· 临床病理讨论 ·

Clinicopathological Conference (the 47th case)

An elderly male patient with pituitary apoplexy complicated with pneumonia and multiple organ failure

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Case presentation

A male patient, 71 years old, was admitted to Department of Respiratory Diseases, Shengjing Hospital of China Medical University on February 20, 2010, with the complaint of headache, nausea and vomiting, accompanied with fever, cough and expectoration for 3 days.

Past history The patient reported previous history of hypertension 50 years ago with the highest 170/60mmHg, type 2 diabetes mellitus 13 years ago(insulin administration for 3 years), tuberculosis 3 years ago(recovered following systemic treatment for 1 year), and chronic renal failure 6 months ago.

Physical examination Body temperature was 36.5℃, pulse rate 78 beats/min, respiratory rate 18 times/min, and blood pressure 200/60 mmHg. The patient was conscious but a little obtuse. No jaundice, rash or hemorrhage was seen on his skin. No superficial lymph node was palpated. Mild facial edema and pale conjunctiva were seen without cyanosis on lips. No rigidity was observed on his neck. The moist rales in right upper lung were heard. His cardiac rhythm was regular at 78 beats/min. His abdomen was flat and soft with no tenderness or rebound tenderness. No mass was palpated. The liver and spleen were not palpable below the costal margin. Murphy's sign was negative. The shifting dullness was negative. There was no edema on his legs. Brudzinski's signs were negative.

Laboratory examination Blood routine test showed that white blood cells(WBC) were 9.4×10^9/ L, neutrophil granulocytes 0.81, and hemoglobin (HGB) 10^4 g/L. Urine routine test showed that urine protein was 2+, red blood cells(RBC) 2.81/HP. The blood biochemical test showed that CK was 366.9U/L, CKMB 24U/L, troponin 0.03μg/L, serum K^+ 5.71mmol/L, Na^+ 131mmol/L, capillary fasting blood glucose 4.3-5.2 mmol/L, postprandial blood glucose 6.0-8.4mmol/L, creatinine 254.6μmol/l, BUN 27.01mmol/l. Coagulation function was normal. Cranial CT scan displayed a mass in sella turcica and multiple lacunar infarction(?) in right frontal and temporal lobes, and cerebral atrophy. Pulmonary CT scan revealed secondary tuberculosis(?) with multiple proliferative lesions at right upper lung, local inflammatory manifestation, pleural effusion on both sides and pleural thickening on the right side.

Admission diagnosis (1) pneumonia; (2) grade 3 hypertension (extremely high risk); (3) type 2 diabetes mellitus; (4) chronic renal failure(CKD4 phase); (5) electrolyte disorder with hyperkalemia and hyponatremia; (6) pleural effusion on both sides with unknown cause; (7) multiple lacunar infarction and cerebral atrophy; (8) pituitary tumor.

Treatment (1) Antiinfection. Piperacillin and Tazobactam Sodium were given intravenously 4.5g q8h. (2) Expectoration. Expectorants were administrated orally 300mg 3 times daily; (3) Antihypertension. Nifedipine controlled-release tablets were administrated orally 30mg pounce daily; (4) Antiplatelet therapy. Aspirin was administrated orally 100mg pounce daily. (5) Lowering the non-protein nitrogen. Activated charcoal tablets(1.2g) and compound α-Ketoacids(4g) were administrated orally three times daily. (6) Regulating the electrolyte disorder. 5% Glucose saline solution 250ml+50% glucose 60ml+insulin 14U *iv* drip + furosemide 40mg were administrated intravenously.

Further investigation (1) Doppler ultrasonography to evaluate the heart function and 24h ECG monitoring to observe the heart rhythm; (2) Color ultrasonography for kidney, ureter and bladder; (3) Close surveillance of serum potassium and renal function and blood gas analysis.

