论著·临床医学

急性前循环大血管闭塞性卒中患者血管内治疗后发生卒中相关性肺炎的危险因素分析

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[**摘要**] **目的** 探究急性缺血性卒中(acute ischemic stroke, AIS)患者经血管内治疗(endovascular treatment, EVT)后发生卒中相关性肺炎(stroke-associated pneumonia, SAP)的影响因素。**方法** 回顾性病例对照分析2017年1月至2021年4月在陆军军医大学第一附属医院神经内科与自贡市第三人民医院神经内科共426例急性前循环大血管闭塞性脑卒中患者的所有资料。根据是否发生SAP将患者分为两组。收集2组患者的人口学信息(性别和年龄)、卒中的病因学TOAST分型(大动脉粥样硬化型、心源性栓塞型、其他)、血管危险因素(高血压、高脂血症、糖尿病、心房颤动、吸烟、既往卒中史、吸烟)、发病后其他临床资料[吞咽困难、低密度脂蛋白胆固醇、白细胞、中性粒细胞、基线NIHSS评分、术后NIHSS评分、血管内操作结果(mTICI分级2b级或3级)、90 d良好预后(mRS 0~1分)等指标],并进行组间比较。将单因素分析中$P<0.1$的参数作为自变量,进一步对AIS患者经EVT治疗后发生SAP的影响因素行多因素Logistic回归分析。**结果** 共纳入426例AIS患者,其中SAP患者194例(45.5%)。在纳入的所有患者中,多因素Logistic回归分析显示,入院白细胞计数(OR=1.125, 95%CI:1.043~1.213,P=0.000 2)、术后NIHSS(OR=1.019, 95% CI:1.001~1.037,P=0.041)、男性(OR=1.687, 95%CI:1.078~2.638,P=0.022)与急性缺血性卒中患者EVT治疗后发生SAP有关。**结论** 入院白细胞计数较高、术后NIHSS评分较高、男性为AIS患者EVT治疗发生SAP的危险因素,应重点关注。

[**关键词**] 急性缺血性卒中;卒中相关性肺炎;血管内治疗;危险因素

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Risk factors for stroke-associated pneumonia after endovasular treatment in acute anterior circulation ischemic stroke patients

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[**Abstract**] **Objective** To investigate the influencing factors for stroke-associated pneumonia (SAP) in acute ischemic stroke (AIS) patients after endovascular treatment (EVT). **Methods** A retrospective case-control trial was conducted on 426 AIS patients with large vessel occlusion (LVO) in anterior circulation admitted in the neurological departments from First Affiliated Hospital of Army Medical University and Zigong Third People's Hospital during January 2017 and April 2021. Based on SAP occurrence or not, they were divided into an SAP group and a non-SAP group. Demographic information (gender and age), TOAST stroke

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subtypes (large artery atherosclerosis type, cardiac embolism type, others), vascular risk factors (hypertension, hyperlipidemia, diabetes, atrial fibrillation, smoking, prior stroke history, smoking), and post-onset clinical data [dysphagia, LDL cholesterol, white blood cells, neutrophils, baseline and postoperative NIHSS scores, endovascular outcomes (mTICI grade 2b or 3), 90-day good prognosis (mRS 0-1)] were collected and compared between the 2 groups. Multivariate logistic regression analysis was performed using the parameters with $P<0.1$ in univariate analysis as independent variables to investigate factors influencing SAP occurrence after EVT in AIS patients. **Results** Among the 426 participants, SAP occurred in 194 cases (45.5%). Multivariate logistic regression analysis revealed that admission white blood cell count (*OR*=1.125, 95% *CI*: 1.043~1.213, *P*=0.000 2), postoperative NIHSS score (*OR*=1.019, 95% *CI*: 1.001~1.037, *P*=0.041), and male (*OR*=1.687, 95% *CI*: 1.078~2.638, *P*=0.022) were associated with SAP occurrence after EVT in AIS patients. **Conclusion** Higher admission white blood cell count, elevated postoperative NIHSS score, and male gender are risk factors for SAP in AIS patients after EVT. These risk factors should be focused on clinical practice to control SAP incidence.

