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· 论著 ·

急性肺栓塞患者下肢深静脉栓塞情况和右心功能改变及其临床意义

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【摘要】 背景 肺动脉栓子主要来源于下肢深静脉血栓, 而栓子阻塞肺动脉及其分支可导致肺血管阻力增加及右心功能改变, 但目前下肢深静脉血栓及右心功能改变对急性肺栓塞 (APE) 患者的影响尚未完全明确。目的 探讨 APE 患者下肢深静脉血栓情况和右心功能改变及其临床意义。方法 选取 2016—2018 年皖北煤电集团总医院收治的疑似 APE 患者 80 例, 根据 CT 肺动脉造影 (CTPA) 检查结果分为 APE 组 32 例和非 APE 组 48 例, 并根据 APE 危险分层标准将 32 例 APE 患者分为低危组 8 例、中危组 18 例和高危组 6 例。比较 APE 组与非 APE 组患者临床症状及体征、基础疾病、实验室检查指标、下肢深静脉血栓情况、右心功能指标, 并比较低危组、中危组、高危组患者下肢深静脉血栓情况、右心功能指标。结果 (1) APE 组与非 APE 组患者咯血、胸痛、胸闷、心悸、意识障碍、咳嗽、发热、低血压、慢性阻塞性肺疾病 (COPD)、高血压、冠心病、心房颤动、肿瘤发生率比较, 差异无统计学意义 ($P>0.05$); APE 组患者晕厥、单侧下肢肿胀、血脂异常发生率, 有近期手术史或骨折者所占比例, 红细胞计数及尿酸、纤维蛋白原、D-二聚体水平高于非 APE 组 ($P<0.05$)。 (2) APE 组患者栓塞血管数目多于非 APE 组, 栓子直径、右心室舒张末期内径 (RVEDD) / 左心室舒张末期内径 (LVEDD) 大于非 APE 组, 双侧深静脉血栓、深静脉血栓、近端深静脉血栓发生率高于非 APE 组, 右心室壁运动幅度 (RVWM)、右房室瓣环收缩期位移 (TAPSE) 小于非 APE 组, 右房室瓣反流速度 (TRV) 快于非 APE 组 ($P<0.05$)。 (3) 高危组患者栓塞血管数目多于低危组、中危组, 栓子直径、RVEDD / LVEDD 大于低危组、中危组, 双侧深静脉血栓、近端深静脉血栓发生率高于低危组、中危组, RVWM、TAPSE 小于低危组、中危组, TRV 快于低危组、中危组 ($P<0.05$); 中危组患者栓塞血管数目多于低危组, 栓子直径、RVEDD / LVEDD 大于低危组, 双侧深静脉血栓、近端深静脉血栓发生率高于低危组, RVWM、TAPSE 小于低危组, TRV 快于低危组 ($P<0.05$)。低危组、中危组、高危组患者深静脉血栓发生率比较, 差异无统计学意义 ($P>0.05$)。结论 APE 患者多存在下肢深静脉血栓及右心功能改变, 且 APE 危险分层越高, 下肢深静脉血栓越严重、右心功能改变越大, 因此针对出现下肢深静脉血栓及右心功能改变的疑似 APE 患者需及时行 CTPA 检查以明确诊断并进行危险分层, 以提高 APE 的防治效果。

【关键词】 肺栓塞; 静脉血栓形成; 危险分层; 右心功能

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Deep Venous Embolism of Lower Extremity and Change of Right Ventricular Function in Patients with Acute Pulmonary Embolism and Its Clinical Significance LI Xiaohua¹, ZHANG Dongguang¹, LI Wei²

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【Abstract】 **Background** Pulmonary artery embolus mainly derive from lower extremity deep venous thrombosis, while embolus blocking in pulmonary artery and its branches may lead to increase of pulmonary vascular resistance and change of right ventricular function, but the impact of venous embolism of lower extremity and change of right ventricular function on patients with acute pulmonary embolism (APE) is not very clear yet. **Objective** To investigate the deep venous embolism of

