行政院原子能委員會
委託研究計畫研究報告

以$[^{99\text{m}}\text{Tc}]$TRODAT-1 及$[^{123}\text{I}]$IBZM 單光子腦部斷層掃瞄追蹤
評估急性中風引起的單側舞蹈症基底核中多巴胺運輸器及
接受器的變化

Longitudinal Study of Striatal Dopaminergic Status in Vascular
Hemichorea-Hemiballism with $[^{99\text{m}}\text{Tc}]$TRODAT-1 and
$[^{123}\text{I}]$IBZM SPECT Imaging

計畫編號：90-NU-7-006-001
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報告日期：中華民國 90 年 11 月 29 日
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（二）中文摘要

由解剖学及药理学的研究发现，急性中风引起的单侧舞蹈症可能是基底核中多巴胺系统机能异常所致。过去利用功能性影像学来研究这一类群病人的报告很少且是在慢性恢复期。这个研究的目的是同时利用多巴胺运输器（[^99mTc]TRODAT-1）及接受器（[^123I]IBZM）单光子脑部断层扫描来长期追踪评估基底核中多巴胺系统的状态，藉以了解血管性单侧舞蹈症的病期生理。由于典型病人极为稀少，我们一共收集了两位血管性单侧舞蹈症患者在不同阶段接受检查：包括急性期（症状出现后一週內，且無藥物治療）與治療期（在haloperidol的治疗下症状完全消失或接近完全消失），并取两名同年龄正常人作对对照组。我们发现病人病肢对侧纹状体之多巴胺运输器及第二型多巴胺接受器的结合能力都有下降的情形，也就是整个多巴胺系统的机能低下。從抗丁頓氏舞蹈症的观察研究，我们知道舞蹈症的产生是因为基底核中“间接路线”的功能低下所造成。而血管性舞蹈症患者多巴胺运输器及第二型多巴胺接受器的研究结果与抗丁頓氏舞蹈症完全相同。我们也都知道多巴胺对基底核中“间接路线”的作用是抑制性的，所以使用抗精神病药物（如haloperidol）可以减少多巴胺对基底核中“间接路线”的抑制能力，进而使得整个原本功能低下的“间接路线”得以接近正常。这也解释了为何haloperidol可以改善血管性单侧舞蹈症的症状。
From anatomical and pharmacological findings, vascular hemichorea-hemibalism (HC-HB) is supposed to be a result of striatal dopaminergic dysfunction. Functional neuroimaging studies on the dopaminergic system of this group of patients are rare and are done at the chronic recovery stage. The purpose of this study is to explore the pathophysiology of vascular HC-HB by evaluating the striatal dopaminergic status at acute, subacute, and chronic stages by concomitant \[^{99m}\text{Tc}]\text{TRODAT-1} and \[^{123}\text{I}]\text{IBZM SPECT imaging.}\]