On February 21, the patient had watery diarrhea (7 times during the night) without abdominal pain. He had persistent headache, somnolence and apathy, while no nausea or vomiting. Blood pressure was 125/60mmHg, pulse rate 80beats/min, and respiratory rate 20times/min. Pupils on both sides were about 2.0 mm in diameter with normal light reflex. No neck rigidity was observed. Signs of Brudzinski, Kernig, Babinski, and Hoffmann reflex were all negative. Laboratory test showed occult blood test ±, disordered bacterial colonies in digestive tracts, serum K^+ 4.62 mmol/L, and Na^+ 129 mmol/L.

In the morning of February 22, the patient had impaired mental status. The causes might included the following ones. (1)Hypothyroidism. Thyroid function test was needed because the patient had facial edema with apathetic expression, and the thyroid dysfunction could not be excluded. (2) Brain lesion. Head MIR + diffused scan +Flair and cerebrospinal fluid (CSF) test were needed because the results of previous CT scan could not explain the patient's mental condition; (3)Metabolic acidosis. Artery blood analysis showed pH 7.315 and HCO^-15.9mmol/L. The metabolic

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acidosis was associated with chronic renal failure and infection. Artery blood analysis showed PCO_2 31mmHg, so pulmonary encephalopathy can be excluded. Although the patient had previous history of diabetes, recently his blood glucose was almost normal without ketones in his urine. Non-ketotic hyperosmolar coma and diabetic ketoacidosis (DKA) were excluded.

On February 23, thyroid function test revealed FT3＜1.54 pmol/L, FT4 8.02pmol/L, TSH 2.25 μIU/L. The diagnosis of hypothyroidism was made and hypopituitarism was suspected. Then the patient was transferred to the Department of Endocrinology.

On February 24, the patient received laboratory examination: pH 7.312, PO_2 117.2 mmHg, PCO_2 26.8 mmHg, factual HCO_3^- 13.3mmol/L, WBC 7.2×10^9 /L, Hb 10.3 g/L, serum total protein 55g/L, albumin 28.6g/L, Cr 315.4 μmol/L, BUN 17.4 mmol/L, serum K^+ 3.02 mmol/L, Na^+ 130 mmol/L. ACTH was 21.15, 16.92, 24.87 pg/ml at 8: 00, 16: 00, and 0: 00 respectively, and cortisol 6.71, >62.8, and 4.15 μg/L respectively. The serum levels of follicular stimulating hormone (FSH), luteinizing hormone(LH), testosterone (T), prolactin, progesterone (P) and estrogen (E) were very low. Cerebrospinal fluid(CSF) biochemistry revealed Cl^- 115 mmol/L, glucose 2.35 mmol/L, protein 0.7 g/L. CSF routine test was normal. No bacterium was detected in CSF. Pleural fluid was an exudate with weak positive Rivalta reaction, leukocytes 65×10^6/L, protein 18.3 g/L, glucose 3.28 mmol/L, Cl^- 95 mmol/L, CY21-1 14.57, CA125 348.1, ADA 9.03U/L, LDH 102.1U/L. No bacterium or fungus was found in the sputum. CT scan of lung showed increased pleural effusion. PDE indicated left ventricular (LV) systolic dysfunction with ejection fraction 35%. MRI of head showed a mass(34mm × 22mm × 19mm) in pituitary gland with hemorrhage. The patient had right temporal hemianopsia.

Diagnosis (1) pituitary apoplexy, pituitary tumor; (2) type 2 diabetes; (3) diabetic nephropathy, chronic renal failure(CKD4 phase); (4) pneumonia; (5) Grade 3 hypertension (extremely high risk); (6) pleural effusion; (7) electrolyte disorders with hyperpotassium, hypopotasium and hyponatremia; (8) coronary heart disease, arrhythmia; (9) heart failure degree Ⅲ; (10) multiple lacunar infarction, cerebral atrophy.

Treatment The patient received low quality-protein and diabetic diet, antibiotics, activated charcoal tablets and compound α-Ketoacids po to lower the creatinine, $NaHCO_3$ to correct metabolic acidosis, fatty emulsion to supply energy, aspirin and Prostaglandin E1 to improve microcirculation, albumin supplement, hydro-cortisone 100mg *q8h iv* for hypopituitary stroke and L-thyroxin 25μg *qd* orally for hypothyroidism. On February 26, the patient's mental status was obviously improved with better appetite. His body temperature dropped to normal with elevated blood glucose. Aspart and glargine insulin analogues were then given to control blood glucose, and his serum sodium gradually returned to normal. Prednisone was given 10mg *tid* orally instead of Hydrocortisone.

