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Case study

Isolated INO as a presentation of midbrain paramedian area lacunar infarction in patients with diabetes

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ABSTRACT

The clinical features and pathogenesis of the pure midbrain infarction need to be described. Of 4257 stroke patients who were on the stroke registry between January 2000 and December 2015, 25 patients with pure midbrain infarctions, as demonstrated on diffusion-weighted magnetic resonance imaging, were enrolled. We analyzed the clinical features, MRI findings, and etiologic mechanisms of the infarctions. According to the distribution of each penetrating artery of the midbrain, we classified all the infarctions into paramedian (PM) area (13 patients), lateral area (10 patients), and PM and lateral areas (2 patients). In the lacunar infarction of the PM area group (8 patients), 6 patients had diabetes mellitus (DM), and 5 patients showed isolated anterior internuclear ophthalmoplegia (INO)—the ratio was much higher than that of the non-PM-lacunar infarction group. Patients with DM were more likely to develop lacunar infarction of the midbrain PM area than that of non-diabetics. Data suggest that isolated INO is a presentation of midbrain PM area lacunar infarction in patients with DM.

1. Introduction

Because of the diverse perfusion territory of the vessels that supply the midbrain, infarction of this portion of the brainstem is often accompanied by infarction elsewhere, such as the pons, thalamus and cerebellum. Therefore, infarctions limited to the midbrain are rare [1]. Although previous studies attempted to describe the clinical aspects and etiology of pure midbrain infarction, no consistent conclusions were obtained [1,2]. In the present study, we investigated the clinical characteristics and etiology of 25 patients whose infarctions were limited to the midbrain. We sought to elucidate the clinical, radiologic, and pathogenesis of pure midbrain infarction in different blood supply areas.

2. Methods

2.1. Patients and methods

Data were retrospectively reviewed from medical records and radiologic images for 4257 consecutive patients who were admitted with acute cerebral infarctions to the Neurology Department at the Beijing Shijitan Hospital between January 2000 and December 2015. Patients who had MRI-verified ischemic lesions were only considered, and those with concomitant infarctions in the other locations such as the pons were excluded. We studied 25 patients admitted to our hospital and diagnosed as having pure midbrain infarction on the basis of magnetic resonance imaging (MRI) findings (Table 1). Data from the case series in this study were gathered retrospectively and were originally obtained for clinical purposes only.

We investigated the symptoms, vascular risk factors, and MRI findings by reviewing the patients' medical records. The infarctions were categorized into three groups (paramedian [PM] area, lateral area, and posterior area) on the basis of previously published templates for the distribution of each penetrating artery supplying the midbrain (Fig. 1) [2]. All patients underwent conventional T2- and T1-weighted MRI, and diffusion-weighted MRI (single-shot echo planar spin-echo sequence, b = 1000 s/mm [2], TR/TE 2798/86 ms) was also performed in these 25 patients.

2.2. Risk factors and etiologic classification of stroke

Diabetes mellitus (DM) was defined on the basis of a previously documented diagnosis, the current use of any oral hypoglycemic agents and/or insulin, or hemoglobin A1c (HbA1c) ≥6.5%. Hypertension (HTN) was defined as a previous diagnosis of elevated blood pressure (systolic ≥140 mmHg or diastolic ≥90 mmHg) or the use of antihypertensive medication. Hyperlipidemia was defined as the current use of lipid-lowering drugs or a previous

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Table 1
Characteristics of the 25 patients with pure midbrain infarction.

No./Sex/Age	MRI lesions	Eye movement disorders	Neurologic abnormalities	Risk factors
1/F/50	rt L	Skew deviation	Hemiplegia, Body sway	HTN, Obesity
2/M/81	lt PM (lacunar)	lt adduction palsy	Dysarthria	DM, Hyperlipidemia
3/M/48	rt L (lacunar)	–	Hemiplegia	HTN, Hyperlipidemia
4/M/67	lt PM (lacunar)	–	–	DM, HTN
5/F/71	bil PM	bil adduction palsy	Dysarthria, Paresis of the extremities	HTN, Hyperlipidemia
6/F/59	rt PM	rt adduction palsy/exotropia	Dysarthria, Hemiplegia	HTN, Smoking
7/M/81	lt L	–	Dysarthria, Hemiplegia	DM, Hyperlipidemia
8/F/78	rt PM (lacunar)	rt adduction palsy	–	DM, HTN, Hyperlipidemia
9/F/63	rt L	–	Dysarthria	HTN, Hyperlipidemia, Obesity
10/F/70	lt L	–	Dysarthria, Hemiplegia	–
11/F/56	rt PM (lacunar)	rt adduction palsy	–	DM, Obesity
12/M/75	lt L	–	Dysarthria	HTN
13/M/64	lt PM	lt adduction palsy	–	DM, HTN
14/F/85	lt PM and L	lt vertical gaze/adduction palsy	Dysarthria, Hemiplegia	HTN, Hyperlipidemia
15/M/78	rt PM (lacunar)	rt adduction palsy	–	Smoking
16/F/59	bil PM	bil adduction palsy, skew deviation, ptosis	Dysarthria, Body sway	DM, Hyperhlipidmea, obesity
17/M/63	lt PM (lacunar)	lt adduction palsy	–	DM, HTN, Obesity
18/M/68	rt L(lacunar)	–	Hemiplegia	Smoking
19/M/69	lt PM (lacunar)	lt adduction palsy	–	DM, HTN
20/M/48	lt L(lacunar)	–	Hemiplegia	DM
21/M/64	lt PM	lt adduction palsy, L ptosis	–	DM, HTN
22/F/71	lt PM (lacunar)	lt adduction palsy	Dysarthria, Body sway	Hyperlipidemia
23/M/79	lt PM and L	lt vertical gaze/adduction palsy	Dysarthria, Hemiplegia	Smoking
24/F/66	lt L	–	Hemiplegia, Body sway	Hyperlipidemia
25/M/75	lt L	lt adduction palsy	Dysarthria	HTN

