·临床病例讨论·

Clinicopathological Conference

Acute pulmonary embolism after percutaneous coronary intervention

(The fourth case)

Case Presentation

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The patient is a man aged 67 yrs. He had a history of hypertension for ten years, had his varicose of great saphenous vein stripped three years ago, and had acute inferior myocardial infarction one year ago.

The patient was admitted to our hospital because of recurrent ischemic chest pain for eight months. The diagnoses were unstable angina pectoris, old inferior myocardial infarction, cardiac function class Ⅲ. After intensive drug treatment, his condition became stable. A coronary angiography examination showed a focal 90% stenosis located in the proximal segment of LAD and another 90% stenosis located in the middle segment of RCA-PL. After predilation, a stent was implanted in his LAD. Repeated angiography showed ideal immediate outcome. No dissection and thrombus were found. Residual stenosis disappeared.

Right femoral artery sheath was removed four hours after last intravenous heparin. The patient was asked to keep the right lower extremity immobile for 12 hours. Routine aspirin (0.3g qd) and ticlopidine (0.25g bid) were administrated. He had ambulatory activity after 24 hours. During defecation, he suddenly felt chest distress, palpitation and short breath. His symptoms continued over one hour and were not relieved. Physical examination: on supine position, the respiratory rate was 24/min, blood pressure was 60/40 mmHg(1mmHg=0.1333kPa), heart rate was 90-140 bpm, the jugular vein was obviously engorged and the extremities were cold and wet. ECG: T waves of leads V_{1-3} were inverted, T wave of lead V_4 was biphasic, S wave of lead Ⅰ became deeper and wider, T waves of leads Ⅲ and aVF were changed from upright to inverted. Emergency blood gas analysis: PaO_2 50mmHg, $PaCO_2$ 34.8mmHg. After oxygen supply through face mask, reasonable blood volume expansion and intravenous dopamine therapy, his condition tended to be stable. Blood pressure was recovered to 100/60 mmHg. Emergency coronary angiography showed that the LAD stent kept patent, no thrombus and dissection were found, RCA angiograph was similar to the previous imaging and coronary blood flow was TIMI class 3 (figure 1). Re-examination of serum myocardial marker after six hours showed CK 180 kU/L, CK-MB 6μg/L and cTnI 0.6 μg/L. An acute coronary event was excluded. A radiography of the chest showed both lungs were roughly normal. Re-examination of blood plasma D-dimer was 17.7mg/L. Echocadiography: right ventricle dimension was obviously enlarged with moderate tricuspid insufficiency, right ventricular wall was apparently hypokinetic (figure 2). The echocadiograph suggested increase in pressure load of right ventricle. Isotope pulmonary perfusion imaging showed widespread reduction of radioactivity distribution in right lung, especially obvious in anterior basal segment, lateral basal segment of lower lobe and middle lobe. Radioactivity distribution in superior lingual segment and partial apicoposterior segment of left lung was reduced. Isotope pulmonary ventilation imaging was normal. Combination of these results suggested that the patient had multiple pulmonary embolisms (figure 3). According to clinical manifestation and laboratory examination, the diagnosis was acute pulmonary thromboembolism. Because his hemodynamics had been stable, thrombolysis was not essential. After oxygen supply and intensive antithrombotic treatment, the patient recovered and was discharged two weeks later.

Clinical Discussion

Dr. Jiang Jie: The patient had sudden chest distress 24 hours after stenting and ECG showed T wave changes in the anterior and inferior leads. We first thought of acute coronary thrombosis or re-infarction of inferior wall. Emergency coronary angiography was a reasonable choice for diagnosis and further treatment. Angiographic data denied our initial supposition. Final diagnosis was acute pulmonary thromboembolism. Why did the patient have acute pulmonary thromboembolism? How can we explain the slight enhancement of the level of serum myocardial markers (CK-MB, cTnI)?

