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巨噬细胞移动抑制因子在阻塞性睡眠呼吸暂停低通气综合征中的表达及与血管内皮损伤的相关性

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[摘　要]　**目的：**探讨巨噬细胞移动抑制因子(macrophage migration inhibition factor，MIF)在阻塞性睡眠呼吸暂停低通气综合征(obstructive sleep apnea hypopnea syndrome，OSAHS)中的表达及与血管内皮损伤的相关性。**方法：**选择2016年9月至2018年9月在秦皇岛军工医院收治的OSAHS患者126例，按病情严重程度分为轻度组(n=42)、中度组(n=42)、重度组(n=42)；另选42例健康人群作为对照组。分析各组中MIF的表达情况及血管内皮损伤标志物的水平差异。**结果：**各组AHI、氧减指数、最低血氧饱和度水平比较，差异均有统计学意义(均P<0.05)。对照组AHI、氧减指数水平最低，随病情加重，逐渐升高，以重度组最高；对照组最低血氧饱和度水平最高，随病情加重，逐渐降低，以重度组最低(P<0.05)。各组MIF，TNF-α，IL-6，sVCM-1，ET，CPR，vWF水平比较，整体差异均有统计学意义(均P<0.05)，对照组MIF，TNF-α，IL-6，sVCM-1，ET，CPR，vWF水平最低，随病情加重，逐渐升高，以重度组最高(P<0.05)。MIF与OSAHS严重程度呈正相关(r=0.691，P<0.001)。MIF与AHI、氧减指数正相关，与最低血氧饱和度负相关(P<0.05)。MIF与TNF-a，IL-6，sVCM-1，ET，CPR，vWF存在不同程度正相关(P<0.05)。**结论：**OSAHS患者血清MIF水平随疾病的严重程度明显升高,与血管损伤相关因子具有正向相关性，提示MIF可能参与了OSAHS的炎性反应，在血管内皮损伤中起一定作用。

[关键词]　阻塞性睡眠呼吸暂停低通气综合征；巨噬细胞移动抑制因子；血管内皮；炎性因子

Expression of macrophage migration inhibitory factor in obstructive sleep apnea hypopnea syndrome and its correlation with vascular endothelial injury

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Abstract　**Objective:** To investigate the expression of macrophage migration inhibition factor (MIF) in obstructive sleep apnea hypopnea syndrome (OSAHS) and its correlation with vascular endothelial injury. **Methods:** From

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September 2016 to September 2018, 126 patients with OSAHS were divided into the mild group (n=42), the moderate group (n=42) and the severe group (n=42) according to the severity of the disease, and 42 healthy people were selected as the control group. The expression of MIF and the level of vascular endothelial injury markers in each group were analyzed. **Results:** There were significant differences in AHI, oxygen reduction index and lowest oxygen saturation among the groups (all P<0.05). The control group had the lowest levels of AHI and Oxygen Reduction Index, and gradually increased with the aggravation of the disease, with the highest level in the severe group; the lowest level of oxygen saturation in the control group was the highest, with the aggravation of the disease, gradually decreased, with the lowest level in the severe group (P<0.05). The diffrences in the MIF, TNF-α, IL-6, sVCM-1, ET, CPR, and vWF levels were all statistically significant (all P<0.05); the MIF, TNF-α, IL-6, sVCM-1, CPR, vWF levels in the control group were the lowest, which gradually increased with the severity of the disease, the levels in the severe group were the highest (P<0.0.5). MIF was positively correlated with OSAHS severity (r=0.691, P<0.001). MIF was positively correlated with AHI and oxygen reduction index, and negatively correlated with the lowest oxygen saturation (P<0.05). MIF was positively correlated with TNF-α, IL-6, sVCM-1, ET, CPR and vWF (P<0.05). **Conclusion:** Serum MIF levels in OSAHS patients is significantly increased with the severity of the disease and positively correlated with vascular injury related factors, suggesting that MIF may participate in the inflammatory response of OSAHS and play a role in vascular endothelial injury.

