Evolving isolated hand palsy: a parietal lobe syndrome associated with carotid artery disease

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Summary

Six patients with cerebral ischaemia who presented evolving isolated hand palsy were studied, five prospectively and one retrospectively. The motor deficit involved only the hand and the wrist in some cases. In almost all cases the motor deficit was pseudo-ulnar. None of them had a Babinski sign, all had mild sensory symptoms or signs in the affected hand. CT and MRI disclosed recent infarctions contralateral to the affected hand, in the white matter of the angular gyrus, in a vascular borderzone. Five had a tight stenosis of the internal carotid artery. The pyramidal tract was anatomically spared in three cases, even considering its parietal origin. Consistent with previous data, our study suggests that the parietal lobe is involved in the control of the motor function of the hand. We propose the existence of a new entity, characterized by an evolving non-pyramidal motor deficit in the hand following infarction of the angular gyrus of the inferior parietal lobe.

Keywords: stroke; motor deficit; pyramidal tract; angular gyrus; somatotopy

Abbreviations: ACA = anterior cerebral artery; MCA = middle cerebral artery; PCA = posterior cerebral artery

Introduction

Isolated hand palsies are observed in patients with stroke, albeit infrequently. The pattern of weakness has been used to attempt to differentiate subtypes of stroke (Chamorro et al., 1991; Timsit et al., 1992; Bogousslavsky and Caplan, 1995). The term fractional arm weakness (Timsit et al., 1992) has been applied when the weakness of the hand differs from that of the shoulder. It has been shown that a fractional weaknesses is a potent predictor of atherosclerotic infarctions that primarily affect borderzones unlike those due to cardiac embolism.

Two types of fractional weakness have been described, one predominantly involving the shoulder, the other the hand. The former, referred to as ‘the man in the barrel’ by Mohr (1969) and later Sage (1986), concerns patients with bilateral shoulder weakness, following cardiac arrest, associated with middle cerebral artery (MCA)–anterior cerebral artery (ACA) borderzone infarction. Freund and Hummelshheim (1985) reported moderate unilateral weakness of shoulder and hip muscles and limb-kinetic apraxia in 11 patients with lesions (infarctions or tumours) in the premotor cortex.

The old term ‘pseudoperipheral palsy’ has been used when weakness predominates in the hand (pseudo-ulnar or pseudo-radial or pseudo-median hand palsy). The first to mention cortical pseudo-radicular hand sensory deficit was apparently J. Lhermitte in 1909, apropos of patients (n = 7) that had either pseudo-peripheral sensory-motor deficits (n = 4) or isolated pseudo-peripheral sensory deficits (n = 3). In 1913 and 1914 Regnard, and later Déjerine in 1918, described patients with brachial monoplegia, involving only the thenar and hypothenar muscles and the adductor interosseus muscles caused by a tumour located in the frontal and parietal regions. In her 1918 thesis, Athanasio-Benisty studied five patients with rolandic lesions after war wounds. All had a sensorimotor pseudo-peripheral hand deficits: in three the tongue (her patient 3), the face (her patients 1, 2 and 3) and the toes (her patient 1) were also slightly involved. The lesions were tentatively located in the pre- and post-central gyrus. No autopsy was performed. In two of her patients (4 and 5) the lesions were only in the post-central gyrus. In 1932 Garcin stressed the importance of stroke in a patient with a pseudo-median deficit.

The aim of this work is to establish the clinical and radiological profiles as well as the pathophysiology of limited evolving hand palsy in such patients with cerebral infarction.
Methods and patients

Five patients, out of 690 (0.7%) hospitalized in the Stroke unit, with fluctuating hand palsy between January 1993 and April 1994 were prospectively included in the study based on the following criteria: (i) a motor deficit of the fingers with or without wrist motor deficit, while the shoulder and the elbow were spared; (ii) the motor deficit was due to clinically and radiologically confirmed ischaemic stroke. Patients who initially presented a motor deficit of the whole arm, but subsequently improved to a limited hand palsy were not included. An additional case was also included, but retrospectively (Patient 3). All patients were fully examined by at least two neurologists (S.T. and M.L.), with a special emphasis on hand motor function. The medical history as well as the risk factors was also recorded. Risk factors were: a history of hypertension, angina pectoris, hypercholesterolaemia, hypertriglyceridaemia, diabetes and cigarette smoking.

Cranial CT and MRI were performed in all patients who were at least in the end of the first week after stroke onset. For MRI, T1- and T2-weighted images in at least two planes (in most cases a sagittal and an axial slice) were obtained from each patient. The infarction and the vascular territory were localized by CT scan and MRI according to the atlases of Talairach and Tournoux (1988) and Damasio and Damasio (1989). The pyramidal tract was also localized with the atlas of Talairach and Tournoux (1988).