[**Key words**] acute ischemic stroke; stroke-associated pneumonia; endovascular treatment; risk factors

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大血管闭塞性卒中患者在卒中发生后24 h内接受血管内治疗可得到临床获益[1]。卒中相关性肺炎(stroke -associated pneumonia, SAP)是缺血性卒中后的常见并发症之一[2-3]。既往研究表明,SAP与急性卒中早期死亡风险高、功能转归较差和住院时间较长相关[4-7]。SAP的发病率介于2.3%~31.3%之间[8-10],而大血管闭塞性卒中患者中SAP的发生率尤为突出。SAP的病理生理机制涉及多因素参与。卒中通过激活自主神经系统和应激轴抑制免疫反应,在SAP的发生发展中起重要作用[11-12]。卒中后的全身性免疫抑制导致SAP的发生风险增高[13]。除卒中导致的免疫抑制外,吞咽障碍也是SAP常见的原因之一[14-17]。因此,为更有效预防接受血管内治疗的患者发生SAP,早期识别显得尤为重要。探索SAP的危险因素并厘清其与SAP的关联,有助于实现早期预测与针对性干预。本研究通过回顾性病例对照分析急性缺血性卒中(acute ischemic stroke, AIS)患者的临床资料、实验室检查、影像学资料,旨在探究AIS患者血管内治疗(endovascular treatment, EVT)后SAP的影响因素,以期为临床改善此类患者预后提供预测依据。

1 资料与方法

1.1 研究对象

选取2017年1月至2021年4月在陆军军医大学第一附属医院神经内科与自贡市第三人民医院神经内科所有急性前循环大血管闭塞性卒中且行血管内治疗的患者。纳入标准:①AIS患者;②年龄18~80岁;③影像学检查确定前循环大血管闭塞;④行血管内治疗。排除标准:①社区获得性肺炎患者;②小血管病型;③临床或影像学资料不齐全患者。本研究经陆军军医大学第一附属医院伦理委员会[(B)KY2021146]及自贡市第三人民医院伦理委员会(2021-01-01)批准。

1.2 治疗方法

头颅急诊CT或CTA检查明确无出血病灶。在溶栓时间窗(4.5 h)内的患者排除相关禁忌证且与家属沟通后行静脉内rt-PA-血管内桥接治疗。对超过4.5 h时间窗,有急诊血管内治疗手术适应证患者,充分告知患者家属风险后,行血管内治疗。采用Solitaire取栓支架行血管内取栓。完成取栓后,行DSA检查明确是否有前向血流以确定开通是否成功。如未成功,可重复取栓,取栓重复不超过4次,直至成功再通[定义为脑梗死的改良溶栓(modified thrombolysis in cerebral infarction, mTICI)评分>2b级]。如果再通失败,则进行补救性措施。

1.3 研究方法

收集患者的临床、血液学、影像学及随访资料。所有患者入院后24 h内由经过培训的护理人员进行吞咽筛查,包括意识水平、姿势、配合试验能力、口腔运动功能和饮水试验[18]。A2DS2评分范围为0~10分(预测SAP的有效工具)[3]。根据卒中相关性肺炎组共识,卒中相关性肺炎被定义为卒中后7 d内发生的肺

炎[19]。将患者分组后,比较2组患者的基线资料。影像资料的评估由2位资深神经介入医师评估。患者良好预后结局是指发病后90 d mRS评分0~1分。随访由专业的医师以电话或门诊面对面的形式进行评估。

1.4 统计学方法

使用SPSS 22.0统计软件进行分析。患者临床、入院时检验、影像学资料描述:符合正态分布定量资料使用$\bar{x}\pm s$表示,不符合正态分布的偏态分布定量资料使用中位数和$M(P_{25},P_{75})$表示,定性资料使用例数和百分比(%)表示。SAP组及非SAP组资料的比较,符合正态分布的连续变量使用t检验,偏态分布的连续变量使用Mann-Whitney U检验,定性资料使用卡方检验或Fisher精确检验。将单因素分析中变量(P<0.1)纳入多因素Logistic回归模型,评估独立预测因子,当P<0.05表示差异具有统计学意义。

2 结果

2.1 患者的临床资料

本研究共纳入426例患者。年龄(69.83±12.08)岁,51.6%为男性。368例(86.4%)患者成功再通。根据是否发生SAP将患者分为2组。194例(45.5%)患者发生SAP(SAP组),其余为非SAP组。

2.2 影响急性缺血性卒中患者EVT治疗后SAP的单因素分析

SAP组患者男性、住院天数、吸烟、术后NIHSS评分、吞咽困难、入院白细胞、入院中性粒细胞的比例高于非SAP组,但90 d良好预后率低于非SAP组(P<0.05),其余临床资料无统计学差异(表1)。