Clinical discussion

Prof. HAN Ping from Department of Endocrinology: The patient had a long history of hypertension and diabetes complicated with pneumonia and multiple organ dysfunction, such as the heart and kidney. At present, the diagnosis of hypopituitarism could be definitely made because the patient had low hormone levels of pituitary-thyroid axis and pituitary-testis axis. The serum cortisol level was slightly elevated instead of reduced, which may be caused by the stress state of the patient. The causes of hypopituitarism might be the compression of pituitary gland by the large and bleeding tumor. It might be non-functional tumor because the prolactin and growth hormone levels were normal as well as other hormones. The patient had hypoglycemia and hyponatremia in recent days, so it was considered that the dosage of glucocorticoid was not enough. Regarding the patient's heart and kidney dysfunction, the amount of fluid infusion should be controlled. Prednisone 10mg *tid* orally was suggested and L-thyroxin dose was increased to 50 μg *qd* orally. The patient's response should be closely observed.

Prof. LIU Yunhui from Department of Neurosurgery: Considering his heart and lung dysfunction, the patient could not tolerate the pituitary tumor resection. According to the MRI image, the most part of the tumor was growing toward sphenoid parasinus. In such situation, the tumor rarely grew outward, so it would seldom induce intracerebral hypertension. Regular re-examination is strongly recommended rather than immediate tumor resection. Besides, because the tumor was in a special place and in stable size, the bleeding may stop spontaneously. The most important point was to treat the dysfunction of pituitary gland so as to improve his immune system and general condition. The patient should be under close surveillance with conservative medical treatment. Hormone replacement therapy is strongly recommended.

Dr. YANG Zhiyong from Department of Cardiology: Diagnosis of heart dysfunction was definitely established for the patient. Fluid intake should be strictly controlled. Furosemide, a diuretic agent, was given 40mg *bid iv*. Dopamine was given *iv* continuously to improve kidney blood circulation. In respect that the patient had hypokalemia, potassium chloride was give 2.0 g *tid* orally and antisterone, 20mg *qd po*, to keep potassium at normal level. Meanwhile, serum potassium should be tested every day to exclude hyper- or hypokalemia. Hemodialysis should be performed if necessary.

Prof. JIAO Guangyu from Department of Respiratory Diseases: The patient had fever at admission. But he had no fever in recent few days, only with mild cough and a small amount of sputum, so it was of no need to change antibiotics. The first thing right now was to control his heart failure. If his

cough and expectoration got worse, or the fever reoccurred, the antibiotics should be changed to more powerful one to cover more bacterial species. Further sputum and blood tests should be performed to exclude fungus infection. Pleural effusion should be associated with hypoproteinemia, so supplement of albumin is necessary. In addition, patient's cardiac function should be closely surveyed, so furosemide should be used.

Dr. LIU Jun from Department of Surgery: The pleural effusion was considered to be associated with heart and renal failure. The patient still had symptom of breath shortness. The artery blood-gas analysis should be performed again to see if there was retention of carbon dioxide. Next, anti-asthma medicine should be given continuously, heart and renal failure be controlled, and the electrolyte disorders be prevented. The pleural fluid of 1000ml had already been drawn out. It was not necessary to manage immediately the closed thoracic drainage.

Summary (1) The patient was still in serious situation, so he should be under close surveillance to monitor the vital signs. (2) The antibiotics should be given continuously to control pulmonary infection. (3) diuretics, dopamine, aspirin and prostaglandin E1 should be administrated to improve heart and renal function. (4) Activated charcoal tablets and compound α-Ketoacids should be given to lower the non-protein nitrogen. (5) Potassium chloride 1. 5g *tid* orally, and antisterone 20mg *qd* orally to regulate blood electrolyte inbalance. (6) Prednisone 10 mg *tid* orally, and thyroxin 50ug to replace hormone deficiency. (7) Albumin supplement be performed.