Keywords obstructive sleep apnea hypopnea syndrome; macrophage migration inhibitory factor; vascular endothelium; inflammatory factor

阻塞性睡眠呼吸暂停低通气综合征(obstructive sleep apnea hypopnea syndrome，OSAHS)是指患者在睡眠过程中，由多种因素造成的上呼吸道的完全或部分阻塞，引发的睡眠打鼾、呼吸暂停等间断性缺氧的一系列症候群[1]。

OSAHS属于一种慢性炎性反应性疾病，白介素-6(interleukin 6，IL-6)、C-反应蛋白(C-reactive protein，CRP)等炎性介质表达增加，可诱导血管内皮损伤和动脉粥样硬化，进而造成OSAHS患者发生心脑血管并发症[2]。巨噬细胞移动抑制因子(macrophage migration inhibition factor，MIF)是一种炎性反应促进因子，参与血管内皮损伤和动脉粥样硬化的病理进程[3]。研究[4-6]表明：MIF与机体糖代谢紊乱、胰岛素抵抗密切相关，可能与OSAHS的严重程度及血管内皮损伤具有相关性，但目前关于MIF是否参与OSAHS合并血管内皮损伤的相关报道仍较少。本研究观察不同程度OSAHS患者血清MIF及血管内皮损伤相关因子的表达水平，探讨MIF的表达情况及其与疾病严重程度和血管内皮损伤标志物的相关性。

1 对象与方法

1.1 对象

选择2016年9月至2018年9月在秦皇岛军工医院收治的126例OSAHS患者。参照《阻塞性睡眠呼吸暂停低通气综合征诊治指南》[7]诊断OSAHS：每次睡眠呼吸暂停低通气指数(apnea-hypopnea index，AHI) >5次/h或每晚呼吸暂停反复发作次数>30次。参照指南按OSAHS严重程度分组，轻度OSAHS患者为轻度组(n=42)，AHI(5~15次/h)；中度OSAHS患者为中度组(n=42)，AHI(16~30次/h)；重度OSAHS患者为重度组(n=42)，AHI(>30次/h)。另匹配同期在秦皇岛军工医院接诊的42例健康体检者为对照组。排除标准：1)严重感染者或免疫系统疾病患者；2)慢性阻塞性肺疾病、中枢性睡眠呼吸暂停综合征者或呼吸窘迫综合征、支气管哮喘等疾病患者；3)严重精神疾病者；4)严重凝血功能障碍者；5)严重肾、肝等脏器疾病者；6)近2周内手术或重大创伤者。所有研究对象及家属均签署知情同意书，本研究经秦皇岛军工医院医学伦理委员会批准通过。所有研究对象性别、年龄、BIM、病程等基线资料差异无统计学意义(P>0.05)，具有可比性(表1)。

1.2 观测指标与方法

1)采用凯迪泰医学科技有限公司多导睡眠监测仪对所有研究对象行睡眠相关指标检查，于检查前24 h均停止服用刺激性饮料或影响本研究相关药物，检测AHI、氧减指数、最低血氧饱和度。2)次

日晨起取空腹静脉血4 mL，抗凝后以3 000 r/min离心，取上清液−80 ℃保存备用。采用ELISA法[8]，分别检测MIF、肿瘤坏死因子α(tumor necrosis factor α，TNF-α)、IL-6、可溶性血管细胞黏附分子(soluble vascular cell adhesion molecule，sVCM)-1、内皮素(endothelin，ET)、CPR、血管性血友病因子(von Willebrand factor，vWF)，严格参照ELISA试剂盒说明书操作(有限公司)。

1.3 统计学处理

采用SPSS 22.0统计软件进行数据分析。检验数据符合正态分布及方差齐性，以均数±标准差($\bar{x}\pm s$)表示，各指标在各组间的差异采用单因素方差分析，各亚组间两两比较采用LSD-t检验，计数资料及率的比较采用χ^2检验，分别以Pearson法分析各指标间相关性。P<0.05为差异有统计学意义。

表1 各组患者基线情况比较(n=42)

Table 1 Comparison of baseline conditions of patients in each group (n=42)

组别	男/女	年龄/岁	BIM/(kg·m^{-2})	病程/年	吸烟/例	饮酒/例
对照组	27/15	42.5 ± 5.3	24.3 ± 4.2	4.04 ± 0.46	17	5
轻度组	24/18	43.1 ± 5.8	25.4 ± 4.5	4.02 ± 0.56	20	6
中度组	23/19	42.8 ± 6.4	25.1 ± 5.6	4.08 ± 0.62	24	8
重度组	25/17	43.4 ± 6.2	25.2 ± 5.8	4.03 ± 0.78	25	7
统计值	0.861	1.066	1.103	1.191	3.907	0.910
P	0.835	0.365	0.349	0.315	0.272	0.823

2 结果

2.1 各组睡眠呼吸相关参数比较

各组AHI、氧减指数、最低血氧饱和度水平比较，差异均有统计学意义(均P<0.05，表2)；随后两两比较发现，各组间差异均有统计学意义(P<0.05，表2)，其中对照组AHI、氧减指数水平最低，随病情加重，逐渐升高，以重度组最高；对照组最低血氧饱和度水平最高，随病情加重，逐渐降低，以重度组最低(P<0.05，表2)。