In addition, ECG, echo-Doppler of neck vessels including carotids and vertebral arteries, extracranial and intracranial angiograms with selective visualization of the carotid arteries and their branches, and transthoracic echocardiograms were performed. In five of the patients, cerebral blood flow was measured by the i.v. Xenon technique. Global and regional basal cerebral blood flow was estimated. Asymmetries in regional blood flow and cerebellar diaschisis were noted. Vessel reactivity was studied by i.v. acetazolamide injection.

Results

Clinical data

Six men aged 46–87 years (mean 65.5 years) were included in this study. All had at least one risk factor: hypertension (four), cigarette consumption (four), hypercholesterolemia (three) and history of angina pectoris (one). None had diabetes. Five had had transient ischaemic attacks in the past or after the discovery of the hand motor deficit. In two patients (1 and 5) transient ischaemic attacks were clearly localized in the vertebro-basilar territory while in three cases the territory could not be defined (Table 1). Symptoms were postural in two cases, involved the right side in three and the left side in three.

The motor deficit started with the extension of the little finger, then extended progressively toward the thumb in three patients. In one patient, it started in the thumb (Table 2). In all cases, the motor deficit spread to all the fingers, but the degree of involvement was unequal: in four, one finger predominated, the little finger in three. The wrist was less affected than the hand and the shoulder, and the elbows were always spared. The time course of evolution of the fluctuating or progressing isolated hand palsy (Table 1) was of several days and, in three cases, >1 month. Two patients had amyotrophy of the hand. The motor deficit was always primary and could be distinguished from hypotonia, motor neglect, ataxia and apraxia. No clinical differences were found between patients with right or left deficits as far as the motor impairment was concerned.

The associated signs are summarized in Table 3. Reflexes were present in all cases, but in two they were bricker on the side ipsilateral to the motor deficit. A unilateral Hoffman sign was also observed in these two cases. None of the patients had Babinski signs. All patients showed either sensory symptoms (reported by the patient) or mild sensory signs (observed by the neurologist) involving at least the whole hand; five had no deep sensory deficits. Two patients had an impairment of object recognition by the weak hand. In addition, one patient exhibited aphasia and another hemianopsia.

The evaluation of Patient 1 is typical of this disorder. He was a 52-year-old male manual labourer, and was hospitalized for a right hemiparesis. A few weeks after mild cerebral trauma, he progressively showed a circumscribed right motor hand deficit that worsened over a 1-month period. Despite this paresis, he was able to work for a few weeks, but later had to stop. The motor deficit started with impaired extension of the right little finger, followed by the middle finger. Abduction and adduction of the four last fingers then became impaired and, finally, all the fingers were affected, including the index–thumb grip. During this first month, the patient had six episodes of three different types: two which lasted 10 min and were characterized by a brachiofacial motor deficit associated with dysarthria; three, which occurred while standing-up, consisted of a sudden weakness in both legs, with a collapse of the body; he was unable to move his legs for ~30 s. A single episode of right hemianopsia, lasting 2–3 min, was also experienced. The patient was admitted to the neurosurgery department for a suspected cervical medullary compression, but both cervical MRI and cervical myelography were normal. Cerebral CT and MRI showed an infarction at the boundary between the territories of the left MCA and left ACA. The angiogram showed an occlusion of the left internal carotid artery, an occlusion of the left posterior cerebral artery (PCA) at its origin, and a moderate stenosis of the right internal carotid artery.

Radiological data

Reconstructions of the lesions from CT scan and MRI are shown in Fig. 1, with more detailed analyses in Tables 4 and 5. Three patients (1, 4 and 6) had left infarctions and three right infarctions (2, 3 and 5) involving the parietal lobe. In five, the white matter of the angular gyrus was certainly...
involved, and it was possibly involved in the other (Patient 1). In two patients (2 and 3) the rolandic region was also involved, and in one patient (1) the rolandic supraventricular area. According to the atlas of Talairach and Tournoux (1988), the pyramidal tract was involved in only one patient (1). However, according to studies in primates (Russel and Demyers, 1961), the motor area (areas 4 and 6) and the parietal lobe [areas 3, 1 and 2 (primary sensory cortex), area 5 (anterosuperior sector of superior parietal lobule) and area 7 (superior parietal lobule)] are included in the origin of the pyramidal tract, which led us to suspect pyramidal involvement in Patients 2 and 3 as well. Even excluding these last two cases, the pyramidal tract was therefore completely spared in three patients (4, 5 and 6). These infarctions corresponded to specific territories (Table 6): In four patients the MCA–PCA, in four patients the ACA–MCA, and in two patients the ACA–PCA boundaries were involved. In five patients, angiograms were abnormal ipsilateral to the infarction: in one patient it showed an occlusion of the left internal carotid artery (ICA) (Patient 1), in three patients severe stenosis >70% (NASCET, 1991) of the ICA (2, 5 and 6) and in one patient (4) a severe stenosis at the carotid siphon associated with a tight kink. In the remaining patient, the angiogram was performed by direct puncture above the right carotid bifurcation (Patient 3). One year later this patient presented an occlusion of the right ICA, shown by a new angiogram. Cardiac work-ups showed no potential cardiac sources of embolisms but one patient had a patent foramen ovale associated with a mitral valve prolapse (Patient 3). Among five patients, three had an abnormally decreased global basal cerebral blood flow, but all had a decreased parietal regional cerebral blood flow (Table 7). Three had cerebellar diaschisis. One patient had no reactivity to acetazolamide and two a poor one.

