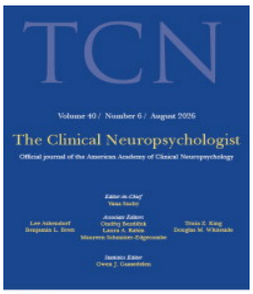

The Clinical Neuropsychologist

ISSN: 1385-4046 (Print) 1744-4144 (Online) Journal homepage: www.tandfonline.com/journals/ntcn20

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To cite this article: Davide Cardile, Serena Campana, Carmelo Mario Vicario, Fabrizio Doricchi, Stefano Lasaponara, Rocco Salvatore Calabrò & Francesco Tomaiuolo (2026) Post-stroke disorders of ownership and agency, alien/anarchic hand syndrome: A longitudinal case analysis and systematic review, The Clinical Neuropsychologist, 40:6, 1760-1778, DOI: 10.1080/13854046.2025.2557973

To link to this article: https://doi.org/10.1080/13854046.2025.2557973

Published online: 04 Oct 2025.

THE CLINICAL NEUROPSYCHOLOGIST
2026, VOL. 40, NO. 6, 1760–1778
https://doi.org/10.1080/13854046.2025.2557973

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Post-stroke disorders of ownership and agency, alien/anarchic hand syndrome: A longitudinal case analysis and systematic review

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ABSTRACT

Objective: Disorders of motor agency and ownership following stroke represent a complex clinical spectrum, ranging from transient phenomena to chronic syndromes. However, the prognostic factors that govern symptom persistence remain poorly defined. **Methods:** We conducted a systematic review of post-stroke cases with uncontrollable hand actions and structural imaging data. Eligible reports were screened for lesion sites, awareness of limb ownership, and clinical courses. The time to the last reported assessment was documented to distinguish acute/subacute from chronic trajectories. Additionally, we present a longitudinal case study of a patient with a lesion extending from the genu to the splenium of the corpus callosum and into the right medial frontal area cortex, with follow-up imaging at both acute and chronic stages. A lesion-based disconnectome analysis was performed to characterize network disconnection. **Results:** Agency disruption was universal, whereas ownership loss occurred selectively, typically associated with parietal, parieto-occipital, fronto-parietal, or combined callosal and medial frontal lesions. Patients with isolated callosal, fronto-parietal, or callosal plus cingulate lesions often achieved complete resolution in the early stages. In contrast, chronic persistence of symptoms was almost invariably linked to combined damage of the corpus callosum and frontal or fronto-parietal cortices. The index case exemplified this pattern, with sustained grasping behavior at long-term follow-up and disconnection of callosal fibers, the superior longitudinal fasciculus, the frontal aslant tract, and cingulum bandle confirmed by tract-based modelling. **Conclusions:** Chronic anarchic hand phenomena primarily result from the combined breakdown of interhemispheric and premotor networks. Early imaging of callosal and frontal pathways is essential for prognosis and therapeutic planning.

ARTICLE HISTORY

Received 24 March 2025
Accepted 2 September 2025
Published online 04 October 2025

KEYWORDS

Anarchic hand syndrome; motor control; corpus callosum; intermanual conflict; neurorehabilitation

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1. Introduction

Alien hand syndrome (AlHS) is a rare neurological syndrome described for the first time by Goldstein in 1908 as a particular kind of motor apraxia (Goldstein, 1908). This syndrome has intrigued researchers for many years due to its distinctive clinical features (Trojano et al., 1993). Typically, patients experience a loss of voluntary control over a limb, which may act autonomously by performing unintended movements or interfering with intentional actions (Pacella et al., 2025). Over time, various labels have been employed to describe this condition, reflecting its complex neuroanatomical underpinnings and heterogeneous symptomatology (Brion & Jedynak, 1972; Marchetti & Della Sala, 1998). Among various terminologies, it is essential to distinguish between the terms AlHS and anarchic hand syndrome (AnHS). While these terms are sometimes used interchangeably, they refer to different clinical phenomena. In AnHS, patients are aware that the limb belongs to them (awarness of ownership), despite its movements occurring without conscious control, whereas in AlHS, movements may also occur involuntarily, but patients fail to recognize the limb as their own (Della Sala et al., 1991).

AlHS lesions' could lead to behaviors such as pathological grasping reflex, involuntary groping, or compulsive object utilization (Doody & Jankovic, 1992), but this syndrome specifically involves a disruption of body ownership. Patients may feel that the limb does not belong to their body, experiencing it as alien or externally controlled. This phenomenon is more common in posterior or callosal lesions (Hassan & Josephs, 2016; Kloesel et al., 2010), where damage to the parieto-occipital or sensory-associative areas distorts body schema and perception. As a result, patients often express astonishment or estrangement toward the limb's movements, describing it as if it were someone else's hand (Graff-Radford et al., 2013). Differently, AnHS is associated with lesions in the medial frontal regions, such as the supplementary motor area and anterior cingulate cortex, of the hemisphere contralateral to the affected limb. Also, in these cases patients lose voluntary control over a limb, leading it to act independently through unintended movements or by disrupting intentional actions, nevertheless they retain awareness of limb ownership (Feinberg et al., 1992). Additionally, involuntary limb movements that interfere with voluntary actions, while awareness of ownership remains intact, are also observed in diagonistic dyspraxia and agonistic dyspraxia. The former typically manifests as intermanual conflict, in which the two hands act at cross-purposes or perform contradictory actions. This phenomenon is most commonly associated with lesions of the CC, resulting in impaired interhemispheric coordination and causing one hand to oppose or undo the actions of the other (Tanaka et al., 1996).

In contrast, agonistic dyspraxia consists in the agonistic movements of the contralateral hand to perform those movements for which the specified hand is apraxic (Lavados et al., 2002). Both phenomena result from disrupted interhemispheric communication, most often affecting the non-dominant (usually left) hand, which may appear to act independently or interfere with motor commands directed at the dominant hand (Aboitiz et al., 2003).

The clinical course of limb movement agency disturbances and/or loss of ownership varies substantially depending on the underlying etiology. Symptoms may be transient

or persistent, largely determined by lesion location and severity. Recent evidence suggests that post-stroke cases of AlHS/AnHS often have a favorable prognosis, with some patients achieving full or partial symptom resolution within weeks or months (Manea et al., 2024). Nevertheless, despite the growing body of knowledge, critical questions persist regarding the long-term prognosis and stroke localization in relation to rehabilitation strategies for AlHS/AnHS.

