·临床研究·

多系统萎缩^{131}I-间碘苄胍心肌显像初步研究

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【摘要】 **研究背景** ^{131}I-间碘苄胍(^{131}I-MIBG)可被心脏交感神经节后纤维摄取,是评价心脏交感神经功能的显像剂。本研究采用^{131}I-MIBG心肌显像方法,探讨多系统萎缩患者自主神经功能障碍。**方法** 共12例符合2008年第2版诊断标准的多系统萎缩患者和7例正常对照者,通过统一多系统萎缩评价量表进行病情严重程度评价,静脉注射^{131}I-MIBG 3 mCi后于不同测量时间点(15 min、4 h和24 h)采集胸部前位平面像,计算^{131}I-MIBG心肌摄取率。**结果** 注射^{131}I-MIBG后15 min和4 h,多系统萎缩组患者^{131}I-MIBG心肌摄取率均低于正常对照组(15 min:1.90±0.41对2.38±0.32,P=0.017;4 h:1.96±0.63对2.60±0.55,P=0.039)。**结论** 多系统萎缩组患者^{131}I-MIBG心肌摄取率低于正常对照组,提示多系统萎缩可以发生心脏交感神经变性。

【关键词】 多系统萎缩; 交感神经系统; ^{131}I-间碘苄胍(非*MeSH*词)

Cardiac ^{131}I-MIBG scintigraphy in patients with multiple system atrophy

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【**Abstract**】 **Background** ^{131}I-metaiodobenzylguanidine (^{131}I-MIBG) can be intaked by cardiac sympathetic postganglionic fibre, thus becomes the imaging agent to evaluate cardiac sympathetic nerve function. The aim of this study is to investigate the autonomic nerve dysfunction of patients with multiple system atrophy (MSA) by using cardiac ^{131}I-MIBG scintigraphy. **Methods** Clinical data of 12 MSA patients conforming to the "secord consensus statement on the diagnosis of MSA" was analyzed by Unified Multiple System Atrophy Rating Scale (UMSARS). ^{131}I-MIBG scintigraphy was performed in 12 MSA patients and 7 age-matched controls. Planar images of the chest were obtained 15 min, 4 h and 24 h after the intravenous injection of 3 mCi ^{131}I-MIBG. Cardiac ^{131}I-MIBG uptake was quantified by comparing region of interest (ROI) over heart/mediastinum (H/M) ratio. **Results** Cardiac ^{131}I-MIBG uptake ratio in MSA group was significantly less than that in control group in 15 min (1.90±0.41 vs 2.38±0.32, P=0.017) and 4 h (1.96±0.63 vs 2.60±0.55, P=0.039). There were significant difference (P<0.05) between MSA group and control group. **Conclusions** Cardiac ^{131}I-MIBG uptake ratio in MSA group was less than that in control group. This finding suggests cardiac sympathetic degeneration may occur in MSA patients.

【**Key words**】 Multiple system atrophy; Sympathetic nervous system; ^{131}I-MIBG (not in *MeSH*)

多系统萎缩(MSA)是一种散发性、成年发病且快速进展的神经变性疾病,临床表型复杂多样,主要包括自主神经功能障碍、帕金森综合征、共济失调和锥体系统功能障碍等,临床表现为各种症状不同程度的重叠组合[1-2]。其共同的病理学特征是少突胶质细胞胞质内包涵体(GCIs)形成,根据症状出现顺序和严重程度分为帕金森型(MSA-P)和小脑萎缩型(MSA-C)[3]。放射性标志物间碘苄胍(MIBG)是假性交感神经递质胍乙啶类似物,与去甲肾上腺素具有相同的摄取和贮存机制,可被心脏交感神经节后纤维摄取,但不被代谢,从而滞留在交感神经末梢,形成局部胞质聚集。因此,可以作为交感神经元显像剂,直观且定量观察心脏交感神经末梢分

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布的完整性和功能状态，目前已成为评价心脏交感神经功能的敏感指标[4-5]。近20年来，^{131}I-MIBG心肌显像在欧洲和日本成功应用于评价心脏交感神经节后神经支配，但国内尚无相关文献报道。在本研究中，我们采用^{131}I-MIBG心肌显像方法，探讨多系统萎缩患者自主神经功能障碍。

