Parkinsonism or Other Movement Disorders Presenting as Stroke Mimics

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Abstract-

Purpose: Patients with parkinsonism or other movement disorders may visit the emergency department due to acute deterioration of neurological status or consciousness disturbance. Under such circumstances, patients may be misdiagnosed as having a hyperacute stroke, i.e. stroke mimic. The purpose of the present study was to explore the clinical features and consequences of patients with parkinsonism or other movement disorders presenting as stroke mimics with activation of a stroke code.

Methods: In this retrospective case-series study, we reviewed the charts and stroke code registry data in two stroke centers with high volume of stroke codes and thrombolytic therapy in the Southern Taiwan.

Results: We found seven male patients (67.0 ± 12.8 years old): one with focal myoclonus, one with focal dystonia, and the other five with parkinsonism. The chief problems for emergency department visit included acute consciousness disturbance in one patient and motor weakness in other six patients. Five of the six patients with motor weakness complained unilateral symptoms. Six patients were evaluated by neurology residents (five by second-year residents, one by a third-year resident) and one by a board-certified neurologist, while a misdiagnosis of a stroke was made in three patients. All patients experienced neurological improvement when follow-up. One patient who received intravenous thrombolytic therapy had no intracranial hemorrhagic complications.

Conclusion: Although rare, parkinsonism or other movement disorders may present as a stroke mimic with activation of a stroke code. Consulting neurologists should clarify the etiology for those patients with acute consciousness disturbance or motor weakness and avoid unnecessary thrombolysis.

Key Words: Parkinson disease, parkinsonism, movement disorders, stroke code, thrombolysis

Acta Neurol Taiwan 2016;25:124-128

INTRODUCTION

Recombinant tissue-type plasminogen activator (rtPA) administered within 3 hours of onset is the only approved pharmacological treatment in Taiwan for acute ischemic stroke[(1)]. Due to its narrow therapeutic time-

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Received April 7, 2016. Revised June 7, 2016.
Accepted July 19, 2016.

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window, only a minority of our stroke patients can receive intravenous rtPA[1]. A stroke code, i.e. activation of the stroke team when the arrival of a possible stroke patient in the hyperacute phase, has been proven to improve the speed of evaluation and treatment with rtPA[2,3]. Nevertheless, patients with other neurologic disorders may also present with acute symptoms mimicking stroke (i.e. stroke mimics)[4]. Stroke mimics may account for about 10.0% of all stroke codes[2] and even lead to unnecessary administration of IV rtPA[5]. Concerning the cost and potential lethal bleeding consequence of rtPA, it is important to distinguish a true hyperacute stroke and a stroke mimic in the emergency setting.

Neurologic disorders commonly presenting as stroke mimics include seizure, migraine, and conversion disorders[4]. The acute deterioration of asymmetric motor symptoms in parkinsonian patients, with or without consciousness disturbance, may also lead to an erroneous diagnosis of stroke[6]. However, the role of parkinsonism or other movement disorders in stroke mimics has been seldom discussed[7]. In the present study, we retrospectively analyzed patients with parkinsonism or other movement disorders presenting as stroke mimics in two comprehensive stroke centers with well-organized stroke code systems and high volumes of stroke codes and thrombolytic therapy for acute ischemic stroke.

PATIENTS AND METHODS

Two comprehensive stroke centers with their stroke code registries were involved in this study. The National Cheng Kung University Hospital (NCKUH) is a 900-bed medical center. Its pre- and in-hospital stroke code systems were set up since 2008[8]. The accuracy and final diagnosis of each patient with activation of stroke code were prospectively registered as a quality control measurement. The Ditmanson Medical Foundation Chia-Yi Christian Hospital (CYCH) is a 1000-bed academic regional hospital and started its stroke code system and registry since 2007[9]. We retrospectively searched those two registries for all stroke code patients with the final diagnoses of parkinsonism or other movement disorders. Medical records of these patients were reviewed. Their baseline demographics, presenting symptoms, neurologic status, treatment courses and outcomes were retrieved from the medical records. Our institutional review boards approved the protocol (Approval NO.: A-ER-101-046).

RESULTS

One (0.2%) patient from the CYCH and six (0.8%) patients from the NCKUH were identified from the two stroke code registries. Their baseline demographics and neurologic symptoms on initial presentation were summarized in Table 1. More than a half of them had parkinsonism at baseline and all of them except one presented with limb motor weakness. Their mean blood pressure and serum glucose levels were both in the normal range.