lower extremity and change of right ventricular function in patients with APE and its clinical significance. **Methods** A total of 80 patients with suspected APE were selected in General Hospital of Wanbei Coal and Power Group from 2016 to 2018, and they were divided into APE group ($n=32$) and non-APE group ($n=48$) according to the CTPA examination results, and then the 32 patients with APE were divided into A group (with low-risk APE, $n=8$), B group (with medium-risk APE, $n=18$) and C group (with high-risk APE, $n=6$) according to APE risk stratification criteria. Clinical symptoms and signs, basic diseases, laboratory examination results, deep venous embolism of lower extremity and index of right ventricular function were compared between APE group and non-APE group, meanwhile deep venous embolism of lower extremity and index of right ventricular function were compared in groups A, B and C. **Results** (1) No statistically significant difference of incidence of hemoptysis, chest pain, chest distress, palpitation, consciousness disorder, cough, fever, hypotension, COPD, hypertension, coronary heart disease, atrial fibrillation or tumour was found between APE group and non-APE group ($P>0.05$); incidence of syncope, unilateral lower limb swelling and dyslipidemia, proportion of patients with recent surgical history or fracture, RBC, UA, FIB and D-dimer in APE group were statistically significantly higher than those in non-APE group ($P<0.05$). (2) Number of embolized vessels in APE group was statistically significantly more than that in non-APE group, diameter of embolus and RVEDD/LVEDD ratio in APE group were statistically significantly greater than those in non-APE group, incidence of bilateral deep venous embolization, deep venous thrombosis and proximal deep venous embolization in APE group were statistically significantly higher than those in non-APE group, RVWM and TAPSE in APE group were statistically significantly smaller than those in non-APE group, moreover TRV in APE group was statistically significantly faster than that in non-APE group ($P<0.05$). (3) Number of embolized vessels in C group was statistically significantly more than that in A group and B group, respectively, diameter of embolus and RVEDD/LVEDD ratio in C group was statistically significantly greater than that in A group and B group, respectively, incidence of bilateral deep venous embolization and proximal deep venous embolization in C group was statistically significantly higher than that in A group and B group, respectively, RVWM and TAPSE in C group was statistically significantly smaller than that in A group and B group, respectively, moreover TRV in C group was statistically significantly faster than that in A group and B group, respectively ($P<0.05$); number of embolized vessels in B group was statistically significantly more than that in A group, diameter of embolus and RVEDD/LVEDD ratio in B group were statistically significantly greater than those in A group, incidence of bilateral deep venous embolization and proximal deep venous embolization in B group was statistically significantly higher than that in A group, respectively, RVWM and TAPSE in B group were statistically significantly smaller than those in A group, moreover TRV in B group was statistically significantly faster than that in A group ($P<0.05$). No statistically significant difference of incidence of deep venous embolization was found in groups A, B and C ($P>0.05$). **Conclusion** There are deep venous embolism of lower extremity and change of right ventricular function in patients with APE, and as risk stratification increase, severity of deep venous embolism of lower extremity aggravates and change of right ventricular function increase, thus it is necessary to carry out CTPA when we found suspected APE patients with deep venous embolism of lower extremity and change of right ventricular function on clinic, to make a definite diagnosis and clear risk stratification, eventually improve the prevention and control ability for APE.

【Key words】 Pulmonary embolism; Venous thrombosis; Risk stratification; Right heart function;

肺栓塞指来源于静脉系统或右心的栓子脱落、阻塞肺动脉及其分支所致的一种临床常见的致命性疾病,主要病理特征为肺循环和呼吸功能障碍^[1]。据报道,急性肺栓塞(APE)年发病率为100/10万~200/10万^[2],但其病情进展快,病死率高^[3],是住院患者死亡的第三位致死原因^[4],仅次于卒中、急性冠脉综合征。大型国际注册登记数据显示,APE 7 d全因死亡率为1.9%~2.9%,30 d全因死亡率为4.9%~6.6%^[5]。APE患者常无特异性临床症状,故其漏诊率、误诊率较高。目前CT肺动脉造影(CTPA)是临床诊断APE的首选检查方法^[6]。下肢深静脉血栓形成(LDVT)与肺栓塞是同一疾病的不同表现形式,因肺动脉栓子主要来源于下肢深静脉,因此早期诊断LDVT可有效预防APE。本

研究旨在探讨APE患者下肢深静脉栓塞情况和右心功能改变及其临床意义,以提高APE的防治效果,现报道如下。

1 资料与方法

1.1 一般资料 选取2016—2018年皖北煤电集团总医院收治的疑似APE患者80例,均存在胸痛、呼吸困难、咯血或单侧下肢疼痛肿胀等临床表现。排除标准:(1)合并急性心肌梗死者;(2)因肾功能不全、对造影剂过敏等而无法行CTPA检查者;(3)临床资料不全者;(4)因胸廓畸形而导致右心功能不全者;(5)合并肝肾功能不全者;(6)伴有抗凝血酶原缺乏、遗传性异常纤维蛋白原血症、高同型半胱氨酸血症、抗心磷脂抗体综合征、蛋白S缺乏、蛋白C缺乏等原发性静脉血

