Ipsilateral hemiparesis in lateral medullary infarction: Clinical investigation of the lesion location on magnetic resonance imaging

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ABSTRACT

Background: In 1946, Opalski reported two cases of Wallenberg syndrome with ipsilateral hemiparesis (IH). His hypothesis seems to be based on the view that IH is caused by post-decussating pyramidal tract damage. Afterwards, other researchers proposed a different hypothesis that ipsilateral sensory symptoms of limbs (ISSL) or ipsilateral limb ataxia (ILA) caused by lateral medullary infarction (LMI) might lead to ipsilateral motor weakness. The present study is aimed to clarify whether IH in LMI patients is attributable mainly to ISSL/ILA or disruption of ipsilateral post-decussating pyramidal tract.
Methods: Thirty-two patients with acute LMI admitted during the last 13 years were divided to IH Group ($n = 7$) and Non-IH Group ($n = 25$). Lesion location/distribution on MRI and neurological findings were compared between the two groups.
Results: LMI involved the lower medulla in all seven IH patients and 12 of 25 Non-IH patients. The lower medullary lesion extended to the cervico-medullary junction (CMJ) in four of seven IH patients and one of 12 Non-IH patients. Definitive extension to upper cervical cord (UCC) was confirmed in none of the patients. ISSL was found in two IH and three Non-IH patients all showing only superficial sensory impairments. ILA or hypotonia was observed in 57% of IH and 60% of Non-IH patients.
Conclusion: IH in LMI appears to be due mainly to post-decussating pyramidal tract damage at the lower medulla instead of ILA or ISSL participation.

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1. Introduction

In 1946, Opalski reported two cases of Wallenberg syndrome associating with spastic hemiplegia on the same side as the lesion [1,2]. He assumed that Wallenberg syndrome in the two cases could be explained with ischemic lesions in the upper cervical cord (UCC), which might disrupt various neural fibers descending from the medulla, such as trigeminal nerve spinal cord fibers, and damage ipsilateral post-decussating pyramidal tract fibers. Since his original cases were described, several researchers reported patients with lateral medullary infarction (LMI) extending to UCC which caused ipsilateral hemiparesis (IH) and began to call them Opalski's syndrome [3–6]. Later, many others found the association of IH in LMI patients, whose lesions did not reach UCC on MRI, and called them also Opalski's syndrome [7–14]. The majority of them simply assumed that IH might be caused by damage of the ipsilateral post-decussating pyramidal tract at the lower medulla. Brochier et al. and Kim [15,16] were, however,

suspicious to such a pyramidal tract interruption theory, because many LMI patients with IH show no pyramidal tract sign on neurological examinations and are associated with ipsilateral deep sensory disturbance. Brochier et al. hypothesized that a loss of ipsilateral deep sensation may lead to the disturbance of motor control resulting in ipsilateral limb weakness [15]. Kim similarly hypothesized that IH in LMI patients may result from proprioceptive disturbance combined with spinocerebellar hypotonia/ataxia [16]. Thus, two possible theories have been proposed about the manifestation of ipsilateral motor weakness in LMI. However, previous LMI studies have not focused this particular point. As Opalski's hypothesis is supported by lower lesion location and Kim's hypothesis by symptoms of ISSL and ILA, we retrospectively examined the prevalence of these phenomena in a group of LMI patients with and without IH to test which hypothesis prevails.

2. Materials and methods

We conducted a single-center hospital-based retrospective study. This study was approved by the local ethics committee at our center. During the period between June 1998 and July 2011, 68 LMI patients were admitted to our hospital within a week after ictus. In all these

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patients, LMI lesions were confirmed by diffusion-weighted magnetic resonance imaging (DW-MRI). On admission, detailed neurological examinations were carried out routinely by two or more neurologists, and the findings were recorded on neurological charts. These neurological charts were carefully reviewed retrospectively by two of the authors (M. Uemura and K. Miyashita) to identify the presence of IH, ipsilateral sensory symptoms of limbs (ISSL), ipsilateral limb hypotonia/ataxia (ILA), and pyramidal signs such as pathological reflexes or hyperreflexia. The DW-MRI lesions were also carefully reviewed by three authors (M. Uemura, H. Uno, and K. Miyashita). We excluded patients with the involvement of other lesions which could affect the status of limb weakness such as medial medulla oblongata or cerebellum and without detailed clinical information. The remaining 32 patients were subjected to the study.

The 32 patients were divided to two groups, such as one with IH (IH Group, $n = 7$) and the other without IH (Non-IH Group, $n = 25$). In the IH Group, the severity of motor weakness for upper and lower limbs was classified into the following three grades according to manual muscle testing scores: 1–2 (severe), 3 (moderate), and 4 (mild). Moreover, we added another grade — very mild weakness (trivial paresis) manifesting only pronation of hand — so-called Barre's sign. Baseline data, including age, gender, and comorbidities (hypertension, diabetes, hyperlipidemia, and atrial fibrillation), were collected for all patients. The diagnosis of stroke subtype was based on the criteria of the trial of Org 10172 in Acute Stroke Treatment (TOAST) [17]. The vertebral arteries (VAs) were evaluated by Doppler ultrasonography and MRA in all patients. Conventional angiography was additionally done in 26 patients. VA dissection was diagnosed on the basis of the following: (1) double-lumen sign or intimal flap in MRA or cerebral angiography, (2) characteristic chronological changes in angiography, or (3) intramural hematoma on T1-weighted MRI, as described in previous reports [18,19].

