高磊医师：患者入院后感染重，白细胞一直不升，反而下降，体温控制不佳，除普通细菌感染外不

排除少见细菌、病毒感染及恶性肿瘤可能。

高伟医师：患者主因发热入院，诊断吸入性肺炎，感染重，病情进展快，持续发热，入院后体温最高达 40.2℃。血象不高，病情进展过程中白细胞下降，可能是严重肺部感染、机体抵抗力差引起。给予持续呼吸机辅助呼吸后脉氧饱和度始终不能上至 90%以上，100%浓度吸氧无效，考虑有成人呼吸窘迫综合征（adult respiratory distress syndrome，ARDS）的可能，严重感染导致感染性休克，低血压休克基础上出现弥散性血管内凝血（disseminated intravascular coagulation，DIC），合并消化道、呼吸道大出血。该患者符合王士雯院士提出的老年多器官衰竭（MODSE）"肺启动"假说。直接死亡原因为严重肺部感染，感染性休克。

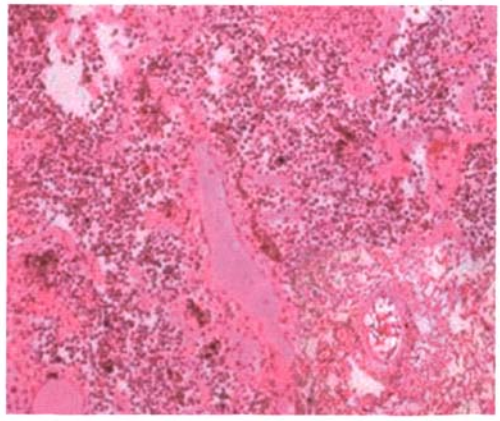
赵玉生医师：患者为老年男性，有脑梗死病史，处于卧床状况，有误吸可能。入院前已反复出现两次高热，病情来势凶猛，反复发作，已在发热门诊排除人禽流感可能。结合病史肺部感染存在，因患者为老年患者，胸片提示肺门增大，不排除肺部肿瘤、结核的可能，在此基础上出现阻塞性肺炎。入院后对患者印象：神志淡漠，精神状况进行性下降，持续高热，用退热药效果不佳，查体两肺呼吸音低，少量干湿性啰音。于 2007 年 12 月 27 日 12:00 出现呼吸窘迫，呼吸频率 30～40 次/min，心率 110～120 次/min，脉氧饱和度降至 85%，全身出现花斑，在纤维支气管镜下给予经鼻气管插管，持续呼吸机辅助呼吸，调整吸氧浓度为 100%，脉氧饱和度始终波动在 77%～85%，造成呼吸窘迫原因首先考虑 ARDS 可能，不排除肺栓塞、痰栓、急性左心衰可能，但结合胸片、既往病史考虑后者可能性不大。给予加强吸痰、翻身、叩背，效果均不理想，血压不能回升，在感染性休克基础上出现 ARDS。患者的临床表现如严重感染、低血压，D-二聚体由 0.61mg/L 升至 7.81mg/L，部分凝血活酶时间延长至＞180s（表 1），血小板减少到 41×10^9/L（表 2），提示出现 DIC，引起消化道、呼吸道大量出血。直接死亡原因考虑 DIC。从该患者吸取经验教训总结如下：对于长期卧床老年患者，经常发生误吸诱发肺部感染，一旦发生感染，治疗较困难。并且老年人肺部感染容易诱发老年多器官功能衰竭。文献提示当发生感染、脓毒症时，可考虑行连续性肾脏替代治疗（continuous renal replacement therapy），滤除致炎细胞因子，控制脓毒症及失衡的炎症反应。

陈明枝医师：该患者病理切片显示两肺下叶肺泡腔内充满中性粒细胞、吞噬细胞及水肿液，个别小支气管腔内充满中性粒细胞。肺间质小血管扩张、充血（图 1）。两肺上叶及右肺中叶肺泡腔内纤维素样渗出物，肺泡扩张、肺泡间隔断裂、肺泡融合（图 2）。心脏切片显示冠状动脉内膜不同程度的纤维性偏心状增厚及钙化，中膜萎缩，外膜无显著变化（图 3）。死亡原因与临床诊断基本一致。双肺下叶弥漫性中性粒细胞浸润，加之右肺中叶透明膜形成，肺功能严重受损，出现呼吸衰竭；结合临床病史，因大叶性肺炎导致的感染性休克，造成了患者末梢循环衰竭，最终导致患者死亡。