Six patients were evaluated by neurology residents (five by second-year residents, one by a third-year resident) and one by a board-certified neurologist, while a misdiagnosis of a stroke was made in three patients. The three misdiagnosed patients were all evaluated by second-year residents. The final diagnosis at discharge was Parkinson disease or other parkinsonian syndrome in four patients, focal left upper limb dystonia in one

Table 1. Clinical demographics characteristics of the seven patients

Characteristics	
Age, mean (SD)	67.0 (12.8)
Male sex, n (%)	7 (100.0)
Hypertension, n (%)	3 (42.9)
Diabetes, n (%)	3 (42.9)
Hyperlipidemia, n (%)	1 (14.3)
Current smoker, n (%)	1 (14.3)
Parkinsonism diagnosis at baseline, n (%)	4 (57.1)†
Systolic blood pressure, mmHg, mean (SD)	139.0 (21.0)
Diastolic blood pressure, mmHg, mean (SD)	83.7 (11.4)
Glasgow Coma Scale, median (IQR)	15 (11-15)
Disturbance of consciousness, n (%)	2 (28.6)
Facial asymmetry, n (%)	2 (28.6)
Focal limb weakness, n (%)	6 (85.7)
Asymmetry of deep tendon reflex, n (%)	0 (0)
Babinski sign, n (%)	0 (0)
NIHSS, median (IQR)	10 (5-15)
Serum glucose, mg/dl, mean (SD)	131.4 (62.4)

SD: standard deviation; IQR: interquartile range; NIHSS: National Institutes of Health Stroke Scale; †The mean duration of parkinsonism diagnosis was 9.9 years (SD: 7.3 years; range: 1.5-19 years).

Table 2. Detailed clinical information of the five patients with acute worsening of parkinsonism

	Prior diagnosis of parkinsonism, duration	Worse side of parkinsonism at baseline	Presence of motor fluctuation at baseline	Baseline anti-parkinsonian medication, levodopa equivalent daily dose	Timing of last anti-parkinsonian medication before stroke code, hours	Initial presentation activating stroke code	Consulting neurologist, correct diagnosis	Comments
Case 1	No (newly diagnosed)	Right	NA	NA	NA	Acute right side weakness	Second-year resident, no	Rest tremor and bradykinesia were present for 1 year, without any medical consultation; misdiagnosed as a stroke
Case 2	Yes, 4 years	Right	Unknown	500 mg	3 hours	Acute left side weakness	Second-year resident, yes	Right subdural hemorrhage with operation two years ago
Case 3	Yes, 15 years	Left	Yes	1360 mg	4 hours	Acute paraparesis	Third-year resident, yes	The difficulty in standing up and walking is related to the "OFF" symptoms
Case 4	Yes, 1.5 years	Right	No	230 mg (irregularly controlled)	Unknown; poor compliance	Acute right side weakness and dysarthria	Second-year resident, no	Poor compliance and under-treatment of his parkinsonian symptoms; misdiagnosed as a stroke; family refused IV rtPA
Case 5	Yes, 19 years	Left	Yes	600 mg	4 hours	Acute consciousness disturbance	Second-year resident, no	Sleep attack due to Parkinson disease and benzodiazepine overdose; misdiagnosed as a stroke ("basilar artery occlusion") and treated with IV rtPA

Note: NA: not applicable; IV: intravenous; rtPA: recombinant tissue-type plasminogen activator.

patient, myoclonus of left upper and lower limbs in one patient, and sleep attack and benzodiazepine overdose in a patient with Parkinson disease, respectively. For the dystonic patient, the potential cause was sulpiride (50 mg/day) prescribed by his pyschiatrist 17 days ago. The focal dystonia improved after one bolus intravenous biperidin 5 mg. For the myoclonic patient, he had past history of "mandible-palatal and left limbs myoclonus" after operation of right trigone meningioma. And his focal myoclonus improved after intravenous piracetam 12 gm.