Two patients with HC-HB were included in this study. Each patient received \[^{99m}\text{Tc}]\text{TRODAT-1} and \[^{123}\text{I}]\text{IBZM SPECT at acute stage (medication -, symptom +) and medical stage (medications +, symptoms -). The results were then compared with age-matched normal controls. We found the binding potential for both \[^{99m}\text{Tc}]\text{TRODAT-1} and \[^{123}\text{I}]\text{IBZM in the contralateral basal ganglia was decreased in vascular HC-HB. This implied that the function of the nigrostriatal dopaminergic system was decreased. The SPECT imaging findings in our patients with vascular HC-HB were the same as those of Huntington's disease. The symptoms of chorea and ballism have been shown to result from underactivity of the indirect pathway in the basal ganglia. Since dopamine exerts an inhibitory function on the indirect pathway, the use of dopamine receptor blocking agent (e.g. haloperidol) can reduce the inhibitory function on the underactive indirect pathway and restore the indirect pathway to a physiologically near normal state. This could explain why haloperidol is effective for vascular HC-HB.\]
From the anatomical and pharmacological points of view, dopaminergic dysfunction is the likely cause for vascular HC-HB (2–5, 6–11, 17, 19, 22–28). More recent evidence shows that experimental chorea and ballism from different lesions may result in the same reduction in subthalamic pallidal activity (5). The present study use the CT, MRI, \[^{123}\text{I}]\text{IBZM (D}_2\text{ receptor ligand)}\) and \[^{99m}\text{Tc}]\text{TRODAT-1 (dopamine transporter ligand)}\) SPECT imaging to investigate the serial structural changes and striatal dopaminergic status in the acute unmedicated, subacute medicated, and chronic unmedicated recovery stages. Effects of haloperidol on choreic/ballistic symptoms and SPECT imaging findings at the three different stages will be evaluated. Longitudinal follow-up of CT and MRI can be helpful to understand the possible causes of signal changes. Longitudinal evaluation of dopamine D\(_2\) receptors and dopamine transporters may provide evidence of dopaminergic dysfunction in acute vascular HC-HB and establish the rationale for clinical use of haloperidol treatment.
Hemichorea-hemiballism (HC-HB) is the most common type of hyperkinetic movement disorders in acute stroke (1). Clinically, the onset is abrupt in the majority of patients with vascular HC-HB. The face is usually spared. Most patients recover spontaneously within 2–4 weeks, although some do continue to have longer duration of choreic movement. Patients respond very well to neuroleptics (low-dose haloperidol, 3–15 mg/day) or to dopamine depleters (2–5). Classically, hemiballism was considered a result of a lesion of the subthalamic nucleus; it is now known that a variety of lesions in the basal ganglia (and in corticostrial pathways as well) that interrupt both afferent and efferent subthalamopallidal pathways, detected both at autopsy and with modern neuroimaging, may cause persistent or paroxysmal choreic or ballistic movements (5). The responsible lesions for vascular HC-HB have been reported to locate in caudate, putamen, thalamus, corona radiata, and subthalamic nucleus (1, 7–10).

Previous studies on vascular HC-HB mainly focus on anatomical changes in computed tomography (CT) and magnetic resonance imaging (MRI) (1, 7, 8, 11). Functional neuroimaging study by positron emission tomography (PET) or single-photon emission computed tomography (SPECT) was rarely done in vascular HC-HB. A $^{18}$F-fluorodeoxyglucose PET study in 2 patients with vascular HC-HB showed decreased glucose metabolism in the contralateral striatum (12). Striatal $^{18}$F-Dopa uptake by PET was done in one case, one year after onset of her symptoms, and showed normal (12). With the advent of the new dopamine transporter ligand ($^{99m}$TcTRODAT-1) and the widespread availability and low
operating costs of SPECT (13–14), we try to elucidate the presynaptic and postsynaptic changes of the nigrostriatal dopaminergic system in vascular HC-HB.
（六）執行方法與進度說明

Subjects

Two patients of first acute vascular hemichorea-hemiballism (HC-HB) were recruited. Patients with a history of parkinsonism, severe depression, or coma at presentation were excluded. Written informed consent was obtained from each patient. The demographic data, risk factors for stroke, and previous history of transient ischemic attack (TIA) or stroke were documented. Complete physical and neurological examinations were done by a movement disorder specialist. The onset time of clinical symptoms and the time of anatomical and functional neuroimaging examinations were recorded. The characteristics of involuntary movement were videotaped and analyzed according to Jankovic's criteria (17). A baseline work-up of electrolytes, plasma glucose, hemogram, urinalysis, electrocardiogram, and chest X-ray were done on each patient. Any significant violation of the metabolic factors were corrected and followed up as needed.

Each patient was scheduled for a brain CT, MRI, $[^{123}]$IBZM/SPECT, and $[^{99mTe}]$TRODAT-1/SPECT within one week of the onset of symptom and before haloperidol treatment. Those patients who suffer from severe HC-HB and have the potential for seriously degrading image quality will be treated with midazolam 15 mg IV 3 minutes before image studies.

Although the abnormal movements were reported to disappear spontaneously in the majority of patients, especially those secondary to non-ketotic hyperglycemia, only in a few instances does near-complete improvement come within days. The exhausting nature of the movements
makes it imperative to reduce its intensity. The patients were treated with haloperidol, starting from a low dose with gradual increase in dosage until complete remission or minimal visible residual movement is achieved. When the maximum effectiveness of the haloperidol was reached, a second set of CT, MRI, $^{[123}]$IBZM/SPECT and $^{[99mTc]}$TRODAT-1/SPECT images were repeated again. Patients were then maintained on effective dosage of haloperidol and regularly followed up at outpatient department. We tried to taper gradually and then fully stop haloperidol if clinically stable. In the absence of recurrence, the third set of CT, MRI, $^{[123}]$IBZM/SPECT and $^{[99mTc]}$TRODAT-1/SPECT images were done after complete discontinuance of haloperidol.