In this study, we report a case of AnHS and present a systematic review of the literature, aiming to examine the neuroanatomical correlates of post-stroke limb movement agency disturbances and/or loss of ownership. It is essential to highlight that, we employ the patient's awareness of hand ownership as a discriminative factor between AlHS and AnHS, without being constrained by the reported diagnosis. Finally, we highlight the potential for early differentiation between persisting-diminishing and resolving symptoms to the rehabilitation team in selecting motor-cognitive or pharmacological interventions.

2. Case report

A 68-year-old right-handed woman with a history of diabetes, hypertension, dyslipidemia, transient ischemic attacks and ischemic heart disease requiring coronary artery bypass surgery. She was admitted to a neuro-rehabilitation unit four weeks post-ischemic stroke in the anterior cerebral artery territory. During hospitalization, the patient was prescribed quetiapine at a dosage of 50 mg per day, in addition to a regimen of non-psychotropic medications, including cardiovascular, antidiabetic, and gastrointestinal agents. At admission, she presented with left lower limb paresis.

2.1. Neuroimaging findings

CT imaging performed 18 months post-stroke (Figure 1) revealed hypodense areas affecting the genu, body, isthmus, and extending up to the splenium of the CC. Based on the sulcal pattern defined in the Petrides atlas (Petrides, 2018), we observed that the cortical lesion also involved the right cingulate gyrus (CG), including the cingulate sulcus, reaching the ascending ramus of the cingulate sulcus (Brodmann areas 24, 33, 31, and 23). Superior to this, the lesion extended into the area surrounding the anterior vertical paracingulate sulcus, up to the paracentral sulcus (Brodmann area 32, and the inferior portions of BA 6 and 4). The most anterior and superior mesial frontal gyrus (MdFG) part of the superior frontal gyrus (SFG) was partially or completely spared (vertex portions of BA 6; and BA 8, and 9).

A further anatomical investigation of the patient's lesion was performed deriving the tract disconnection measures using the lesion quantification toolkit (Griffis et al., 2021). More specifically, the streamline trajectories of all 87 tracts from the HCP-1065 streamline tractography atlas are combined into a single aggregate whole-brain streamline atlas. The patient's lesion is then embedded into the whole-brain streamline atlas and intersected with the streamline trajectories to identify the subset of streamlines whose trajectories intersect the volume occupied the lesion, producing a percentage probability of disconnection for each (white-matter) association tracts. Successively, the disconnected streamline map shown is output as a tract density image (TDI)

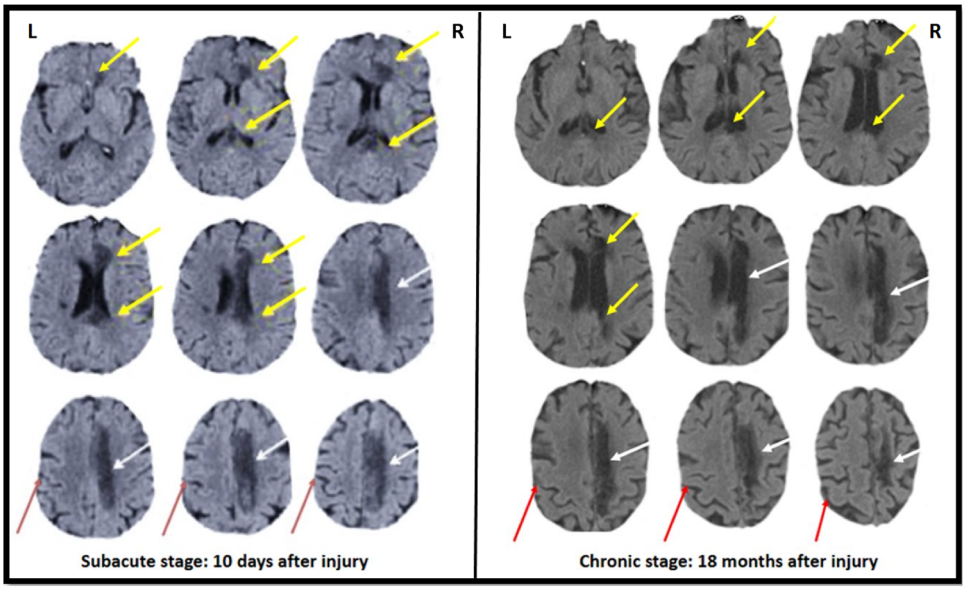

Figure 1. Axial brain CT scans illustrating the neuroanatomical disruptions observed in patients with AnHS. The series progresses from left to right and top to bottom, displaying sequential axial slices at different brain levels. For each level, images acquired at T0 (acute phase) are presented on the left, and those at T1 (chronic phase) on the right. Yellow arrows highlight lesions of the CC, the principal commissural tract connecting the left and right cerebral hemispheres. White arrows indicate damage involving the CG or the inferior portion of the SFG/MdFG. Lesions in these regions are implicated in the disruption of voluntary motor control (agency), while awareness of limb ownership remains preserved. L = left; R = right.

volume in voxel-space and converted to a voxel-wise percent disconnection map smoothed with a 2 mm full-width half-maximum Gaussian kernel (see Figure 2).

Our interpretation of these maps (Figure 2) suggests that damage to interhemispheric and intrahemispheric association pathways underlies the motor and cognitive symptoms observed in our AnHS patient, particularly the loss of agency (voluntary action). Specifically, the Superior Longitudinal Fasciculus I (SLF I) appears to be involved in higher-order aspects of motor behavior requiring information about body part localization (Petrides & Pandya, 2002). The cingulum bundle, as a key limbic pathway, is essential for integrating emotional, cognitive, and executive processes across medial brain regions, especially those that include action regulation (Paus, 2001; Petrides & Pandya, 2002). We also interpret the involvement of the Frontal Aslant Tract as contributing to deficits in the initiation of voluntary movements (Budisavljevic et al., 2017). Furthermore, based on tractography analysis using the Yeh atlas (Yeh, 2022), we identified a unilateral disruption of the Extreme Capsule, a critical structure for ventral stream communication between the frontal and temporal lobes (Schmahmann & Pandya, 2006). Collectively, these interpretations support the view that not only the cingulate area cortex and superior frontal area cortex but also the integrity of their connections and networks including the CC play a fundamental role in explaining the complex behavioral symptoms observed in our investigated patient.