Dr. Ding Wenhui: The patient had high risk of deep venous thrombosis because he had history of great saphenous varices. Impeded venous return might induce deep vein thrombosis after lower extremity immobilization for extended time. During ambulatory activity and exertion of defecation, thrombus from deep vein of lower extremities might shed and result in acute pulmonary thromboembolism.

Dr. Gao Wei: Combined effect of mechanical obstruction and neuroendocrine factors quickly made pulmonary artery pressure rise. Right ventricular afterload was rapidly enhanced. Hypoxemia and hypotension were serious in acute phase. Because the patient had stenotic lesions in coronary arteries, hypoperfusion and hypoxemia might aggravate myocardial ischemia and finally result in focal necrosis and slight enhancement of serum myocardial markers (CK-MB, cTnI). Active treatment including improvement of blood pressure and oxygen avoided critical coronary events. With disappearance of neuroendocrine factors, activation of fibrinolysis system, and intensive anti-coagulation therapy, pulmonary vasospasm was relieved, thrombus dissolved gradually and pulmonary artery pressure recovered to normal.

Dr. Chen Ming: Vascular complications including hematoma, internal hemorrhage, external hemorrhage, pseudo-aneurysm and arterio-venous fistula are common after catheter intervention. Perfect hemostasis of puncture sites is our important mission. So we usually pay more attention to vascular complications than to thrombosis. What can we do for solving the contradiction between hemorrhage and thrombosis?

Dr. Huo Yong: In recent decade, the number of cases with catheter examination and intervention treatment through femoral access is rising rapidly. The associated vascular complications are becoming more and more common and various. To prevent vascular complications such as hemorrhage and pseudo-ancurysm, compression of femoral arterial puncture site and immobilization of corresponding lower extremity for adequate time are required after removal of femoral artery sheath. Because femoral vein and artery are anatomically located close together, compression and immobilization of lower extremity certainly cause hampered venous return, stasis of blood flow and tendency of occurrence of deep vein thrombosis. It is usually known that the incidence of major vascular complications is 5%-10%. So it is reasonable that we think highly of vascular complications. Although acute pulmonary thromboembolism resulting from deep vein thrombosis is uncommon, it may be fatal. So close attention should be paid to the complicated condition. For the purpose of sufficient hemostasis, strict immobilization and compression for at least six hours are required. The compression time should be extended in the patients with following conditions: the aged, female, weak tissue around femoral artery, diabetes mellitus with peripheral vascular disease, thrombolytic therapy, and intensive antiplatelet and anti-coagulation therapy. The patients at high risk of vascular complications and those at high risk of deep vein thrombosis may overlap. To solve the contradiction between hemorrhage and thrombosis, the following strategies and measures may be taken: ① the possibility of vascular complications and deep vein thrombosis should be estimated before catheterization, and individual programs of compression hemostasis are made for special cases. ② For those cases at high risk of both hemorrhage and thrombosis, pressure bandages are used to compress puncture site. If hemorrhage does not occur, lower extremity exercise in

bed 6-12 hours after sheath removal and ambulatory activity with pressure bandages not removed 12-24 hours after sheath removal may be permitted. If the time of lower extremity immobilization needs to be extended in case of hemorrhage, the lower extremities should be massaged intermittently and the patients are asked to contract the muscles of lower extremities *in situ*. The methods can facilitate venous return and inhibit thrombosis. Exertions that may result in increase in intraabdominal pressure should be avoided as far as possible, such as keeping defecation easy and smooth, preventing cough. The symptoms and signs should be closely observed and blood plasma D-dimer should be monitored, so that the deep vein thrombosis can be found in time. ③ In recent years, new generation of percutaneous vascular suture (PVS) device and technique is becoming more and more perfect. The hemostasis method can make the patients free of pressure dressing. Early ambulatory activity and anti-coagulation therapy can reduce the risk of deep vein thrombosis and pulmonary thromboembolism.