Two age-matched healthy subjects were selected as controls for MRI, $^{[123}]$IBZM/SPECT, and $^{[99mTc]}$TRODAT-1/SPECT imaging.

**Methods**

**Radiopharmaceutical**

$^{[99mTc]}$TRODAT-1

Each 10 ml vial contained a pre-dispensed sterile, non-pyrogenic, lyophilized mixture of 126 µg TRODAT-1·3HCl (2-[[2-[[3-(4-chlorophenyl)-8-methyl-8-azabicyclo[3.2.1]oct-2-yl]methyl][2-mercaptopropyl]amino]-ethyl]-amino]ethane-thiolato(3-)-N2,N2',S2,S2')oxo-[1R-(exo-exo) hydrogen chloride], 320 µg sodium glucoheptonate, 930 µg Na₂ EDTA 2H₂O (disodium ethylenediaminetetraacetate dihydrate), 32 µg stannous chloride dihydrate, 20 mg mannitol, 4.1 mg anhydrous sodium
phosphate dibasic and 460 µg sodium phosphate monobasic, sealed under nitrogen atmosphere with a rubber closure. No bacteriostatic preservative was present. A technetium Tc-99m generator was eluted within 24 hours prior to obtaining any elute for reconstitution with the INER TRODAT-1 kit.

\[ ^{123}I \] IBZM

Iodine-123-IBZM was prepared using a modification of a previously described procedure by directly oxidative iodination of the phenolic precursor BZM [(S)(-)-N-[1-ethyl-2-pyrrolodinyl]methyl]-2-hydroxy-6-methoxybenzamide) with high purity \(^{123}\)I-sodium iodide (obtained from the Institute of Nuclear Energy Research, Lung-Tan, Taiwan). \(^{123}\)I-IBZM with radiochemical purity > 90%, as determined by a radio-thin layer chromatography was obtained. The yield of \(^{123}\)I-IBZM was 50-60% with specific activity > 10,000 Ci/mmol. The final formulation of \(^{123}\)I-IBZM—containing 18% (v/v) ethanol and 1.3 mg of ascorbic acid in 3.5 ml 0.9 % sodium chloride—was filtered through a sterile 0.22 µm membrane filter and was tested for sterility and apyrogenicity to meet the requirements of injection.

\[ ^{99m}Tc \] TRODAT-1 and \[ ^{123}I \] IBZM SPECT Imaging

\[ ^{99m}Tc \] TRODAT-1 SPECT

A dose of 25 mCi of \(^{99m}\)TcTRODAT-1 was injected intravenously into each patient. The binding to dopamine transporter was assessed 4 hours
after injection with SPECT. A rotating three-headed gamma camera with 
fan-beam collimator (Multi SPECT 3, Siemens, Germany) and a 
commercially available computer system were used for data acquisition and 
processing. Data were collected for 120 projections (360° rotation) in a 128 
× 128 matrix. The acquisition time was 40 seconds per projection. 
Attenuation correction was performed in selected transverse slices 
according to a modified Chang's method. In-plane resolution of the 
reconstructed images was 8.5 mm FWHM, and slice thickness was 
approximately 6 mm.

[123I]IBZM SPECT

For the 123I-IBZM SPECT examination, every patient was given an 
intravenous injection of 5 mCi 123I-IBZM (Institute of Nuclear Energy 
Research, Taiwan). The IBZM binding was assessed by SPECT 2 h later. 
While in the scanner, the patient's head was immobilized with a comfortable 
molded head holder. Patients were aligned with the orbitomeatal lines 
parallel to the detector rings. A rotating three-head gamma camera with a 
fan-beam collimator (Multi SPECT 3, Siemens, Munich, Germany) and a 
commercially available computer system were used for data acquisition and 
processing. Data were collected for 60 projections (360° rotation) on a 128 
× 128 matrix. The acquisition time was 40 sec per projection. Attenuation 
correction was performed in selected transverse slices according to Chang's 
method.
**CT and MRI imaging**

1. The first axial routine head CT scan was done at the beginning of symptoms and before haloperidol treatment. The second scan was done at the subacute medicated stage, during which symptoms were effectively controlled by haloperidol. The third scan was done at the chronic unmedicated recovery stage, i.e., after complete discontinuance of haloperidol for two weeks and complete or near-complete remission of clinical symptoms.

