LETTER TO THE EDITOR

OVERUSE OF COMMUNICATION TECHNOLOGY, A POTENTIAL RISK FACTOR FOR GENERALIZED ANXIETY DIORDER

AAMIR ABBAS
Marie Stopes Society, Pakistan

Anxiety is a physiological response to daily life events. Though some level of anxiety is good but if it is disproportionate to the source of worry, then it is classified as Generalized Anxiety Diorder (GAD). GAD is an unconstructive consequence of different types of repetitive thoughts (RT) – like preservative cognition, cognitive and emotional processing. RTs are defined as the process of thinking attentively and repetitively about one's own self and one's world. If this pattern of thinking gets out of control then it results in a number of unconstructive consequences, GAD being one of them.

GAD is characterized by a persistent, pervasive and uncontrollable state of worry which is disproportionate to its source and lasts for at least six months.[1] It is also associated with decreased functional ability in daily routine. The clinical presentation can be diverse. The most common reasons for help seeking are complains of muscle tension, restlessness, irritability, fatigability, dyspepsia and sleep disturbance.[2]

The prevalence of GAD is reported to be around 1.7% to 7.0% in different countries.[3-6] In Europe and United States, the life time risk of GAD was estimated to be 3.9% in women and 1.7% in men.[3] A Community based prevalence study from United Kingdom reports it to be around 3%.[4] A population based study from Asia (Hong Kong) reports the 12-month prevalence of GAD to be 3.4–4.0%.[6] There is no nationally representative survey on GAD from Pakistan. However a center-based cross-sectional survey from Karachi reports the prevalence of pathological anxiety to be 28%.[7]

Some studies in the recent years have established a temporal relationship between different psychiatric morbidities and over use of different types of communication technology. This include excessive mobile use and internet surfing. A cohort study carried out in Sweden reports an association between high mobile usage and psychiatric morbidities.[8] The undisciplined pattern of mobile phone use puts lot of stress on the users. Repeated external stimulation results in increased mental fatigue and functional compromise. Conditions like sleep disturbance, depressed mood and stress predispose individuals to the GAD.[3]

More research needs to be done to explore the relationship between overuse of communication technology and GAD. The prevalence of newly classified disorders like mobile phone addiction and internet addiction needs to be found out. In addition, qualitative as well as quantitative studies are needed to collect evidence related to the extent and severity of internet and mobile addiction. It is also high time to think of some interventions to manage disorders like internet addiction and problematic mobile phone use.

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GENETICS OF MENTAL DISORDERS: BENCH TO BEDSIDE APPLICATION OF RESEARCH

HIRA WASEEM
Aga Khan University

The future of mental health research lies in genetics.[1] A century of research in to the etiology of mental disorders has failed to elucidate the genetics of major mental disorders. Conditions like schizophrenia and bipolar affective disorder have remained an enigma.[2] The absence of a classical Mendelian model of genetic risk transmission for mental disorders was noted very early.[4] It was conjectured that mental disorders have polygenic etiology, as evidenced by numerous family studies, undertaken at the turn of the century.

Family studies are less laborious to carry out but their interpretations are limited. In general, closer the relationship the greater the shared culture and environment; first-degree relatives (i.e., parents, siblings and offspring) share an average of 50% of their genetic material, and second degree relatives share approximately 25%. Studies carried out on twin registry have given estimates on the concordance and discordance of schizophrenia in monozygotic (MZ) and dizygotic twins (DZ); approximately pair-wise concordance rate of MZ twins is 50% and DZ twains is 10%.[5] In other studies concordance rates in MZ pairs have varied considerably but they appear to be higher than the concordance rates in DZ pairs. Gender-difference studies in the age at onset of schizophrenia have highlighted some important familial morbidity patterns.[6,7]

Attention has been focused recently towards association studies which focus on single nucleotide polymorphisms (SNPs) within possible 'candidate genes'. Few genes replicated in multiple population models and having supportive neurobiological data for schizophrenia are: Neuregulin 1(NRG-1) located on chromosome 8p12-p23 which is involved in the NMDA pathway. The DTNBP1 (Dystrobrevin binding protein 1) located on chromosome 6p22.3 which actively plays a role in synaptic vesicle trafficking and neurotransmitter release and may also be required for normal dopamine homeostasis. Gene located (G72) on chromosomal region 13q33, which is associated with the modulation of prefrontal cortex and hippocampus. The COMT (catechol-O-methyltransferase),which is located on chromosome 22 between positions 11.21-to-11.23 and provides instructions for production of an enzyme called catechol-O-methyltransferase by the nerve cells in the brain. Another implicated gene is 'Disrupted in Schizophrenia 1' (DISC1), loci on chromosome 1q42, producing a protein that is coupled with the synaptic function and synaptic plasticity of the brain which underlies the processes of learning and memory. The KCNH2 gene sequence on chromosome 7q coding for a potassium channels is also known to contribute to the etiology. It is highly expressed in the prefrontal cortex and hippocampus and could be implicated in the neuropathology of schizophrenia.[8, 9, 10, 11]

Researchers believe that the illness is caused by multiple genes acting together or many single genes acting separately in to heterogeneous pattern. The emerging evidence also points towards the role of epigenetic factors contributing to the risk of psychosis. Researchers have discovered that human DNA is coated with a second code, transferred during meiotic cell division, with variable stability which regulates gene expression but is not based on DNA sequence rather it is based on factors which cause heritable, potentially reversible changes in the DNA or chromatin structure. These epigenetic factors could be influenced by environmental risk factors like exposure to toxins, chemicals and behavioral patterns like parenting, stressful life circumstances.[12] In short we have come full circle, albeit with more insight, on the debate on the etiology of major psychosis. This calls for greater focus and collaboration on part of the researchers with bench-to-bedside application looking to unravel the scientific mystery of disease.

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