The patient's pneumonia had already recovered for 1 week, and his heart and renal function was obviously improved with normal electrolytes and good blood glucose control. He was discharged from the hospital on March 8. Oral hormone replacement therapy (prednisone and thyroxin) and regular follow-up visit were ordered.

(Translator: HAN Ping)

垂体卒中伴肺炎和多脏器功能衰竭 1 例

1 病历摘要

患者, 男, 71岁, 主因"头痛、恶心、呕吐伴发热、咳嗽、咳痰3天"于2010年2月20日收入我院呼吸内科住院治疗。

既往史: 50年前患高血压, 最高达170/60 mmHg（1 mmHg=0.133 kPa）; 13年前患糖尿病, 3年前应用胰岛素, 血糖控制良好; 3年前患肺结核, 已治愈; 半年前发现肾功能不全。

查体: 体温37.3°C, 脉搏78/min, 呼吸18/min, 血压200/60 mmHg, 神志清, 反应迟钝, 查体合作。周身浅表淋巴结未触及, 颜面轻度水肿, 睑结膜苍白, 巩膜无黄染, 口唇无发绀, 无颈强直, 右肺上野可闻及少量湿啰音, 心率78/min, 节律齐, 腹软, 无压痛, 肝脾肋下未及, 肝肾区无叩痛, 双下肢无水肿, 巴氏征阴性。

辅助检查: （1）血常规: WBC 9.4×10^9/L, N 80.10%, Hb: 10^4g/L; (2) 尿常规: 尿蛋白＋＋, RBC: 2.81/HP; (3) 心肌酶谱: CK 366.9 U/L, CKMB 24 U/L; （4）肌钙蛋白0.03 μg/L; （5）血清K^+5.71 mmol/L, Na^+131 mmol/L; (6) 末梢空腹血糖4.3～5.2 mmol/L, 三餐后2 h血糖6.0～8.4 mmol/L; 7）肾功能: Cr 254.6 μmol/L, BUN 27.01 mmol/L; (8) 凝血五项: 未见明显异常; （9）头颅CT: 鞍区改变, 右额、颞叶梗死？脑白质疏松、脑萎缩; （10）肺CT: 右肺尖多发增殖为主病变, 继发型肺结核？双肺散在炎症, 双侧胸腔积液、左侧略多, 右侧胸膜增厚。

入院诊断: （1）肺炎; （2）高血压病3级（极高危险组）; (3) 2型糖尿病; (4) 肾功能不全（CKD4期）; （5）离子紊乱（高钾血症、高钠血症）; （6）胸腔积液性质待定; （7）多发性腔隙性脑梗死、脑萎缩; （8）垂体瘤。

给予处置: （1）抗感染: 0.9%生理盐水100 ml＋哌拉西钠-他唑巴坦（商品名: 联邦他唑仙）静脉滴注, 每8小时4.5 g; (2) 化痰: 稀化黏素（商品名: 吉诺通）300 mg, 一日3次, 口服; (3) 控制血压: 硝苯地平（商品名: 拜新同）30 mg, 一日1次, 口服; (4) 抑制血小板聚集: 阿司匹林100 mg, 一日1次, 口服; (5) 减少非蛋白氮: 药用炭1.2 g, 一日3次, 口服; 复方α-酮酸片（商品名: 开同）4g, 一日3次, 口服; (6) 降低血钾: 5%葡萄糖氯化钠溶液250 ml＋50%葡萄糖溶液60 ml＋胰岛素14U静脉滴注, 呋塞米40 mg, 静脉注射。进一步完善检查: （1）多普勒超声心动图, 动态观察心电图变化; (2) 完善双肾输尿管、膀胱、前列腺彩超; (3) 注意监测血钾, 肾功能及血气分析。

