Belief and awareness: reflections on a case of persistent anosognosia

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Abstract

Persisting anosognosia after acute lesions is relatively rare, and no case studies to date have reported functional scanning investigation of this disorder. This is a case report of an 85-year-old right-handed Scottish woman, EN, who showed persistent anosognosia for hemiplegia following a haemorrhagic stroke. Extensive damage in the right hemisphere caused left upper and lower limb flaccid hemiplegia and severe left-sided neglect. Lack of awareness for her deficits was still present 2 years after the stroke, when neurological, neuropsychological, and SPECT examinations were performed. Testing revealed severe left unilateral neglect and poor performance on verbal fluency tasks. EN had age normal memory performance, and her object recognition and praxic abilities were preserved. She showed no global reasoning or language problems apart from her abnormal beliefs. SPECT scan showed marked hypoperfusion in the right parietotemporal cortex and this extended to the associative cortex in the right frontotemporal regions.

The persistence of anosognosia in this patient cannot be explained by memory impairments or global cognitive decline. A possible account might be that alteration in awareness was maintained by contingent right frontal and/or parietal dysfunction causing a suspension or change in the ability to monitor and check the ‘real’ and especially to assess the veracity of mental contents.

Keywords: Anosognosia; Hemiplegia; Neglect; SPECT; Persistent; Chronic; Reality monitoring; Delusions

1. Introduction

A failure of awareness of deficit or anosognosia is frequently reported following right hemisphere damage and can also be seen in patients who have global cognitive impairment from degenerative causes like Alzheimer’s disease (AD). Lack of awareness of functional decline in AD, when this symptom makes its appearance, is persistent and its severity increases with disease progression. When anosognosia appears following an acute lesion it is usually shortlived, in most cases involves unawareness of hemiplegia, and when the acute or post-acute state is over patients usually gain awareness of their condition. Persisting anosognosia after acute lesions is less frequent, although a few cases have been described (e.g. Berti, Ladavas, & Della Corte, 1996; Berti, Ladavas, Straciari, Giannarelli, & Osuola, 1998; Cocchini, Beschin, & Della Sala, 2002; Marcel, Tegné, & Nimmo-Smith, in press). In the context of global cognitive impairments anosognosia can be seen as an inevitable consequence of multiple and increasingly severe cognitive failures, but the need for a theoretical explanation seems both more necessary and potentially achievable where this symptom appears in the absence of global deficits. Several such theoretical accounts have been put forward. These can be fairly divided into psychodynamic or motivational theories (Weinstein & Kahn, 1955) and cognitive theories (Bisiach, Meregalli, & Berti, 1990; McGlynn & Schacter, 1989). Pure psychodynamic theories, involving for example denial or depression, are challenged to some degree as comprehensive explanations of disturbances in awareness by the more frequent link between anosognosia and right hemisphere dysfunction. It is also perhaps hard to understand how mechanisms of denial might be specific to individual aspects of awareness when these disturbances are dissociated in individual patients (Berti et al., 1996). Motivational explanations are also less convincing given the usual parallel resolution of the symptom and the acute phase of brain damage, although it is conceivable that certain forms of brain damage might allow the appearance of otherwise suppressed defence mechanisms.

The existing cognitive theories are numerous and must attempt to account for the various syndromes of
anosognosia, and the different observed patterns of neuro-
ological and cognitive disturbance. No theory appears to
account for the phenomenon in all its forms, its association
with the right hemisphere, and the appearance of anosog-
nosia as both a transient and occasionally as a persistent
symptom.

This paper reports a case of persisting anosognosia for
hemiplegia and left-sided neglect in the absence of global
mental deterioration. It is suggested that the associated
phenomena of false beliefs and misidentifications may
be integral components of persistent anosognosia in this
case. Similar mechanisms may be of importance, albeit
transiently, in association with more acute disturbances of
awareness. It is important for our argument, and not avail-
able in other published material, that functional as well as
structural imaging is available for analysis in this case.