栓栓塞危险因素者。根据 CTPA 检查结果将所有患者分为 APE 组 32 例和非 APE 组 48 例；根据《急性肺栓塞的诊断和治疗指南》^[7] 中的 APE 危险分层标准将 32 例 APE 患者分为低危组 8 例、中危组 18 例、高危组 6 例。APE 组和非 APE 组患者男性比例、年龄、体质指数、吸烟率比较，差异无统计学意义 ($P>0.05$ ，见表 1)，具有可比性。本研究经皖北煤电集团总医院医学伦理委员会审核批准，所有患者及其家属对本研究知情并签署知情同意书。

表 1 APE 组与非 APE 组患者一般资料比较

Table 1 Comparison of general information between APE group and non-APE group

组别	例数	男性 [n (%)]	年龄 ($\bar{x} \pm s$, 岁)	体质指数 ($\bar{x} \pm s$, kg/m^2)	吸烟 [n (%)]
APE 组	32	21 (65.6)	65.5 $\pm$ 9.2	23.4 $\pm$ 2.8	19 (59.4)
非 APE 组	48	27 (56.3)	64.1 $\pm$ 10.4	22.7 $\pm$ 2.3	21 (43.8)
t (χ^2) 值		0.70 ^a	0.62	1.23	1.88 ^a
P 值		0.40	0.54	0.23	0.17

注：APE= 急性肺栓塞；^a为 χ^2 值

1.2 方法

1.2.1 CTPA 检查 所有患者入院后采用 64 层螺旋 CT (美国 GE 公司生产) 进行肺动脉检查，先行胸部平扫，再行增强扫描，使用双筒高压注射器经患者肘静脉注射碘普罗胺 (370 mg I/ml) 60 ml，速率为 5 ml/s；扫描结束后将图像传至 AW 4.5 工作站行多平面重建、曲面重建、容积重建等，CTPA 检查结果示肺动脉主干及其各级分支管腔内充盈缺损、血管壁不规则者可确诊为 APE。

1.2.2 超声检查 采用 GE Vivid E9 彩色超声诊断仪 (美国 GE 公司生产) 检查患者下肢深静脉栓塞情况及右心功能指标，探头频率为 3~15 MHz，采用灰阶超声技术、常规横向切面、间断加压法^[8] 检查下肢深静脉栓塞情况，包括栓塞血管数目、栓子直径及双侧深静脉栓塞、深静脉栓塞、近端深静脉栓塞发生情况；采用实时三维超声心动图定量分析右心功能指标，包括右心室舒张末期内径 (RVEDD)、左心室舒张末期内径 (LVEDD)、右心室壁运动幅度 (RVWM)、右房室瓣反流速度 (TRV)、右房室瓣环收缩期位移 (TAPSE)，均检测 5 个心动周期并取平均值，计算 RVEDD/LVEDD。

1.3 观察指标 比较 APE 组与非 APE 组患者临床症状及体征、基础疾病、实验室检查指标、下肢深静脉栓塞情况、右心功能指标，比较低危组、中危组、高危组患者下肢深静脉栓塞情况、右心功能指标。临床症状及体征包括咯血、胸痛、胸闷、心悸、晕厥、意识障碍、咳嗽、发热、低血压、单侧下肢肿胀等；基础疾病包括慢性阻塞性肺疾病 (COPD)、高血压、冠心病、心房颤动、

血脂异常、肿瘤、近期手术史或骨折；实验室检查指标包括红细胞计数、尿酸、纤维蛋白原、D-二聚体。

1.4 统计学方法 应用 SPSS 22.0 统计学软件进行数据分析，符合正态分布的计量资料以 ($\bar{x} \pm s$) 表示，两组间比较采用两独立样本 t 检验，多组间比较采用单因素方差分析，组间两两比较采用 q 检验；不符合正态分布的计量资料以 M (QR) 表示，采用非参数检验；计数资料分析采用 χ^2 检验。以 $P<0.05$ 为差异有统计学意义。