2.3 影响急性缺血性卒中患者EVT治疗后SAP的多因素Logistic回归分析

将表1中P<0.1的变量作为自变量,包括年龄、男性、住院天数、吸烟史、术后NIHSS、吞咽困难、A2DS2、入院白细胞计数、入院中性粒细胞计数进行多变量Logistic回归分析。结果显示,入院白细胞计数(OR=1.125, 95%CI: 1.043~1.213,P=0.0002)、术后NIHSS(OR=1.019, 95% CI :1.001~1.037,P=0.041)、男性(OR=1.687, 95% CI: 1.078~2.638,P=0.022)与急性缺血性卒中患者EVT治疗后发生SAP有关(P<0.05),见表2。

3 讨论

卒中相关性肺炎可增加急性卒中患者的早期死亡风险,导致更差的功能转归和更长的住院时间。接受EVT的患者因麻醉、长期卧床等原因易发生SAP,降低EVT的获益。因此,早期识别SAP是必要的。在本研究中观察到在接受EVT治疗的AIS患者中,SAP与性别、EVT后较高的NIHSS评分、较高的白细胞计数密切相关。对于存在这些危险因素的患者,应保持警惕,防范SAP的发生。

3.1 EVT后神经功能缺损严重程度是SAP的核心预测因素

NIHSS评分是衡量缺血性卒中后神经功能损伤程度的重要指标,也是影响SAP发生风险的已知因素。既往研究表明,较高的基线NIHSS评分与SAP风险增加相关[20-21]。这是因为NIHSS评分高的患者神经功能严重缺损常伴随意识障碍(如昏迷)和吞咽困难,更可能需要留置鼻胃管,这直接增加了误吸风险。意识障碍程度深、昏迷时间长的患者,气道保护能力持续削弱,是SAP发生的极高危因素。此外,神经功能严重受损导致患者卧床时间延长,从而增加吸入性肺炎的风险。肢体瘫痪严重、活动受限时间延长,也使得坠积性肺炎的发生风险增高,最终导致SAP发生率上升。然而,本研究发现,基线NIHSS评分与SAP无显著相关性;相反,EVT治疗后较高的NIHSS评分才是SAP的独立预测因素。这一差异的可能原因在于,EVT能显著改善卒中患者的神经功能,且EVT后的NIHSS评分更能准确反映患者住院期间的临床状态。本研究建立了AIS患者EVT后的NIHSS评分和SAP之间的关联。

3.2 入院时白细胞计数升高提示SAP风险增加

既往研究表明,AIS发生后可诱发局部炎症免疫反应,进而导致免疫功能抑制,增加SAP的发生风险[22]。白细胞计数是反映机体炎症状态的重要指标[13,23]。AIS后持续的炎症反应可过度或长时间持续性消耗免疫系统资源,引发继发性全身免疫抑制,从而提高患者的感染易感性。本研究发现,白细胞计数与接受EVT治疗的AIS患者术后SAP的发生显著相关。鉴于白细胞计数检测便捷,其可作为早期识别SAP潜在风险的辅助指标。

3.3 男性性别是SAP的独立危险因素

本研究中,SAP组男性比例显著高于非SAP组,且男性是AIS患者接受EVT治疗后发生卒中相关性肺炎的独立危险因素。这一发现与既往研究结果一致。有研究表明,男性发生SAP的风险高于女性,这可能部分源于生活习惯的差异[24]。现有证据提示,男性更高的SAP风险或与其吸烟、饮酒、作息紊乱及精神压力等不良生活方式相关;长期吸烟所致的慢性肺损伤亦进一步增加了患病概率[24]。但更主要的机制可能

表1 非卒中后相关肺炎组与卒中后相关肺炎组人口统计学和临床特征比较[$\bar{x}\pm s$,例(%),$M(P_{25},P_{75})$]

Table 1 Comparison of demographic and clinical characteristics between the non- and stroke-associated pneumonia (SAP) group [$\bar{x}\pm s$, n(%), $M(P_{25},P_{75})$]