2. Case report

EN1 is an 85-year-old right-handed Scottish lady with a
history of hypertension, atrial fibrillation and asthma. She
was taking aspirin. In February and March 1999 she had
two transient ischemic attacks. In the first, she reported
mild and shortlived speech disturbance, while in the sec-
ond, she had left upper limb paraesthesia which resolved
spontaneously after 1 h. During the second episode, her
case notes record that she felt that her arm was weakened
and difficult to move. By the time she was neurologically
examined, however, there were no focal signs. Warfarin
was prescribed after the first episode. In June 2000 she was
admitted to general medical care following a haemorrhagic
stroke, with extensive damage in the right hemisphere. The
MRI images, 3 days after the stroke, showed a haemor-
rhage measuring 2 cm × 3 cm × 3 cm involving the right
posterior temporal and parietal regions. The lesion was
surrounded by a rim of cerebral oedema, and there was
some effacement of the posterior horn of the right lateral
ventricle. Routine neurological examination indicated a
left-sided flaccid paralysis of the upper and lower limb.
There was a mild paresis of the left lower facial muscles,
which quickly resolved. She also had some difficulty swal-
lowing with impaired tongue movements. She was mildly
dysarthric with severe left visuospatial neglect and profound
anosognosia for her hemiplegia and unilateral neglect. No
visual field defect was elicited. No specialist neurological
or neuropsychological assessments were carried out at this
time.

The patient had physical rehabilitation but remained un-
able to walk and was mobilised only with a wheelchair. She
was discharged after 4 weeks to permanent nursing home
care.

4 Patient’s initials have been changed to preserve anonymity and both
the patient and her relatives gave their consent to the present report.

2.1. Neuropsychiatric assessment

In November 2001, 17 months after the stroke, she was
referred for psychiatric assessment because of behavioural
problems. The nursing home staff reported her spontaneous
claims following admission that she was able to stand and
walk, and to look after herself. She accused staff of being
cruel, torturing and poisoning her and leaving her out in
the rain. At times she had requested that her mother be infor-
med of her whereabouts. She had discussed sexual matters with
her son in an inappropriate and disinhibited way.

EN presented as an alert and emotionally serene lady who
smiled readily and showed no language disorder. There was
no evidence of subacute confusion or alteration in the level
of consciousness at any point during the examinations. She
claimed that she lived in a new house in the city, although
she was presently visiting another city where her sister lived.
She had frequent visitors at home and was perfectly able to
travel by train between her present residence and that of her
sister. She continued to hold the belief that her left side was
fully functional and that she was physically independent.
At different times the staff reported she had said that either
her left hand or both of her hands had disappeared. Neu-
ropsychiatric inventory was completed and the retrospective
assessment showed that she had frequent delusions, persist-
ent apathy interspersed with frequent episodes of aggres-
sive behaviour, all of moderate severity. There was evidence
of left-sided neglect and inattention. She was referred for
functional and structural brain scan and neuropsychological
assessment, because of concerns about more global cogni-
tive decline due to progressive degenerative brain disease
together with a requirement to counsel and fully inform her
family and nursing staff about her symptoms. Risperidone
0.5 mg daily was prescribed with benefit for the hostility
and aggressive behaviours associated with paranoid beliefs.

2.2. Neuropsychological assessment

EN was tested in December 2001 and August 2002. On
both occasions comprehensive testing as detailed below was
carried out, including assessment of the persistent anosog-
nosia. There were only minor differences in psychomet-
ric test scores and no changes in her behaviour or mental
state between examinations. Mini Mental State Examination
(Folstein, Folstein, & McHugh, 1975) showed that EN was
partially oriented in time and place. She knew the month
and the year, and the city and country where she lived.
She could readily understand and follow test instructions
and was very cooperative. She had no oral or written compre-
hension deficits nor grammatical comprehension deficits nor
expressive language problems. Her spontaneous speech was
well organised with good intonational contour, grammatic-
al and logical structure and rich in information content. On
confrontation naming she showed no naming difficulties, al-
though on two occasions she produced visual paraphasias
(e.g. thermometer → pen, compasses → stepladder). There

1 EN
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