2月21日夜内共腹泻7次, 为黄色水样便, 无腹痛。患者仍持续头痛, 嗜睡, 神志淡漠, 无恶心呕吐。监护显示: 血压125/60 mmHg, 脉搏80/min, 呼吸20/min, 双侧瞳孔直径约为2.0 mm, 对光反射存在, 颈软无抵抗, 布氏征、克氏征、巴氏征、霍夫曼征均为阴性。粪便常规: 潜血±, 余值正常; 化验显示: 肠道菌群, 中度失调; 血清K^+4.62 mmol/L; Na^+129 mmol/L。

2010年2月22日上午, 呼吸科焦光宇主任查房分析患者神志淡漠原因: (1) 患者现有颜面部轻度水肿, 且神志淡漠, 不能排除甲状腺功能减低症, 需行甲状

腺功能系列检查；（2）不能排除颅脑病变：现头CT结果不能解释患者目前的神志状态，需行头MIR平扫＋弥散＋Flair，同时腰椎穿刺行脑脊液检查；（3）今日血气分析示PH 7.315，实际碳酸氢盐15.9 mmol/L，考虑代谢性酸中毒与肾功不全有关；患者尽管现有肺炎，但患者血气分析PCO_2 31 mmol/L，因此可排除肺性脑病。患者虽有糖尿病史，但近日血糖无明显增高，尿常规显示酮体阴性，可排除糖尿病所致高渗昏迷和酮症酸中毒。

2月23日，甲状腺功能检查：游离三碘甲状腺素原氨酸（FT_3）＜1.54 pmol/L，游离甲状腺素（FT_4）：8.02 pmol/L，促甲状腺激素（TSH）2.25 μIU/L。诊断为“甲状腺功能减低，腺垂体功能减退?”，随即转入内分泌继续诊治。

2月24日化验结果回报：血气分析，PH 7.312，PO_2 117.2 mmHg，PCO_2 26.8 mmHg，实际HCO_3^-13.3 mmol/L；血常规，WBC 7.2×10^9/L，Hb 10.3g/L；总蛋白55 g/L，白蛋白28.6g/L；肾功能：Cr 315.4μmol/L，BUN 17.4mmol/L；血清K^+3.02 mmol/L；Na^+130 mmol/L。ACTH（8:00，16:00，0:00）分别为21.15，16.92，24.87 pg/ml，皮质醇（8:00，16:00，0:00）分别为6.71，＞62.8，4.15（μg/dl），卵泡刺激素（FSH）、黄体生成尿素（LH）、睾酮（T）、孕酮（P）、雌激素（E）和泌乳素（PRL）均很低；脑脊液生化：氯化物115 mmol/L，糖2.35 mmol/L，蛋白质0.7 g/L，脑脊液常规未见异常，脑脊液细菌涂片未找到细菌，结核菌培养阴性。胸水：胸水常规，黄色透明，李凡它反应弱阳性，细胞总数260×10^6/L，白细胞65×10^6/L，蛋白质18.3 g/L，糖3.28 mmol/L，氯化物95 mmol/L，CY21-1 14.57，CA125 348.1，ADA 9.03U/L，LDH 102.1U/L，痰细菌及结核菌培养阴性涂片，未找到真菌。肺CT示胸腔积液较前增多，心脏彩超提示左心收缩功能减退，EF 35%。头增强MR：垂体占位34 mm×22 mm×19 mm，伴出血。右眼视野颞侧偏盲。诊断：（1）垂体出血，垂体肿瘤；（2）2型糖尿病；（3）糖尿病肾病，肾功能不全（CKD4期）；（4）肺炎；（5）高血压病3级（极高危）；（6）胸腔积液；（7）离子紊乱（低钠血症、高钾血症、低钾血症）；（8）冠心病,心律失常；（9）Ⅲ度心力衰竭；（10）多发腔隙性脑梗死，脑萎缩。

