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个案与短篇

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系统性红斑狼疮合并伤寒1例

刘会芳,张　憬,赵景宏　（400037 重庆,第三军医大学新桥医院肾内科）

[**关键词**]　系统性红斑狼疮;伤寒;发热;狼疮性脑病
[**中图法分类号**]　R516.3; R593.24　[**文献标志码**]　B

系统性红斑狼疮(systemic lupus erythematosus, SLE)一种全身性自身免疫病。由于免疫功能紊乱及长期免疫抑制剂的应用,易继发感染,出现发热、腹泻等症状,严重时常危及生命[1]。伤寒是由伤寒杆菌引起,以持续发热、相对缓脉、神情淡漠、脾大、玫瑰疹和血白细胞减少等为特征的急性消化道传染病[2],在临床症状不典型且合并有其他疾病时很难鉴别。SLE合并伤寒极其少见,易误诊,现将我科收治的1例SLE合并伤寒报告如下。

1　临床资料

患者,男性,26岁,因"反复水肿、颜面红斑9年,发热、头痛3 h"于2012年7月17日入院。患者9年前受凉后发热,体温最高40 ℃,常规抗感染治疗无明显好转,且出现全身水肿、颜面蝶形红斑、畏光、口腔多发溃疡,尿常规示蛋白尿,入我科住院治疗。经肾活检、查抗核抗体谱等,诊断:①慢性肾功能衰竭(氮质血症期);②系统性红斑狼疮,狼疮性肾炎。予以激素、环磷酰胺及保肾排毒等治疗,病情稳定后出院。院外长期服用激素、环磷酰胺等。

3 h前因受凉出现头痛、发热,自查体温37.8 ℃,伴咳嗽,无腹痛、腹泻、恶心、呕吐等症状,服用塞来昔布头痛缓解,但仍感发热,遂入院。查体:体温37.5 ℃,慢性病容,腹两侧及双腿多发紫色斑纹。心、肺、腹无阳性体征,双下肢无水肿。实验室检查:白细胞计数 15.5×10^9/L, 中性粒细胞百分率84.9%,补体 C_3 0.63 (参考值0.9~2.1)。尿蛋白+++,24 h尿蛋白定量1.6 g。血沉、抗核抗体谱正常、PPD试验阴性。胸部CT未见异常。结合既往病史,诊断:系统性红斑狼疮、狼疮性肾炎。但发热原因尚不明确,予以激素、降尿蛋白、改善循环、物理降温等治疗[3],并行痰、血培养,结果均无异常。患者有受凉病史,考虑发热为病毒性感冒引起,但不排除狼疮活动所致,在物理降温的基础上予以利巴韦林抗病毒治疗。治疗过程中患者仍间断出现发热,体温最高39 ℃,伴头痛,无腹痛、腹泻、皮疹等。头颅CT示:脑室系统扩张,余未见异常。再行多次血培养,结果均阴性。考虑发热系狼疮活动、头痛因狼疮性脑病所致,故予以激素冲击治疗。3 d后复查血常规示血象高、补体 C_3 0.77,且患者再次出现发热,体温39 ℃,伴严重头痛,尿蛋白无明显增加、补体 C_3 无明显减低。故除外狼疮活动,予以小剂量激素治疗,行血培养。8月9日血培养结果示:伤寒沙门氏菌,对美罗培南、环丙沙星等敏感,查肥达反应示阴性,美罗培南抗感染治疗。患者发热、头痛症状逐渐缓解,抗感染治疗有效,诊断系统性红斑狼疮合并伤寒。待患者未诉发热,血常规恢复正常,一般情况可,于8月23日出院。

2　讨论

该患者青年男性,诊断系统性红斑狼疮成立。狼疮患者发

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[**通信作者**]　赵景宏,电话:(023)68774321,E-mail:zhaojh73@yahoo.com.cn
[**优先出版**]　http://www.cnki.net/kcms/detail/51.1095.R.20121211.1307.001.html(2012-12-11)

能力者,选择拨打120的比例也较低。今后需采取措施进一步提高社区居民对卒中警示症状的认知水平,同时必须增强社区居民对卒中发病后优先选择拨打120的急救意识,才能更有效地减少卒中患者的院前延迟。

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热常见,主要因狼疮活动所致;病程中伴头痛,查尿常规蛋白无明显增加、补体C_3无明显减低故除外狼疮活动,狼疮性脑病暂不能除外,予以激素冲击治疗后头痛未明显缓解,考虑头痛系其他原因所致。另外,SLE患者本身免疫功能紊乱及长期使用免疫抑制剂使机体抵抗力下降,感染机会上升,也常出现感染性发热,但该患者无腹泻、腹痛等,多次血培养结果阴性,鉴别发热原因较困难。最终血培养中发现伤寒沙门氏菌(诊断伤寒的重要依据)[4],抗感染治疗,发热、头痛症状好转出院。

临床上,系统性红斑狼疮合并伤寒极其少见,容易漏诊。本例患者无伤寒特有的脾大、玫瑰疹、白细胞降低等典型症状,反复血培养结果阴性。因为该类患者免疫力低下,机体的抗原抗体反应很弱,导致其感染伤寒后症状不典型,且随感染时间增长,作为诊断标准的血培养阳性率逐渐降低,本例多次血培养阴性,是延误诊治的重要原因。通过本例报告得出,针对系统性红斑狼疮合并伤寒患者,临床症状不典型且多次血培养结果阴性的情况下,需积极结合其他实验室依据,尽早明确诊断。尽管血培养出伤寒杆菌是确诊伤寒的论据,但其阳性率随病程延长而降低,可通过骨髓、尿或粪便培养鉴定,且骨髓培养阳性率较血培养高,尤适合于已用抗菌素药物治疗,血培养阴性。除外细菌学诊断依据,血清特异性抗体阳性也是诊断伤寒的依据之一;肥大反应"O"抗体凝集效价≥1∶80。针对此罕见病例,我们有以下体会:临床发热,在治疗原发疾病的基础上,不应忽略合并疾病(常见为感染性疾病),需积极找出病原学依据,予以敏感抗生素治疗。

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