对象与方法

一、研究对象

1. 多系统萎缩组　选择2013年3–9月在北京大学人民医院和卫生部中日友好医院神经内科就诊并明确诊断的多系统萎缩患者共12例。(1)纳入标准：符合2008年Gilman等[6]在*Neurology*发表的第2版多系统萎缩诊断标准；入组时被详细告知本研究目的和检查流程，并签署知情同意书。(2)排除标准：发病年龄<30岁；有家族史，具有明显的遗传模式；存在影响自主神经功能的内科系统疾病；存在其他中枢神经系统疾病，如进行性核上性麻痹(PSP)、皮质基底节变性(CBD)等；存在影响检查结果的其他神经精神疾病，如路易体痴呆(DLB)、精神分裂症等；正在服用可能导致帕金森病症状或干扰^{131}I-MIBG摄取的任何药物；拒绝接受^{131}I-MIBG心肌显像的患者。

2. 正常对照组(对照组)　健康志愿者7例，均符合以下条件：(1)年龄与多系统萎缩组相匹配。(2)无心脏病症状，神经系统检查(无震颤、运动迟缓及其他神经系统疾病)和心电图检查均无异常。(3)均被详细告知本研究目的和检查流程，并签署知情同意书。

二、研究方法

1. 临床资料　(1)一般资料：详细询问病史，包括首发和主要症状、病情进展顺序、服药情况，以及既往脑卒中、颅脑创伤、脑炎、特殊药物应用、毒物接触、一氧化碳中毒和家族史等，并进行全面的神经系统检查。(2)量表评价：采用统一多系统萎缩评价量表(UMSARS)进行评价[7-8]，其中UMSARS-Ⅰ为病史回顾，包含12种检查项目；UMSARS-Ⅱ为运动检查评分，包括14种检查项目；UMSARS-Ⅲ和Ⅳ分别为自主神经功能和整体失能评分。UMSARS-Ⅰ和Ⅱ中每一项均对0(正常)~4(严重异常)分的特征进行定义。UMSARS量表已在欧洲多所神经病学医疗中心进行临床实践，能够较好地反映疾病严重程度之变化[7]。

2. MRI检查　常规头部MRI检查，T_2WI表现为壳核“裂隙征”、壳核萎缩、壳核后部低信号、脑桥“十字征”、小脑萎缩和小脑中脚高信号等多系统萎缩特征性影像学图像[9]。除矢状位观察脑干和小脑萎缩程度外，重点观察T_2WI上脑桥“十字征”、壳核“裂隙征”和壳核异常低信号。

3. ^{131}I-MIBG心肌显像　于受试者肘静脉注射^{131}I-MIBG 3 mCi，应用PHLIPS Precedence 6 SPECT/CT成像系统，分别于注射药物后15 min、4 h和24 h采集胸部前位平面像，通过勾画兴趣区(ROI)计算心脏/纵隔(H/M)放射性计数比值，并作为^{131}I-MIBG心肌摄取率。

4. 统计分析方法　采用SPSS 17.0统计软件对所获取数据进行处理与分析。计量资料以均数±标准差($\bar{x} \pm s$)表示，行两独立样本的t检验。以$P \leqslant 0.05$为差异具有统计学意义。

结　果

一、临床资料

多系统萎缩组12例患者，男性9例，女性3例；年龄53~80岁，平均(63.92±9.59)岁；病程1~5年，平均(2.54±1.21)年(表1)。其中很可能与可能多系统萎缩病例各6例，MSA-C型3例、MSA-P型9例。

二、神经影像学特征

1. 常规MRI　多系统萎缩患者MRI均呈现异常，10例矢状位T_1WI出现脑干和小脑萎缩，6例横断面T_2WI显示“十字征”，4例表现为壳核“裂隙征”，3例合并脑桥“十字征”和壳核“裂隙征”，5例出现壳核异常低信号(图1)。