Factors	SAP(n=194)	Non-SAP(n=232)	$t/Z/\chi^2$	P_{Value}
Age/years	70.0(61.7,79.0)	72.0(64.0, 79.0)	−1.710[c]	0.087
Male	113 (58.2)	107 (46.1)	9.148[b]	0.002
Length of hospital stay/d	17.0 (9.0,30.0)	9.5(3.0,14.0)	−5.715[c]	<0.001
Hypertension	88 (45.4)	110 (47.4)	0.179[b]	0.672
Hyperlipidaemia	104 (53.6)	122 (52.6)	0.044[b]	0.833
Diabetes mellitus	14 (7.2)	19 (8.2)	0.140[b]	0.708
Atrial fibrillation	40 (20.6)	51 (22.0)	0.117[b]	0.732
Previous stroke	15 (7.7)	25 (10.8)	1.151[b]	0.283
Smoking	71 (36.6)	62 (26.7)	4.797[b]	0.029
Baseline NIHSS	18.0 (14.0,23.0)	17.0(12.0,24.0)	−1.153[c]	0.682
NIHSS after EVT	10.0 (5.0,20.0)	6.0(2.0,16.0)	−3.564[c]	<0.001
NIHSS stratification after EVT			17.649[b]	<0.001
0~4	45 (23.2)	97 (41.8)		
5~15	73 (37.6)	75 (32.3)		
>15	76 (39.2)	60 (25.9)		
Dysphagia	121 (62.4)	118 (50.9)	5.683[b]	0.017
A2DS2	6.21±1.42	5.95±1.54	−1.750[a]	0.081
Cause of stroke			0.248[b]	0.883
Atherosclerosis	94(48.5)	107(46.1)		.
Cardiac embolism	87(44.8)	108(46.6)		
Other	13(6.7)	17(7.3)		
Leukocyte count /(10^9/L)	9.69 (7.47,11.90)	8.07 (6.47,9.81)	−3.717[c]	<0.001
Neutrophil count/(10^9/L)	7.77 (5.46,10.01)	6.14 (4.67,7.93)	−2.564[c]	<0.001
NLR	5.16 (3.01,8.26)	4.97(3.12,7.85)	−0.123[c]	0.902
LDL/(mmol/L)	2.50±0.77	2.40±0.81	−1.128[a]	0.260
Glucose/(mmol/L)	8.10±2.94	7.84±3.05	−0.840[a]	0.401
mTICI 2b or 3	165 (85.0)	203 (87.5)	0.316[b]	0.579
Intra-arterial thrombolysis	26 (13.4)	33 (14.2)	0.060[b]	0.807
90-day mRS score 0-2	70 (36.1)	147 (63.4)	31.461[b]	<0.001
90-day mortality	53 (27.3)	42 (18.1)	5.179[b]	0.023

NIHSS: National Institutes of Health stroke scale; EVT: Endovascular treatment; NLR: Neutrophil to lymphocyte ratio; LDL: Low-density lipoprotein; mTICI: Modified thrombolysis in cerebral infarction; mRS: Modified Rankin scale. a: t; b: χ^2; c: Z

表2 卒中后相关肺炎的多因素Logistic回归分析

Table 2 Multivariate logistic regression analysis of stroke-associated pneumonia (SAP)

Variables	β	SE	OR (95% CI)	P_{Value}
Leukocyte count	0.117	0.039	1.125(1.043~1.213)	0.002
NIHSS after EVT	0.018	0.009	1.019(1.001~1.037)	0.041
Male	0.523	0.228	1.687(1.078~2.638)	0.022

与性激素相关,DZIENNIS等[25]研究表明,雄激素(如睾酮)及其代谢物二氢睾酮会加剧卒中后外周免疫抑制状态,从而增加SAP发生风险。相反,COLBERT等[26]研究发现女性卒中后肺炎感染率相对较低,这可能与雌激素的保护作用相关。大量实验证据表明,雌激素具有固有的神经保护效应,能显著减轻雌性动物的缺血性脑损伤[27]。这种神经保护作用可减少脑损伤程度,进而降低卒中后感染风险。此外,性类固醇(包括雌激素)对卒中后免疫抑制具有调节作用。鉴于性类固醇受体广泛存在于中枢神经系统的各类细胞中,雌激素还可能直接调节卒中后的免疫反应[28]。REID等[29]研究则进一步证实,即使在校正潜在混杂

因素后,男性仍是SAP的独立预测因素,这也与本研究结论相符。

3.4 研究局限性

本研究存在一定局限性。首先,其为回顾性设计,且样本量相对较小,导致统计效力可能不足。因此,研究结果需通过更大规模的前瞻性研究加以验证。其次,AIS患者接受EVT治疗后发生SAP可能是多因素共同作用的结果。本研究未能进行更深入的分层分析以探索所有潜在影响因素。未来有必要收集更全面的临床数据,以期识别更多相关因素并得出更精确的结论。

综上所述,本研究表明入院白细胞计数升高、术后NIHSS评分较高以及男性性别是AIS患者接受EVT治疗后发生SAP的独立危险因素。针对存在这些特定危险因素的患者,实施早期针对性干预对于降低死亡及致残风险、改善患者预后至关重要。

利益冲突 本文所有作者声明不存在利益冲突

参考文献:

[1] 杨清武,资文杰.急性颅内大血管闭塞血运重建:直接还是桥接取栓?——研究回顾及展望[J].第三军医大学学报,2021,43(14):1299-1303. DOI:10.16016/j.1000-5404.202104153.
YANG Q W, ZI W J. Revascularization in acute intracranial large vessels occlusion: direct or bridging thrombectomy?[J]. J Third Mil Med Univ, 2021, 43(14): 1299-1303. DOI: 10.16016/j.1000-5404.202104153.