2 结果

2.1 APE 组与非 APE 组患者临床症状及体征、基础疾病、实验室检查指标比较 APE 组与非 APE 组患者咯血、胸痛、胸闷、心悸、意识障碍、咳嗽、发热、低血压、COPD、高血压、冠心病、心房颤动、肿瘤发生率比较，差异无统计学意义 ($P>0.05$)；APE 组患者晕厥、单侧下肢肿胀、血脂异常发生率，有近期手术史或骨折者所占比例，红细胞计数及尿酸、纤维蛋白原、D-二聚体水平高于非 APE 组，差异有统计学意义 ($P<0.05$ ，见表 2)。

2.2 下肢深静脉栓塞情况、右心功能指标

2.2.1 APE 组与非 APE 组 APE 组患者栓塞血管数目多于非 APE 组，栓子直径、RVEDD/LVEDD 大于非 APE 组，双侧深静脉栓塞、深静脉栓塞、近端深静脉栓塞发生率高于非 APE 组，RVWM、TAPSE 小于非 APE 组，TRV 快于非 APE 组，差异有统计学意义 ($P<0.05$ ，见表 3)。

2.2.2 低危组、中危组、高危组 三组患者栓塞血管数目、栓子直径、双侧深静脉栓塞发生率、近端深静脉栓塞发生率、RVEDD/LVEDD、RVWM、TRV、TAPSE 比较，差异有统计学意义 ($P<0.05$)；其中高危组患者栓塞血管数目多于低危组、中危组，栓子直径、RVEDD/LVEDD 大于低危组、中危组，双侧深静脉栓塞、近端深静脉栓塞发生率高于低危组、中危组，RVWM、TAPSE 小于低危组、中危组，TRV 快于低危组、中危组，差异有统计学意义 ($P<0.05$)；中危组患者栓塞血管数目多于低危组，栓子直径、RVEDD/LVEDD 大于低危组，双侧深静脉栓塞、近端深静脉栓塞发生率高于低危组，RVWM、TAPSE 小于低危组，TRV 快于低危组，差异有统计学意义 ($P<0.05$)。三组患者深静脉栓塞发生率比较，差异无统计学意义 ($P>0.05$ ，见表 4)。

3 讨论

肺栓塞是各种栓子阻塞肺动脉及其分支的一组疾病或临床综合征，包括肺血栓栓塞症、脂肪栓塞综合征、羊水栓塞、空气栓塞、肿瘤栓塞等，其中肺血栓栓塞症是肺栓塞的常见类型^[9]。据报道，APE 3 个月全因死亡率为 8.6%~17.0%^[10]，而尽早诊断并有效治疗可降低 APE 患者病死率，进而改善患者预后^[11]。研究表明，

表2 APE组与非APE组患者临床症状及体征、基础疾病、实验室检查指标比较

Table 2 Comparison of clinical symptoms and signs, underlying diseases and laboratory examination results between APE group and non-APE group

组别	例数	临床症状及体征 [n (%)]									
		咯血	胸痛	胸闷	心悸	晕厥	意识障碍	咳嗽	发热	低血压	单侧下肢肿胀
APE组	32	4 (12.5)	7 (21.9)	20 (62.5)	3 (9.4)	6 (18.8)	1 (3.1)	11 (34.4)	3 (9.4)	4 (12.5)	6 (18.8)
非APE组	48	1 (2.1)	13 (27.1)	28 (58.3)	5 (10.4)	2 (4.2)	1 (2.1)	17 (35.4)	5 (10.4)	6 (12.5)	2 (4.2)
χ^2 (Z) 值		3.56	0.28	0.14	0.05	4.54	0.09	0.01	0.05	0.00	4.54
P 值		0.06	0.60	0.71	0.82	0.03	0.77	0.92	0.82	1.00	0.03

组别	基础疾病 [n (%)]							实验室检查指标 [M (QR)]			
	COPD	高血压	冠心病	心房颤动	血脂异常	肿瘤	近期手术史或骨折	红细胞计数 ($\times 10^{12}/L$)	尿酸 ($\mu mol/L$)	纤维蛋白原 (g/L)	D-二聚体 ($\mu g/L$)
APE组	2 (6.3)	12 (37.5)	2 (6.3)	3 (9.4)	20 (62.5)	3 (9.4)	7 (21.9)	5.06 (1.52)	357 (139)	3.74 (1.59)	234 (352)
非APE组	3 (6.3)	23 (47.9)	4 (8.3)	5 (10.4)	18 (37.5)	5 (10.4)	2 (4.2)	4.43 (1.08)	349 (187)	3.14 (1.22)	128 (186)
χ^2 (Z) 值	0.00	0.85	0.12	0.02	4.81	0.02	6.03	4.26 ^a	3.58 ^a	2.25 ^a	11.35 ^a
P 值	1.00	0.36	0.73	0.88	0.03	0.88	0.01	<0.01	<0.01	0.02	<0.01