2. ^{131}I-MIBG心肌显像　与正常对照组相比，多系统萎缩组患者^{131}I-MIBG心肌摄取率降低(图2，3)。多系统萎缩组有2例患者因病情严重未进行24 h ^{131}I-MIBG心肌显像。多系统萎缩组患者15 min和4 h心肌显像^{131}I-MIBG心肌摄取率均低于正常对照组，且差异具有统计学意义($P<0.05$，表2)。

讨　论

多系统萎缩的诊断主要依靠临床表现，包括病史和体格检查。2008年第2版多系统萎缩诊断标准还包括特征性影像学改变，提出3项诊断等级：可能的、很可能的和确定的多系统萎缩[6]。确定的多系

表1 12例多系统萎缩患者临床资料

Table 1. Clinical data of 12 MSA patients

No	Sex	Age (year)	Duration (year)	Initial symptom	Parkinsonian signs	Cerebellar dysfunction	Autonomous dysfunction	Babinski sign	UMSARS (score)
1	Male	80	2	Dizziness	Resting tremor, postural tremor, hypermyotonia, bradykinesia	—	Orthostatic hypotension, frequent urination, constipation, erectile dysfunction	–	25
2	Male	53	1.50	Bradykinesia	Resting tremor, rigidity, bradykinesia	Dysarthria	Frequent urination, constipation	+	44
3	Male	67	1	Walking instability	Postural tremor, hypermyotonia	Limb and gait ataxia	Frequent urination, urinary incontinence, constipation	+	18
4	Female	54	4	Bradykinesia	Postural tremor, rigidity, bradykinesia, postural instability	Dysarthria, drinking cough, limb and gait ataxia	Orthostatic hypotension, frequent urination, urinary incontinence, constipation, anhidrosis under the chest	+	49
5	Male	77	5	Bradykinesia	Rigidity, bradykinesia, postural instability	Dysarthria, astasia	Orthostatic hypotension, frequent urination, urinary incontinence, constipation	+	61
6	Male	74	3	Dizziness	Postural tremor, rigidity, bradykinesia, postural instability	Dysarthria, astasia	Orthostatic hypotension, frequent urination, urinary incontinence, constipation, erectile dysfunction	+	58
7	Male	61	1.50	Bradykinesia	Postural tremor, rigidity, bradykinesia, postural instability	Limb ataxia	Orthostatic hypotension, syncope, urinary incontinence, constipation, erectile dysfunction, horner in right	+	48
8	Male	59	3	Walking instability	Hypermyotonia, bradykinesia	Dysarthria, limb and gait ataxia	Constipation, dysuresia	+	34
9	Male	54	1	Bradykinesia	Bradykinesia, hypermyotonia	Limb ataxia	Orthostatic hypotension, frequent urination, constipation	+	21
10	Male	56	2.50	Bradykinesia	Postural tremor, rigidity, bradykinesia, postural instability	Dysarthria, drinking cough, gait ataxia	Orthostatic hypotension, frequent urination, urinary incontinence, erectile dysfunction	–	34
11	Female	61	3	Bradykinesia	Postural tremor, bradykinesia, rigidity, can not stand up	Dysarthria, dysphagia, Astasia	Orthostatic hypotension, frequent urination, urinary incontinence, constipation	+	81
12	Female	71	3	Walking instability	Postural tremor, hypermyotonia, bradykinesia	Nystagmus, dysarthria, drinking cough, limb and gait ataxia	Orthostatic hypotension, frequent urination, urinary incontinence, constipation, anhidrosis under the chest	–	38

+, positive, 阳性; –, negtive, 阴性; UMSARS, Unified Multiple System Atrophy Rating Scale, 统一多系统萎缩评价量表

统萎缩为病理证实的少突胶质细胞胞质内包涵体伴黑质纹状体和橄榄-脑桥-小脑通路变性。可能的和很可能的多系统萎缩是基于临床和影像学特征。本研究多系统萎缩组患者以男性居多，诊断为MSA-P型多于MSA-C型，与文献报道相一致[3]。多系统萎缩的特征性MRI表现包括壳核萎缩、脑桥萎缩、第四脑室扩张和小脑中脚萎缩[10]。此外，脑桥“十字征”[11]和壳核“裂隙征”用于诊断多系统萎缩具有较高的阳性预测值[12]。本研究多系统萎缩组患者常规MRI表现为脑桥、小脑萎缩，以及脑桥“十字征”、壳核“裂隙征”和壳核背外侧低信号，然而这些信号改变缺乏特异性。

