Transient Anosognosia for Episodic Hemiparesis: A Singular Manifestation of TIAs and Epileptic Seizures

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ABSTRACT: Anosognosia is a well-known manifestation of non-dominant parietal lobe lesions and typically lasts a few days. That anosognosia may last only a few minutes to a few hours, as observed in six patients, has not been reported. In five patients, transient anosognosia for equally brief left-sided hemiparesis was a manifestation of transient ischemic attacks (TIAs). In the sixth patient, anosognosia for both a left-sided motor seizure and a subsequent brief left hemiparesis could best be explained by an epileptic ictal and post-ictal transient dysfunction of the non-dominant parietotemporal cortex. Prompt recognition of transient anosognosia, whether ischemic or epileptic, is mandatory for proper diagnosis and for rapid initiation of specific therapy.

RÉSUMÉ: Anosognosie transitoire: une manifestation inedite de l'ischémie cérébrale transitoire et de l'épilepsie L'anosognosie, manifestation caractéristique des lésions pariétales de l'hémisphère mineur, est généralement durable. Qu'elle puisse, comme chez six de nos malades, ne durer que quelques minutes ou quelques heures, relève de l'inédit; l'anosognosie transitoire mérite néanmoins d'être traitée avec toute la diligence que commande la prévention de complications redoutables. Car, chez cinq de nos malades, des épisodes d'hémiparésie gauche et d'anosognosie sont à rapprocher de l'ischémie cérébrale transitoire. Chez le sixième malade, des manifestations identiques, néanmoins précédées de clonies hémicorporelles gauches également ignorées malgré un état de conscience par ailleurs préservé, tiennent d'une dysfonction épileptique critique et post-critique de l'hémisphère mineur; à pareille dysfonction et à elle seule, convient l'appellation "épilepsie anosognosique" de Schwab. A côté de l'ischémie transitoire et de l'épilepsie partielle identifiées chez ces six malades, d'autres conditions incluant la migraine et les "tumor attacks" de Ross pourraient trouver dans l'ictus anosognosique une autre forme d'expression clinique plausible.

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Anosognosia is a well-known phenomenon in which a patient fails to recognize his own functional deficit. Anosognosia for hemiplegia, the most common form, is often associated with an acute and extensive ischemic infarct in the territory of the non-dominant middle cerebral artery. Explicit denial of the hemiplegia typically lasts a few days and usually subsides long before significant recovery from the associated motor deficit.[1]

That anosognosia for hemiparesis may occur as a transient phenomenon of a few minutes to a few hours duration, as observed in six patients, has not previously been reported. That such transient anosognosia for equally brief hemiparesis may represent the cardinal manifestation either of a transient ischemic attack (TIA) or of an epileptic seizure deserves acknowledgement, since the resulting potentially baffling behaviour may cause delay in proper referral, early diagnosis and institution of appropriate therapy.

Case Reports

Patient 1

This 68 year old right-handed man had always enjoyed good health and was active in his retirement. One afternoon in October 1987, he came out from his room unaware of being improperly dressed with his buttons and belt undone and his shirt-tail hanging out. He nevertheless declared himself ready to attend a planned choir practice at church. Upon inquiry, he denied being sloppily dressed despite confrontation before a mirror. Intrigued at first, his wife became alarmed after noticing, a few minutes later, an obvious dysarthria and left-sided weakness. Pointing out these deficits to him met emphatic denial once more. The only complaint he readily acknowledged was drooling at the mouth while drinking coffee. Despite insistence from relatives, he initially refused transfer to the local hospital arguing that he felt quite well. Upon arrival at the hospital, he had completely recovered from this initial one-hour episode. An identical 30 minute episode was later witnessed the same evening in our emergency room. Examination towards the end of this second attack disclosed reduced left-sided proprioception and left extinction to bilateral simultaneous tactile stimulation. A medium grade right carotid bruit was heard.

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An emergency head computed tomography scan was normal and treatment with heparin was immediately started. Angiography revealed bilateral internal carotid fibromuscular dysplasia. The patient could hardly be convinced of the necessity for further observation and he repeatedly requested dismissal despite cogent explanations. He argued that drooling at the mouth, although admittedly intriguing, did not justify admission and treatment. He remained asymptomatic under anticoagulants and was discharged seven days after admission.

Patient 2

This 55 year old woman was quietly chatting when she suddenly developed a left-sided hemiparesis. She repeatedly refused to acknowledge the motor deficit which was obvious to her children. She insisted on being perfectly well and objected to the transfer by ambulance. She completely recovered during the transfer. While being examined in the emergency room, a second episode of left hemiparesis occurred which she again adamantly denied. This second attack lasted two hours. Later, upon direct questioning, she clearly recalled that day's events but again denied the paresis. She did, however, remember having noticed left sided numbness. Cerebral computed tomography, electroencephalography and right carotid arteriography were normal. Platelet inhibiting agents were begun; she has remained asymptomatic.

Patient 3

As witnessed by his daughter, this 55 year old man denied a left hemiparesis of three hours' duration, during which he complained of a visual disturbance suggestive of a left homonymous hemianopsia. Examination within twenty-four hours was normal, but an electroencephalogram showed right temporoparietal intermittent theta activity. Three days later, a control EEG recording was normal. Head computed tomography was not obtained. A platelet inhibiting drug was begun; he has remained asymptomatic.

Patient 4

This 74 year old lady presented two brief episodes of left hemiparesis evident to her children, during which she persistently refused to acknowledge the motor deficit. On the following day, neurological examination was normal. Head computed tomography, EEG, and an echocardiogram were normal. Carotid arteriography was not obtained. Platelet inhibiting agents were begun; she has remained asymptomatic.

