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Unusual sensory disturbance in the thoracic region after stroke: relationship to cheiro-oral and cheiro-oral-pedal syndrome

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Abstract

Three patients with unusual unilateral sensory disturbances in the thorax, in addition to hand and mouth, hand and foot, and hand, mouth and foot regions, respectively, after stroke, are described. Magnetic resonance imaging (MRI) showed a causative lesion in the ventral posteromedial and ventral posterolateral nucleus of the contralateral thalamus in two patients and the contralateral thalamocortical projections in one patient. Symptoms in three patients were due to infarction. Objective sensory loss was not found, but subjective paresthesia was observed in the thorax, in addition to the hand and mouth, hand and foot, and hand, mouth and foot regions, respectively. Paresthesia recovery time was evaluated in these three patients in addition to seven patients with cheiro-oral syndrome and eight patients with cheiro-oral-pedal syndrome. All thirteen patients who demonstrated recovery showed regional improvement of paresthesia in the following sequences: thorax, foot, mouth and then hand, which suggests that the detection threshold, from highest to lowest, occurs in the following sequences: thorax foot>mouth>hand. These three cases with thoracic sensory disturbance form the explanatory links between the cheiro-oral syndrome and the cheiro-oral-pedal syndrome; cases with sensory disturbance in the hand, mouth, foot or thorax could be regarded as cases of sensory hemisyndrome in which the subjective experience of symptoms was restricted to the hand, mouth, foot or thorax. These symptoms may possibly be attributable to differential detection thresholds. © 1997 Elsevier Science B.V.

Keywords: Paresthesia; Threshold; Somatotopy; Thorax; Foot; Mouth; Hand

1. Introduction

Cheiro-oral syndrome is a well-known sensory disturbance that affects the hand unilaterally and the mouth region ipsilaterally. This syndrome was first described by Sittig (1914) and was attributed to a lesion of the parietal lobe. Cortical cheiro-oral syndrome, however, has since been reported in conjunction with brain tumors (Sittig, 1914; Schuster, 1917), infarction (Bogousslavsky et al., 1991), migraines (Bruyn and Weenink, 1966) and epilepsy (Sittig, 1914; Schuster, 1917). Cheiro-oral syndrome can also occur in association with lesions in the thalamus (Strauss, 1925; Garcin and Lapresle, 1954, 1960) and brainstem (Tawara et al., 1974; Ono and Inoue, 1985; Araga et al., 1987; Yasuda et al., 1988; Holter and Tijssen, 1988), and cases associated with lesions in the thalamocortical projections have also been reported (Omae et al., 1992; Isono et al., 1993).

Recently, unusual sensory disturbances in the foot region, in addition to the hand and mouth on the same side (cheiro-oral-pedal syndrome) have been reported in conjunction with lesions of the brainstem (Yasuda et al., 1992), thalamus (Yasuda et al., 1993) and thalamocortical projections (Yasuda et al., 1994). Classical cheiro-oral syndrome can be largely explained by the anatomical proximity of sensory nuclei or fibers from the hand and mouth at various levels in the parietal lobe, thalamus, brainstem and thalamocortical projections. This explana-

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tion, however, cannot account for sensory disturbances in sensory pathways associated with the foot region, because the sensory nuclei or fibers from the feet and hands are not in close proximity in the brainstem (Brodal, 1981), thalamus (Hassler and Riechert, 1961; Lenz et al., 1988) or thalamocortical projections.

Three patients with unusual unilateral sensory disturbances in the thorax, in addition to the hand and mouth, hand and foot, and hand, mouth and foot regions, respectively, after a stroke, were studied. By observing the recovery of sensory disturbances in the thorax, foot, mouth and hand regions in these three patients, and in seven patients with cheiro-oral syndrome and eight patients with cheiro-oral-pedal syndrome, the pathogenesis of sensory disturbance after stroke was evaluated.

2. Materials and methods

2.1. Patients with thoracic sensory disturbances

The clinical features of the three patients (all men) with thoracic sensory disturbances (patients 1-3) are summarized in Table 1. Their mean age was 53.0 years (range: 51-57 years).