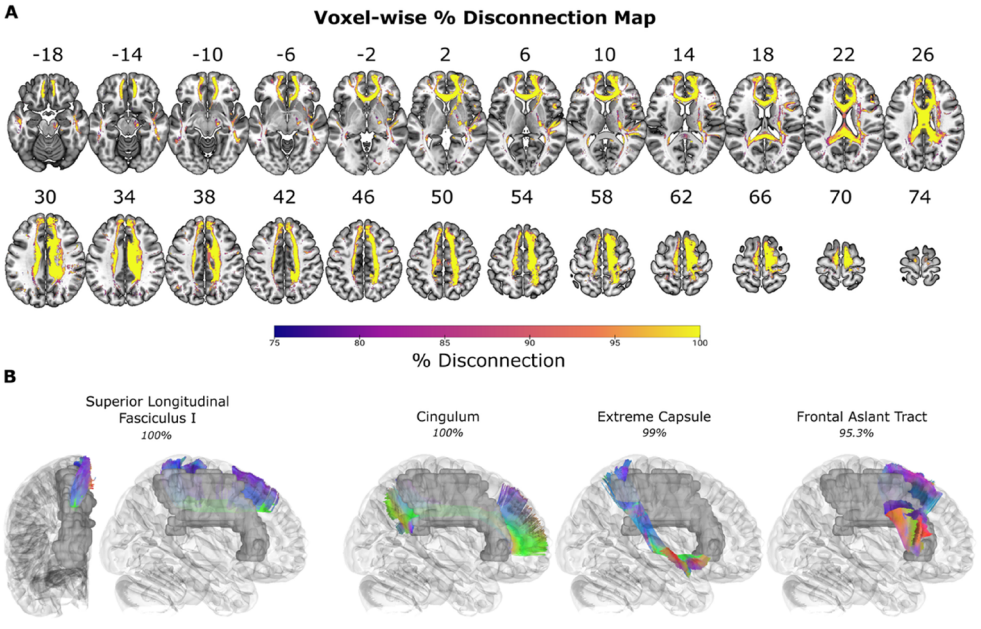

Figure 2. Panel A) Voxel-wise percentage disconnection map derived from a tract density image (TDI), smoothed using a 2 mm full-width at half-maximum (FWHM) Gaussian kernel. Yellow voxels indicate regions with the highest estimated probability of disconnection (100%). Note the involvement of the CC. Panel B) Three-dimensional reconstruction of the white matter association tracts, color-coded by orientation (red: x-axis, green: y-axis, blue: z-axis), showing those significantly affected (>95%) by the patient's lesion (depicted in semi-transparent dark grey). Tractography data were extracted from the HCP-1065 atlas (Yeh, 2022) and visualized using DSI Studio (http://dsi-studio.labsolver.org).

2.2. Clinical and neuropsychological evaluation

Upon admission, the patient was oriented in both space and time but exhibited persistent goal-directed involuntary actions of the left hand: object-driven grasping, repetitive groping of clothing and bed linen, and episodes of intermanual conflict. During hospitalization, she attempted self-strangulation but immediately reported that her left hand had acted against her will and that she was unable to control it. She expressed significant fear and confusion regarding this loss of control, describing it as though the hand was not under her control. However, no undirected or non-purposeful movements such as levitation, elevation in the air (floating hand) or mirror movements were ever recorded, nor did the patient report alien tactile sensations or loss of limb ownership. The patient consistently recognized the left hand as her own and actively attempted to counteract its movements by physically restraining it or verbally addressing it as if communicating with another person. Additionally, the patient exhibited tactile anomia, showing an inability to name objects placed in her left hand when palpated without visual input, whereas no such deficit was observed in the right hand, which also retained normal praxis abilities. However, she could describe certain physical characteristics such as temperature, size, and similarity to previously encountered items. Signs of left spatial neglect were also observed,

limited to extra-personal space, with extinction to double simultaneous stimulation on her left side. Behavioral observations revealed functional impairments in daily activities. The patient required assistance with dressing, meal preparation, and general safety, primarily due to involuntary grasping and groping movements of the left hand, which interfered with bimanual tasks. Overall, the clinical presentation is indicative of AnHS, as the patient recognized the limb as her own despite exhibiting goal-directed well executed but unintended movements, in other words awareness of ownership with loss of agency.

The neuropsychological assessment was performed in a single 60-min session in a quiet room. The performed tests were selected from the Esame Neuropsicologico Breve (ENB), and the normative values used were the Italian reference values (Mondini et al., 2011). The performed tests included a modified forms of the Token Test, Digit Span, Phonemic Verbal Fluency, Short Story Immediate and Delayed Recall, Verbal Abstraction, Cognitive Estimation, Copy of a Drawing, Drawing of a Deasy, Clock Drawing Test. Additionally, Visual neglect for the extra personal space was assessed using: (a) the Line Cancellation Task (Albert, 1973), Reading of Sentences (Pizzamiglio et al., 1989).

The Token Test was administered to assess subtle deficits in receptive language comprehension. Using colored tokens of varying shapes, the task required participants to follow increasingly complex verbal commands. The patient was instructed to indicate the correct sequence of actions as specified. The Digit Span Test measured verbal short-term memory capacity. Digits were read aloud at a rate of one per second, increasing in length until the participant failed to recall sequences correctly on two consecutive attempts. Phonemic Verbal Fluency tasks evaluated lexical access. In the phonemic task, individuals generated as many words as possible beginning with specific letters (C, P, S) in one minute per letter. The Short Story task assessed both Immediate and Delayed recall of a short story read aloud by the examiner. Immediate recall was tested immediately after the first reading. Soon after the immediate recall, the examiner read the story a second time, followed by a delayed recall test after a five-minute interval filled with a non-verbal distractor task. Performance was quantified by counting the number of correctly recalled informative units. The Verbal Abstraction Task assessed reasoning and conceptual thinking by requiring participants to identify abstract categories uniting two concrete concepts (e.g. "bread and milk"). The Cognitive Estimation test assesses the individual's ability to respond to prompts that do not necessarily require a single, precise answer, but instead must be estimated and evaluated based on general world knowledge. The Copy of a Drawing Task assesses the patient's copying abilities when the model is a complex yet highly familiar figure (a house with a fence). The Drawing of a Daisy evaluates the subject's praxic abilities and access to visual representation in relation to a simple figure. The Clock Drawing Test provided insight into constructive praxis, visuo-spatial planning, and executive functioning. Participants drew a clock showing a specific time (e.g. 2:45), and errors were categorized to derive a performance score.

To evaluate extra-personal neglect, the Line Cancellation Test and the Reading Sentences Test were administered. In the Line Cancellation Test, the patient is instructed to cross out every line they can see on the page. A difference in the number of omissions between the left and right sides of the page indicates the presence of

visual neglect. In the Reading Sentences Test, the patient is asked to read one of six sentences at a time from a designated board. Patients with neglect typically omit or misread words on the left side of the text, leading to characteristic reading errors.