(Translator CHEN Ming)

经皮冠状动脉介入治疗后并发急性肺栓塞
(第4例)

1 病例摘要

患者,男性,67岁,汉族,干部。因反复发作缺血性胸痛8个月,加重1个月入院。1年前曾患急性下壁心肌梗死。既往有高血压病史10年,并有右侧大隐静脉曲张剥脱手术史。入院后心电图示陈旧下壁、后壁心肌梗死。冠状动脉造影示左前降支第1对角支发出后局限性狭窄90%,右冠状动脉左室后侧支中段90%狭窄。行左前降支中段经皮冠状动脉腔内成形术(percutaneous transluminal coronary angioplasty,PTCA)及支架置入术,术后无残余狭窄及撕裂,结果满意。术后4h拔除动脉鞘管,右下肢制动12h。同时口服阿司匹林0.3g qd和噻氯匹定(抵克力得)0.25g bid。患者于术后24h下床活动。在大便过程中突感胸闷、气短、心悸,持续>1h不缓解。查体:平卧位,呼吸24次/min,血压60/40mmHg(1mmHg=0.1333kPa),心率90~140次/min,四肢湿冷,颈静脉明显充盈,其余查体无异常发现。心电图示Ⅰ导联S波加深加宽,Ⅲ导联呈QR型,Ⅲ、aVF导联T波由直立变为倒置,$V_{1\sim3}$导联T波倒置,V_4导联T波双向。急查动脉血气示PaO_2 50mmHg,$PaCO_2$ 34.8mmHg。经面罩吸氧、适量扩容及静脉滴注多巴胺后血压恢复至100/70mmHg,病情趋于平稳。考虑到患者支架术后24h突发胸闷,前壁和下壁导联心电图T波改变,不能排除支架内血栓形成或下壁再梗死,决定紧急重复冠脉造影。结果显示,原支架处管腔无狭窄,无血栓和撕裂,血流正常(图1),右冠脉造影与前无差别。6h后复查CK 180kU/L,CK-MB 6μg/L,cTnI 0.6μg/L。以上结果基本排除了急性冠脉事件。考虑到患者曾有大隐静脉曲张病史,支架术后右下肢加压包扎、制动并卧床24h,可能出现下肢血栓而导致肺栓塞。复查静脉血D-二聚体(D-dimer)17.7mg/L,超声心动图示左房室内径正常,下后壁运动减低,右房室明显扩大,右室压力负荷增加,右室壁运动明显低下,三尖瓣中度关闭不全(图2)。同位素肺灌注扫描显示,右肺放射性分布普遍减低,以右肺下叶前基底段、侧基底段、中叶为著;左肺上舌段、部分尖后段放射性分布减低。肺通气扫描正常,提示多发性肺栓塞(图3)。结合临床表现和实验室检查肺栓塞诊断明确。考虑到患者血流动力学已稳定,不需要积极溶栓治疗。经吸氧,加强抗凝治疗,病情逐渐平稳出院。

2 临床讨论

本例患者既往有下肢静脉曲张病史,属于深静脉血栓(deep vein thrombosis,DVT)高发人群,在长时间下肢制动和静脉回流受阻的情况下出现深静脉血栓,下床活动和用力排便后血栓脱落造成急性肺栓塞。由于机械性阻塞和神经体液因素的共同作用,肺动脉压力迅速增高,右心室负荷急剧加重,急性期出现显著的低血压和低氧血症。患者冠状动脉存在狭窄病变,低灌注和低氧血症可加重心肌缺血,结果造成了心肌的局灶性坏死,表现为CK-MB和