2.2. Patient 1

This patient was a 51-year-old man who suddenly developed paresthesia in the lower face, hand and lateral thorax on the left side. Diplopia, headache, nausea and vomiting were not observed. He was admitted to the neurology ward as an emergency patient. Upon admission, his blood pressure was 150/98 mm Hg, and he was alert. His cognitive abilities were normal and he was not disoriented. Eye movement was normal with no nystagmus. The tongue did not deviate. Other tests for cranial nerve functions were normal. Hemiparesis was not evident. Deep tendon reflexes were normal and a pathological reflex was not elicited. Paresthesia near the corner of the mouth and in the hand and lateral thoracic region at dermatome Th5–9 was seen on the left side (Fig. 1A). Objective sensory loss was not present.

Routine blood analysis, cerebrospinal fluid (CSF) examination, electroencephalogram and auditory-evoked potentials all yielded normal results. Somatosensory-evoked potentials (SEPs) from the left side showed a decrease in amplitude of N20 to 25.5% of the normal side. Latencies were normal. Magnetic resonance imaging (MRI) revealed a causative infarction in the ventral posteromedial and ventral posterolateral nucleus of the right thalamus (Fig. 2).

Paresthesia in the lateral thoracic region, near the corner of the mouth and in the hand region improved in three weeks, one month and one year, respectively.

2.3. Patient 2

This patient was a 57-year-old man who suddenly developed paresthesia in the hand, foot and lateral thorax on the left side. Diplopia, headache, nausea and vomiting were not observed. He was admitted to the neurology ward as an emergency patient. Upon admission, his blood pressure was 170/100 mm Hg. He was alert. His cognitive

Table 1

Clinical findings in patients with thoracic sensory disturbances (TSD), cheiro-oral-pedal (COP) and cheiro-oral syndrome (COS)

Patient no.	Symptom	Age/sex	Causative lesion	Etiology	Paresis	Superficial sensation	Deep sensation	Paresthesia recovery time			
								Hand	Mouth	Foot	Thorax
1	TSD	51/M	VPM, VPL	Infarct	No	Normal	Normal	1Y	1M	_	3W
2	TSD	57/M	Project	Infarct	Yes	Normal	Normal	NC	-	NC	NC
3	TSD	51/M	VPM, VPL	Infarct	Yes	Normal	Normal	9M	4M	3W	2W
4	COP	53/M	PPT	Infarct	Yes	Normal	Normal	7M	2W	4D	-
5	COP	81/F	PPT	Bleeding	Yes	MD	MD	NC	NC	2W	-
6	COP	76/M	PPT	Infarct	No	Normal	Normal	NC	3W	3W	-
7	COP	63/F	VPM, VPL	Infarct	Yes	Normal	Normal	3W	7D	4D	-
8	COP	73/F	VPM, VPL	Infarct	No	Normal	Normal	3M	3W	2W	_
9	COP	72/F	VPM, VPL	Infarct	No	Normal	Normal	NC	NC	NC	-
10	COP	65/F	VPM, VPL	Infarct	No	Normal	Normal	NC	NC	NC	-
11	COP	47/F	Project	Bleeding	Yes	Normal	SD	NC	NC	NC	-
12	COS	68/M	VPM, VPL	Infarct	No	Normal	Normal	3W	1W	-	-
13	COS	68/M	VPM, VPL	Infarct	No	Normal	Normal	8M	1M	-	-
14	COS	66/F	VPM, VPL	Infarct	No	Normal	Normal	2Y	9M	-	-
15	COS	56/F	VPM, VPL	Infarct	No	Normal	Normal	4M	1M	_	-
16	COS	45/F	VPM, VPL	Infarct	No	Normal	MD	8M	2M	-	-
17	COS	68/F	VPM, VPL	Infarct	No	Normal	Normal	8D	3H	_	-
18	COS	47/F	Cortex	Infarct	Yes	MD	SD	NC	NC	_	_

TSD, patients with thoracic sensory disturbances; COP, cheiro-oral-pedal; COS, cheiro-oral syndrome; VPM or VPL, ventral posteromedial or posterolateral nucleus; PPT, paramedian pontine tegmentum; Project, thalamocortical projections; MD or SD, mildly or slightly disturbed; NC, not cured; Y, year; M, month; W, week; D, day and H, hour.



Fig. 1. Paresthesia was seen in the thoracic region in addition to the hand and mouth in patient 1 (A), hand and foot in patient 2 (B), and hand, mouth and foot in patient 3 (C).