The patient underwent cognitive and motor evaluations at two time-points: 4 wk and 18 months post-injury. Table 1 summarizes her performance.

Overall, her performance was within normal limits on most tests. At the initial assessment conducted four weeks post-stroke, the patient demonstrated preserved language function. Performances on the Token Test (comprehension) and Phonemic Word Fluency (lexical retrieval) were above Italian normative thresholds. Similarly, basic attentional span (Digit Span) and episodic memory (both immediate and 5-min delayed recall of a short story) fell within normal limits. The evaluated executive functions were preserved, as evidenced by normative scores on the Verbal Abstraction and Cognitive Estimation tests. In contrast, deficits emerged in visuo-constructive and visuospatial domains. The patient's performance on the Copy of a Drawing and the Daisy Drawing fell below the criterion, indicating constructional apraxia and impaired spatial analysis without evident neglect signs. Finaly Performance on the Line Cancellation Test showed a significant left-sided omission pattern, supported by a pathological between side different score, also the Reading of Sentences test was below the normal score. These findings collectively pointed to a clinical picture of constructional apraxia associated with left-sided extra-personal neglect.

2.3. Follow-up assessment

Eighteen months post-stroke, the patient underwent a follow-up evaluation (Table 1) that was performed using the tests that had scored below the cut-off at the time of the initial assessment. At this time, the patient maintained the same pharmacological regimen prescribed during hospitalization, except for quetiapine (50 mg/day), which had been discontinued. The patient recognized the examiner, and accurately recalled episodes from her rehabilitation stay. The frequency of involuntary and overt grasping movements of the left hand had reduced from "multiple daily" to "sporadic daily" occurrences. However, voluntary control over the limb remained limited. The patient

Table 1. Patient's neuropsychological test results at two time-points: 4 wk and 18 months post-injury.

Test	4 wk from injury	18 months from injury	Cut-off score
Verbal Digit Span	5/8	n.a.	4
Word Fluency	10	n.a.	8
Short Story immediate recall	10/28	n.a.	6
Short Story delay recall	12/28	n.a.	9
Token Test	5/5	n.a.	5
Verbal Abstraction	5/6	n.a.	3
Cognitive Estimation	5/5	n.a.	4
Drawing (copy of a house)	0/2*	1/2*	2
Drawing of Design (daisy)	1/2*	2/2	2
Clock Drawing Test	2/10*	7/10	7
Extra-personal Neglect			
Reading of sentences	2/6*	6/6	5
Line Cancellation Task (between side difference)	10*	0	1

It compares the scores with the cut-off scores for each test to determine impairments. * = indicate the score below the cut-off; n.a.: not assessed.

continued to exhibit involuntary grasping during meals and made conscious but only partially effective attempts to inhibit the behavior. Tactile anomia persisted in the left hand, whereas right-hand haptic functions remained intact as well as praxia. In terms of daily living, the patient still required occasional assistance from her family, particularly for tasks involving fine motor control, such as fastening buttons, tying shoelaces, and handling delicate objects, due to the reemergence of grasp reflexes in the affected hand. We re-administered the tests that had previously yielded scores below the normal range at the first observation, conducted 4 wk after onset. Performance on the Daisy Drawing task met the adequacy threshold, although the copy of a house remained marginally below standard. The Clock Drawing Test revealed residual difficulties in planning complex figures. No signs of left-sided extrapersonal neglect were observed. Performance on the Letter Cancellation task and Reading Sentences tests had returned to normative levels. Overall, mild persistent impairment in high-level visuo-constructive functions remains.

3. Systematic review

3.1. Search strategy

A systematic review was conducted on PubMed, Web of Science, Embase, and the Cochrane Library (search date: 01.10.2024). The search was carried out using the following search string: *(alien hand) OR (anarchic hand) OR (agonistic dyspraxia) OR (diagonistic dyspraxia)*.

Two authors (D.C. and R.S.C.) independently reviewed and extracted data from the studies. Discrepancies were resolved by discussion, with input from a third author (F.T.) when necessary. This multi-step process guaranteed that each article was assessed by at least three researchers. In case of persistent disagreement, the final decision was made collectively by all authors. This study was registered on Open Science Framework (OSF) at: https://doi.org/10.17605/OSF.IO/MPXG9.

The PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) diagram was added to describe the sequence of steps (identification, screening, eligibility, and inclusion) for the collection and determination of qualified studies (Moher et al., 2009) as shown in Figure 3.

3.2. Inclusion/exclusion criteria

Only original studies published in English from 1981 to 2024 and describing patients who suffered from AlHS, AnHS, diagonistic or agonistic dyspraxia were included. Each study had to report age and sex of patients, time elapsed since stroke onset (acute: 1–7 days, subacute: 1 week-6 months, chronic: >6 months) and to contain a neuroanatomical image showing the brain lesion (CT or MRI). We only included articles with MRI or CT scans, because we were interested in localizing the anatomical stroke region. The extent and site of the lesion were better defined using both analog and digital brain atlases (Duvernoy & Cabanis, 1991; Petrides, 2018). A study was excluded if: (I) it describes subjects who suffered from neurodegenerative disorders, inflammatory brain syndromes, traumatic brain injury, lesion resulting from brain tumors and

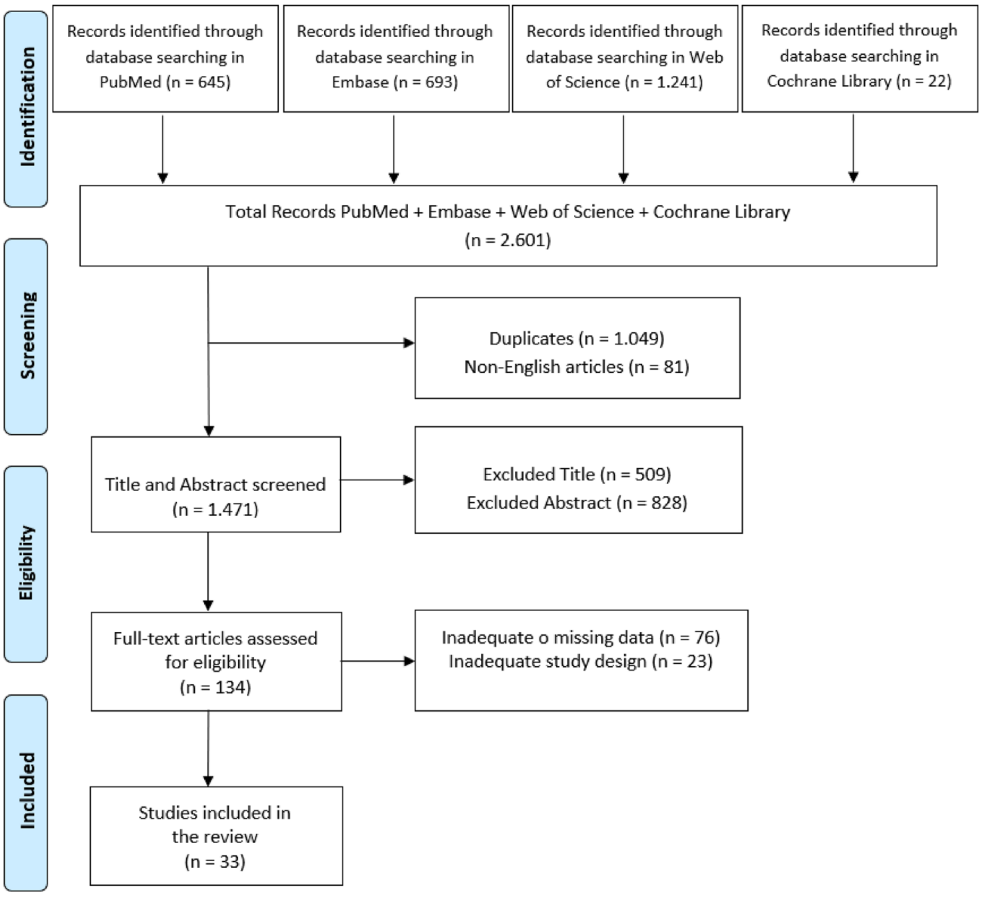

Figure 3. PRISMA flow diagram for the current review.

damage of the thalamus or the basal ganglia; (II) it is a systematic, narrative, or integrative review, even if their reference lists were checked and included when appropriate.

3.3. Results of the systematic review

The initial electronic data search yielded a total of 2601 potentially relevant studies on PubMed, Embase, Web of Science and Cochrane Library. Of these, 1049 were duplicated and 81 were non-English articles. A total of 1337 articles were excluded due to title or abstract (Figure 3).

Of the resulting 134 articles, 33 fully met the inclusion criteria comprising 42 patients who revealed significant variations in symptom presentation based on the lesion location and phase of evaluation.

The images declared by the authors of each study were identified and, if necessary, specified by comparing them with brain atlas (Duvernoy & Cabanis, 1991; Moher et al., 2009). The features of the studies are summarized in Table 2 and include the

Table 2. Summary of cases included in the review.

Studies	Age/Sex	Impaired Hand	Ownership	Hemisphere	CC	CG	SFG	MdFG	Other FL areas	PL	TL	Time to last reported assessment	Symptoms Outcome
(Faber et al., 2010)	56/M	R	YES	L	X		X					24 months	Persisting
(Brainin et al., 2008)	53/F	R	YES	L	X	X	X	X				24 months	Persisting
(Della Sala et al., 1991)	56/F	R	YES	L+R	X		X	X				24 months	Persisting
(Pouget et al., 2011)	37/M	L	YES	L+R	X							24 months	Persisting
(McNabb et al., 1988)	75/F	R	YES	L	X	X	X					12 months	Persisting
(Kischka et al., 1996)	66/M	R	YES	L	X	X	X	X		X		8 months	Persisting
(Moro et al., 2015)	47/F	L	YES	R	X	X	X	X		X		6 months	Persisting
(Goldberg & Bloom, 1990, c1)	53/F	L	NO	R	X				X			12 months	Decreased
(Pappalardo et al., 2004)	60/M	R	NO	L				X				9 months	Decreased
(Nowak et al., 2014, c1)	54/F	R	YES	L	X	X				PL/OL		7 months	Decreased
(Lavados et al., 2002)	40/F	R	NO	L+R	X							7 months	Decreased
(Pacella et al., 2021)	71/F	L	YES	R								6 months	Decreased
(Hanakita & Nishi, 1991)	43/M	L	YES	L	X	X	X			X	X	6 months	Decreased
(Nowak et al., 2014, c2)	71/F	R	YES	L	X	X						9 months	Resolved
(Tanaka et al., 1990)	51/M	L	YES	L+R	X	X	X					7 months	Resolved
(Sarkar et al., 2022)	36/F	L	NO	R	X			X				3 months	Persisting
(Paliwal et al., 2013)	65/M	L	YES	R	X	X						2 months	Persisting
(Mahawish, 2016)	58/F	L	YES	L	X							2 months	Persisting
(Biran et al., 2006)	56/M	R	YES	L	X	X		X				2 months	Persisting
(Park et al., 2012, c1)	72/M	R	YES	L	X	X						7 weeks	Persisting
(Goldberg & Bloom, 1990, c2)	76/F	R	YES	L				X				5 weeks	Persisting
(Goldberg et al., 1981, c1)	63/F	R	YES	L	X			X				5 weeks	Persisting
(Goldberg et al., 1981, c2)	76/F	R	YES	L	X			X				3 weeks	Persisting
(Park et al., 2012, c2)	47/F	L	YES	R	X	X			X			3 weeks	Persisting
(Amalnath et al., 2013)	45/M	R	YES	L	X	X		X				2 months	Decreased
(Bundick & Spinella, 2000)	73/F	L	NO	R				X	X	PL/OL	X	2 months	Decreased
(Murdoch et al., 2021)	74/M	R	YES	L	X					PL/OL		5 weeks	Decreased
(Qureshi et al., 2016)	84/F	L	YES	L						X		1 month	Decreased
(Ma et al., 2023, c2)	63/F	R	YES	L	X	X						17 days	Decreased
(Ma et al., 2023, c5)	61/F	R	YES	L	X	X			X			12 days	Decreased
(Huang & Jia, 2013)	26/F	L	YES	L+R	X							3 months	Resolved
(Nishikawa et al., 2001)	41/M	L	YES	L+R	X							2 months	Resolved
(Wan Yusoff et al., 2021)	62/M	R	YES	R					X			2 months	Resolved
(Yuan et al., 2011)	71/F	L	NO	L+R	X							2 weeks	Resolved
(Martí-Fàbregas et al., 2000)	81/F	L	NO	R						X		5 months	Resolved
(Qu et al., 2021)	57/F	R	YES	L	X	X			X			3 months	Resolved
(El Nahas et al., 2024)	66/M	R	YES	L						X		2 days	Persisting
(Lin et al., 2007)	67/M	R	YES	L	X	X						1 week	Decreased
(Ma et al., 2023, c3)	52/F	R	YES	L	X	X						5 days	Decreased
(Ma et al., 2023, c1)	69/M	L	YES	R	X	X						5 days	Resolved
(Ma et al., 2023, c4)	47/M	L	YES	R	X	X						4 days	Resolved
(Panda, 2010)	68/M	L	NO	R					X	X		2 hours	Resolved

Cases were grouped according to the Time to last reported assessment and to the symptoms. Legend: Male (M), Female (F), Right (R), Left (L), Corpus Callosum (CC), Superior Frontal Gyrus (SFG), Cingulate Gyrus (CG), Medial Frontal Gyrus (MdFG), Frontal Lobe (FL), Parietal Lobe (PL), Occipital Lobe (OL), Temporal Lobe (TL), Case 1 (c1), Case 2 (c2), Case 3 (c3), Case 4 (c4), Case 5 (c5). "X" indicates the lesional site reported by the authors of the different articles. The "X" instead takes the form of a note from the authors of this paper.

patient's age, sex, impaired hand side, awareness of ownership of the impaired hand, lesion site (i.e. the damaged hemisphere(s) and affected lobes or areas), time to last reported assessment and symptom outcome. Importantly, we employ the patient's sense of hand ownership as a discriminative factor between the included patients, without being constrained by the reported diagnosis.

A total of 55 patients were identified in the reviewed literature; however, not all met the inclusion criteria, and only 42 were ultimately included in the present analysis (Table 2). The sample comprised 18 males and 24 females, with ages ranging from 26 to 84 years (mean = 59.3; SD = ±13.18). All patients were right-handed. The agency symptoms affected the right hand in 23 cases (55%) (Amalnath et al., 2013; Biran et al., 2006; Brainin et al., 2008; Della Sala et al., 1991; El Nahas et al., 2024; Faber et al., 2010; Goldberg et al., 1981, c1–c2; Goldberg & Bloom, 1990, c2; Kischka et al., 1996; Lavados et al., 2002, c1–c2; Lin et al., 2007; Ma et al., 2023, c2–c3; 2023, c5; McNabb et al., 1988; Murdoch et al., 2021; Nowak et al., 2014; Pappalardo et al., 2004; Park et al., 2012; Qu et al., 2021; Wan Yusoff et al., 2021) and the left hand in 19 cases (45%) (Bundick & Spinella, 2000; Goldberg & Bloom, 1990, c1; Hanakita & Nishi, 1991; Huang & Jia, 2013; Ma et al., 2023; 2023, c1; 2023, c4; Mahawish, 2016; Martí-Fàbregas et al., 2000; Moro et al., 2015; Nishikawa et al., 2001; Pacella et al., 2021; Paliwal et al., 2013; Panda 2010; Park et al., 2012, c2; Pouget et al., 2011; Qureshi et al., 2016; Sarkar et al., 2022; Tanaka et al., 1990; Yuan et al., 2011).

Regarding the lesion characteristics, injuries involving the CC were reported in 33 patients (79%) (Amalnath et al., 2013; Biran et al., 2006; Brainin et al., 2008; Della Sala et al., 1991; Faber et al., 2010; Goldberg et al., 1981, c1–c2; Goldberg & Bloom, 1990, c1; Hanakita & Nishi, 1991; Huang & Jia, 2013; Kischka et al., 1996; Lavados et al. 2002; Lin et al., 2007; Ma et al., 2023, c1–c5; Mahawish, 2016; McNabb et al., 1988; Moro et al., 2015; Murdoch et al., 2021; Nishikawa et al., 2001; Nowak et al., 2014, c1–c2; Paliwal et al., 2013; Park et al., 2012, c1; 2012, c2; Pouget et al., 2011; Qu et al., 2021; Sarkar et al., 2022; Tanaka et al., 1990; Yuan et al., 2011). However, isolated CC lesions were relatively rare, occurring in only 7 cases (17%) (Huang & Jia, 2013; Lavados et al., 2002; Mahawish, 2016; Nishikawa et al., 2001; Pouget et al., 2011; Sarkar et al., 2022; Yuan et al., 2011). Among the patients with CC involvement, the CG was affected in 20 cases (61%) (Amalnath et al., 2013; Biran et al., 2006; Brainin et al. 2008; Hanakita & Nishi, 1991; Kischka et al., 1996; Lin et al., 2007; Ma et al., 2023, c1–c5; McNabb et al., 1988; Moro et al., 2015; Nowak et al., 2014, c1; 2014, c2; Paliwal et al., 2013; Park et al., 2012, c1; 2012, c2; Qu et al., 2021; Tanaka et al., 1990). In 9 of these, the injuries were limited only to CC and CG (Lin et al., 2007; Ma et al., 2023, c1–c4; Nowak et al., 2014, c1; 2014, c2; Paliwal et al., 2013; Park et al., 2012, c1). The remaining 11 cases showed additional involvement of the SFG/MdFG, and/or other frontal areas (Amalnath et al., 2013; Biran et al., 2006; Brainin et al., 2008; Hanakita & Nishi, 1991; Kischka et al., 1996; Ma et al., 2023, c5; McNabb et al., 1988; Moro et al., 2015; Park et al., 2012, c2; Qu et al., 2021; Tanaka et al., 1990). Lesions affecting the CC and frontal areas but sparing the CG were observed in 5 cases (Della Sala et al., 1991; Faber et al., 2010; Goldberg et al., 1981, c1–c2; Goldberg & Bloom, 1990, c). Only one patient had a lesion involving both the CC and the parieto-occipital areas cortex (Murdoch et al., 2021).

Among the 9 patients without CC involvement, the majority exhibited parietal lesions (Bundick & Spinella, 2000; El Nahas et al., 2024; Martí-Fàbregas et al., 2000; Pacella et al., 2021; Panda, 2010; Pappalardo et al., 2004; Qureshi et al., 2016), either in isolation (El Nahas et al., 2024; Martí-Fàbregas et al., 2000; Qureshi et al., 2016) or combined with frontal (Bundick & Spinella, 2000; Panda, 2010) and/or temporo-occipital cortical areas (Bundick & Spinella, 2000; Pacella et al., 2021; Pappalardo et al., 2004). Only two patients had an isolated frontal lesion (Goldberg & Bloom, 1990; Wan Yusoff et al., 2021).

Considering both acute-subacute and chronic cases with reference to the awareness of ownership, it was preserved in 34 patients (81%) (Amalnath et al., 2013; Biran et al., 2006; Brainin et al., 2008; Della Sala et al., 1991; El Nahas et al., 2024; Faber et al., 2010; Goldberg et al., 1981, c1–c2; Goldberg & Bloom, 1990, c2; Hanakita & Nishi, 1991; Huang & Jia, 2013; Kischka et al., 1996; Lin et al., 2007; Ma et al., 2023, c1–c5; Mahawish, 2016; McNabb et al., 1988; Moro et al., 2015; Murdoch et al., 2021; Nishikawa et al., 2001; Nowak et al., 2014, c1–c2; Pacella et al., 2021; Paliwal et al., 2013; Park et al., 2012, c1; 2012, c2; Pouget et al., 2011; Qu et al., 2021; Qureshi et al., 2016; Tanaka et al., 1990; Wan Yusoff et al., 2021), while 8 patients (19%) exhibited impaired ownership (Bundick & Spinella, 2000; Goldberg & Bloom, 1990, c1; Lavados et al., 2002; Martí-Fàbregas et al., 2000; Panda, 2010; Pappalardo et al., 2004; Sarkar et al., 2022; Yuan et al., 2011). Within this latter group with impaired ownership, two patients had parietal or parieto-occipital lesions (Martí-Fàbregas et al., 2000; Pappalardo et al., 2004), two had fronto-parietal lesions (Bundick & Spinella, 2000; Panda, 2010), one presented with a lesion involving both the CC and the MdFG (Goldberg & Bloom, 1990, c1), and three had isolated CC lesions (Lavados et al., 2002; Sarkar et al., 2022; Yuan et al., 2011).

As regards the symptom duration, the clinical status of symptoms at the last available assessment was classified as persistent, decreased or resolved. Stroke phases were defined according to the criteria proposed by Bernhardt et al. (Bernhardt et al., 2017): in 15 cases (Brainin et al., 2008; Della Sala et al., 1991; Faber et al., 2010; Goldberg & Bloom, 1990; Hanakita & Nishi, 1991; Kischka et al., 1996; Lavados et al., 2002; McNabb et al., 1988; Moro et al., 2015; Nowak et al., 2014, c1–c2; Pacella et al., 2021; Pappalardo et al., 2004; Pouget et al., 2011; Tanaka et al., 1990), follow-up was conducted during the chronic phase (≥6 months post-onset), while in 27 cases (Amalnath et al., 2013; Biran et al., 2006; Bundick & Spinella, 2000; El Nahas et al., 2024; Goldberg et al., 1981, c1–c2; Goldberg & Bloom, 1990, c2; Huang & Jia, 2013; Lin et al., 2007; Ma et al., 2023, c1–c5; Mahawish, 2016; Martí-Fàbregas et al., 2000; Murdoch et al., 2021; Nishikawa et al., 2001; Paliwal et al., 2013; Panda, 2010; Park et al., 2012, c1; 2012, c2; Qu et al., 2021; Qureshi et al., 2016; Sarkar et al., 2022; Wan Yusoff et al., 2021; Yuan et al., 2011) assessments took place during the acute or subacute phase (<6 months). Since persistence or attenuation of symptoms cannot be regarded as definitive when measured in the acute/subacute phase, these outcomes were considered stable only when documented during the chronic phase. In contrast, complete resolution of symptoms was considered definitive regardless of timing, including when it occurred early after stroke. This methodological approach reduces temporal bias while allowing meaningful recovery trajectories to be captured.

Among the 9 patients who achieved complete resolution of symptoms in the acute/subacute phase, three presented with isolated CC lesions (Huang & Jia, 2013; Nishikawa

et al., 2001; Yuan et al., 2011), three had fronto-parietal lesions without CC involvement (Martí-Fàbregas et al., 2000; Panda, 2010; Wan Yusoff et al., 2021), and three exhibited combined CC and CG lesions (Ma et al., 2023, c1; 2023, c4; Qu et al., 2021).

For the 15 patients evaluated during the chronic phase, only two (both with preserved awareness of limb ownership) achieved complete resolution (Nowak et al., 2014, c2; Tanaka et al., 1990). One of these had a lesion involving the anterior CC, the left rostral CG, and a small portion of the right rostral SFG/MdFG (Tanaka et al., 1990), while the other showed lesions confined to the CC and CG (Nowak et al., 2014, c2).

In the remaining 13 patients, symptoms persisted or showed only partial improvement when the follow-up was performed between 6 and 24 months post stroke onset (Brainin et al., 2008; Della Sala et al., 1991; Faber et al., 2010; Goldberg & Bloom, 1990, c1; Hanakita & Nishi, 1991; Kischka et al., 1996; Lavados et al., 2002; McNabb et al., 1988; Moro et al., 2015; Nowak et al., 2014, c1; Pacella et al., 2021; Pappalardo et al., 2004; Pouget et al., 2011). Among these subjects, two had partial CC lesions, presenting with either diagonistic dyspraxia (Pouget et al., 2011) or agonistic dyspraxia (Lavados et al., 2002). One patient had isolated parieto-temporal lesions with retained limb ownership awareness (Pacella et al., 2021), while another one had isolated parieto-occipital lesions, with impaired limb ownership awareness (Pappalardo et al., 2004). The remaining nine patients exhibited persistent symptoms associated with CC lesions, in combination with frontal (Brainin et al., 2008; Della Sala et al., 1991; Faber et al., 2010; Goldberg & Bloom, 1990, c1; Hanakita & Nishi, 1991; Lavados et al., 2002; Moro et al., 2015; Nowak et al., 2014, c1; Pouget et al., 2011) or fronto-parietal damage (Kischka et al., 1996; McNabb et al., 1988).

Summarizing the results, across 42 eligible right-handed patients suffering from with aberrant hand agency, neuroimaging revealed a consistent anatomical pattern: CC involvement in four-fifths of cases, yet pure callosal injury in only one-sixth. When the CC was affected, CG co-lesions predominated, whereas CC-sparing lesions clustered in the posterior parietal region cortex, occasionally extending to frontal or temporo-occipital regions. Limb ownership was preserved in 81 % of patients; the minority with ownership loss exhibited parietal/parieto-occipital, fronto-parietal, CC and MdFG, or isolated CC lesions. Outcome analyses showed that complete recovery within the acute/subacute phase occurred chiefly in isolated CC, fronto-parietal, or CC + CG lesions, while persistent or only attenuated symptoms at 6–24 months were almost exclusively associated with combined CC and frontal or fronto-parietal damage. Collectively, these findings indicate that chronic AnHS presentations require concurrent disruption of interhemispheric CC fibres and anterior sensorimotor networks, whereas isolated CC or parietal lesions tend to spare ownership and resolve more readily.

Discussion

Our study identified two predominant lesion profiles underlying post-stroke disorders of limb agency and ownership: isolated injury to a single node (e.g. CC or cingular, SFG/MdFG or parietal cortex areas alone) often produces transient or partial symptoms (Huang & Jia, 2013; Ma et al., 2023, c1; 2023, c4; Martí-Fàbregas et al., 2000; Nishikawa et al., 2001; Nowak et al., 2014, c2; Panda, 2010; Qu et al., 2021; Tanaka et al., 1990;

Wan Yusoff et al., 2021; Yuan et al., 2011), whereas simultaneous disruption of inter-hemispheric fibers and anterior motor-intentional areas cortices is required for full-blown, persistent alien or anarchic hand phenomena (Brainin et al., 2008; Della Sala et al., 1991; Faber et al., 2010; Goldberg & Bloom, 1990, c1; Hanakita & Nishi, 1991; Kischka et al., 1996; Lavados et al., 2002; McNabb et al., 1988; Moro et al., 2015; Nowak et al., 2014, c1; Pacella et al., 2021; Pappalardo et al., 2004; Pouget et al., 2011). Two rare agency variants, diagonistic and agonistic dyspraxia, arose only after selective callosal fiber disruption (Lavados et al., 2002; Nishikawa et al., 2001; Pouget et al., 2011), showing how the precise topography of inter-hemispheric disconnection shapes behavior (e.g. Tomaiuolo et al., 2001). When lesions spared the CC but involved parietal or temporo-occipital areas cortex, the clinical picture was dominated by disturbed spatial/body awareness (neglect or hemisomatognosia) rather than persistent motor conflict (Bundick & Spinella, 2000; Martí-Fàbregas et al., 2000; Panda, 2010; Pappalardo et al., 2004). Clinically, this implies that early MRI evidence of dual-network damage (callosal with cingular and SFG/MdFG) should raise concern for a protracted course.

Regarding lesion topography and long-term outcome, fifteen patients were followed beyond six months (6–24 months) (Brainin et al., 2008; Della Sala et al., 1991; Faber et al., 2010; Goldberg & Bloom, 1990; Hanakita & Nishi, 1991; Kischka et al., 1996; Lavados et al., 2002; McNabb et al., 1988; Moro et al., 2015; Nowak et al., 2014, c1–c2; Pacella et al., 2021; Pappalardo et al., 2004; Pouget et al., 2011; Tanaka et al., 1990). Two of them achieved complete recovery despite the chronic time frame: one with a mixed lesion involving the anterior CC, left rostral CG, and a small right rostral SFG/MdFG region (Tanaka et al., 1990), and one with a lesion confined to the CC and CG (Nowak et al., 2014, c2). In the remaining 13 patients, symptoms persisted or improved only partially. Unfavorable patterns were: Partial CC lesions producing diagonistic (Pouget et al., 2011) or agonistic dyspraxia (Lavados et al., 2002), isolated parieto-temporal lesions (ownership preserved in one (Pacella et al., 2021) and lost in another (Pappalardo et al., 2004)), combined lesions of CC with frontal or fronto-parietal damage (9/13 cases) (Brainin et al., 2008; Della Sala et al., 1991; Faber et al., 2010; Goldberg & Bloom, 1990, c1; Hanakita & Nishi, 1991; Kischka et al., 1996; Lavados et al., 2002; McNabb et al., 1988; Moro et al., 2015; Nowak et al., 2014, c1; Pouget et al., 2011).

Overall, chronic AnHS was almost always linked to dual-site damage, particularly CC injury with medial frontal (CG, SFG/MdFG) and/or fronto-parietal area cortex involvement, whereas isolated CC or parietal or temporo-parietal lesions usually resolved.

Our 68-year-old patient illustrates the chronic form. Dual-time-point CT (10 days and 18 months post-stroke) showed a lesion spanning the genu to splenium of the CC and extending into the right CG and SFG. Tract-based disconnection mapping confirmed lasting disruption of the CC, superior longitudinal fasciculus I, frontal aslant tract, cingulum bundle, and extreme capsule, resulting in a persistent grasping and groping behavior at 18 months. This case indicates that chronic AnHS results not only from callosal severance but from a broader breakdown of premotor control circuits.

Our findings align with and extend the Dual Premotor System Theory (Goldberg, 1991), which posits that voluntary action depends on a balance between a medial, internally driven network (supplementary motor area and cingulate cortex) and a lateral, externally guided network (lateral premotor cortex), synchronized *via* callosal

fibers. Severing these links disrupts inter-hemispheric coordination, producing involuntary goal-directed movements; additional damage within either premotor network collapses compensatory pathways, fostering chronicity.

Early, high-resolution imaging that assesses both callosal integrity and premotor connectivity is crucial for prognosis. By integrating structural, functional, and behavioral data, AI-based lesion–symptom mapping could (i) distinguish transient from chronic phenotypes, (ii) predict specific residual deficits, and (iii) guide targeted rehabilitation strategies.

Our study provides one of the few longitudinal imaging datasets extending 18 months post-stroke and an atlas-guided synthesis of 42 cases, yet several constraints remain: rarity-driven small sample size, heterogeneous imaging protocols, limited follow-up intervals, absence of volumetric datasets (hindering fine sub-regional analyses), and lack of a formal risk-of-bias assessment, considered of limited applicability for single-case reports.

Future ultra-high-field MRI and voxel-wise symptom-mapping studies will be required to pinpoint the micro-anatomical nodes underlying agency and ownership.

Even within these constraints, converging evidence shows that combined callosal and premotor cortical injury is the principal driver of chronic AnHS, whereas isolated parietal damage, particularly with an intact CC, primarily disrupts ownership without causing persistent motor conflict.

Conclusion

Most post-stroke disorders of ownership and agency considered in this systematic review are transient when the damage is focal, on the contrary CC injury with CG, SFG/MdFG and/or fronto-parietal area cortex result in persistent syndromes. Recognizing the lesion pattern and separately assessing agency and ownership enables clinicians to better anticipate recovery trajectories and tailor conservative management strategies. These findings refine the Dual Premotor System model by demonstrating that compensatory networks fail only when both callosal and premotor areas are affected. In the future, multimodal predictive models, integrating neuroimaging, behavioral data, and rehabilitation parameters, may represent the next step toward personalized, multidisciplinary care for this rare but disabling condition.

Ethics statements

Ethics approval was granted by the National Research Ethics Committee of Messina (notified 05.15.2024). Written informed consent was obtained from the patient.

Disclosure statement

No potential conflict of interest was reported by the author(s).

Funding

This research was supported by PRIN 2022 [CUP J53D23008320006].

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