[2] 王拥军,陈玉国,吕传柱,等.卒中相关性肺炎诊治中国专家共识(2019更新版)[J].中国卒中杂志,2019,14(12):1251-1262. DOI:10.3969/j.issn.1673-5765.2019.12.011.
WANG Y J, CHEN Y G, LYU C Z, *et al*. An updated Chinese consensus statement on stroke-associated pneumonia 2019[J]. Chin J Stroke, 2019, 14(12): 1251-1262. DOI: 10.3969/j.issn.1673-5765.2019.12.011.

[3] HOFFMANN S, MALZAHN U, HARMS H, *et al*. Development of a clinical score (A2DS2) to predict pneumonia in acute ischemic stroke[J]. Stroke, 2012, 43(10): 2617-2623. DOI: 10.1161/STROKEAHA.112.653055.

[4] KOENNECKE H C, BELZ W, BERFELDE D, *et al*. Factors influencing in-hospital mortality and morbidity in patients treated on a stroke unit[J]. Neurology, 2011, 77(10): 965-972. DOI: 10.1212/WNL.0b013e31822dc795.

[5] LANGHORNE P, STOTT D J, ROBERTSON L, *et al*. Medical complications after stroke: a multicenter study[J]. Stroke, 2000, 31(6): 1223-1229. DOI: 10.1161/01.str.31.6.1223.

[6] FINLAYSON O, KAPRAL M, HALL R, *et al*. Risk factors, inpatient care, and outcomes of pneumonia after ischemic stroke[J]. Neurology, 2011, 77(14): 1338-1345. DOI: 10.1212/WNL.0b013e31823152b1.

[7] SELLARS C, BOWIE L, BAGG J, *et al*. Risk factors for chest infection in acute stroke: a prospective cohort study[J]. Stroke, 2007, 38(8): 2284-2291. DOI: 10.1161/STROKEAHA.106.478156.

[8] HUANG G Q, LIN Y T, WU Y M, *et al*. Individualized prediction of stroke-associated pneumonia for patients with acute ischemic stroke[J]. Clin Interv Aging, 2019, 14: 1951-1962. DOI: 10.2147/CIA.S225039.

[9] JI R J, SHEN H P, PAN Y S, *et al*. Novel risk score to predict pneumonia after acute ischemic stroke[J]. Stroke, 2013, 44(5): 1303-1309. DOI: 10.1161/STROKEAHA.111.000598.

[10] CUGY E, SIBON I. Stroke-associated pneumonia risk score: validity in a French stroke unit[J]. J Stroke Cerebrovasc Dis, 2017, 26(1): 225-229. DOI: 10.1016/j.jstrokecerebrovasdis.2016.09.015.

[11] ZHU Y B, GAO J, LV Q S, *et al*. Risk factors and outcomes of stroke-associated pneumonia in patients with stroke and acute large artery occlusion treated with endovascular thrombectomy[J]. J Stroke Cerebrovasc Dis, 2020, 29(11): 105223. DOI: 10.1016/j.jstrokecerebrovasdis.2020.105223.

[12] MEISEL C, SCHWAB J M, PRASS K, *et al*. Central nervous system injury-induced immune deficiency syndrome[J]. Nat Rev Neurosci, 2005, 6(10): 775-786. DOI: 10.1038/nrn1765.

[13] WESTENDORP W F, DAMES C, NEDERKOORN P J, *et al*. Immunodepression, infections, and functional outcome in ischemic stroke[J]. Stroke, 2022, 53(5): 1438-1448. DOI: 10.1161/STROKEAHA.122.038867.

[14] AL-KHALED M, MATTHIS C, BINDER A, *et al*. Dysphagia in patients with acute ischemic stroke: early dysphagia screening may reduce stroke-related pneumonia and improve stroke outcomes[J]. Cerebrovasc Dis, 2016, 42(1/2): 81-89. DOI: 10.1159/000445299.