^{131}I-MIBG心肌显像提供了一种评价心肌交感神经分布与功能的无创性检查方法。国外研究通常进行早期（15 min）和延迟（4 h）两次显像，将心脏/纵隔放射性计数比值作为^{131}I-MIBG心肌摄取率，判断心肌对^{131}I-MIBG的摄取能力[4]。^{131}I-MIBG通过两条途径进入交感神经节后突触前神经元：神经元特异性摄取（Ⅰ型摄取，由去甲肾上腺素转运体将其摄入神经末梢）和非神经元性摄取（Ⅱ型摄取，为主要依靠被动扩散机制的生理性摄取）。正常组织（除心肌、肾上腺）摄取^{131}I-MIBG主要依靠被动扩散机制，^{131}I-MIBG的释放主要通过溢出与胞吐作用，早期显像来自Ⅰ和Ⅱ型摄取，延迟显像包含极少的Ⅱ型摄取，可更准确地代表心脏交感神经活动，显示心肌交感神经分布特征[13]。为此本研究特意增加了24小时显像。

目前的研究显示，帕金森病患者自主神经功能障碍主要由于外周交感神经节后纤维受损，包括心肌交感神经；而多系统萎缩患者则是中枢交感神经节前纤维受损。因此，应用^{131}I-MIBG显像研究心脏交感神经节后神经元功能完整性，可用于二者的鉴别诊断。Druschky等[14]以及Rascol和Schelosky[15]的研究显示，与正常对照组相比，帕金森病组患者

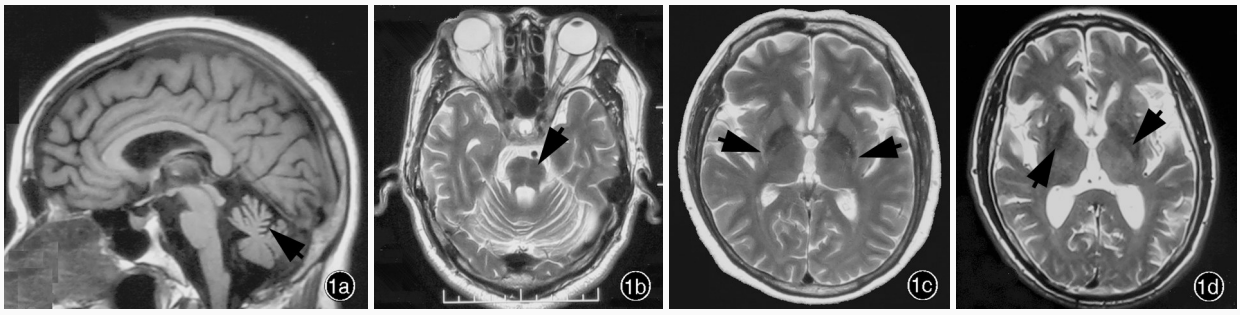

图1　多系统萎缩典型MRI表现　1a　矢状位T_1WI显示橄榄-脑桥-小脑萎缩(箭头所示)　1b　横断面T_2WI显示脑桥"十字征"(箭头所示)　1c　横断面T_2WI显示壳核"裂隙征"(箭头所示)　1d　横断面T_2WI显示壳核异常低信号(箭头所示)

Figure 1　Typical MRI signs consistent with regional atrophy in MSA. Sagittal T_1WI showed olivopontocerebellar atrophy (arrow indicates, Panel 1a). Axial T_2WI showed "hot cross bun" sign of pons (arrow indicates, Panel 1b). Axial T_2WI showed putaminal "slit" sign (arrows indicate, Panel 1c). Axial T_2WI showed abnormally low signal in putamen (arrows indicate, Panel 1d).