Fig. 2. MRI (left: T1-weighted sequence, TR 500, TE 18, axial section; right: T2-weighted sequence, TR 2300, TE 90, coronal section) in patient 1. A causative lesion (arrow) existed in the ventral posteromedial and ventral posterolateral nucleus on the right side.

abilities were normal and he was not disoriented. Eye movement was normal with no nystagmus. The tongue deviated to the left. Other tests for cranial nerve functions were normal. The patient showed mild left hemiparesis, and deep tendon reflexes were increased on the left side with Babinski's sign. There was no objective sensory loss, but paresthesia in the hand, foot and lateral thoracic region at dermatome Th5–10 was seen on the left side (Fig. 1B).

Routine blood analysis, CSF examination, electroencephalogram and auditory-evoked potentials all yielded normal results. SEPs from the left side showed a decrease in amplitude of N20 to 28.7% of the normal side. Latencies were normal. MRI revealed a causative infarction in the posterior portion of the posterior limb of the internal capsule and at the border between the corona radiata and the posterior limb of the internal capsule on the right side, sparing the thalamus (Fig. 3).

Paresthesia in the hand, foot and thoracic region remained for three years.

2.4. Patient 3

This patient was a 51-year-old man who suddenly developed paresthesia in the lower arm, lower face, foot and frontal thorax on the left side. He also showed dysarthria and left hemiparesis. Headache, nausea and vomiting were absent. He was admitted to the neurology ward as an emergency patient. Upon admission, his blood pressure was 200/110 mm Hg. He was alert, his cognitive conditions were normal, and he was not disoriented. No cranial nerve palsy was found. He showed mild left hemiparesis. Deep tendon reflexes were increased on the left side with Babinski's sign. Paresthesia was found in the lower arm, lower face, foot and frontal thoracic region at the dermatome Th7–9 on the left side (Fig. 1C). Objective sensory loss was not seen.

The electroencephalogram and auditory-evoked response were normal. SEPs from the left side showed a decrease in amplitude of N20 to 67.5% of the normal side. Latencies were normal. MRI revealed a causative infarction in the ventral posteromedial and ventral posterolateral nucleus of the right thalamus (Fig. 4).

Paresthesia in the thoracic region, foot, lower face and lower arm improved in two weeks, three weeks, four months and nine months, respectively.

2.5. Patients with cheiro-oral-pedal syndrome

The clinical features of the eight patients (six women, two men) with cheiro-oral-pedal syndrome are summarized in Table 1 (patients 4–11). Their mean age was 66.3 years (range: 47–81 years). The causative lesion in these cases of cheiro-oral-pedal syndrome was found in the contralateral paramedian pontine tegmentum of three patients, the

Fig. 3. MRI (left: T1-weighted sequence, TR 500, TE 18, axial section; right: T2-weighted sequence, TR 2300, TE 90, coronal section) in patient 2. A causative lesion (arrowhead) existed in the posterior portion of the posterior limb of the internal capsule and at the border between the corona radiata and the posterior limb of the internal capsule on the right side, sparing the thalamus.



Fig. 4. MRI (left: T1-weighted sequence, TR 500, TE 18, axial section; right: T2-weighted sequence, TR 2300, TE 90, coronal section) in patient 3. A causative lesion (arrow) existed in the ventral posteromedial and ventral posterolateral nucleus on the right side.

ventral posteromedial and ventral posterolateral nucleus of the contralateral thalamus of four patients and in the contralateral thalamocortical projections of one patient. The clinical features of patients 5 and 6 (Yasuda et al., 1992), patients 9 and 10 (Yasuda et al., 1993) and of patient 11 (Yasuda et al., 1994) have already been published. In one of the three patients, the pontine lesion was due to haemorrhage and in two, the lesion was due to infarction. The four cases of thalamic lesion were due to infarction and the single case of lesions within the thalamocortical projections was due to haemorrhage. Both patients with haemorrhage (patients 5 and 11) showed mild hemiparesis, but only two cases (patients 4 and 7) with infarction showed hemiparesis. Patients with haemorrhage showed mild objective sensory loss, while those with infarction did not. SEPs from the disturbed side showed a decrease in amplitude of N20 to $40.1\pm9.4\%$ (mean \pm SD, n=8) of the normal side. Latencies were normal.