Patient 5

This 78 year old man was admitted to our institution twenty-four hours after relatives had witnessed three brief episodes of left hemiparesis, all of which were denied by the patient during the attacks and thereafter. A fourth attack was witnessed during the initial examination. Explicit denial of left hemiparesis was striking, and according to relatives present, identical to that observed in the earlier three episodes. However, anosognosia in this fourth attack persisted three days. Cerebral CT scanning was consistent with a recent right parieto-occipital infarct. Anticoagulants were begun on the day of admission and continued after discharge. At a six month follow-up visit, he has remained asymptomatic.

Patient 6

This 70 year old man was initially referred in 1981 for left hemiplegia and anosognosia, both of which recovered completely within weeks. Investigation disclosed a right posterior parietal hematoma which was drained. Six months later, he developed left sided motor seizures. In 1983, two episodes of transient (10 and 25 minutes) anosognosia for left hemiparesis were witnessed and were thought to represent a postictal (Todd) phenomenon even though their very onset was not observed.

In 1987, however, a house officer witnessed an entire one-hour episode of transient anosognosia for left hemiparesis which clearly followed a left sided clonic seizure. In addition, during the seizure, anosognosia for the motor manifestations could be demonstrated; he was otherwise lucid and well oriented. The interictal EEG showed spikes in the right temporo-parietal region. Treatment on both occasions consisted of readjustment of the anticonvulsive therapy.

Discussion

In the first 5 patients, one or more episodes of transient anosognosia for equally transient left hemiparesis were witnessed. Clinical profile, investigation and clinical course are consistent with TIAs in the territory of the right middle cerebral artery. In patient 5, a subsequent longer lasting ischemic event accounted for the left hemiparesis and anosognosia of the three days' duration. It therefore appears that anosognosia may be encountered within the entire range of severity of cerebrovascular ischemia.

Anosognosia for left hemiparesis does not necessarily preclude recognition of other concomitant neurological symptoms. Patient 2 complained of left-sided paresthesia and patient 3 of a left visual field defect while both ignored the associated but obvious left hemiparesis. The occurrence of such a dissociation strengthens the hypothesis that anosognosia is not merely a failure to recognize disease, as etymologically implied, but an imperception of selective neurological deficits.

Accordingly, Alajouanine and Lhermitte[2] have proposed the term "anosognosies electives de fonction" (selective anosognosias) with the implication that anosognosia should always be qualified in relation to the non-perceived neurological deficit, such as "anosognosia for hemiplegia" or "anosognosia for cortical blindness".

The pathophysiology of anosognosia remains enigmatic. A parallel may be drawn with sensory neglect, a related phenomenon, in which dysfunction of a hypothetical corticolimbic reticular formation loop has been proposed as the pathophysio-

Table 1: Transient Anosognosia for Left Hemiparesis in Six Patients

Patient No/Age/Sex	Number of Episodes	Duration of Episodes (minutes)	Associated Features	Presumed Underlying Mechanism
1/68/M	2	30, 60	—	TIA à
2/55/F	2	30, 120	Left-sided numbness	TIA
3/55/M	1	180	Left homonymous hemianopsia	TIA
4/74/F	2	10, 20	—	TIA
5/78/M	3*	20, 30, 30	—	TIA
6/70/M	2 (1983)	25, 10	—	Ictal and post-ictal
	1 (1987)	60	Left-sided motor seizures	non-dominant parietal cortex dysfunction

à TIA indicates transient ischemic attack.

* A cerebral infarct followed, with anosognosia lasting 72 hours.

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logical basis.[6] Transient anosognosia for hemiparesis may correspond to a time-limited dysfunction of a modality-specific non-dominant parietotemporal association area within this loop combined with additional impairment of the precentral cortex responsible for the motor deficit.

Although anosognosia for hemiparesis was also a dominant feature of the attacks of patient 6, the preceding partial motor seizure suggests a different underlying pathophysiological mechanism. The observed anosognosia in this patient for both the initial clonic movements and subsequent left hemiparesis can best be explained by an epileptic ictal and post-ictal transient dysfunction of the minor hemisphere.

"Anosognosic seizure", a term coined by Schwab[3] in 1959, is an appropriate designation to characterize a seizure such as the one witnessed in the sixth patient, during which full consciousness is preserved despite concurrent agnosia of motor manifestations. However, the expression is a misnomer when used to describe transient global alteration in consciousness as in Schwab's report of 13 patients with typical absence and complex partial seizures.

Although anosognosia as such has never been reported to occur as a brief phenomenon, Heilman and Howell[4] in 1980, described an epileptic patient in whom features of the neglect syndrome could be demonstrated only during his seizures or in the immediate post-ictal period. In addition, although not commented upon, the authors observed that the patient denied the ictal motor manifestations despite preserved consciousness. Therefore, their patient not only presented seizure-induced ictal and post-ictal neglect but also seizure-induced ictal anosognosia.

Conceivably, transient anosognosia could well be encountered with episodic cerebral dysfunction in conditions other than TIAs and epilepsy, such as migraine or Ross's tumour attacks.[5]

In conclusion, although anosognosia is a well-known phenomenon of classical neurology, transient anosognosia, epileptic or not, has never been properly singled out. Early recognition of transient anosognosia, despite the patient's potentially misleading behaviour, is mandatory for proper diagnosis of the underlying condition and for prompt initiation of specific therapy. Widespread awareness of this phenomenon among physicians is paramount if potentially longer lasting crippling deficits are to be prevented.

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