rt = Right; lt = left; PM = paramedian; L = lateral; bil = bilateral; HTN = hypertension; DM = diabetes mellitus.

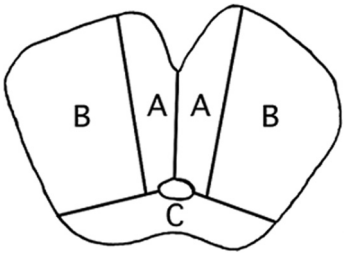

Fig. 1. Classification of the midbrain according to the distribution of each penetrating artery. The midbrain is subdivided into three areas: PM area (A), lateral area (B), and posterior area (C).

diagnosis based on the following values: low-density lipoprotein (LDL) ≥140 mg/dL or cholesterol ≥200 mg/dL) [3].

2.3. Statistical analysis

Gender; age at onset of the stroke; and presence of risk factors such as HTN, DM, hyperlipidemia, cigarette smoking were designated as clinical variables. Differences between the groups were assessed using the Chi-square exact probability method for categorical data and Student's *t*-test for continuous data. Logistic regression analysis for PM-lacunar infarction was conducted, and the coefficients, odds ratios (OR), 95% confidence intervals (CIs), and *p* values were obtained. Probability values were two-tailed, and the threshold for statistical significance was accepted as a *p* value ≤0.05.

3. Results

3.1. Background of the patients

There were 25 patients (14 men and 11 women), ranging in age from 48 to 85 years (mean 67.52 years). The demographic and clinical characteristics of the patients are summarized in Table 1. The most common risk factor was HTN in 14 (56%) patients, followed by DM in 11 (44%) patients, and hyperlipidemia in 10 (40%) patients (Table 1). Among 11 patients with DM, 6 (54.55%) had stroke in the territory of the paramedian.

3.2. Location of the infarctions and clinical features

The infarctions were identified with diffusion imaging, FLAIR imaging, and T2-weighted MRI. The clinical features in the 25 patients are listed in Table 1. Based on clinical presentation and neuro-radiological findings, lacunar infarction was diagnosed in the presence of one of the five classic clinical syndromes associated with magnetic resonance imaging (MRI) evidence of a lacunar lesion measuring ≤1.5 cm in a territory supplied by deep or superficial small perforating arteries consistent with the clinical findings [4]. The infarctions of 13 patients were located in the PM area (including 8 unilateral PM area lacunar infarctions, 3 unilateral PM area nonlacunar infarctions, and 2 bilateral PM area infarctions). Further, the infarctions of 10 patients were located in the lateral area. In 2 patients, the infarctions were located in the PM and lateral area. There were no patients with infarctions located in the posterior area (Table 1, Fig. 2).

Eight of the 25 patients (3 women, 5 men) had PM-lacunar infarctions and 17 had non-PM-lacunar infarctions. There were no differences between the two groups with regard to mean age at stroke, body sway, dysarthria (Table 2, $p > 0.05$). Hemiplegia was frequent among those with a lesion in the non-PM-lacunar infarction (Table 2, $p < 0.05$). Eye movement disorders were observed in 15 patients. Seven patient showed only adduction palsy, which was more common in patients with PM-lacunar infarction than in patients with a non-PM-lacunar infarction (Table 2, $p < 0.05$). Two patients showed biladduction palsy. Adduction palsy and exotropia were found in one patient. Adduction palsy and vertical gaze were found in two patients Table 3.

About 48% percent of all the patients with midbrain infarction had DM. Up to 75% of patients with PM-lacunar infarction had DM. On the contrary, only 29.4% of patients with non-PM-lacunar infarction had DM. Logistic regression analysis for PM-lacunar infarction showed that DM was an independent factor associated