For the five patients with parkinsonism (Table 2), one patient's Parkinson disease was newly diagnosed during this hospitalization. And his motor function improved significantly after L-dopa treatment. The other four patients had parkinsonism for 1.5-19 years, while symptoms of motor fluctuation were present according to medical records. Only the one patient with benzodiazepine overdose was treated as a hyperacute ischemic stroke (basilar artery occlusion) with intravenous rtPA. Retrospectively, the results of cranial nerve examinations, including bilateral pupil sizes, light reflexes, and ocular movements of this patient were all normal. The muscle tones were hypertonus and the muscle power was ≥ 3 for upper and lower limbs bilaterally and symmetrically. A diagnosis of metabolic coma, rather than "basilar artery occlusion", might be easily made by a board-certified neurologist. No bleeding complication, including intracranial hemorrhage, happened in that patient. The treatment courses and outcomes of the seven patients were listed in Table 3. And all but one of them had neurologic improvement when discharge.

Table 3. Treatment course and clinical outcome of the patients

Intravenous rtPA, n (%)	1 (14.3)
Length of stay, days, median (IQR)	2 (1-3)
Discharge modified Rankin Scale, median (IQR)	2 (1-3)
Neurologic improvement when discharge, n (%)	6 (85.7)

rtPA: recombinant tissue-type plasminogen activator; IQR: interquartile range

DISCUSSION

In two stroke centers with high volumes of stroke code and thrombolytic therapy, parkinsonism or other movement disorders may present as a stroke mimic and account for less than 1% all stroke code activated. Most of the patients had improved neurological status at discharge. One patient was inadvertently treated with intravenous rtPA, though no subsequent bleeding complications happened.

Parkinsonian patients usually have motor symptoms such as bradykinesia, rigidity, and tremor, while it may be difficult to distinguish them from motor paresis. Patients may also complain sensory symptoms like stiffness of limbs or joint. All those motor and sensory manifestations may be asymmetrical, deteriorate acutely, and lead to emergency room admission[6]. Our analysis of case series suggests several clues which may be useful for consulting neurologist. First, we should be alert to a past history of Parkinson disease or other parkinsonian syndrome. Second, the blood pressures were not as high as those in acute stroke patients. Third, the deep tendon reflexes are symmetrical, while a Babinski's sign is rare. Finally, a detailed review of past medical history, including drug history, was also very important.

It may sometimes be difficult to correctly diagnose an ischemic stroke at its hyperacute phase. The initial presentation may not be typical, while the statement of symptoms may not be clear due to the old age of the patients. A non-contrast of computed tomography (CT) usually fails to shows definite evidence of an acute cerebral infarct during the hyperacute stage. For a consulting neurologist, the diagnosis of ischemic stroke sometimes is usually made in a tentative form and should be confirmed later by using magnetic resonance image (MRI). But due to Taiwan's hospital accreditation guidelines requiring us to administrate intravenous rtPA[10,11] within 60 minutes, a neurologist may fall into such dilemma whether to give rtPA in a patient potentially with stroke mimic. In our series, the drug-overdose patient was erroneously diagnosed as a basilar artery occlusion by a second-year resident. This case may argue the current practice pattern in some stroke centers that after activation of a stroke code, a neurology resident or emergency physician can decide whether to administer rtPA without consulting a board-certified neurologist. And in the era of endovascular therapy, further image modality like CT angiography will become routine for patients with suspected hyperacute stroke[12] and thus may be helpful in such a circumstance.

Myoclonus, dystonia, and other movement disorders may happen acutely, with focal onset and caused by an

acute stroke[6,13]. There's a similar case report[14] about a 32-year-old woman who took prochlorperazine and then developed hemidystonia, which triggered a stroke code response from prehospital, emergency medicine and neurology providers. They would be a diagnostic challenge in the emergency setting, especially when the brain CT is non-revealing and MRI is also unavailable. We suggest that response to specific treatment (e.g. piracetam for myoclonus) and past history of brain structural lesion (e.g. contralateral meningioma in our myoclonic patient) may be helpful when differential diagnosis.

Limitations existed in the present study. First, this was a retrospective study and we did not have information other than medical chart (e.g. video for movement disorders). Second, the sample size was quite small because we only have data from two stroke centers. Further study collaborating data from other centers is needed for better understanding of this topic.

CONCLUSION

Although rare, parkinsonism or other movement disorders may present as a stroke mimic with erroneous activation of a stroke code. Consulting neurologists should clarify the etiology for those patients with acute consciousness disturbance or motor weakness and avoid unnecessary thrombolysis.

ACKNOWLEDGEMENT

The study was supported by grants from the National Science Council NSC 99-2628-B-006-033-MY3.

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