Diffusion-weighted imaging (DWI), fluid-attenuated inversion recovery (FLAIR) imaging, and time-of-flight magnetic resonance angiography (MRA) were routinely performed at 1.5 T (Magnetom Sonata or Magnetom Vision, Siemens Medical Solutions, Erlangen, Germany). DWI images were obtained using the following parameters: repetition time/echo time, 4000/100 ms; matrix, 128×128; field of view, 23 cm; section thickness, 4 mm; intersection gap, 2 mm; and b values, 0 and 1000 s/mm^2.

3. Theory and calculation

Lesion distribution on MRI, including DWI, was evaluated in coronal and axial directions. In the vertical evaluation, medullary regions were divided into upper, middle, lower areas and cervico-medullary junction (CMJ), according to the classification of Bassetti et al. [20]. The extension of LMI lesion to UCC was also carefully evaluated. Lesions in the upper, middle, lower medulla and CMJ were drawn schematically as shaded zones on stereotyped figures, for each patient, to assess regions potentially responsible for IH manifestation. Statistical analyses were performed using the Student's t-test, Fisher exact test or Pearson's chi-square test in JMP®9.0.0. P-value < 0.05 was considered to be significant.

4. Results

Clinical profiles of two groups including age, gender, comorbidities, association of ILA or ISSL, and levels of involved medullary lesions are shown in Table 1. On DWI, the lower medulla was involved in all IH patients (100%) but only in 12 of 25 Non-IH patients (48%). The involvement of the lower medulla was significantly more common in the IH Group than in the Non-IH Group ($P = 0.025$). The lower medullary lesions reached CMJ in four of the seven IH patients (57%) and only in one of the 12 Non-IH patients (8%). The incidence of CMJ involvement was significantly higher in the IH Group compared with the Non-IH Group ($P = 0.004$). In none of 32 patients, definitive extension of LMI

Table 1
Basic characteristics of LMI patients with IH and without IH.

	LMI with IH	LMI without IH	p
	$n = 7$	$n = 25$	
Age	58.6 ± 15.5	56.9 ± 13.4	0.78
Sex (male)	4 (57.1)	21 (84.0)	0.16
Etiology			
DA	4 (57.1)	11 (44.0)	0.64
LAA	3 (42.9)	9 (36.0)	
CES	0	0	
SVD	0	1 (4.0)	
Und	0	4 (16.0)	
Risk factors			
HT	5 (71.4)	17 (68.0)	1.0
DM	4 (57.1)	2 (8.0)	*0.01
DL	1 (14.3)	10 (40.0)	0.37
AF	0	1 (4.0)	1.0
Smoking	4 (57.1)	13 (52.0)	1.0
Level of involved lesions			
Upper medulla	0	7 (28.0)	0.30
Middle medulla	3 (42.9)	19 (76.0)	0.17
Lower medulla	7 (100)	12 (48.0)	*0.025
CMJ	4 (57.1)	1 (4.0)	*0.004
Neurological findings in ipsilateral to the lesion			
ISSL	2 (28.6)	3 (12.0)	0.30
ILA	4 (57.1)	15 (60.0)	1.0

DA = dissecting artery, LAA = large artery atherosclerosis, CES = cardiogenic or aortogenic embolism, SVD = small vessel disease, Und = undetermined, HT = hypertension, DM = diabetes mellitus, DL = dyslipidemia, AF = atrial fibrillation, CMJ = cervicomedullary junction, ISSL = ipsilateral sensory symptoms in limb/body, ILA = ipsilateral limb ataxia/hypotonia. Asterisks indicate statistical significance.

lesions to UCC was identified. ISSL was observed in two of the seven IH patients (28.6%) and three of the 25 Non-IH patients (12.0%). ILA was found in four of the seven IH patients (57%) and 15 of the 25 Non-IH patients (60%). There was no significant difference in the incidence of ISSL association and ILA association between the two groups.

Fig. 1 summarizes neurological findings, such as the severity of motor weakness, the presence or absence of ISSL or ILA and MRI findings in each IH patient. In all the seven IH patients, the severity of motor weakness was mild both for the upper and lower limbs. None of them had pyramidal tract signs such as pathological reflexes or hyperreflexia, although not indicated in Fig. 1. Monoparesis, cruciate hemiparesis, or contralateral hemiparesis was not observed. It should be, however, noted that two patients (Cases 1 and 2) had crural dominant hemiparesis evidenced by trivial weakness in the upper limb and mild weakness in the lower limb. The lower medullary lesions in these two IH patients were somewhat smaller than those in the other five IH patients and reached the most lateral area of CMJ. In the other five IH patients, the severity of weakness was the same in the upper and lower limbs. In two of these five IH patients (Cases 3 and 4) also, the lower medullary lesions reached CMJ. Two IH patients (Cases 4 and 5) had ISSL. In these two IH patients, however, the type of ISSL was numbness and not deep sensory impairment. In Case 4, numbness was detected in the ipsilateral upper and lower limbs. In Case 5, numbness was detected only in the ipsilateral hand, while motor weakness was observed in the ipsilateral upper and lower limbs. ILA was observed in 4 of 7 IH patients (Cases 3, 4, 6 and 7).

Fig. 2 displays the presence or absence of ISSL/ILA and MRI findings in each Non-IH patient. LMI lesions involved the lower medulla in 12 patients (Cases 8–19) and reached CMJ in one patient (Case 15). In the three Non-IH patients with ISSL (Cases 14, 15, and 22), the type of ISSL was all numbness without deep sensory impairments. Two of them (Cases 14 and 15) had lower medullary lesions, whereas the remainder (Case 22) had no lesions in the lower medulla. Fifteen of the 25 Non-IH patients had ILA. The incidence of ILA association was the same whether the lesions involved the lower medulla (8/13, 62%) or not involved the lower medulla (7/12, 58%).