

# Alien Hand Sign after a Right Parietal Infarction

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## Key Words

Alien hand · Cerebral infarction · Stroke · Ischemic stroke

## Abstract

Involuntary movements are unusual during or after acute stroke, and alien hand sign has not been reported after single parietal infarction. We report on a woman who presented with involuntary movements and a sense of alienness of her left upper limb following a right parietal infarction. This combination, that can meet the criteria for the 'alien hand syndrome', is rarely seen after acute lesions sparing the corpus callosum and the mesial frontal areas.

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## Introduction

Alien hand sign (AHS) is a term introduced by Brion and Jedynak [1] to describe the sense of alienness of one limb experienced by patients with callosal lesion. More recently, this sign was revisited to include autonomous and observable motor activity of the hand [2]. AHS is usually considered a type of disconnection syndrome resulting from a corpus callosum and mesial frontal lesion, either alone or in combination [2–4], but its local-

izing value is controversial as it has also been reported after lesions sparing the corpus callosum [5–8]. We describe a patient who developed AHS and involuntary movements after a single right parietal infarction, a topography, to our knowledge, previously not reported.

## Case Report

An 81-year-old right-handed woman was admitted to the emergency room because of the acute onset of disorientation. She had arterial hypertension and long-standing type II diabetes mellitus, complicated by chronic renal insufficiency and requiring peritoneal dialysis during the last 2 years, and a right ductal carcinoma of the breast, diagnosed 1 month before admission and staged as T<sub>4</sub>N<sub>2</sub>M<sub>0</sub>. Previously unknown atrial fibrillation was detected. At admission she was disoriented and unaware that she had had a stroke. She also had left hemiasomatognosia, left spatial neglect, mild sensory deficit for tactile and pain modalities, left Babinski sign and bilateral Achilles tendon areflexia. Position and vibration sensations were intact. She presented spontaneous movements in the left arm that were repetitive and consisting on large amplitude wrist pronosupination and wrist and finger flexion-extension. These movements seemed to explore repeatedly the left side, were sometimes combined with grasping the railing of the bed or the drip pole, and often hit these or other nearby objects. The left arm sometimes struck her face or the wall. Although highly patterned, these movements did not have any discernible objective. The patient scornfully considered that this arm was 'silly' and that it 'liked to dance' by itself when asked about. She many times denied it belonged to her. Optic ataxia was not present. When performing the maneuver recommended to

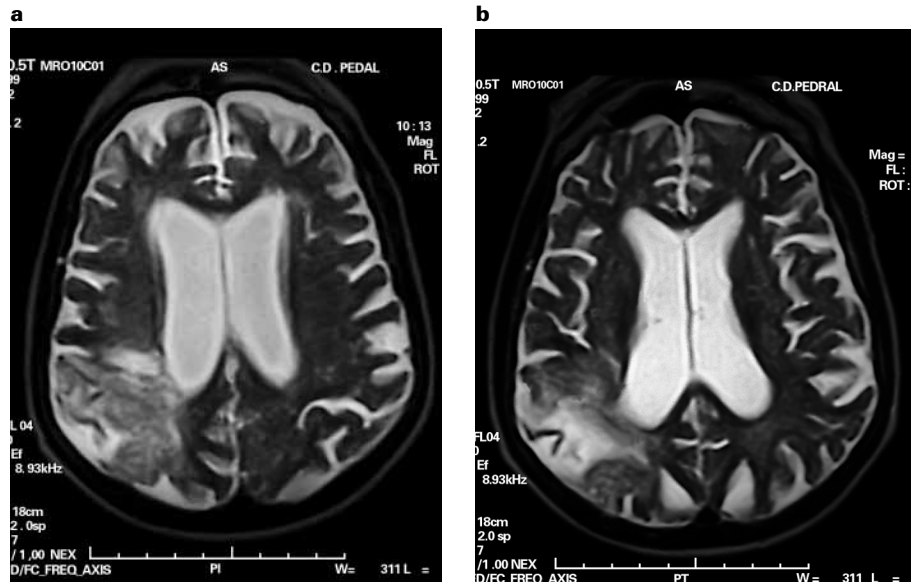
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**Fig. 1a, b.** T2-weighted brain MRI showing a single right parietal infarction.

explore alienness [1], she confused her hand with the examiner's hand. The movements could not be diminished voluntarily and, although almost continuous, did not seriously interfere with voluntary ones, and she could point, catch and manipulate objects without obvious difficulty. Intermanual conflict, diagonalistic dyspraxia and mirror movements were not observed. Except for disorientation that vanished, these neurologic abnormalities persisted unchanged on day 4 when a CT scan demonstrated a right inferior parietal infarction. An MRI confirmed this lesion without disclosing other additional lesions (fig. 1). Mainly affected cerebral areas were 39 and 40. No lesions were detected in the corpus callosum or other cerebral regions. She received antiaggregant therapy with ticlopidine.

During the following weeks, asomatognosia improved but abnormal movements and the sense of alienness of the hand persisted unchanged. She was discharged 2 months after stroke onset and died 3 months after discharge. According to her family, the abnormal hand movements persisted until shortly before death.