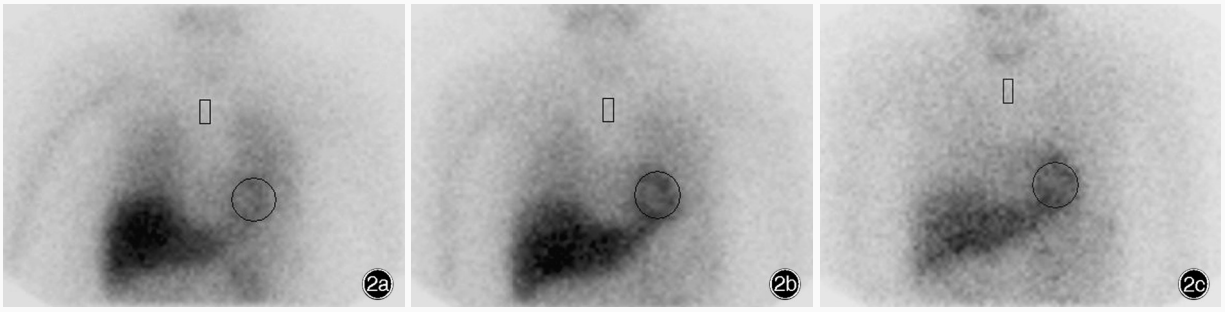

图2　正常对照组受试者^{131}I-MIBG心肌显像所见(圆形所示区域为心脏、矩形所示区域为纵隔)　2a　15 min像可见唾液腺、肝脏、心脏和肺摄取^{131}I-MIBG,属于生理性摄取　2b　4 h像仍可见心肌摄取^{131}I-MIBG,放射性分布均匀　2c　24 h像仍可见心肌摄取^{131}I-MIBG,放射性分布均匀

Figure 2　Normal uptake pattern of cardiac ^{131}I - MIBG scintigraphy (round areas indicate heart and oblong areas indicate mediastimum). The uptake of the radiopharmaceutical in salivary glands, liver, heart and lungs was considered as physiological uptake at 15 min after injection (Panel 2a). Visible uptake of the radiopharmaceutical was observed in the heart at 4 and 24 h after injection (Panel 2b, 2c).

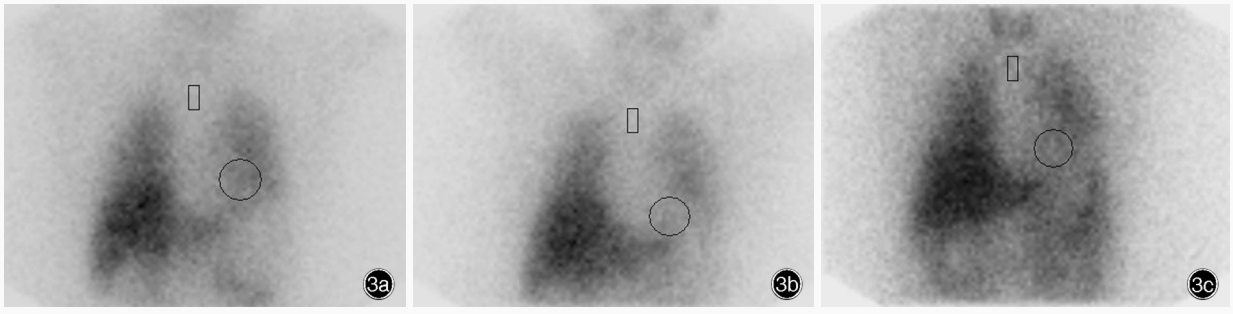

图3　多系统萎缩组患者^{131}I-MIBG心肌显像显示,各测量时间点心肌摄取率均显著低于正常对照组(圆形所示区域为心脏、矩形所示区域为纵隔)　3a　15 min像　3b　4 h像　3c　24 h像

Figure 3　Cardiac ^{131}I - MIBG scintigraphy in MSA patients (round areas indicate heart and oblong areas indicate mediastimum). Cardiac ^{131}I-MIBG uptake in MSA group was less than that in control group in 15 min (Panel 3a), 4 h (Panel 3b) and 24 h (Panel 3c).