2.6. Patients with cheiro-oral syndrome

The clinical features of the patients (five women, two men) with cheiro-oral syndrome are summarized in Table 1 (patients 12–18). Their mean age was 59.7 years (range: 47–68 years). The causative lesion in these cases of cheiro-oral syndrome was in the ventral posteromedial and ventral posterolateral nucleus of the contralateral thalamus in six patients and in the contralateral parietal cortex in one patient. The symptoms in all patients were due to infarction. Only one patient with cortical infarction (Case 18) showed mild hemiparesis. Objective sensory loss, which was severe, particularly in the case of deep sensation, was found in one patient with cortical infarction, and mild disturbance of deep sensation was observed in one patient with thalamic infarction. SEPs from the disturbed side showed a decrease in amplitude of N20 to $35.8\pm36.5\%$ (mean \pm SD, n=7) of the normal side. Latencies were normal.

3. Results

Patients with haemorrhage showed hemiparesis and mild objective sensory loss in addition to paresthesia, and patients with infarction demonstrated only subjective paresthesia, with the exception of objective sensory loss in two cases and hemiparesis in five cases.

In the patients with thoracic sensory disturbances, paresthesia remained in one patient who had lesions of the thalamocortical projections, however, in two patients with thalamic infarction paresthesia improved in the following sequence: thorax, foot, mouth and then hand. In the patients with cheiro-oral-pedal syndrome, paresthesia did not improve in two patients with a thalamic infarction and in one patient with a lesion of the thalamocortical projections. In two patients with thalamic infarction and in three patients with pontine lesions, paresthesia improved in the following sequence: foot, mouth and then hand. In the patients with cheiro-oral syndrome, paresthesia remained in one patient with a cortical infarction, but in all patients



Fig. 5. Paresthesia recovery time. Paresthesia improvement occurred in the following sequence: thorax, foot, mouth and then hand, in all patients who experienced recovery. (Open squares: cases with thoracic sensory disturbances; open circles: cheiro-oral-pedal syndrome; filled triangles: cheiro-oral syndrome).

with thalamic infarction, it improved in the mouth region first, followed by the hand.

Fig. 5 is a schematic presentation of paresthesia recovery time over the two years period following a stroke. Paresthesia in the various regions improved in the following sequence: thorax, foot, mouth and then hand in the thirteen patients who experienced recovery. Table 2 indicates the mean paresthesia recovery time in the foot $(1.7\pm1.0 \text{ weeks})$, mouth $(6.8\pm10.1 \text{ weeks})$ and hand $(29.1\pm29.2 \text{ weeks})$. Paresthesia recovery time was sig-

Table 2 Mean paresthesia recovery time for the foot, mouth and hand regions

	Foot	Mouth	Hand
Number	6	12	11
Recovery time (mean±SD, weeks)	1.7 ± 1.0^{a}	6.8 ± 10.1^{b}	29.1±29.2

Data were analyzed using Tukey's multiple comparisons. One way ANOVA was applied to determine the significance of differences between groups.

 $^{a,b}P = 0.0107$ compared with hand.

nificantly different between the hand and foot, and between the hand and mouth.

4. Discussion

Cheiro-oral syndrome has been attributed to the close proximity of the sensory areas of the hand and mouth in the parietal lobe (Penfield and Boldrey, 1937; Merzenich et al., 1978; Nelson et al., 1980). In the ventral posteromedial nucleus of the thalamus, the sensory nuclei of the tongue, mouth, cheek and occiput are situated from the inside to the outside, and in the ventral posterolateral nucleus, the sensory nuclei of the hand, upper extremity, trunk and lower extremity are also situated sequentially from the inside to the outside (Hassler and Riechert, 1961; Lenz et al., 1988). The nuclei of the hand and mouth are close together in the thalamus. In the pons and midbrain, the medial part of the medial lemniscus, which transmits sensory fibers from the upper extremity to the ventral posterolateral nucleus, and the ventral secondary ascending tract of the trigeminal nerve, which transmits sensory fibers from the face to the ventral posteromedial nucleus, are very closely situated in the ventromedial part of the tegmentum (Brodal, 1981). The sensory pathways from the thalamus to the cortex are thought to be located near the pyramidal tract, behind the posterior limb of the internal capsule (Bertrand et al., 1965), or in the posterior fourth of the posterior limb of the internal capsule (Groothuis et al., 1977), with the cortical and thalamic innervation of the hand and mouth being in close proximity. As such, somatotopy may well apply also to the innervation of the thalamocortical projections. It has also been suggested that the sensory fibers from the hand and mouth area probably travel in close proximity to the cortical sensory area after leaving the posterior ventral thalamic nucleus. Thus, classical cheiro-oral syndrome can be largely explained by the closeness of the sensory nuclei or fibers from the hand and mouth at any level in the parietal lobe, thalamus, brainstem and thalamocortical projections.