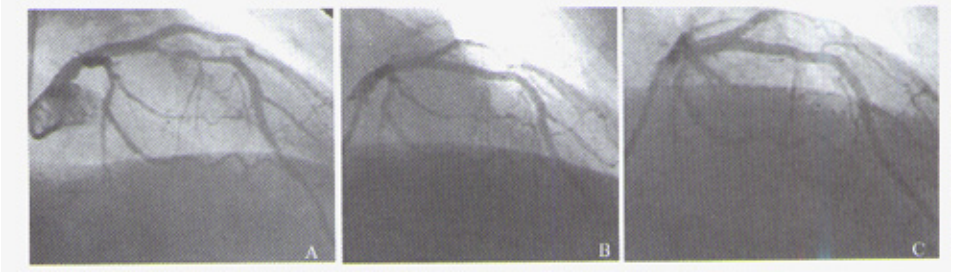

图 1 A:术前左前降支冠状动脉病变;B:支架术后的即刻造影结果;C:紧急重复冠状动脉造影的左前降支冠状动脉

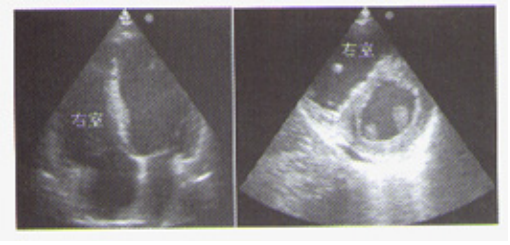

图 2 超声心动图显示右房室明显扩大,右室压力负荷增加,右室壁运动明显低下

cTnI 轻度增高。经过升高血压和提高血氧的积极处理,避免了严重的冠状动脉事件。随着神经体液因素逐渐恢复、体内纤溶系统的激活以及强化抗凝治疗,肺血管痉挛缓解、血栓逐步溶解,肺动脉压力恢复正常,病情趋于稳定。

临床讨论:近 10 年来国内经股动脉穿刺导管检查和介入治疗的病例数迅速增加,由此带来的血管并发症也层出不穷。通常在拔除鞘管后,股动脉穿刺部位需加压止血并制动相应下肢一段时间,以防止出血、假性动脉瘤等血管并发症。由于解剖上股静脉紧邻股动脉,加压止血和下肢制动必然导致静脉回流障碍,血流淤滞,容易形成深静脉血栓。一般认为术后的主要血管并发症可达 5%～10%,理应受到重视。而术后 DVT 导致的急性肺血栓栓塞(pulmonary thromboembolism,PTE)虽然少见,却可能致命,所以也应受到重视。为了达到充分止血的目的,患者至少需要严格制动和沙袋压迫 6h。对于老年、女性、股动脉周围组织薄弱、糖尿病合并外周