注: COPD=慢性阻塞性肺疾病; ^a 为 Z 值

表3 APE组与非APE组患者下肢深静脉血栓情况、右心功能指标比较

Table 3 Comparison of deep venous embolism of lower extremity and index of right heart function between APE group and non-APE group

组别	例数	下肢深静脉栓塞情况					右心功能指标 ($\bar{x} \pm s$)			
		栓塞血管数目 ($\bar{x} \pm s$, 条)	栓子直径 ($\bar{x} \pm s$, mm)	双侧深静脉栓塞 [n (%)]	深静脉栓塞 [n (%)]	近端深静脉栓塞 [n (%)]	RVEDD/ LVEDD	RVWM (mm)	TRV (m/s)	TAPSE (mm)
APE 组	32	1.48 ± 0.61	14.35 ± 1.25	12 (37.5)	24 (75.0)	18 (56.3)	0.93 ± 0.15	7.26 ± 0.46	3.55 ± 0.38	15.26 ± 0.93
非 APE 组	48	0.46 ± 0.25	6.24 ± 1.03	0	20 (41.7)	6 (12.5)	0.78 ± 0.08	9.26 ± 0.59	1.51 ± 0.34	18.47 ± 0.75
t (χ^2) 值		10.38	31.66	21.18 ^a	8.62 ^a	17.50 ^a	5.82	16.17	25.08	17.02
P 值		<0.05	<0.05	<0.05	<0.01	<0.05	<0.05	<0.05	<0.05	<0.05

注: RVEDD=右心室舒张末期内径, LVEDD=左心室舒张末期内径, RVWM=右心室壁运动幅度, TRV=右房室瓣反流速度, TAPSE=右房室瓣环收缩期位移; ^a 为 χ^2 值

表4 不同危险分层 APE 患者下肢深静脉血栓情况、右心功能指标比较

Table 4 Comparison of deep venous embolism of lower extremity and index of right heart function in APE patients with different risk stratification

组别	例数	下肢深静脉血栓情况					右心功能指标 ($\bar{x} \pm s$)			
		栓塞血管数目 ($\bar{x} \pm s$, 条)	栓子直径 ($\bar{x} \pm s$, mm)	双侧深静脉栓塞 (n/N)	深静脉栓塞 (n/N)	近端深静脉栓塞 (n/N)	RVEDD/LVEDD	RVWM (mm)	TRV (m/s)	TAPSE (mm)
低危组	8	0.68 ± 0.54 ^{ab}	7.63 ± 1.38 ^{ab}	0 ^{ab}	6/8	1/8 ^{ab}	0.74 ± 0.06 ^{ab}	9.84 ± 0.54 ^{ab}	1.65 ± 0.31 ^{ab}	20.34 ± 2.14 ^{ab}
中危组	18	1.56 ± 0.67 ^a	14.32 ± 1.25 ^a	7/18 ^a	13/18	11/18 ^a	0.96 ± 0.17 ^a	7.15 ± 0.32 ^a	3.48 ± 0.32 ^a	16.43 ± 1.24 ^a
高危组	6	1.90 ± 0.72	15.46 ± 1.19	5/6	5/6	6/6	1.25 ± 0.18	6.24 ± 0.44	3.86 ± 0.33	13.24 ± 0.75
F (χ^2) 值		7.22	91.91	10.19 ^c	0.30 ^c	11.06 ^c	19.06	167.28	112.17	42.48
P 值		<0.05	<0.05	0.01	0.86	<0.05	<0.05	<0.05	<0.05	<0.05

注: 与高危组比较, ^aP<0.05; 与中危组比较, ^bP<0.05; ^c 为 χ^2 值

LDVT 是 APE 的独立危险因素^[12]。栓子阻塞肺动脉及其分支可导致肺血管阻力增加、右心室后负荷增大, 因此 APE 患者行超声心动图检查可见右心室后负荷过重征象^[13-14]。CTPA 是临床诊断 APE 的“金标准”, 具有无创、扫描速度快、图像清晰等特点, 可直观判断肺动脉栓塞程度、形态及累及部位和范围, 目前已广泛用于临床, 但 CTPA 检查费用昂贵并存在辐射暴露等缺点^[15]。因此, 临床需优化 APE 诊断流程, 以快速、准确筛选疑似 APE 患者, 提高 APE 诊断率, 节省医疗资源。

