## Aphasia and Psychiatric disturbances in Cerebrovascular accident patients

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A phasia is a condition in which there is a defect or loss of the power of expression by speech, writing, or signs, or a defect or loss of the power of comprehension of spoken or written language. 21-24% of patients admitted to hospital with acute stroke are aphasic shortly after their stroke and in the long-term it is likely that 10-18% of survivors are left with significant aphasia. Among these patients, a variety of neuropsychiatric symptoms arise as a complication of stroke but their diagnosis is often delayed by the presence of speech and language problems.

Aphasia is the considered to be the most important. discnostic symptom in predicting the location of a brain lesion. Prefrontal lesions are generally supposed to cause personality and emotional disorders, the most distinctive being disinhibition. Parietal lesions cause aprada and subtle sensory disturbances. The occipital lobes are concerned with vision but evidence for location of specific types of psychovisual disturbance is often conflicting. Dementia in which there is slowing down of thought processes rather then memory loss, is considered to be subcortical. Brain stem lesions characteristically produce deep come and localised neurological signs rather than mental symptoms but can also lead to confusional states or hallucinations. Mental disturbance is much more obvious if the damage is in the right. hemisphere rather than in the left since a left-sided infarct leading to right-sided hemiparesis and loss of speech makes it difficult to detect certain deficits.

Most patients with stroke related aphasia improve mostly in the first 10 weeks and may keep improving till the 18th month. Speech and music therapy have been proven effective in recovery but improvement is dependent on a patient's decision to learn. A personalised multidisciplinary approach is essential for management of an aphasic patient and must include elements of education, support and communication skills conducted ideally by a speech and language pathologist, social worker and clinical psychologist. Involvement of family members and friends can further increase the efficacy of rehabilitation. Moreover, the use of bromocriptine as an edjuvant to traditional speech language theraples is associated with aignificant improvement in non-fluent sphasia. Also, dopamine and its agonists not only improve the dopaminergic atimulation dependent language functions (i.e. verbal latency and reading comprehension), but they also seem to improve the mood of patients. Stimulants such as amphetamines and amphatamine salts might also help recovery after stroke.

Aphasia and depression commonly coexist and develop as a consequence of stroke; moreover, having aphasia increases the risk of developing depression. Other neuropsychiatric conditions (e.g. dementia, andety disorders, and psychotic disorders) and symptomatic correlates (e.g. apathy or fatigue), may further obscure the diagnosis and the care of stroke patients. These conditions arise, in part, from the size and location of the lesion. There is evidence that depression is caused by both biological factors provoked by brain injury, associated with left anterior and basel ganglis

lesions and lesions close to the frontal pole, and a secondary psychological response to the physical, cognitive, and social Impairments produced by the stroke, 30-50% of stroke survivors suffer post-stroke depression with the same signs. and symptoms as minor and major depressive disorders, characterized by lethargy, irritability, sleep disturbances, lowered self esteem and withdrawal. Depression can reduce motivation and worsen outcome. Initiation of somatic therapies such as antidepressants, stimulants or electroconvulsive therapy can contribute to symptomatic relief and help to ensure sufficient co-operation of the patient. Controlled studies have demonstrated that various classes of antidepressants are effective in treating post-stroke depression. Behavioural therapy such as cognitive behaviour therapy or other forms of talk therapy, especially interpersonal therapy can also help.

The discovery of specific neurotransmitter-producing areas and distribution pathways has suggested that a strategically situated infarct might produce a specific deficiency of that neurotransmitter. This might be a way in which stroke leads to depression. In brain injury, an excitotoxic effect takes place at the NMDA receptors resulting in an excessive inflow of calcium into the receptor nerve cell and a sequence of chemical changes killing the cell. One approach to treating acute stroke has therefore been to use drugs that counteract glutamic acid, and drugs that block NMDA receptors have been used in atroke treatment although they might themselves cause mental disturbances.

There is also a direct effect of stroke in producing fatigue. Post-stroke emotional problems including enviety, penic attacks, flat affect, mania, spathy and psychosis can result from direct damage to emotional centree in the brain or from frustration and difficulty adapting to new limitations. Delusions are more often associated with lesions in the right hemisphere, posterior areas and left temporal lobe. Anxiety and nervousness are more frequent in the first year after a atroke but thereafter, tend to improve. Treatments for endety Include psychotherapy and hypnotic-arcidolytic medication like benzodlazephea which might cause drowsiness as they act at the GABA-A receptor. Buspirone, by altering serotonergic transmission can reduce aredety without causing drowsiness. Antipaychotic medication is useful but very paranoid patients might refuse it and side-effects of drowsiness and parkinsonism might cause major difficulties in post-stroke patienta.

Studies show that communication problems of cerebrovascular accident patients due to apheala affect the quality of life not only of patients but also of their cerers. Failure to rehabilitate properly might be due to increased apathy after a stroke. In fact, 20% of stroke patients develop apathy and lack the motivation to return to mobility. Young patients are often intolerant of rehabilitation procedures that they cannot perceive as immediately leading to desired objectives. In conclusion patients' motivation and ceregivers' patience are crucial in a quick and effective rehabilitation process.

Bibliography
1. Clean AJ, Hearing S, Allen K et al. Depression after stroke and betth iconfort: a systematic review. Lancet 2000; 368(#22-0):122-6. 2. Buildin SS, Civid LS, Stern TA. Aphaetic Associated Disturbances in Affect, Behavior, and Cognition in the Setting of Speech and Language Difficulties. Psychosometics 2007; 48:258-64. 8. Thing VMC, Chen VMC, Lu JY et al. Microbiects and post-stroke emotional liability. Journal of Neurology Alexansurpery and Psychiatry 2008; 80:1082-8. 4. Microbiect and consequences of post-stroke depression. Curr Opin Neurology Psychiatry 2008; 16:86-9. 6. Tenorit LNRI, Prégues R, Lubie M et al. Importance of setroicion and feligianithment domains for the degracies of major depressive episode effer abolies a four months prospective et al. Psychiatry 1996; 49:11-9.

Psychiatry 1996; 49:11-9.