[15] DENNIS M S, LEWIS S C, WARLOW C, *et al*. Effect of timing and method of enteral tube feeding for dysphagic stroke patients (FOOD): a multicentre randomised controlled trial[J]. Lancet, 2005, 365(9461): 764-772. DOI: 10.1016/S0140-6736(05)17983-5.

[16] ZHANG R H, JI R J, PAN Y S, *et al*. External validation of the prestroke independence, sex, age, national institutes of health stroke scale score for predicting pneumonia after

stroke using data from the China national stroke registry[J]. J Stroke Cerebrovasc Dis, 2017, 26(5): 938-943. DOI: 10.1016/j.jstrokecerebrovasdis.2016.10.043.

[17] SMITH C J, BRAY B D, HOFFMAN A, *et al*. Can a novel clinical risk score improve pneumonia prediction in acute stroke care? A UK multicenter cohort study[J]. J Am Heart Assoc, 2015, 4(1): e001307. DOI: 10.1161/JAHA.114.001307.

[18] HIGASHIDA R T, FURLAN A J, ROBERTS H, *et al*. Trial design and reporting standards for intra-arterial cerebral thrombolysis for acute ischemic stroke[J]. Stroke, 2003, 34(8): e109-37. DOI: 10.1161/01.STR.0000082721.62796.09.

[19] SMITH C J, KISHORE A K, VAIL A, *et al*. Diagnosis of stroke-associated pneumonia: recommendations from the pneumonia in stroke consensus group[J]. Stroke, 2015, 46(8): 2335-2340. DOI: 10.1161/STROKEAHA.115.009617.

[20] KWON H M, JEONG S W, LEE S H, *et al*. The pneumonia score: a simple grading scale for prediction of pneumonia after acute stroke[J]. Am J Infect Control, 2006, 34(2): 64-68. DOI: 10.1016/j.ajic.2005.06.011.

[21] CHUMBLER N R, WILLIAMS L S, WELLS C K, *et al*. Derivation and validation of a clinical system for predicting pneumonia in acute stroke[J]. Neuroepidemiology, 2010, 34(4): 193-199. DOI: 10.1159/000289350.

[22] FLURI F, MORGENTHALER N G, MUELLER B, *et al*. Copeptin, procalcitonin and routine inflammatory markers-predictors of infection after stroke[J]. PLoS One, 2012, 7(10): e48309. DOI: 10.1371/journal.pone.0048309.

[23] FAURA J, BUSTAMANTE A, MIRÓ-MUR F, *et al*. Stroke-induced immunosuppression: implications for the prevention and prediction of post-stroke infections[J]. J Neuroinflamm, 2021, 18(1): 127. DOI: 10.1186/s12974-021-02177-0.

[24] FRIEDANT A J, GOUSE B M, BOEHME A K, *et al*. A simple prediction score for developing a hospital-acquired infection after acute ischemic stroke[J]. J Stroke Cerebrovasc Dis, 2015, 24(3): 680-686. DOI: 10.1016/j.jstrokecerebrovasdis.2014.11.014.

[25] DZIENNIS S, AKIYOSHI K, SUBRAMANIAN S, *et al*. Role of dihydrotestosterone in post-stroke peripheral immunosuppression after cerebral ischemia[J]. Brain Behav Immun, 2011, 25(4): 685-695. DOI: 10.1016/j.bbi.2011.01.009.

[26] COLBERT J F, TRAYSTMAN R J, POISSON S N, *et al*. Sex-related differences in the risk of hospital-acquired sepsis and pneumonia post acute ischemic stroke[J]. J Stroke Cerebrovasc Dis, 2016, 25(10): 2399-2404. DOI: 10.1016/j.jstrokecerebrovasdis.2016.06.008.

[27] ROOF R L, HALL E D. Gender differences in acute CNS trauma and stroke: neuroprotective effects of estrogen and progesterone[J]. J Neurotrauma, 2000, 17(5): 367-388. DOI: 10.1089/neu.2000.17.367.

[28] MEISEL C, MEISEL A. Suppressing immunosuppression after stroke[J]. N Engl J Med, 2011, 365(22): 2134-2136. DOI: 10.1056/NEJMcibr1112454.

[29] REID J M, DAI D W, GUBITZ G J, *et al*. Gender differences in stroke examined in a 10-year cohort of patients admitted to a Canadian teaching hospital[J]. Stroke, 2008, 39(4): 1090-1095. DOI: 10.1161/STROKEAHA.107.495143.

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