本研究结果显示, APE 组患者晕厥、单侧下肢肿胀、

血脂异常发生率, 有近期手术史或骨折者所占比例, 红细胞计数及尿酸、纤维蛋白原、D-二聚体水平高于非 APE 组, 提示 APE 好发于有近期手术史、骨折患者并伴有红细胞计数、纤维蛋白原、D-二聚体水平升高。相关研究表明, 肺栓塞患者主要表现为胸闷、咯血或痰中带血、晕厥及 D-二聚体水平升高, 且 D-二聚体水平对 APE 具有一定预测价值^[16-17], 分析其原因主要为机体出现急性血栓形成时凝血和纤溶系统激活, 进而导致血浆 D-二聚体水平升高。

LDVT 是静脉血栓栓塞症的表现形式之一。有研究

报道,LDVT患者易并发肺栓塞,且近期预后差^[4]。因此,提高LDVT早期诊断率可有效预防APE发生。相关研究表明,下肢血管彩色超声检查诊断LDVT的灵敏度为88%~98%,特异度为97%~100%,且该检查操作简单,现已成为诊断LDVT的主要检查方法^[18]。本研究结果显示,APE组患者栓塞血管数目多于非APE组,栓子直径大于非APE组,双侧深静脉栓塞、深静脉栓塞、近端深静脉栓塞发生率高于非APE组,表明APE患者下肢深静脉栓塞发生率高且病情严重。有研究表明,约70%的肺栓塞患者发生下肢深静脉栓塞,约50%的近端深静脉栓塞患者存在症状性或无症状肺栓塞^[19],本研究结果与之相似。

右心功能改变是APE的常见并发症,可进展为心力衰竭甚至导致患者死亡。临床可通过心电图、胸部X线、超声心动图、放射性核素显像、右心导管检查等检查右心功能,其中超声心动图是检查心脏结构及心功能改变的常用手段,且经济、简便、易行^[20]。RVEDD/LVEDD、RVWM、TRV、TAPSE均是评价右心功能的重要指标,本研究结果显示,APE组患者RVEDD/LVEDD大于非APE组,RVWM、TAPSE小于非APE组,TRV快于非APE组,表明APE患者存在右心功能改变。ROY等^[20]研究表明,肺栓塞患者超声心动图检查可见右心室后负荷过重征象,本研究结果与之相似。

本研究结果显示,高危组患者栓塞血管数目多于低危组、中危组,栓子直径、RVEDD/LVEDD大于低危组、中危组,双侧深静脉栓塞、近端深静脉栓塞发生率高于低危组、中危组,RVWM、TAPSE小于低危组、中危组,TRV快于低危组、中危组;中危组患者栓塞血管数目多于低危组,栓子直径、RVEDD/LVEDD大于低危组,双侧深静脉栓塞、近端深静脉栓塞发生率高于低危组,RVWM、TAPSE小于低危组,TRV快于低危组,表明下肢深静脉栓塞及右心功能改变对APE危险分层有一定提示作用,且APE危险分层越高则下肢深静脉栓塞及右心功能改变越严重。

综上所述,APE患者多存在下肢深静脉栓塞及右心功能改变,且APE危险分层越高,下肢深静脉栓塞及右心功能改变越严重,因此针对出现下肢深静脉栓塞及右心功能改变的疑似APE患者需及时行CTPA检查以明确诊断并进行危险分层,以提高APE的防治效果;但本研究为单中心研究,且样本量较小、观察时间较短,今后需联合多中心、扩大样本量、延长观察时间以明确APE的发病机制。

作者贡献:李晓花、张东光进行文章的构思与设计,研究的实施与可行性分析;李为进行数据收集、整理、分析;李晓花进行结果分析与解释、撰写论文、进行论文的修订;李晓花、李为负责文章的质量控制及审校,

