·临床研究·

双侧延髓内侧梗死三例并文献复习

方嵘　吴斌　邓伟平　王晓丹

【摘要】 **目的**　双侧延髓内侧梗死易误诊，本研究总结其临床特点以及诊断与治疗经验，以为临床诊断与鉴别诊断疾病提供参考。**方法与结果**　上海交通大学医学院附属瑞金医院北部院区2017年1月至2022年1月收治3例双侧延髓内侧梗死患者，均为急性起病，分别以急性脊髓炎样表现发病（快速进展性四肢瘫，1例）、顽固性呃逆发病（1例）、偏侧肢体麻木乏力首发伴舌下神经瘫（1例）。头部MRI显示双侧延髓内侧梗死（1例早期MRI未见梗死灶）。2例因出现下运动神经元损害体征或症状不典型而误诊（例1、例2）。予以抗栓治疗后症状好转。**结论**　急性双侧延髓内侧梗死临床表现多样，早期易误诊，明确诊断需依靠临床表现及头部MRI检查，应早期准确诊断，及时予以针对急性脑梗死的抗栓治疗。鉴别要点主要为是否具有脑卒中危险因素，有无上运动神经元损害体征，治疗是否有效。

【关键词】 延髓；　脑梗死；　脑血管障碍

Bilateral medial medullary infarction: three cases report and literatures review

FANG Rong[1], WU Bin[2], DENG Wei-ping[1], WANG Xiao-dan[1]

[1]*Department of Neurology and Institute of Neurology, Ruijin Hospital, Shanghai Jiaotong University School of Medicine, Shanghai 200025, China*

[2]*Department of Neurology, Huashan Hospital, Fudan University, Shanghai 200040, China*

FANG Rong and WU Bin contributed equally to the article

Corresponding author: WANG Xiao-dan (Email: starbuck000731@163.com)

【**Abstract**】 **Objective**　Bilateral medial medullary infarction (MMI) is prone to misdiagnosis. This study summarizes the clinical characteristics of MMI, in order to provide reference to clinicians. **Methods and Results**　The clinical data of the 3 patients with acute bilateral MMI treated in Ruijin Hospital North Campus affiliated to Shanghai Jiaotong University School of Medicine from January 2017 to January 2022 were collected. All 3 patients had acute onset, one case had acute myelitis-like manifestations (rapidly progressive quadriplegia), one case had intractable hiccup, and the other one had unilateral limb numbness and fatigue as the first manifestation, accompanied by hypoglossal palsy. The head MRI showed acute bilateral MMI (one case without acute lesions on early MRI). Two cases were misdiagnosed because of either presenting lower motor neuron damage signs or having atypical symptoms. All patients improved after antithrombotic therapy for acute cerebral infarction. **Conclusions**　The clinical manifestations of acute bilateral MMI are diverse, and it's easy to be misdiagnosed in the early stage. The diagnosis is mainly based on clinical manifestations and head MRI, which requires early recognition and timely antithrombotic treatment for acute cerebral infarction. The main distinguishing points are whether there are risk factors for stroke, upper motor neuron damage signs, and the treatment was effective.

【**Key words**】 Medulla oblongata;　Brain infarction;　Cerebrovascular disorders

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作者单位：200025 上海交通大学医学院附属瑞金医院神经内科 上海交通大学医学院神经病学研究所（方嵘，邓伟平，王晓丹）；200040 上海，复旦大学附属华山医院神经内科（吴斌）