2. T1 and T2-weighted MR imaging (Magneton 1.5 Tesla scanner) with thin cuts in basal ganglia were done at the same timetable as CT scan.

3. A template of the region of interest (ROI) derived from co-registered MR images was used for analyzing activity in the basal ganglia.

**SPECT Quantitative and Statistical Analysis**

For semiquantitative evaluation of specific tracer uptake, regions of interest (ROIs) are placed over the basal ganglia and the frontal lobe for \[^{123}\text{I}]\text{IBZM}\) and over the basal ganglia and the occipital cortex for \[^{99m}\text{Te}]\text{TRODAT-1}\). The size of all ROIs is at least twice the FWHM. The basal ganglia/frontal cortex and basal ganglia/occipital cortex for 2 ligands were calculated on both sides. Data from contralateral side to clinical symptoms were added, averaged, and expressed as mean ± S.D. Those data from the ipsilateral side to clinical symptoms were dealt with the same way. Differences for 2 ligands were compared by Student's t-test between patients and normal controls and the significance level was set at p < 0.05.
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| 預定進度累計百分比(%) | 5 | 10 | 20 | 25 | 30 | 40 | 50 | 60 | 70 | 80 | 90 | 100 |
The sudden onset of clinical symptoms, the distribution on one side of the body, and the presence of risk factors for cerebrovascular disease in our patients suggest that a vascular insult is the most likely cause for hemichorea-hemiballism (HC-HB). The serum glucose level (133 mg/dl, 235 mg/dl respectively) was mildly elevated and the choreic and ballistic symptoms persisted for a long time even after the serum glucose level returned to normal. Besides, hyperglycemia is a risk factor for cerebrovascular disease and elevation of serum glucose level in acute stress condition is common. These observations suggest that the episodes were not purely secondary to hyperglycemia. The CT scan showed hyperdense lesion in contralateral putamen in the 68-year-old man and MRI imaging (T1WI) showed hyperintensity in bilateral corpus striatum and globus pallidus in the 72-year-old woman with right HC-HB. The signals in CT/MRI images subsided or showed marked improvement in the follow-up scan 3 months later. The sequential changes of CT/MRI in our cases including the development of significant internal brain atrophy are the same as previous reports. Specifically, there were no lesions identified in the subthalamic nucleus in both patients.

Both the $^{99m}$TcTRODAT-1 and $^{[123]}$IIBZM SPECT of the 68-year-old man showed decreased binding potential in bilateral basal ganglia (Table 1). The decrease of $^{[123]}$IIBZM binding in the second scan is most likely related to the D$_2$ blocking effect of haloperidol. The $^{[123]}$IIBZM was not available
when the 72-year-old woman was recruited. The first dopamine transporter scan showed increased binding potential which could be compensatory to a primary deficit of decreased D₂ receptors. In summary, the nigrostriatal dopaminergic activity decreased in patients with vascular HC-HB.

Both patients showed an excellent response to haloperidol (5 mg/day for the man; 10 mg/day for the woman). The older lady developed confusion in addition to HC-HB. She needed an increased dose of haloperidol for symptom control during the acute phase (30 mg/day). The adverse effects from haloperidol treatment were fatigue and excessive daytime drowsiness. We tried to taper and stop the haloperidol treatment, but the involuntary movement recurred again. Patients were maintained on haloperidol treatment and there were only mild intermittent distal choreic movements.

Two control subjects with comparable ages were studied with both [⁹⁹ᵐTc]TRODAT-1 and [¹²³I]IβZM SPECT. The studies were done smoothly. All the subjects experienced no adverse effects from the SPECT study.
Table 1: \([^{99m}Tc]TRODAT-1\) and \([^{123}I]IBZM\) SPECT imaging in 2 patients with vascular HC-HB and 2 control subjects.