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本文无利益冲突。

参考文献

- [1] 徐巧莲, 万献尧. 急性肺栓塞的诊断和治疗[J]. 中国呼吸与危重监护杂志, 2011, 10(3): 308-312.DOI: 10.3969/j.issn.1671-6205.2011.03.028.
- [2] HEIT J A. The epidemiology of venous thromboembolism in the community[J]. Arterioscler Thromb Vasc Biol, 2008, 28(3): 370-372.DOI: 10.1161/ATVBAHA.108.162545.
- [3] BERTOLETTI L, LE GAL G, AUJESKY D, et al. Prognostic value of the Geneva prediction rule in patients in whom pulmonary embolism is ruled out[J]. J Intern Med, 2011, 269(4): 433-440.DOI: 10.1111/j.1365-2796.2010.02328.x.
- [4] GOLDBABER S Z, BOUNAMEAUX H. Pulmonary embolism and deep vein thrombosis[J]. Lancet, 2012, 379(9828): 1835-1846.DOI: 10.1016/S0140-6736(11)61904-1.
- [5] JIMÉNEZ D, DE MIGUEL DÍEZ J, GUIJARRO R, et al. Trends in the management and outcomes of acute pulmonary embolism: analysis from the riete registry[J]. J Am Coll Cardiol, 2016, 67(2): 162-170.DOI: 10.1016/j.jacc.2015.10.060.
- [6] 中华医学会心血管病学分会肺血管病学组, 中国医师协会心血管内科医师分会. 急性肺血栓栓塞症诊断治疗中国专家共识[J]. 中华内科杂志, 2010, 49(1): 74-81.DOI: 10.3760/cma.j.issn.0578-1426.2010.01.026.
- [7] TORBICKI A, PERRIER A, KONSTANTINIDES S, et al. Guidelines on the diagnosis and management of acute pulmonary embolism: the Task Force for the Diagnosis and Management of Acute Pulmonary Embolism of the European Society of Cardiology (ESC)[J]. Eur Heart J, 2008, 29(18): 2276-2315.DOI: 10.1093/eurheartj/ehn310.
- [8] 中国成人血脂异常防治指南修订联合委员会. 中国成人血脂异常防治指南(2016年修订版)[J]. 中国循环杂志, 2016, 31(10): 937-950.DOI: 10.3969/j.issn.1000-3614.2016.10.001.
- [9] 葛均波, 徐永健, 王辰. 内科学[M]. 北京: 人民卫生出版社, 2018.
- [10] LAPORTE S, MISMETTI P, DÉCOUSUS H, et al. Clinical predictors for fatal pulmonary embolism in 15, 520 patients with venous thromboembolism: findings from the Registro Informatizado de la Enfermedad TromboEmbolica venosa (RIETE) Registry[J]. Circulation, 2008, 117(13): 1711-1716.DOI: 10.1161/CIRCULATIONAHA.107.726232.
- [11] MARTIN RIEDEL. Emergency diagnosis of pulmonary embolism[J]. Heart, 2001, 85: 607-609.
- [12] MARSTON N, BROWN J P, OLSON N, et al. Right ventricular strain before and after pulmonary thromboendarterectomy in patients with chronic thromboembolic pulmonary hypertension[J]. Echocardiography, 2015, 32(7): 1115-1121.DOI: 10.1111/echo.12812.
- [13] LANKHAAR J W, WESTERHOF N, FAES T J, et al. Quanti-