^{123}I-MIBG心肌摄取率明显下降,多系统萎缩组和进行性核上性麻痹组患者则正常或略下降;特别是在疾病早期,帕金森病组患者^{123}I-MIBG心肌摄取率明显低于多系统萎缩组。因此,帕金森病组患者早期MIBG心肌摄取率减少为其特征性表现。Nagayama等[16]研究发现,约有30%的多系统萎缩患者也可出现^{131}I-MIBG摄取率降低。Raffel等[17]以^{11}C-HED作为交感神经显像剂,注射40分钟后行心脏显像可反映交感神经节后纤维末梢密度,其结果显示,与正常对照组相比,帕金森病组患者^{11}C-HED心肌摄取率明显下降,交感神经末梢密度明显减少,发生心脏去交感神经支配。本研究10例多系统萎缩患者中4例^{11}C-HED心肌摄取率下降,提示多系统萎缩患者发生心脏去交感神经支配的可能较先前文献报

表2 多系统萎缩组与正常对照组患者^{131}I-MIBG心肌摄取率的比较($\bar{x} \pm s$)

Table 2. Comparison of ^{131}I-MIBG uptake between control and MSA groups ($\bar{x} \pm s$)

Group	N	Age (year)	UMSARS (score)	H/M ratio		
				15 min	4 h	24 h
Control	7	57.29 ± 9.08	—	2.38 ± 0.32	2.60 ± 0.55	2.56 ± 0.47
MSA	12	63.92 ± 9.59	42.58 ± 18.27	1.90 ± 0.41	1.96 ± 0.63	1.96 ± 0.62
t value		-1.535		2.644	2.235	2.027
P value		0.143		0.017	0.039	0.062

MSA, multiple system atrophy, 多系统萎缩; UMSARS, Unified Multiple System Atrophy Rating Scale, 统一多系统萎缩评价量表; H/M ratio, heart/mediastinum, 心脏/纵隔比值

道更高。本研究多系统萎缩组患者15分钟和4小时^{131}I-MIBG心肌摄取率均较正常对照组降低,与文献报道基本一致。^{131}I-MIBG心肌摄取率降低可切实反映心脏交感神经末梢密度降低。由于本研究样本量较小,其与病程和病情严重程度之间的关系尚不明确,有待进一步扩大样本量加以证实。本研究24小时显像显示,多系统萎缩组患者^{131}I-MIBG心肌摄取率低于正常对照组,但未达统计学意义。

多系统萎缩自主神经衰竭与中枢和节前传出神经自主神经功能障碍相关,其病理表现为中枢交感神经节前神经元受累,节后神经元通常幸免,因此,MIBG心肌摄取率并不下降。然而,有些多系统萎缩患者MIBG心肌摄取率降低,意味着节后突触前心脏交感神经末梢密度降低[16]。Orimo等[18]对15例多系统萎缩患者进行尸检,发现了^{123}I-MIBG摄取率轻微下降的病理生理学机制,所有多系统萎缩患者交感神经节后神经元缺失并不十分明显,其中6例心外膜酪氨酸羟化酶(TH)免疫性神经纤维数目轻至中度减少,4例交感神经节后酪氨酸羟化酶免疫性神经元数目减少,提示多系统萎缩交感神经节神经元损伤可能与心脏交感神经受累有关;1例心脏酪氨酸羟化酶和神经微丝蛋白(NF)免疫性神经纤维几乎完全消失,并在交感神经节和迷走神经背核发现路易小体(LB)。提示多系统萎缩可以发生轻度心脏交感神经变性,与交感神经节后神经元病理改变密切相关,导致MIBG心肌摄取率轻微下降,尤其是病程较长的患者。此外,交感神经节或中枢神经系统出现路易小体可能会加速心脏去交感神经支配。

综上,多系统萎缩患者^{131}I-MIBG心肌摄取率较正常对照组降低,意味着心脏交感神经末梢密度减少,提示多系统萎缩可发生心脏交感神经变性。

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Time: June 22–26, 2014

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