处理：优质低蛋白糖尿病饮食；继续抗炎；利尿降钾，药用炭（商品名：爱西特）及复方α-酮酸片（商品名：开同）降肌酐，碳酸氢钠纠正酸中毒；脂肪乳补充能量，拜阿司匹林抗血小板凝聚，前列地尔（商品名：凯时）改善循环，对症补充白蛋白治疗；予氢化可的松100 mg，静脉滴注，每8小时1次，给予左旋甲状腺素钠25 μg，一日1次，口服替代治疗；监测各项生命指标及血糖、离子。2月26日，患者加用糖皮质激素治疗后，精神状态明显改善，食欲好转，体温恢复正常，血糖开始升高，血钠逐渐恢复正常，应用门冬及甘精胰岛素控制血糖。氢化可的松改为泼尼松10 mg，一日3次，口服。

2 临床病例讨论

内分泌科韩萍主任：患者病情复杂严重，存在高血压、糖尿病及严重心、肾并发症和肺炎。目前垂体前叶功能低下已明确，垂体-甲状腺，垂体-性腺轴激素水平均下降，垂体肾上腺轴激素水平虽然没有下降甚至有升高，考虑与患者处于应激状态有关。垂体功能减退与垂体肿瘤体积较大压迫正常组织和瘤体出血有关。泌乳素水平不高，因此不考虑泌乳素瘤，考虑此瘤为无功能性瘤可能性大。从患者近2日出现低血糖，血钠降低上看，糖皮质激素用量不够，但由于患者心肺功能的关系，限制了入液量，建议将静脉滴注氢化可的松，改为口服泼尼松10 mg，一日3次，左旋甲状腺素加量至50 μg，一日1次，口服，观察病情和激素水平变化。

脑外科刘云会主任：患者垂体肿瘤手术需考虑心、肺功能，患者目前状态不能耐受手术。从头磁共振分析，患者的垂体肿瘤大部分向蝶窦内生长，这种情况向外生长的可能性小，不易出现颅内高压，可暂缓手术，定期复查。至于患者垂体占位出血是否可再复发，鉴于垂体的特殊位置及大小固定，一般出血达到一定量可自行停止。目前主要的问题是垂体低功，手术并不能纠正垂体低功，且垂体低功本身可导致免疫功能低下，加重肺炎及心功能不全，建议内科继续保守治疗，补充激素替代治疗。

心血管内科杨志勇医师：患者目前明确诊断心功能不全，建议严格控制入液量，继续利尿治疗，呋塞米可调整为40 mg，一日2次，静脉注射，继续应用多巴胺改善肾血流，鉴于患者低钾，需注意血钾变化，将氯化钾缓释片调整至2.0 g，一日3次，口服，加用螺内酯20 mg，一日1次，口服，利尿保钾，尽量保证血钾在正常范围，但鉴于患者肾功差，需一日复查血钾，以免出现高钾或低钾血症。必要时可行血液透析治疗。

呼吸内科焦光宇主任医师：患者入院时有明显发热，近来无发热，咳痰量无明显增多，暂不给予抗生素升级，建议继续控制心力衰竭，若咳嗽、咳痰不缓解或出现发热，建议抗生素升级治疗，注意真菌感染的可能，胸腔积液与患者蛋白低有一定关系，建议继续补充白蛋白，但需注意心功能变化，配合呋塞米利尿治疗。

胸外科刘军主治医师：患者目前胸腔积液考虑与心、肾功能不全有关，仍有喘息，建议复查血气分析，观察有无二氧化碳潴留，继续平喘治疗，纠正心、肾功能不全，注意离子紊乱，患者昨日已抽取胸

水1000 ml左右，暂无必要行胸腔闭式引流。

科室综合意见：（1）患者病情重，需密切关注病情变化；（2）继续应用抗生素控制肺部感染；（3）给予利尿剂、多巴胺、阿司匹林和前列地尔改善心、肾功能。（4）药用炭和复方α-酮酸降低非蛋白氮；（5）将氯化钾缓释片调整至1.5 g，一日3次，口服，加用螺内酯20 mg，一日1次，口服，调节离子紊乱；（6）调整泼尼松至10 mg，一日3次，口服，左旋甲状腺素增至50 μg，日一次口服；（7）对症补充白蛋白。