方嵘与吴斌对本文有同等贡献

通讯作者：王晓丹，Email：starbuck000731@163.com

延髓内侧梗死(MMI)是一类由椎动脉(VA)、脊髓前动脉(ASA)及其分支堵塞导致的危重型缺血性脑血管病,由Spiller教授于1908年首次报告[1]。延髓内侧梗死临床少见,占所有脑卒中的0.5%~1.5%;双侧延髓内侧梗死更加罕见,仅占0.07%~0.21%[2-3]。双侧延髓内侧梗死临床表现多样,主要为四肢瘫、舌下神经瘫、深感觉障碍、延髓麻痹等;影像学典型表现为MRI延髓"Y"形或"心"形梗死灶,但由于部分患者早期病灶不易显影,易误诊为吉兰-巴雷综合征(GBS)、脑干脑炎、急性脊髓炎等疾病[4],治疗方法同缺血性卒中[5]。目前关于双侧延髓内侧梗死病因、临床特点及诊断与治疗的报道较少。本研究回顾分析上海交通大学医学院附属瑞金医院北部院区神经内科收治的3例易误诊的双侧延髓内侧梗死患者的临床过程,并复习相关文献,总结其临床特点以及诊断与治疗经验,以期提高临床医师对疾病的认识及警惕性。

病例资料

例1 女性,56岁,因头晕伴肢体乏力6天,于2017年3月19日入院。入院前6天无明显诱因突发眩晕,约3分钟后自行缓解,未予重视;1天前自觉左下肢乏力,进行性加重,无法站立,并逐渐出现左上肢乏力,急诊至我院就诊。急诊查体:血压为190/100 mm Hg(1 mm Hg=0.133 kPa);伸舌居中,双眼右视时可见细微水平眼震;左上肢肌力3级、右上肢肌力正常,双下肢肌力1~2级,四肢肌张力2级,四肢腱反射减弱,双下肢振动觉减退,双侧Babinski征阳性。实验室检查:血清胆固醇水平为8.02 mmol/L(<5.18 mmol/L),低密度脂蛋白胆固醇(LDL-C)为5.39 mmol/L(<2.58 mmol/L),血糖10.88 mmol/L(3.90~6.10 mmol/L)。头部CT和MRI均未见明显异常。急诊考虑"急性上升性脊髓炎或周围神经病",遂收入院。既往有糖尿病病史2年,长期规律服用二甲双胍250 mg/次(3次/d),自述空腹血糖波动于6~7 mmol/L。入院后即予以静脉注射甲泼尼龙(IVMP)500 mg/d和静脉注射免疫球蛋白(IVIg)0.40 g/(kg·d),连续3天,以及对症支持治疗,但病情持续恶化。入院第3天查体时可见饮水呛咳、吞咽困难、构音障碍,双侧咽反射减退,右上肢肌力降至4级;改良Rankin量表(mRS)评分为5分;腰椎穿刺脑脊液检查均于正常值范围。头部MRI平扫和增强扫描可见双侧延髓内侧"Y"形DWI高信号影(图1a),增强扫描病灶可见低信号影(图1b),提示急性脑梗死。临床诊断为急性双侧延髓内侧梗死,改为口服阿司匹林负荷剂量300 mg后调整为100 mg/d抗血小板以及阿托伐他汀40 mg/d强化调脂治疗。入院第10天头部MRA显示左大脑中动脉(MCA)和双侧大脑前动脉(ACA)A1段狭窄,左大脑后动脉(PCA)P1段和P2段狭窄,左椎动脉末端管壁轻度狭窄(图1c,1d)。经抗血小板、强化调脂以及康复治疗2周后,四肢乏力和吞咽障碍改善。患者共住院17天,出院时可饮水、吞咽,双下肢肌力2+级。出院后遵医嘱继续服用阿司匹林100 mg/晚和阿托伐他汀20 mg/晚。出院后1个月(5月4日)电话随访,仍行康复治疗,mRS评分降至3分。