- fication of right ventricular afterload in patients with and without pulmonary hypertension [J]. *Am J Physiol Heart Circ Physiol*, 2006, 291 (4): H1731-1737. DOI: 10.1152/ajpheart.00336.2006.
- [14] 叶静, 陆友金, 赵卉. 70 例肺栓塞的临床特征分析 [J]. *临床肺科杂志*, 2019, 24 (3): 393-396. DOI: 10.3969/j.issn.1009-6663.2019.03.002.
- YE J, LU Y J, ZHAO H. Clinical features of 70 patients with pulmonary embolism [J]. *Journal of Clinical Pulmonary Medicine*, 2019, 24 (3): 393-396. DOI: 10.3969/j.issn.1009-6663.2019.03.002.
- [15] 中华医学会心血管病学分会, 中华心血管病杂志编辑委员会. 右心衰竭诊断和治疗中国专家共识 [J]. *中华心血管病杂志*, 2012, 40 (6): 449-461. DOI: 10.3760/cma.j.issn.0253-3758.2012.06.001.
- [16] SHAHRIAR Z, STEPHAN R, SHWETA M, et al. Could the number of CT angiograms be reduced in emergency department patients suspected of pulmonary embolism? [J]. *World J Emerg Med*, 2012, 3 (3): 172-176. DOI: 10.5847/wjem.j.1920-8642.2012.03.002.
- [17] 徐燕. 纤维蛋白原和 D-二聚体检测在肺栓塞诊断中的应用研究 [J]. *临床医药文献电子杂志*, 2017, 4 (84): 16477, 16479. DOI: 10.3877/j.issn.2095-8242.2017.84.021.
- [18] 范译. 下肢深静脉联合腹部大血管彩超诊断深静脉血栓的临床效果 [J]. *智慧健康*, 2017, 3 (16): 5-6. DOI: 10.19335/j.cnki.2096-1219.2017.16.03.
- FAN Y. Clinical Effect of Deep Vein Combined with Abdominal Large Blood Vessel Ultrasonography in the Diagnosis of Deep Venous Thrombosis [J]. *Smart Healthcare*, 2017, 3 (16): 5-6. DOI: 10.19335/j.cnki.2096-1219.2017.16.03.
- [19] 陈斌, 来永飞. 超声心动图与外周血管超声诊断肺栓塞的临床价值分析 [J]. *医学影像学杂志*, 2013, 23 (11): 1800-1802.
- CHEN B, LAI Y F. Ultrasonic and the clinical value of peripheral vascular ultrasound in the diagnosis of pulmonary embolism [J]. *Journal of Medical Imaging*, 2013, 23 (11): 1800-1802.
- [20] ROY P M, COLOMBET I, DURIEUX P, et al. Systematic review and meta-analysis of strategies for the diagnosis of suspected pulmonary embolism [J]. *BMJ*, 2005, 331 (7511): 259. DOI: 10.1136/bmj.331.7511.259.
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- [8] SEEMUNGAL T A, LUN J C, DAVIS G, et al. Plasma homocysteine is elevated in COPD patients and is related to COPD severity [J]. *Int J Chron Obstruct Pulmon Dis*, 2007, 2 (3): 313-321. DOI: 10.2147/copd.s2147.
- [9] LI J, HUANG Y, FEI G H. The evaluation of cognitive impairment and relevant factors in patients with chronic obstructive pulmonary disease [J]. *Respiration*, 2013, 85 (2): 98-105. DOI: 10.1159/000342970.
- [10] BOZINOVSKI S, HUTCHINSON A, THOMPSON M, et al. Serum amyloid A is a biomarker of acute exacerbations of chronic obstructive pulmonary disease [J]. *Am J Respir Crit Care Med*, 2008, 177 (3): 269-278. DOI: 10.1164/rccm.200705-678OC.
- [11] GALIMBERTI D, SCHOONENBOOM N, SCARPINI E, et al. Chemokines in serum and cerebrospinal fluid of Alzheimer's disease patients [J]. *Ann Neurol*, 2003, 53 (4): 547-548. DOI: 10.1002/ana.10531.
- [12] LI J G, CHU J, BARRERO C, et al. Homocysteine exacerbates β -amyloid pathology, tau pathology, and cognitive deficit in a mouse model of Alzheimer disease with plaques and tangles [J]. *Ann Neurol*, 2014, 75 (6): 851-863. DOI: 10.1002/ana.24145.
- [13] HU H, WANG C, JIN Y, et al. Alpha-lipoic acid defends homocysteine-induced endoplasmic reticulum and oxidative stress in HAECs [J]. *Biomedicine Pharmacother*, 2016, 80: 63-72. DOI: 10.1016/j.biopha.2016.02.022.
- [14] ALSHAIKH B, SCHALL J I, MAQBOOL A, et al. Choline supplementation alters some amino acid concentrations with no change in homocysteine in children with cystic fibrosis and pancreatic insufficiency [J]. *Nutr Res*, 2016, 36 (5): 418-429. DOI: 10.1016/j.nutres.2015.12.014.
- [15] GARCIA-RIO F, MIRAVITLES M, SORIANO J B, et al. Systemic inflammation in chronic obstructive pulmonary disease: a population-based study [J]. *Respir Res*, 2010, 11: 63. DOI: 10.1186/1465-9921-11-63.
- [16] RAVAGLIA G, FORTI P, MAIOLI F, et al. Plasma homocysteine and inflammation in elderly patients with cardiovascular disease and dementia [J]. *Exp Gerontol*, 2004, 39 (3): 443-450. DOI: 10.1016/j.exger.2003.11.005.
- [17] CHEN S, DONG Z, ZHAO Y, et al. Homocysteine induces mitochondrial dysfunction involving the crosstalk between oxidative stress and mitochondrial pSTAT3 in rat ischemic brain [J]. *Sci Rep*, 2017, 7 (1): 6932. DOI: 10.1038/s41598-017-07112-z.
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