2.2 各组血管损伤相关因子比较

各组MIF，TNF-α，IL-6，sVCM-1，ET，CPR，vWF水平比较，差异均有统计学意义(均P<0.05，表3)；随后两两比较，各组间差异均有统计学意义(P<0.05，表3)；其中对照组MIF，TNF-α，IL-6，sVCM-1，ET，CPR，vWF水平最低，随病情加重，逐渐升高，以重度组最高(P<0.05，表3)。

表2 各组睡眠呼吸相关参数比较(n=42)

Table 2 Comparison of related parameters of sleep and breathing in each group (n=42)

组别	AHI/(次·h^{-1})	氧减指数/(次·h^{-1})	最低血氧饱和度/%
对照组	2.41 ± 0.32	1.62 ± 0.41	94.23 ± 14.25
轻度组	12.71 ± 3.84*	17.74 ± 2.61*	83.52 ± 12.26*
中度组	22.60 ± 4.52*#	34.63 ± 7.31*#	77.56 ± 11.52*#
重度组	59.83 ± 7.28*#&	56.51 ± 8.13*#&	65.41 ± 12.37*#&
统计值	1 623.930	755.505	39.063
P	0.001	0.001	0.001

与对照组比较，*P<0.05；与轻度组比较，#P<0.05；与中度组比较，&P<0.05。

Compared with the control group, *P <0.05; compared with the mild group, #P<0.05; compared with the moderate group, &P<0.05.

表3 各组血管损伤相关因子比较(n=42)

Table 3 Comparison of vascular injury related factors in each group (n=42)

组别	MIF	TNF-α/(ng·L^{-1})	IL-6/(ng·mL^{-1})	sVCM-1/(ng·mL^{-1})	ET/(pg·mL^{-1})	CPR/(mg·L^{-1})	vWF/(U·mL^{-1})
对照组	20.5 ± 4.2	56.5 ± 5.3	27.3 ± 4.2	1.34 ± 0.26	8.3 ± 1.2	5.3 ± 1.2	1.09 ± 0.12
轻度组	22.8 ± 5.3*	63.1 ± 6.8*	29.7 ± 5.6*	1.42 ± 0.16*	12.6 ± 1.4*	12.4 ± 1.5*	1.17 ± 0.17*
中度组	25.6 ± 5.8*#	68.8 ± 7.4*#	36.7 ± 5.8*#	1.68 ± 0.32*#	17.5 ± 2.7*#	18.1 ± 2.6*#	1,96 ± 0.25*#
重度组	32.4 ± 6.4*#&	117.4 ± 13.2*#&	52.6 ± 6.4*#&	2.33 ± 0.28*#&	25.6 ± 4.3*#&	32.2 ± 2.8*#&	2.68 ± 0.43*#&
统计值	23.654	405.295	169.298	196.182	352.914	1277.002	362.779
P	0.001	0.001	0.001	0.001	0.001	0.001	0.001

与对照组比较，*P<0.05；与轻度组比较，#P<0.05；与中度组比较，&P<0.05。

Compared with the control group, *P<0.05; compared with the mild group, #P<0.05; compared with the moderate group, &P<0.05.

2.3 MIF 与呼吸睡眠相关指标相关性分析

MIF与OSAHS严重程度呈正相关(r=0.691，P<0.001)，MIF与AHI、氧减指数正相关，与最低血氧饱和度负相关(表4)。MIF与TNF-α，IL-6，sVCM-1，ET，CPR，vWF存在不同程度正相关(表5)。

表4 MIF与呼吸睡眠相关指标相关性分析

Table 4 Correlation analysis between MIF and respiratory sleep related indexes

因子	r			
	MIF	AHI	氧减指数	最低血氧饱和度
MIF	1.000	0.364*	0.683*	−0.442*
AHI	0.364*	1.000	0.377*	−0.517*
氧减指数	0.683*	0.377*	1.000	−0.415*
最低血氧饱和度	−0.442*	−0.517*	−0.415*	1.000

*P<0.05.

表5 MIF与血管损伤相关因子相关性分析

Table 5 Correlation analysis between MIF and vascular injury related factors

因子	r						
	MIF	TNF-α	IL-6	sVCM-1	ET	CPR	vWF
MIF	1.000	0.364*	0.683*	0.442*	0.441*	0.422*	0.521*
TNF-α	0.364*	1.000	0.377*	0.517*	0.575*	0.533*	0.502*
IL-6	0.683*	0.377*	1.000	0.415*	0.377*	0.415*	0.683*
sVCM-1	0.442*	0.517*	0.415*	1.000	0.517*	0.431	0.475
ET	0.441*	0.575*	0.377*	0.517*	1.000	0.547*	0.741*
CPR	0.422*	0.533*	0.415*	0.431*	0.547*	1.000	0.531*
vWF	0.521*	0.502*	0.683*	0.475*	0.741*	0.531*	1.000

*P<0.05.