3 月 8 日患者肺炎已控制一周，心、肾功能明显改善，各项离子水平均恢复到正常范围，顺利出院，继续口服激素替代治疗（泼尼松及左旋甲状腺素片），定期复诊。

（参加讨论医师：韩 萍，刘乐桐，孙 贺，张咏言，刘云会，焦光宇，杨志勇）

（韩 萍 整理）

（编辑：任开环）

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表1 导管长期留置的并发症及其确诊方式

并发症	*n*	确诊方式	*n*
导管血栓形成	16	根据临床表现拟诊	5
		导管动脉或静脉端抽出血栓	4
		经长期留置导管造影	6
		血管彩超	1
导管相关感染	3	血培养阳性	1
		血培养阴性，临床拟诊	2
导管血流量低*	15	透析过程中透析仪显示	15
中心静脉狭窄	1	血管造影和静脉彩超	1
无任何并发症	14		

注：*血流量低限定为任何 1 次透析血流量大部分时间不超过150 ml/min

表2 导管长期留置的并发症处理及其转归

并发症	*n*	处理方式	*n*	转归	
				好转	失败
导管血栓形成	16	尿激酶填充	4	3	1
		肝素填充	1	1	0
		导丝再通	4	1	3
		注射器抽出	4	4	0
		未处理	3	1	2
导管相关感染	3	拔除导管并予抗生素治疗	1	1	0
		抗生素治疗	2	2	0
导管血流量低	15	变换体位	8	6	2
		导管动脉端与静脉端反接	10	8	2
中心静脉狭窄	1	未处理	1		

注：透析充分性评估：28 例患者 Kt/V 值为 0.63～1.9，平均(1.13±0.28)

3 讨 论

带涤纶套中央静脉留置导管具有柔韧性好、对组织损伤小、皮下有一涤纶套组织包裹生长后利于固定且能阻止感染、可长期留置等多种优点，而且导管留置后即刻可以使用，与AVF相比，对心脏的血流动力学影响较小，同时还能避免每次透析穿刺的痛苦，使患者易于接受。

穿刺部位首选颈内静脉，右颈内静脉插入大约占80%[1]，其血流量＜150 ml/min的发生率、透析充分性、导管留置时间以及导管感染率均较锁骨下静脉、股静脉理想[2]。

本组患者血液透析时体外循环平均血流量满足国内一般最低血流量标准，即200～250 ml/min。中心静脉留置导管的透析效果是可以肯定的，与内瘘透析患者相比，单次透析的KT/V值无显著性差异[3]。

本组病例中拔除导管最常见原因是AVF成熟，其次，导管血栓形成是导管拔除的第二位原因。

长期留置导管最常见的并发症是导管内血栓形成。对于大多数病例，导管内填充尿激酶可能是最佳的选择[4]。对于缺乏尿激酶使用经验的医院，在数字化减影仪辅助下导丝再通也是一种选择。北京协和医院4例患者因导管血栓形成选择导丝再通，成功仅1例，成功率偏低，但病例数少，并不能反应导丝再通的整体成功率。预防导管相关血栓形成可通过药理途径(使用抗凝药)减少血栓形成[5]。最常用的方法是在透析间期用肝素（≥5×10^6 IU/L）封管。对容易发生血流不畅或预防血栓复发的患者，可常规选择血小板抑制剂治疗[3,4]。本组服用阿司匹林者血栓发生率与未服用者比较，无统计学差异，这可能与研究对象选择有倾向性以及入组病例数量有限，不能排除系统误差对阿司匹林抗栓作用的干扰等因素有关。导管内感染也是较常见的并发症，预防感染首先要严把操作关，对深静脉长期留置导管是否需要抗生素预防性封管一直存在争议[6]。

中央静脉留置导管一般不作为长期血管通路的首选，但对于内瘘尚处于成熟期或内瘘难以建立，同时腹膜透析有禁忌、高龄、有心肺基础疾病、对透析穿刺疼痛有顾虑、长期预后不良，尤其是合并肿瘤的维持性血液透析患者来说，仍是一种良好的选择。

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