This explanation, however, cannot account for sensory disturbances in the sensory pathways associated with the foot region for the following reasons: In the medial lemniscus of the pons and midbrain, the sensory fibers of the upper extremity, trunk and lower extremity are situated from the inside to the outside (Brodal, 1981), and in the ventral posterolateral nucleus of the thalamus, the sensory nuclei of the hand, upper extremity, trunk and lower extremity are situated from the inside to the outside (Hassler and Riechert, 1961; Lenz et al., 1988). Thus, the sensory fibers or nuclei of the hand and foot are separately situated in the medial lemniscus, thalamus and probably projections. the thalamocortical Cheiro-oral-pedal syndrome can be explained by partial involvement of the medial lemniscus, thalamus or thalamocortical projections, as indicated by the SEPs showing normal latencies and

decreased amplitudes of the N20 potentials (primary sensory cortical potential) elicited by stimulation on the disturbed side (Yasuda et al., 1992, 1993, 1994). The normal latency and decreased amplitude of the N20 potentials has also been reported in cases of pure sensory stroke (Labor et al., 1991).

Patient 1 showed paresthesia in the lateral thoracic region in addition to the hand and mouth regions (cheirooral syndrome) on the same side, and MRI showed a causative lesion in the ventral posteromedial and ventral posterolateral nucleus of the contralateral thalamus. Patient 2 showed paresthesia in the lateral thoracic region in addition to the hand and foot regions on the same side, and MRI showed a causative lesion in the contralateral thalamocortical projections. Patient 3 showed paresthesia in the anterior thoracic region, in addition to the hand, mouth and foot regions (cheiro-oral-pedal syndrome) on the same side, and MRI showed a causative lesion in the ventral posteromedial and ventral posterolateral nucleus of the contralateral thalamus. To date, such sensory disturbances have not been reported.

Anatomical proximity of nuclei or fibers conveying sensory input from the hand, thorax and foot cannot account for sensory disturbances in the sensory pathways associated with the thoracic region because the sensory fibers or nuclei from the hand, thorax and foot are separately situated in the thalamus and probably in the thalamocortical projections. Patients 1-3 showed normal latency and decreased amplitude of the N20 potential, elicited by stimulation on the disturbed side. This indicates the partial involvement of the thalamus or thalamocortical projections in cheiro-oral-pedal syndrome (Yasuda et al., 1992, 1993, 1994). Paresthesia recovery occurred in the following sequence: thorax, foot, mouth and then hand, in all of the patients who experienced recovery, which suggests that the detection threshold, from highest to lowest, occurs in the following sequence: thorax>foot> mouth>hand. The high detection threshold of the thorax and foot may be the reason why the patients with thoracic sensory disturbances, such as patients 1-3, and those with cheiro-oral-pedal syndrome, are more rare than patients with classical cheiro-oral syndrome. Patients 1-3 form the explanatory links between cheiro-oral syndrome and cheiro-oral-pedal syndrome.

Like cheiro-oral or cheiro-oral-pedal syndrome, the sensory level is not specific for certain lesions, as reported in a case of a limited parietal lesion (Schmid et al., 1993) and in cases of limited brainstem lesions (Matsumoto et al., 1988). If the degree of involvement in a limited lesion is severe, the sensory level, depending on the size of the lesion, may be determined because sensory nuclei or fibers have somatotopy. If the degree of involvement in a limited lesion is slight, sensory disturbance may appear in the region with a low detection threshold.

The cases with sensory disturbance in the hand, mouth, foot or thorax could be regarded as cases of sensory

hemisyndrome, in which the subjective experience of symptoms was restricted to the hand, mouth, foot or thorax. These symptoms may be attributable to differential detection thresholds, possibly related to partial involvement of sensory nuclei or fibers having somatotopy.

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