**Discussion**

In this study, we reported six patients who had evolving isolated hand palsy. They had a prior history of vascular
risk factors, a symptomatic severe carotid stenosis and an infarction at the boundaries between anterior, middle and posterior cerebral territory. All but one had an infarction of the angular gyrus.

In the following we will discuss three main points: the evolving isolated hand motor deficit; the mechanism of infarction associated with this clinical description; the localization of the infarction and its relationship to the pyramidal tract.

**The evolving, isolated motor hand-deficit**

Unlike the pseudo-peripheral deficit previously reported (see Introduction), most of our patients had an evolving motor deficit that predominated over the sensory deficit, although all did exhibit mild sensory symptoms or signs. In almost every patient the motor deficit was pseudo-ulnar. Since all

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**Table 4** Detailed anatomical localization of infarction

<table>
<thead>
<tr>
<th>Patients</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
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<tr>
<td>Frontal</td>
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<td>FO8, 10</td>
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<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Temporal</td>
<td>TO4, 9</td>
<td>-</td>
<td>-</td>
<td>TO9</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Superior parietal</td>
<td>PO4, 5, 6</td>
<td>PO6</td>
<td>PO3, 4, 6</td>
<td>PO5</td>
<td>-</td>
<td>PO1, 6</td>
</tr>
<tr>
<td>Inferior parietal</td>
<td>-</td>
<td>PO2</td>
<td>PO2</td>
<td>PO1, 2</td>
<td>PO2</td>
<td>-</td>
</tr>
<tr>
<td>Occipital</td>
<td>O2, 5, 6, 7</td>
<td>-</td>
<td>O2, 5, 7</td>
<td>O6, 7</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

FO2 = cingulate gyrus; FO8 = rolandic region; FO10 = supraventricular area; TO4 = posterior middle temporal gyrus; TO9 = posterior to auditory region; PO1 = supramarginal gyrus; PO2 = angular gyrus; PO3 = lateral superior parietal lobule; PO4 = mesial superior parietal lobule; PO5 = paraventricular area; PO6 = supraventricular area; O2 = supracalcarine; O5 = superior lateral aspect; O6 = paraventricular area; O7 = forceps major (according to Damasio and Damasio, 1989).

**Table 5** Anatomical localization of infarction: summary

<table>
<thead>
<tr>
<th>Patients</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
</tr>
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<tbody>
<tr>
<td>Rolandic region</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Supraventricular area</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
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<tr>
<td>Parietal lobe*</td>
<td>+, Ag</td>
<td>+, Ag</td>
<td>+, Ag</td>
<td>+, Ag</td>
<td>+, Ag</td>
<td>+, Ag</td>
</tr>
<tr>
<td>Temporal lobe</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>Occipital lobe</td>
<td>-</td>
<td>+</td>
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<td>+</td>
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<td>+</td>
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</tbody>
</table>

Ag = angular gyrus; *In this table, parietal lobe does not contain the post-central gyrus.
had an elementary deficit, it was not possible to assess whether they had an apraxia when studied during the acute phases. Follow-up a year later, however, showed that, as far as elementary motor functions are concerned, such patients improve dramatically. The Babinski sign, typical of pyramidal disorders, was never observed. However, reflexes were brisker in two patients ipsilateral to the affected hand.

The mechanism of infarction
Clinical, radiological and functional evaluations suggested haemodynamic infarction. The variability of the deficit, the postural symptoms, as well as the frequency of transient ischaemic attacks, all favoured distal field involvement, confirmed by radiological data. In all patients, the infarctions were detected at the boundary between the ACA, MCA and PCA ipsilateral to a tight stenosis or an occlusion of the carotid artery, resulting in decreased basal cerebral blood flow in the parietal lobe. In three patients reactivity decreased as well.

