Transient alien hand syndrome, in a chronic depressive patient with recent Anterior Corpus Callosum Infarct.

Author: NATARAJAN
Department of Psychiatry, STANLEY MEDICAL COLLEGE AND HOSPITAL

Abstract:
Introduction The term alien hand refers to a variety of clinical conditions characterized by the uncontrolled behavior or the feeling of strangeness of one extremity, most commonly the left hand. A common classification distinguishes it as the anterior motor form (1) and posterior sensory form. Symptoms following callosal lesions may or may not be accompanied by hemispheric damage, especially in the frontal medial region however Della Sala et al (15) Goldberg et al (14) have argued that, frontal lesion alone is sufficient to explain alien hand syndrome and callosal lesion need not be present. Case Report 59 year old female, a known case of depression with CVA, Hypertension and Diabetes mellitus presented with irrelevant talk, altered behavior, inability to recognize familiar faces and objects and decreased self care. Examination revealed stable vitals, positive neurological findings were 7th nerve UMN palsy on left side with left sided hemiparesis, ill sustained attention, slurred speech, decline in constructional ability. Lobar function tests revealed dyscalculia, right- left disorientation, hemi neglect, finger agnosia, left limb apraxia and difficulty in bimanual coordination. Clinical diagnosis was CVA-multi infarct predominantly involving fronto parietal with left hemiparesis. Neuroimaging showed anterior corpus callosum infarct with gliosis in right parietal region confirmed the clinical features and Transient Alien hand syndrome. Neurologist concurred with same findings and patient showed improvement within few weeks of initiating treatment. Discussion and Conclusion In this patient the presence of limb apraxia and difficulty in bimanual coordinaton pointed towards the need for detailed work up and MRI revealed anterior corpus callosum infarct. Alien hand syndrome may be presenting with wide spectrum of motor and sensory deficits which may be caused by parietal, frontal or callosal lesions. This patient is a case of Transient Alien hand syndrome, so that callosal lesion need not be present. Diagonistic dyspraxia is a symptom sometimes seen in patients with callosal damage, in which there is apparent conflict between the desired action and the actually performed act. Internmanual conflict is also a symptom of the callosal syndrome, in which one hand is acting at cross purposes to the other. Cerebrovascular disease has played a major part in the understanding of callosal disconnection syndromes and enabled Liepmann and Maas to show the role of the callosal lesion in the genesis of unilateral apraxia.

Case Report:
Mrs. x, a 59 yrs old female, treated as depression at various govt hospitals by psychiatrists, for the past 15 yrs duration, referred from Stanley Medical College Medicine Opd to our Psychiatric Opd on 11.08.2012,for the complaint of altered behaviour, irrelevant talk, inability to recognize familiar faces and objects and decreased self care for the past 8 days duration, with h/o cerebrovascular accident and developed weakness of left upper
lower limb 6 yrs back and found to have diabetes mellitus and hypertension, for which she has been on regular treatment. With h/o picking up quarrels easily, decreased house hold activities, anger outbursts/irritability on & off for the past 6 months duration for which she had taken treatment from private hospital. On examination, pt was conscious, afebrile, pt dressed adequate, eye contact made and maintained. BP110/80 mmHg, Cardiovascular and Respiratory systems were normal. During examination of Nervous system-the following findings noted : pt is a right handed individual, 7th nerve UMN type palsies on the left side with other cranial nerves normal. No neck stiffness found. Slurring of speech with diminished facial expression noted and the attention was ill sustained. Pt’s comprehension, ability to name the objects and to name the colours were intact. Examination of memory showed the impairment in the recent memory. The general fund of knowledge adequate and the calculation ability was intact. Motor system-left hemiparesis with power of 2/5 in left upper limb with decreased bulk and tone. There was 3/5 power in left lower limb with hypertonia. The right upper and lower limbs had weakness with initial difficulty, exaggerated reflexes in both the sides. Gait was hemiplegic. MMSE scored 25/30.