3 讨论

OSAHS常合并全身多系统及多器官障碍，其机制可能与睡眠呼吸暂停间断性缺氧所引发的低氧血症、高碳酸血症、氧化应激、炎症级联反应等密切相关[9]。有学者[10]发现OSAHS可能是心血管疾病的危险因素，其中MIF及各种心血管损伤炎性因子可能参与病情的发生发展，并扮演重要角色。本研究中TNF-α，IL-6，sVCM-1，ET，CPR，vWF水平随着OSAHS疾病的严重程度的增加而增高，提示这几个因子参与了OSAHS的炎性反应，并与病情严重程度存在正向相关性。MIF是受下丘脑-垂体控制的多功能细胞因子，与多种心血管及代谢性疾病密切相关，与糖代谢及氧化应激反应的水平有关[11]。MIF可通过NF-κB等多种信号途径参与信号转导并发挥生物学效应，MIF广泛表达于多种器官、组织的细胞中，具有多种生物学功能，与多种急性、慢性炎症反应相关[12]。近来研究证明MIF也与动脉粥样硬化、糖尿病等心血管及代谢疾病相关[13]。

本研究中患者的MIF水平随OSAHS严重程度逐渐升高，且均明显高于健康人群，这可能由于随病情加重，OSAHS患者受到炎性反应、生理功能紊乱等多种因素共同刺激，使MIF基因表达谱发生改变所致。这与袁平等[13]的研究结果相似，该研究发现血浆中MIF表达水平在重度OSAHS组明显高于对照组，MIF与AHI和氧减指数呈正相关，与最低血氧饱和度呈负相关，这可能是因为OSAHS患者的间断缺氧状态可诱导MIF高表达。MIF可抑制巨噬细胞游走，促进巨噬细胞聚集、增生、活化及分泌一些炎性因子；MIF抑制淋巴细胞凋亡，对抗糖皮质激素的作用，调节炎症反应[11-13]。

本研究中SLT90%，ODI，LSpO水平随OSAHS病情加重逐渐上升。相关性分析发现：OSAHA患者的血清MIF水平与OSAHA严重程度正相关，与AHI呈正相关，与LSpO负相关，说明MIF水平和OSAHS严重程度有关，OSAHS越重，MIF水平越高。这是因为SLT90%，ODI，LSpO均是评价OSAHS严重程度的指标，SLT90%越高，说明机体睡眠时血氧饱和度低于90%的时间越长；ODI越高，说明OSAHS患者夜间低氧血症发生次数越多；LSpO越低说明OSAHS患者缺氧越重[14]，本研究结果与其他学者[13-15]的研究结果一致，其机制可能是间断缺氧导致氧化应激，大量的炎症因子及脂肪因子诱导巨噬细胞及脂肪细胞中的MIF表达增多。OSAHS患者血清MIF水平明显升高，并与AHI呈显著正相关，缺氧刺激血管内皮细胞及单核细胞的MIF释放。

本研究发现：OSAHS患者的血管内皮损伤标志物TNF-α，IL-6，sVCM-1，ET，CPR，vWF水平随OSAHS程度加重而上升，且与MIF呈正相关，这是因为各种炎性细胞因子所致的炎症反应是冠心病的重要影响因素，TNF-α，IL-6，sVCM-1，ET，CPR，vWF水平升高与心血管疾病密切相关，血管内皮的炎性反应可致血管内皮损伤[16-18]。CRP可指示全身炎症，并与动脉粥样斑块及粥样硬化形成相关[16-18]。IL-6是炎症过程的关键介质，其活性可能与心血管疾病有明关，高水平的IL-6可预测心肌梗死的病死率[16-18]，本研究结果与曹江等[16]的研究结论相似，分析原因可能为OSAHS患者间歇性缺氧后MIF水平升高，刺激了TNF-α，IL-6，sVCM-1，ET，CPR，vWF等炎性因子的大量释放，引起了机体的炎性反应，使血管内皮细胞发生损伤，导致血浆中TNF-α，IL-6，sVCM-1，ET，CPR，vWF水平增高，进而出现心脑血管并发症，提示MIF与OSAHS患者病情严重程度相关，可能参与了OSAHS血管内皮细胞损伤的病理过程[15]。

综上所述，OSAHS患者血清MIF水平随疾病的严重程度明显升高，与血管损伤相关因子具有正向相关性，提示MIF可能参与OSAHS的炎性反应，在血管内皮损伤中起一定作用。

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