例2 男性,72岁,主诉突发顽固性呃逆伴行走不稳4天,于2022年1月11日入院。入院前4天无明显诱因突发顽固性呃逆,左侧肢体乏力伴行走不稳。3天前出现饮水呛咳,言语模糊,休息后无明显好转,至我院门诊就诊。门诊分诊至消化科,胃镜检查提示胃大部切除术后,转至神经内科就诊。门诊查体:伸舌居中,咽反射消失,左侧软腭上抬欠佳,左侧肢体轻瘫试验阳性,右侧肢体肌力正常,四肢腱反射阳性,右侧掌颌反射阳性,双侧Babinski征阳性,左侧指鼻试验、跟-膝-胫试验欠稳准;美国国立卫生研究院卒中量表(NIHSS)评分为2分。实验室检查:血糖7.50 mmol/L。头部MRI显示,双侧延髓内侧亚急性梗死灶(图2a);头部MRA显示,左颈内动脉虹吸段局部狭窄,双侧大脑中动脉M1段和M2段、大脑后动脉局部轻度狭窄,多发颅内动脉硬化,双侧椎动脉和基底动脉局部轻度狭窄迂曲(图2b,2c)。门诊以"双侧脑干(延髓及脑桥)内侧梗死"收入院。既往有糖尿病、高血压、哮喘病史20年,近10余年服用格列美脲2 mg/次(2次/d)和苯磺酸氨氯地平5 mg/d、吸入布地奈德福莫特罗320 μg/d,症状控制可;20余年前曾行胃大部切除术。入院后即口服阿司匹林100 mg/d和氯吡格雷75 mg/d抗血小板治疗、阿托伐他汀20 mg/d和依折麦布10 mg/d强化调脂治疗,以及对症支持治疗。患者共住院9天,出院时呃逆、饮水呛咳、言语模糊好转,左上肢轻瘫试验仍阳性但较前缓解,双侧指鼻试验、跟-膝-胫试验稳准。出院后继续服用阿司匹林100 mg/晚、氯吡格雷75 mg/d(5天后停用)、阿托伐他汀20 mg/晚和依折麦布10 mg/d。出院后3个月(4月12日)门诊复查,左上肢轻瘫试验阴性,四肢肌力正常,双侧指鼻

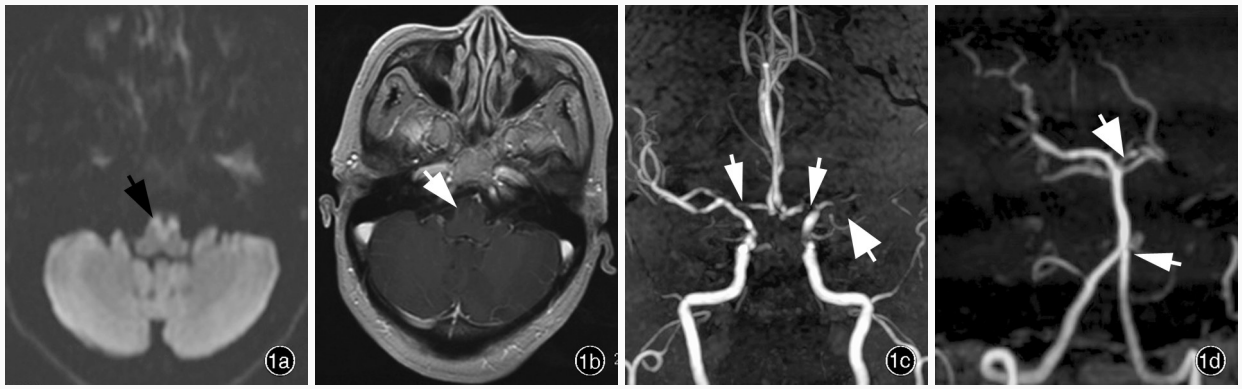

图1　例1头部影像学检查所见　1a　横断面DWI显示延髓腹侧"Y"形高信号影(箭头所示)　1b　横断面增强T_1WI显示延髓腹侧病灶无明显强化(箭头所示)　1c　前循环MRA显示,左大脑中动脉M1段流空消失(粗箭头所示)、双侧大脑前动脉A1段起始部多发狭窄(细箭头所示)　1d　后循环MRA显示,左大脑后动脉P1段和P2段狭窄(粗箭头所示),右椎动脉末端管壁轻度狭窄(细箭头所示)

Figure 1　Head imaging findings of Case 1　Axial DWI showed hyperintensity with "Y" shape in medial medullary (arrow indicates, Panel 1a). Axial enhanced T_1WI showed hypointensity in medial medulla with no obvious enhancement (arrow indicates, Panel 1b). MRA for anterior circulation showed flowing void in left MCA disappeared (thick arrow indicates), and multiple stenosis in initial part of bilateral ACA A1 segment (thin arrows indicate, Panel 1c). MRA for posterior circulation showed stenosis in left PCA P1 and P2 segments (thick arrow indicates), and mild stenosis vessel wall of the end of left VA (thin arrow indicates, Panel 1d).

试验、跟-膝-胫试验稳准。

例3　男性,71岁,主诉突发右侧肢体麻木、乏力2小时,于2021年12月15日急诊入院。患者于2021年12月15日19:00突发右侧肢体麻木、乏力,休息后无明显缓解,21:03急诊至我院就诊。急诊查体:血压为157/74 mm Hg;伸舌偏右,右上肢肌力3级、右下肢肌力4级,左侧肢体轻瘫试验阳性,右侧肢体针刺觉和温度觉减退,右侧Babinski征阳性;NIHSS评分为4分。实验室检查:血糖7.60 mmol/L,血清胆固醇5.63 mmol/L,低密度脂蛋白胆固醇为3.77 mmol/L,糖化血红蛋白(HbA1c)10.80%(4%~6%),尿葡萄糖强阳性。头部CT未见明显异常,考虑"急性脑梗死",发病时间在溶栓时间窗内,但患者拒行静脉溶栓治疗,予口服阿司匹林100 mg/d和氯吡格雷75 mg/d抗血小板、阿托伐他汀40 mg/d强化调脂治疗,以及对症支持治疗。头部MRI检查(12月16日)显示,双侧延髓内侧梗死(图3a);头部MRA显示,双侧大脑中动脉远端及其分支、大脑后动脉管壁充盈缺损,左大脑中动脉部分狭窄闭塞,多发颅内动脉硬化,右椎动脉管壁充盈缺损、左椎动脉管腔粗细不均(图3b,3c)。临床诊断为急性双侧延髓内侧梗死,维持原治疗方案。既往有糖尿病病史25年,未规律服药,血糖控制欠佳;高脂血症和冠心病病史10余年,自述已痊愈,近3年未服药;吸烟史50年(40支/d),已戒烟6年;饮酒史40年,常有酗酒,近5~6年偶少酌。患者共住院10天,出院时右上肢肌力3$^+$级、右下肢4$^+$级,右侧肢体针刺觉仍减退,左侧肢体轻瘫试验阴性。出院后继续服用阿司匹林100 mg/d、氯吡格雷75 mg/d(4天后停用)以及阿托伐他汀20 mg/d,继续康复治疗。出院后3个月(3月15日)门诊复查,右上肢肌力4级、右下肢肌力正常,右侧肢体针刺觉减退但较前缓解。

讨　论

笔者以"bilateral medial medullary infarction"、"brainstem infarction"及"medulla oblongata"为关键词,检索美国国立医学图书馆生物医学文献数据库(PubMed,2000年1月1日至2022年5月11日),共获得双侧延髓内侧梗死相关英文文献41篇计43例患者[4-44],结合本文3例病例,共计46例双侧延髓内侧梗死患者。男性34例,女性11例,1例未明确性别;发病年龄13~81岁,平均(57.42±14.79)岁;临床表现为四肢瘫39例(84.78%),其中包括痉挛性瘫痪5例(10.87%)、弛缓性瘫痪17例(36.96%)及不明类型瘫痪17例(36.96%),构音障碍37例(80.43%),吞咽障碍17例(36.96%),呼吸障碍15例(32.61%),舌下神经瘫、眩晕、眼震各14例(30.43%),深感觉减退11例(23.91%),浅感觉减退8例(17.39%),恶心呕吐8例(17.39%),共济失调4例(8.70%),呃逆和颈部疼痛各2例(4.35%),晕厥1例(2.17%)。38例患者行椎基底动脉影像学检查(包括CTA、MRA或者DSA),椎基底动脉系统无异常6例(15.79%),椎

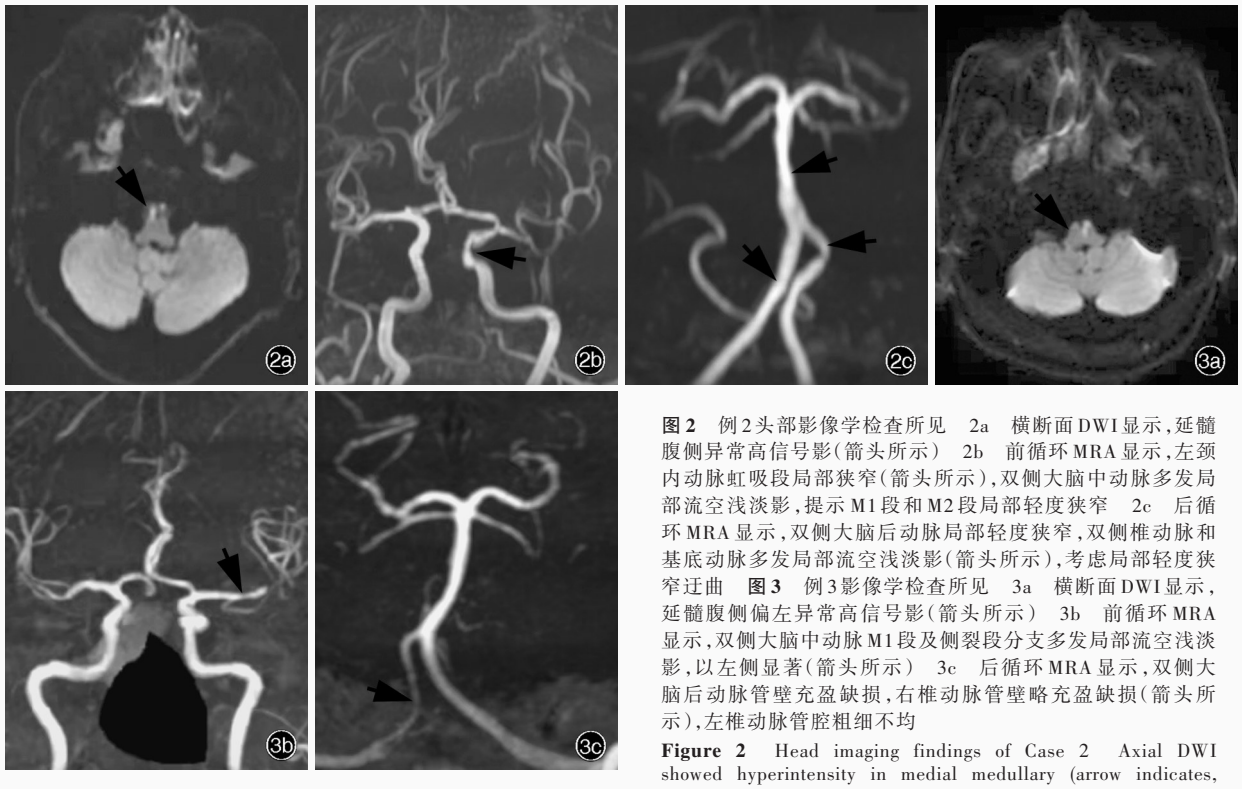

图2 例2头部影像学检查所见 2a 横断面DWI显示，延髓腹侧异常高信号影（箭头所示） 2b 前循环MRA显示，左颈内动脉虹吸段局部狭窄（箭头所示），双侧大脑中动脉多发局部流空浅淡影，提示M1段和M2段局部轻度狭窄 2c 后循环MRA显示，双侧大脑后动脉局部轻度狭窄，双侧椎动脉和基底动脉多发局部流空浅淡影（箭头所示），考虑局部轻度狭窄迂曲 **图3** 例3影像学检查所见 3a 横断面DWI显示，延髓腹侧偏左异常高信号影（箭头所示） 3b 前循环MRA显示，双侧大脑中动脉M1段及侧裂段分支多发局部流空浅淡影，以左侧显著（箭头所示） 3c 后循环MRA显示，双侧大脑后动脉管壁充盈缺损，右椎动脉管壁略充盈缺损（箭头所示），左椎动脉管腔粗细不均

Figure 2 Head imaging findings of Case 2 Axial DWI showed hyperintensity in medial medullary (arrow indicates, Panel 2a). MRA for anterior circulation showed local stenosis in left ICA siphon (arrow indicates) and multiple flowing void shadow in bilateral MCA, suggesting mild stenosis in M1 and M2 segments (Panel 2b). MRA for posterior circulation showed mildly local stenosis in bilateral PCA, multiple flowing void shadow in bilateral VA and BA (arrows indicate), suggesting mild stenosis (Panel 2c). **Figure 3** Head imaging findings of Case 3 Axial DWI showed hyperintensity in medial medullary (arrow indicates, Panel 3a). MRA for anterior circulation showed multiple flowing void shadow in distal part and branches of bilateral MCA M1 segment, especially in left MCA (arrow indicates, Panel 3b). MRA for posterior circulation showed filling defect of vessel walls in bilateral PCA, a mildly filling defect of vessel wall in right VA (arrow indicates), and an uneven thickness of vessel wall in left VA (Panel 3c).

动脉粥样硬化性狭窄11例（28.95%），椎动脉闭塞10例（26.32%），基底动脉粥样硬化性狭窄6例（15.79%），动脉夹层5例（13.16%），椎动脉发育不良3例（7.89%），双侧椎动脉不可见、椎动脉延长扩张、椎动脉瘤、脊髓前动脉闭塞各1例（2.63%）。45例患者有发病早期头部MRI资料，其中37例（82.22%）发病后首次MRI即可见急性梗死灶。发病2年内死亡7例，其中2例死于脑梗死致呼吸衰竭、2例死于肺炎、1例死于心力衰竭、1例死于腹膜炎、1例死因不详。35例患者有随访资料，其中13例遗留中度以上残疾（mRS评分＞2分）。

双侧延髓内侧梗死是临床罕见的后循环缺血性卒中，发病率低，但病死率高达23.8%[45]。Akimoto等[8]纳入2727例脑梗死患者，仅1例为双侧延髓内侧梗死。双侧延髓内侧梗死的临床表现多样，通常与梗死部位相关（图4），典型症状为四肢瘫、舌下神经瘫和深感觉障碍的“三联征”[6]。Pongmoragot等[45]总结38例双侧延髓内侧梗死患者的临床特点，指出除典型的“三联征”外，还可表现为构音障碍、眼震和吞咽困难等症状。本研究总结的46例双侧延髓内侧梗死患者以四肢瘫最常见，且大多数为弛缓性瘫痪，该现象可由“脊髓休克”解释，在重度脑卒中如双侧延髓内侧梗死急性期，由于上运动神经元失去正常的下行促进作用，脊髓或脑干离断节段以下的神经元超极化且不易兴奋，使其支配的骨骼肌紧张性和腱反射减弱或消失，最终表现为四肢弛缓性瘫痪[46]；而症状相对较轻的单侧延髓内侧梗死则主要为痉挛性瘫痪，表现为肌张力增高及病理征阳性[9]。本文例1同时存在痉挛性瘫痪（Babinski征阳性）及弛缓性瘫痪（腱反射减退及肌张力下降），提示锥体束受损严重。构音障碍、吞咽困难可能与病灶自延髓腹侧延伸至背侧并逐渐累及疑核、孤束核及网状结构致语言障碍、吞咽障碍及呃逆有关[47]。呼吸障碍亦为双侧延髓内侧梗死常见临床表现，且急性期出现呼吸障碍可能与预后不良密切相关，本研究总结的20例死亡或中度以

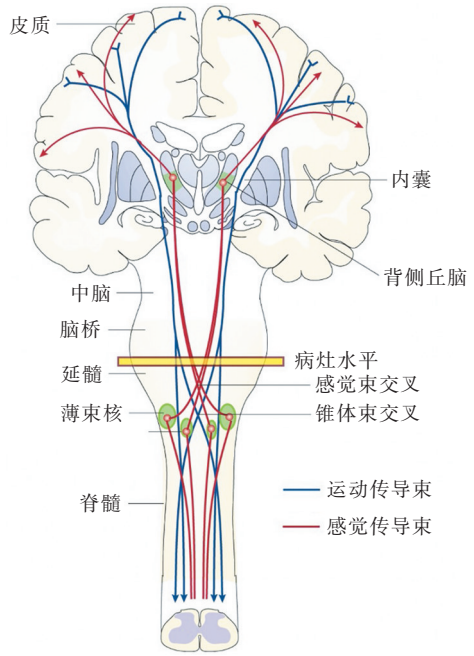

图4　梗死灶致相关传导束损害示意图

Figure 4　Infarction lesions lead to damage of associated conduction tracts.

上残疾(mRS评分>2分)患者中8例急性期出现呼吸障碍。本研究总结14例出现舌下神经瘫,可能与舌下神经核位于延髓背内侧有关[48]。本文例1和例2患者均未出现舌肌麻痹,可能由于舌下神经在内侧丘系与锥体束外侧出颅,可以避开延髓内侧梗死灶[10]。眩晕和眼震的病理生理学机制尚不明确,可能与半规管、耳石器官和前庭小脑等前庭系统损害相关[49],下橄榄核位于延髓上部,发出橄榄小脑纤维投射至对侧小脑[50],该环路可影响眼动[4],因此推测,例1的细微水平眼震系病灶累及下橄榄核,影响眼球振荡节律所致。此外,例1还存在双下肢振动觉减退、痛温觉保留,本研究总结的46例患者中深感觉障碍(11例)多于浅感觉障碍(8例),深浅感觉分离障碍可能是内侧丘系损伤所致[9],而例3出现偏身浅感觉减退则提示病灶累及延髓外侧脊髓丘脑束。

DWI序列显示延髓"Y形征"或"心形征"为双侧延髓内侧梗死的特征性影像学表现[11,44],受累部位包括双侧延髓前内侧区(锥体束和内侧丘系)以及双侧延髓前外侧区(下橄榄核、疑核和孤束核)。然而在临床实践中,早期MRI并无异常。本研究总结的43例患者中8例早期MRI未见急性梗死灶,数日后复查MRI方显示病灶。考虑到部分患者虽早期MRI呈阴性,但可能存在症状缓解而未复查的情况,因此MRI假阴性率较高。双侧延髓内侧梗死早期极易误诊,易与急性脊髓炎、吉兰-巴雷综合征[6-7,43]、脑干脑炎[44]等相混淆。本研究总结的46例患者早期误诊率达13.04%(6/46),主要是由于疾病早期MRI无明显异常以及临床表现多样且呈非特异性,例如,例1为中年女性,入院时双下肢肌力减退,而非单侧受累,3天内快速进展至双上肢,进展为四肢弛缓性瘫痪并累及脑神经,易误诊为急性上升性脊髓炎,因未考虑脑卒中而未评估NIHSS评分,但入院后采用mRS量表评估神经功能在一定程度上弥补这一不足。应注意鉴别急性上升性脊髓炎与急性缺血性卒中:(1)急性脊髓炎早期无眼震等脑神经症状。(2)急性脊髓炎特征性表现为病变节段以下感觉缺失和大小便异常等自主神经障碍,本文误诊患者中有1例(例1)存在深感觉障碍。(3)急性脊髓炎经激素及静脉注射免疫球蛋白后症状缓解,本文误诊患者中有1例(例1)治疗后症状加重[51]。还应注意鉴别四肢弛缓性瘫痪与周围神经病:(1)吉兰-巴雷综合征发病时四肢无力较对称,本文误诊患者中有1例(例1)肌无力发展顺序为双下肢、左上肢、右上肢。(2)吉兰-巴雷综合征双侧病理征阴性,本文误诊患者中有1例(例1)双侧Babinski征阳性。(3)吉兰-巴雷综合征可伴"手套袜套"样针刺觉减退,发病后10天腰椎穿刺脑脊液检查可见蛋白细胞分离现象[5]。本文例1发病早期即出现眩晕及眼震脑神经症状,且伴长束体征(感觉运动障碍、双侧Babinski征阳性),应注意与脑干脑炎相鉴别,脑干脑炎好发于儿童和青壮年,症状严重者可伴意识改变如嗜睡,脑神经损害更广泛,如复视、面瘫,且通常为交叉性瘫痪或交叉性感觉障碍[52]。本文例2以顽固性呃逆发病,早期误诊为消化系统疾病而行胃镜检查,发病早期即出现神经系统症状和阳性体征,但被呃逆症状掩盖,提醒临床医师应对某些疾病的非特异性症状提高警惕,注意仔细询问病史。本研究总结的46例患者中误诊的6例均为中老年,平均发病年龄为57.42岁,存在脑卒中危险因素,且未予抗栓治疗,症状无缓解甚至加重。

大动脉粥样硬化是双侧延髓内侧梗死的最常见病因[8],长期高血压、糖尿病、大量吸烟等脑血管病危险因素可引起血管平滑肌损伤,形成粥样硬化斑块,导致管腔狭窄和闭塞[53];后循环先天性血管变异同样发挥重要作用,延髓部位血供来自成对血

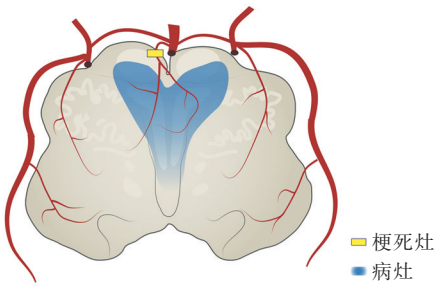

图5 横断面延髓示意图可见脊髓前动脉变异型旁正中动脉分支堵塞导致的梗死灶，双侧锥体束、内侧丘系、下橄榄核、疑核及孤束核均包含在“Y”形病灶区域

Figure 5 Axial medullary illustrating showed the site of the lesion following thrombosis of the variable paramedian branch of ASA. Bilateral pyramidal tracts, medial lemnisci, inferior olivenucleus, ambiguous nucleus and solitary nucleus are all involved in the "Y" shape lesion area.

管，包括椎动脉、小脑后下动脉（PICA）、脊髓前动脉及脊髓后动脉（PSA），延髓内侧主要由椎动脉、脊髓前动脉及其穿支供血[2]，血管变异如两侧脊髓前动脉变异型旁正中动脉穿支来源于一侧椎动脉或脊髓前动脉[12]（图5）或者一侧脊髓前动脉变异型旁正中动脉穿过中线供血对侧区域[13]，则可由单侧动脉堵塞导致双侧延髓梗死。受限于MRA分辨率，双侧延髓内侧梗死难以直接观测到责任血管，椎动脉或脊髓前动脉粥样硬化是常见病因[44]。本文3例患者均有脑血管病危险因素，头部MRA可见颅内动脉粥样硬化性狭窄；主动脉弓以上MRA显示，例1左椎动脉末端轻度狭窄，例2双侧椎动脉局部轻度狭窄迂曲，例3右椎动脉管壁充盈缺损、左椎动脉管腔粗细不均，因此推测3例均为单侧椎动脉或脊髓前动脉穿支变异和动脉粥样硬化性狭窄所致，但遗憾的是均未见责任血管。

综上所述，急性双侧延髓内侧梗死临床表现多样，部分患者早期头部MRI可无异常表现，易误诊为急性上升性脊髓炎、吉兰-巴雷综合征、脑干脑炎甚至其他系统疾病。临床诊断与治疗中应关注是否存脑卒中相关危险因素、脑神经受累症状，治疗后有无预期疗效。临床医师应提高警惕，早期准确诊断并予以积极治疗。

利益冲突 无

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