Lobar functions revealed: Apathetic facial expression with normal release reflexes. Dyscalculia , right left disorientation were present. Hemineglect found on the draw a clock test and finger agnosia were present. There was decline in the constructional abilities. On further examination pt showed difficulty in bimanual coordination like opening and closing the fist alternatively and taking something out of a container as well as abnormalities in somesthetetic transfer like cross localising fingertip stimulation. No abnormality detected in stereognosis and graphasthesia . During examination for Apraxia, on verbal commands, pt started showing the right hand, for instructions to the left hand and she showed improvement in doing the act by repeated instructions. The pt looked as if she was not caring her left upper limb and moved the limb on repeated instructions. The pt was able to carry out these instructions correctly, while on eyes open and not able to do on closing the eyes. While examining for visual naming of fingers- patient was not able to do with left hand on closure of eyes. Mental state examination- pt was conscious, alert, cooperative, rapport established with initial difficulty. Psycho motor activity normal, talk –relevant and coherent, mood- blunted and there was no thought or perception disturbances. Insight- denied illness, graded II. In this patient all the basic blood investigations found with in normal limits except for blood sugar level of 191mg. CT Brain Imaging done 6 yrs back, showed right parietal infarct. The probable clinical diagnosis of depressive disorder with comorbid CVA-multi infarct states with diagnostic suspicion of tumours and later found to have infarction on biopsy reports. The patient may present with sensory & motor symptoms due to corpus callosal infarct. It is revealed in a previous study that clinical manifestations and lesions localized with motor symptoms due to the anterior corpus callosal infarct and sensory symptoms related to the body and posterior infarction of corpus callosum. The clinical symptoms may be left hand ideomotor apraxia, left hand agraphia, left hand tactile anomia, left alien hand syndrome (with three characteristics-inability to recognise the arm as one’s own, when held by the other arm with eyes closed, a feeling of loss of control of left arm movements and personification of left arm), left visual anomia, left auditory extraction and alexia without agraphia. There may be presence of gnosia’s of familiar faces, color, right left and finger test defects and visuospatial and visuo constructive tests, memory function disturbances and neglect may be present. Parkin(19), suggested that, normally the hemisphere ipsilateral to the intended hand is inhibited by the contralateral hemisphere via the corpus callosum. In patient with callosal damage, this inhibition is blocked, and the ipsilateral hemisphere becomes engaged in the task. In this case, pt developed CVA& left sided weakness 6 yrs back, with DM & HTN was on regular treatment , presented with symptomatology of gradual onset of inability to recognise familiar faces and objects (anosognosia). The pt was not able to carry out household works which were in need of bimanual coordination(previously patient was able to do so) and irrelevant talk for the past the 8 days duration. On examination showed dyscalculia, right-left disorientation, hemineglect and finger agnosia. Initially, with the above presentation a diagnosis of CVA- multi infarct involving frontal parietal areas was thought considering the diabetes and hypertension, but the pt presented with limb apraxia and the pt was responding frequently with right hand for instruction given to the left hand. There was difficulty in pointing to the body parts with left hand and presence of inability to recognise the left upper limb as her own, on and off. These findings- made us to go for further examination with MRI and the MRI revealed the anterior corpus callosum infarct and confirmed the above clinical features.
These findings were present only for few weeks and started subsiding after treatment. The classical Alien hand sign was not present in this case, might be masked by the presence of weakness of left upper limb due to CVA. As evidenced from previous studies, anterior corpus callosum may be present only with motor symptoms which is the presenting feature in our case. Previous studies did not establish evidences with clarity about the anterior corpus callosums because of imaging restrictions during that time. Marchetti and Della sala proposed that alien hand syndrome is caused by posterior callosal lesion which may include parietal areas. However the sensation of Alienness of left limb has also been associated with contralateral parietal damage with no callosal involvement. The inability of the patient to respond with left hand may perhaps the classical callosal disconnection syndrome but the presence of weakness of the left upper limb might have masked the feature in this case, so in this syndrome the instruction to the right hand controlled by the left hemisphere and therefore not requiring callosal transfer and these responses may be as fast as normal response. Clinically infarct of corpus callosum are frequently associated with neuropsychiatric symptoms mainly inter hemispheric disconnection syndrome. In addition there may be dyspraxia contralateral to the paired limb and Alien hand syndrome. In this case the exacerbation of neuropsychiatric manifestations might be due to the infarct of the anterior corpus callosum in addition to the already existing parietal infarct which might mimicked the psychiatric manifestation of the depression.

Conclusion:
Alien hand syndrome may be presenting with wide spectrum of motor and sensory deficits which may be caused by parietal, frontal or callosal lesions. In this case, alien handedness probably of motor involvement due to anterior corpus callosum infarct, as evidenced in the previous studies. But the regions and cerebral mechanisms involved in this process not known completely. Further research may be needed to conclude these findings.

References: