Akinetic Mutism

Case reports and aetiological considerations

Akinetic mutism (AM) is defined as a form of stupor, characterised by severe apathy. In AM the patient is alert, conscious of surroundings and able to see and hear, but unable to move (akinetic) and unable to communicate (mutism). Minimal motor responses to painful stimuli are typically preserved.[1-3]

Case Reports

Case 1: An 18-year-old female presented following carbon monoxide intoxication. Clinically she was akinetic and mute. A positron emission tomography (PET) scan showed markedly decreased cerebral metabolism in the frontal region.

Case 2: This 51-year-old male farmer was referred to an emergency department with sudden onset of stupor while working. On examination he was awake, motionless and mute. Computed Tomography (CT) and Magnetic Resonance Imaging (MRI) scans of the brain revealed a unilaterally occluded anterior cerebral artery with consequent globus pallidus - cingulate gyrus disconnection.

Discussion

Contemporary neurology and psychiatry textbooks fail to clearly and accurately delineate the localisation and causes of AM. For instance, Adams et al. argue that "The localization … is quite imprecise. It has been attributed to bilateral lesions in the ventromedial frontal region or fronto-diencephalic connections… In general, we agree that the greatest cognitive intellectual deficits relate to lesions in the dorsolateral parts of the prefrontal lobes and the personality, mood and behavioural changes to lesions of the medial orbital parts, although the two types of disorder often merge with one another".[4]

Furthermore, also in other current sources like journals and web sites, no certain factor(s), is (are) specified. Some of the described cases lack a diagnosis and clinical signs are not discussed with respect to mechanisms and connections. Cairns and colleagues, more than 60 years ago, described originally a patient who appeared to be awake and unresponsive; following each of several evacuations of a third ventricular cyst, the patient would become aware and responsive.[5]

In our patients, frontal cortical - subcortical disconnection was the responsible cause for the AM. Other factors or causes quoted in the literature include subarachnoid haemorrhage, cerebral tumours, viral diseases, infectious encephalitis, and intracranial high pressure. These factors, although considered as "causes" of AM in the textbooks, impact mainly on the cerebral cortex and not on the cortical- subcortical connecting pathways, and can thus not justify the severe executive dysfunction seen in AM. The cause of the clinical signs in AM is a lowered or lost level of motivation, without interfering with the development of any emotion (e.g. fear, anger, sadness, happiness). In conclusion, among the above diseases or syndromes, only those that can result in frontal cortical- subcortical disconnection, can produce AM.

References

1. *Kaplan & Sadock's Comprehensive Textbook of Psychiatry, Eighth Ed.; p.846; 2005; Lippincott Williams & Wilkins.*
2. *Frank J, Ayd J. Lexicon of Psychiatry, Neurology, and the Neurosciences; p.434; 1995; Williams & Wilkins.*
3. *Bradley W.G,,Daroff R.B, Fenichel G.M, Jankovic J. Neurology in Clinical Practice, Fourth Ed.; p.67; 2004; Butterworth, Heinemann.*
4. *Adams R.D, Victor M, Ropper A.H. Principles of Neurology, Sixth. Ed.; pp.347-348, 447; 1997; Mc Graw-Hill.*
5. *Cairns H, Oldfield R.C, Pennybacker J.B. et al. Akinetic mutism with an epidermoid cyst of the third ventricle. Brain. 1941; 64: 273.*

Z Ashtari
Department of Psychiatry, University of Social Welfare & Rehabilitation (USWR), Koodakyar Bld.,Eveen,Tehran,I.R.Iran
email: zabih_ashtari@yahoo.com