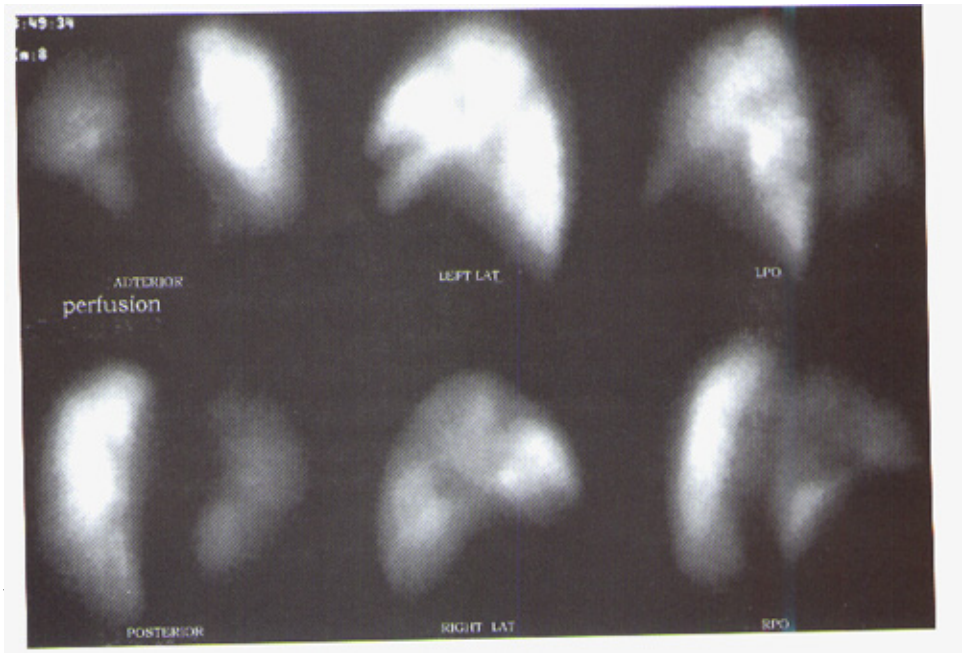

图 3 同位素肺灌注扫描提示多发性肺栓塞

血管疾病、溶栓治疗、强化抗血小板抗凝治疗的患者，可能需要延长加压止血的时间。而这些患者与DVT高危人群有所重叠。为了解决出血和血栓的矛盾，可以采取以下几方面的措施：① 评价患者发生血管并发症和DVT的可能性，对于特殊病例制定术后加压止血的个体方案；② 对于较有可能出血和血栓的病例，选用弹力绷带加压止血。若无出血，则术后6～12h可活动下肢，24h后可带弹力绷带下床活动。保持大便通畅，防治咳嗽，尽量避免导致腹压增高的用力。若因为出血而延长下肢制动时间，则应定时按摩下肢并嘱患者做下肢肌肉原位收缩动作，促进静脉血液回流。严密观察下肢的症状和体征，监测静脉血D-二聚体，以便及时发现DVT形成；③ 近几年，新一代经皮血管缝合器械和技术日益完善。对于DVT高危患者，这种方法可使患者术后不必加压包扎，尽早活动和抗凝，可有效减少DVT和PTE的风险。

（参加讨论医师：蒋捷，陈明，丁文惠，高伟，霍勇）

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·病例报告·

右心房巨大血栓死亡1例报告

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1 病例摘要

患者，男，52岁，退休教师。因双下肢浮肿1年，加重伴腹胀1个月，于2002年3月14日在南方医院住院治疗。患者于1年前出现下肢浮肿，可凹性，无心前区不适。在当地医院B超检查：右心房粘液瘤，三尖瓣关闭不全，未做特殊治疗。入院前1个月，双下肢浮肿明显加重，伴有腹胀，活动后心慌、气短、乏力、纳差。入院后查体：血压135/80mmHg（1mmHg=0.1333kPa），皮肤淤斑，牙龈出血，口唇轻度紫绀，颈静脉怒张，两肺无啰音，三尖瓣听诊区可闻及Ⅲ/Ⅳ级收缩期杂音，肝脏于右肋缘下4cm，可触及，脾大，腹水征阳性，下肢重度浮肿。心脏超声示右心房显著增大，三尖瓣重度关闭不全，少量心包积液。血常规：血小板37.5×10^9/L，白蛋白15g/L，血红蛋白60g/L。胸片示心胸比为0.86，心影普遍增大，呈烧瓶状，心后食管三角消失，少量胸腔积液。B超示：肝脾大，腹腔积液。心电图示：心律失常、房颤、不完全右束支传导阻滞。诊断：右心房占位性病变，粘液瘤？三尖瓣关闭不全、房颤、心力衰竭。于3月26日转胸外科手术治疗，手术中发现，病变占据整个右心房，心房右侧上部有蒂0.83cm×0.83cm，病变质硬，表面不规则，凹凸不平，呈淡黄色，大小约10cm×5cm×4cm。切除病变及病变附着处心房壁，并行三尖瓣成型术，术后常规缝合切口，心脏复苏。复苏成功后出现室速、室颤，抢救无效死亡。病理报告：血栓机化并钙化。

2 讨 论

心房纤颤患者容易形成心房血栓，以左心房多见，此患者在右心房发现巨大血栓，而且时间较长，心腔扩大，严重影响下腔静脉回流，下腔静脉压增大，肝脾淤血，脾功能亢进，贫血、血小板减少，手术为首选治疗手段。患者病程较长，处于衰竭状态，手术风险大，术后死亡。此例提示，对于性质不明的心脏占位性病变，临床上应对患者进行反复检查和尽可能到有条件的医院做病理检查，及早明确诊断以及手术治疗。术前给患者做好充分的准备，掌握治疗时机，为治疗成功创造条件，尤其是对体质消耗的患者尤显重要。

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