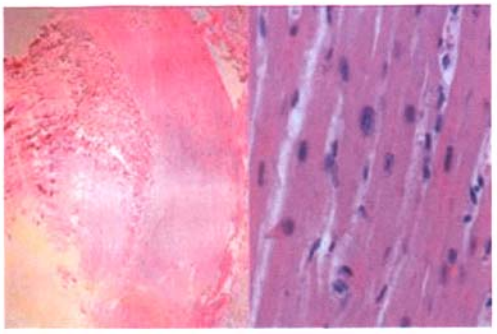
显示肺泡腔内充满中性粒细胞、吞噬细胞及水肿液，个别小支气管腔内充满中性粒细胞。肺间质小血管扩张、充血。右下角插图：视野中央小动脉内见骨髓组织，周围肺组织个别肺泡腔内见吞噬细胞，间质小血管轻度扩张、充血

图 1 肺病理切片（1）



视野中央显示肺泡腔内纤维素样渗出物，周围肺泡腔内见吞噬细胞及中性粒细胞。右下角：肺泡扩张、肺泡间隔断裂、肺泡融合

图 2 肺病理切片（2）



左侧：显示冠状动脉内膜不同程度的纤维性偏心状增厚及钙化，中膜萎缩，外膜无显著变化。右侧：显示心肌细胞排列规整，可见横纹，大部分心肌细胞胞浆内见脂核素沉积。部分心肌细胞核增大，略具异型性

图3 冠状动脉及心肌病理切片

主要病理诊断：(1)双肺下叶大叶性肺炎。(2)双肺上叶、右肺中叶肺气肿。(3)右肺中叶弥漫性肺泡损伤伴透明膜形成。(4)高血压病Ⅲ级：①高血压性心脏病，左心室肌壁厚2.5cm；②肾脏、胰腺、肾上腺被膜小动脉硬化。(5)冠状动脉硬化性心脏病：冠状动脉粥样硬化，狭窄程度：左前降支Ⅰ～Ⅲ级，右主干Ⅰ～Ⅱ级。

赵玉生医师：尽管我们现在对于脓毒症病理生理过程的了解以及开发新的治疗方法等方面已经取得了很大进展，但临床上脓毒症的死亡率还是高得惊人。要改善这种现状，准确的实验室诊断是必不可少的。很长时间以来，作为主要的急性期蛋白的C-反应蛋白的测定以及白细胞分类计数一直被用作炎症及感染性疾病诊断依据。由于对免疫失调在这种疾病中的作用已经有了更好的了解，目前有几种新的标志物已经常规应用于临床实验室诊断中，包括白介素-6、白介素-8、原降钙素和脂多糖结合蛋白。这些新的标志物用于临床可能有助于早期发现、早期诊断脓毒症和全身炎症反应综合征，提高治愈率和生存率。

（参加讨论医师：朱冰坡、高磊、高伟、赵玉生、陈明枝）

（高伟 整理）

（上接第430页）

体，在DIC早期给予小剂量肝素持续泵入，间断输注血小板，因而及时纠正了DIC。(6)抓主要矛盾：本例病情复杂多变，治疗矛盾重重，如：应用抗菌素与诱发真菌感染、肠道菌群失调；静脉用药及营养与心衰；消化道出血与进食；低血压与利尿；肾衰与药物的肾损害；高血糖与激素等。病情的变化是动态的，矛盾的主次也随之而变。在某个特定阶段要明确目前对患者危害最大的是什么，并采取强有力措施，对其毒副作用应尽可能防范，但不必考虑过多。例如：患者有多年糖尿病史，在感染性休克阶段血糖波动很大，为了纠正休克间断应用小剂量地塞米松，同时监测8个时段的血糖，随时调整胰岛素用量，这样既有助于纠正休克，也控制了血糖；患者血压靠药物维持，无尿又需要用利尿剂，在加大升压药剂量维持血压的前提下，持续交替泵入呋塞米和特苏尼，使尿量逐渐增加，对血压也无明显影响。

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