<table>
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<th>No.</th>
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<th>TRODAT-1 (BG/OC) 2nd**</th>
<th>IBZM (BG/FC) 1st*</th>
<th>IBZM (BG/FC) 2nd**</th>
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<td>L 1.92</td>
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* = male, F = female, L = left, R = right, NA = not applicable

*no treatment, **continued treatment with haloperidol
Fig 1. A 68-year-old man with left hemichorea-hemiballism.
Apr, 2001  
(No haloperidol)

Aug, 2001  
(Haloperidol 10 mg/day)

CT without contrast

[\textsuperscript{99m}Tc]TRODAT-1

Fig 2. A 72-year-old woman with right hemichorea-hemiballism.
[\textsuperscript{99m}Tc]TRODAT-1 \hspace{2cm} [\textsuperscript{123}I]IBZM

68 y/o, male
left hemichorea-hemiballism

72 y/o, female
right hemichorea-hemiballism

62 y/o, male
normal control

72 y/o, male
normal control

Fig 3. The [\textsuperscript{99m}Tc]TRODAT-1 and [\textsuperscript{123}I]IBZM imaging in two patients with hemichorea-hemiballism (before haloperidol treatment) and two normal controls.
Previous reports showed that hemichorea-hemiballism (HC-HB) are most commonly due to lesions in the contralateral subthalamic nucleus or the connections of the subthalamic nucleus, although some reports showed ipsilateral basal ganglia lesion (15, 16). Our patients show no demonstrable structural changes in the subthalamic nucleus and the continuous, violent, coordinated movement of the axial and proximal appendicular musculature are due to lesions in the corpus striatum, especially putamen is the common lesion for our patients.

The cause of striatal hyperintensity in T1-weighted MRI image or hyperdense lesion in noncontrast CT scan is still unknown. Reactive astrocytosis or the presence of gemistocytes has been considered to be the cause of the striatal hyperintensity (7). However, striatal hyperintensity on T1WI disappeared in these patients after several months. There was no hemosiderin deposition noted on follow-up T2-weighted images in our patients or previous series, thus the possibility of striatal hemorrhage or ischemic injury with hemorrhagic transformation was unlikely (21).

In this study, we showed a marked decrease of dopaminergic function in the affected basal ganglia, both in pre- and post synaptic SPECT studies, in acute stage in all patients. These findings can explain the clinical symptoms of hemichorea on the contralateral limbs. In the patient 1, the dopamine transporter activity and D2 receptor density recovered in medicated stage when symptoms were under controlled, while MRI or CT showed no apparent change. This finding implies that the pathologic process of HB-HC is reversible and that dopaminergic functional images may
reflect the underlying pathology earlier that MRI or CT. The second patient could not completely finish the IBZM SPECT due to the availability of IBZM from INER was lacking at that period. The findings of \(^{99m}\text{Tc}\) TRODAT-1 and \(^{123}\text{I}\) IBZM SPECT imaging in vascular HC-HB were similar to those of Huntington’s chorea.

In summary, the functional imaging study shows decreased dopaminergic activity in contralateral basal ganglia in patients with vascular HC-HB. Chorea or ballistic movement has been found to be secondary to decreased activity of the indirect pathway in the basal ganglia circuit (29). The dopamine, derived from substantia nigra, has an inhibitory function on the indirect pathway. The effectiveness of haloperidol or tetrabenazine in relieving choreic or ballistic symptoms is most likely due to decreased inhibitory function of dopamine on the indirect pathway and restores the decreased activity of indirect pathway to a physiologically near normal state. The lesions in the basal ganglia are permanent which lead to a negative pressure effect on follow-up CT or MRI scan. The need for continued haloperidol treatment in order to control the HC-HB also suggests a permanent structural lesion has been produced. Dopamine may exert an excitatory or inhibitory influence on various physiological functions. The clinical excellent response to haloperidol does not always imply an increased activity of the dopaminergic system.
建議

1. $[^{123}\text{I}]$ IBZM 的供應請儘量穩定持續。單側舞蹈症是一種罕見但很特殊的疾病，對於它的病態生理的探討不僅有助於基底核神經迴路及神經傳導物質的基礎研究，也可提供臨床治療一個學理上的根據。

2. 因為七月中至十月中有三個月沒有$[^{123}\text{I}]$ IBZM，所以造成整個研究進度落後，殊屬可惜。目前世界上以多巴胺運動器及接受器單光子電腦斷層掃描來長期追蹤研究這個疾病的團隊可說少之又少。我們將繼續完成這個計劃，並寫成原著論文發表。


T1-weighted MR images in a case of chorea with hyperglycemia. 


*Parkinsonism and Related Disorders* 2001;7:319–21.