The localization of the infarction and its relationship to the pyramidal tract: clinical and radiological data
The centre of the infarction was in the posterior part of the inferior parietal lobe, i.e. the white matter of the angular gyrus, concordant with the sensory signs or symptoms, as well as the absence of Babinski signs. The latter was one of the six criteria described by Critchley (1953) as defining disorders of limb motility involving the parietal lobe. The presence of amyotrophy in two out of the six patients also suggests parietal dysfunction according to Silverstein (1931). In his patients, however, amyotrophy was mostly due to a progressively invasive tumour of the parietal lobe. Parietal lobe lesions have already been associated with motor disorders in animal models as well as in humans. In monkeys, parietal lesions impair reaching and/or grasping and skilled movement of the hand (for reviews, see Sakata and Taira, 1994; Mouncastle, 1995). This is particularly true when the inferior parietal lobe is involved, as described by Haaxma et Kuypers (1975), after lesion of the inferior parietal lobe. In humans, parieto-occipital lesions have been associated with optic ataxia, ideational apraxia, ideomotor-apraxia and constructional apraxia. After parietal excisions, in humans, Foerster (1936) observed a transient deficit that he called Tastlähmung (tactile paresis). This term was first used by Wernicke in 1889 to specify that not all motor deficits could be explained by the sensory deficit alone. Sensitivity could be relatively well preserved in cases where tactile exploration and recognition of objects were severely affected. A PET study (Decety et al., 1994) showed that the focus of activity when a patient imagines himself grasping objects is in area 40 of the inferior parietal lobes, as well as in the premotor and prefrontal areas.

In three patients, radiological data excluded any involvement of the pyramidal tract, whatever its origin. Furthermore, the other three patients with partial involvement of the pyramidal tract had no motor signs that distinguished them from the others. This suggests that the six cases of motor deficit might be due to the parietal location of the infarction. Finally, earlier studies (Timsit et al., 1992) showed that the size of the infarction does not modify the profile of weakness in patients with fractional arm weakness. If fractional weakness, in particular isolated hand palsy, was due to a partial pyramidal involvement, the increase of volume of infarctions would have modified the weakness profile. In large infarctions, patients would have a non-fractional weakness. However, this was not the case. In addition, pyramidal fibres from the sensory cortex (Daroff, 1990) tend to project to the more dorsal regions of the spinal cord terminating mainly in the dorsal horn. It is therefore difficult to relate pyramidal involvement originating from the parietal lobe and motor pyramidal deficit.

The major role of certain regions of the parietal cortex is thought to be motor organization (Critchley, 1953), and particularly perceptuomotor co-ordination. The first indication that the parietal cortex is involved in the visual control of hand movement was the Balint syndrome (1909), caused by lesions of the parieto-occipital cortex. Jeannerod (1986) found that patients with parieto-occipital lesions could not form a finger grip correctly before reaching

<table>
<thead>
<tr>
<th>Table 7 Cerebral blood flow in patients with evolving isolated hand palsy</th>
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<tr>
<td>Patients</td>
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<tr>
<td>-----------------</td>
</tr>
<tr>
<td>Basal CBF</td>
</tr>
<tr>
<td>Asymmetry</td>
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<tr>
<td>Decreased rCBF</td>
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<tr>
<td>Crossed cerebellar diaschisis</td>
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<tr>
<td>CO₂ reactivity</td>
</tr>
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(ipsi) = ipsilateral to the infarction.
target objects. More recently, Jeannerod et al. (1994) described a patient with a bilateral grasping deficit and bilateral posterior lesions who had no difficulty in reaching. In these experiments, the hand can be considered as a ‘sensorimotor macula’. However, the hand involvement was mostly functional, whereas our patients had true palsies. This apparent discrepancy might be due to the time of examination. Our patients were seen during the acute phase, while those of Jeannerod were studied later in the chronic phase. It is also possible that the inferior parietal lobe contains a somatotopic representation of the hand and that only the motor aspect of the perceptuomotor function was affected in our patients. The parietal lobe seems to be particularly important for the control of the fingers since digital agnosia can be observed in the Gerstmann syndrome.

We propose the existence of a new entity, characterized by an evolving non-pyramidal motor deficit of the hand related to lesion of the inferior parietal lobe. If verified, it should modify the way we examine patients with motor deficits. The hand would have to be considered as an independent entity and not simply as part of a limb, modifying the notion of pattern of weakness. For example, it would be more relevant, clinically, to analyse the hand itself, rather than to try to define proportional or non-proportional weakness on the basis of comparisons of the entire upper and lower limbs. Finally, if motor functions of the hand are controlled by brain regions that are widely distributed throughout the hemispheres (Mohr et al., 1993), then not only the location but also the volume of the infarction would have to be considered, which in turn differs according stroke subtypes (Timsit et al., 1993).

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References