## Discussion

To our knowledge, this is the first reported patient in whom an isolated nondominant parietal infarction without corpus callosum involvement caused a clinical picture compatible with contralateral AHS. Recently, Doody and Jankovic [2] suggested that the AHS should include the presence of both the feeling of foreignness and involuntary movements. Patients with nondominant parietal infarction can exhibit asomatognosia, in the sense that he or she denies being the owner of his or her limb and can make scornful comments about this limb in accordance with the first condition of the AHS, namely the feeling of foreignness or alienness. However, in the typical case, the

limb is not out of control and it does not carry out involuntary movements. This second condition for the AHS, the autonomous and observable involuntary movement of the left arm, was remarkable in our patient.

Hyperkinetic disorders during and after acute stroke are uncommon [9, 10]. In recent series, 29 out of 2,500 patients (1%) [9] and 9 out of 1,099 patients (0.8%) [10] presented a movements disorder, but AHS was not diagnosed in any of them. The hyperkinetic involuntary movements presented by our patients did not fit the definition of chorea, ballismus, tics, or myoclonus. Although stereotypes have been described after a cerebral infarction [11, 12], our patient did not exhibit the repetitive and almost identical movements typical of stereotypes.

AHS is usually secondary to a corpus callosum lesion either alone or in combination with a lesion in left, right or both medial frontal areas, sometimes extending to neighboring cortical regions, thalamus or basal ganglia [2–4]. However, it has also been reported after localized lesions sparing the corpus callosum: right capsulothalamic hemorrhage [5], right posterior cerebral artery territory infarction [6, 13], bilateral parietal infarctions [7] and border-zone frontoparietal infarction [8], as well as in diffuse degenerative diseases [2]. It is clear from these exceptions that a corpus callosum lesion is neither necessary nor sufficient to explain all reported cases of AHS and that AHS does not have a definite localizing value.

Moreover, the etiology of AHS is also variable, and includes degenerative diseases [2], cerebral ischemia (infarct in anterior cerebral artery territory [2], border zone

infarction [8], posterior cerebral artery territory [6, 13], middle cerebral artery territory [7]), cerebral hemorrhage (of corpus callosum [4], capsulothalamic [5], rupture of an anterior communicating artery aneurysm [2, 4]), traumatic and surgical injuries (callosotomy) [2], tumors [4, 14], encephalitis [4] and epilepsy [14]. AHS in these situations is usually transient, but can persist for months [4].

AHS has never been reported after a single right parietal infarction. Dolado et al. [7] reported a patient with AHS and with an old left parietal infarction and an acute right parietal infarction. Levine and Rinn [6] described a patient with a right posterior cerebral artery infarction with AHS and interpreted the clinical manifestation as an optic-sensory ataxia. Ay et al. [13] reported a patient with AHS after a right posterior cerebral artery infarction involving the splenium of the corpus callosum, in whom PET scans showed a focally reduced uptake in right fronto-parietal-temporal regions and attributed the clinical findings to a triple ataxia (sensory, cerebellar and optic) and the PET findings to diaschisis. In one reported patient with AHS after a right capsulothalamic hemorrhage, cortical hypometabolism was detected by PET and was most pronounced in the sensorimotor area [5]. Leiguarda et al. [14] reported paroxysmal AHS after a right parietal hematoma. All these cases argue about the existence of a sensory or posterior type of AHS that should be distinguished from the more common motor or anterior AHS [3, 13].

The prevailing explanation for AHS indicates a dysfunction of one or both supplementary motor areas or its connections, with release of contralateral and/or ipsilateral movements [2, 4]. Parietal lobes carry motor function,

related to active movements of the contralateral hand and arm, and receives cortical afferents from primary and secondary sensorimotor areas and has reciprocal cortical connections with the premotor and prefrontal cortices [14]. The right parietal infarction in our patient could cause a loss of sensory integration and feedback interfering with motor function. The 'isolated' parietal lobe would then release these exploratory movements in the absence of appropriate stimuli. An alternative explanation is the combination of a neglect syndrome and loss of visual guidance of the affected limb (optic ataxia) that give rise to AHS [2, 6, 13], but our patient had neither a clear optic ataxia nor an occipital lesion.

We agree with other authors who believe that AHS can result from different anatomic lesions and pathological processes and that the combination of lesions necessary to produce this sign is not certain. A right parietal infarction alone may be sufficient to produce AHS, a fact previously predicted by Doody and Jankovic [2], and should be added to the list of rare lesions sparing the corpus callosum and causing AHS. However, caution should be needed when labeling lesions outside the corpus callosum as an AHS. In the sense given by Doody and Jankovic [2], our patient met the main criteria of 'sense of alienness plus involuntary movements'. Labeling these patients as true AHS may be an oversimplification for a previous useful (in the sense of localizing value) concept [1], as such patients may lack other typical accompanying manifestations of 'callosal AHS', such as dyspraxia, intermanual conflict or mirror movements. The AHS concept needs a re-evaluation.

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