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Schizophrenia and Autoimmunity; How hot the link is?

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Schizophrenia, one of the most debilitating and baffling mental illnesses, defines a group of disorders that cause distorted thought and perception. Thoughts can be scrambled or jump from subject to subject. Perception can be distorted beyond reality, causing people to hear or see things that are not there. The etiology of schizophrenia is unknown and the pathophysiology is complex, most of the patients need treatment and care for the rest of their lives (Umadevi and Chinnasamy 2008). People with schizophrenia go through periods of getting better and worse – remission and relapse. They can go for long periods of time without any symptoms, but because schizophrenia is often a chronic illness it requires ongoing medical attention, like hypertension and diabetes (Homel et al. 2002).

The substantial burden of disease is a reflection of two features of schizophrenia: (a) the disorder usually has its onset in early adulthood, and (b) despite optimal treatment, approximately two-thirds of affected individuals have persisting or fluctuating symptoms (Bhugra 2005). The symptoms of schizophrenia fall into three broad categories: Positive, negative and cognitive symptoms (Frances 2006).

Schizophrenia is a multifactor disorder of mind with a constant prevalence of 1-2% in the population. It causes not only significant physical morbidity and social incompatibility to the patients, but also invites major economic hardship for its lengthy diagnostic procedure, devastating course, frequent treatment failures and very difficult rehabilitation measures (Bhugra 2005). Such a debilitating picture of schizophrenia has made it an enticing research topic in psychiatry. The specific cause of schizophrenia is still unknown, but research has shown that the brains of people with schizophrenia are different from the brains of people without the illness (KenDuckworth 2007). Like many other medical illnesses such as cancer or diabetes, schizophrenia seems to be caused by a combination of problems including various social, psychological, developmental, environmental, anatomic, genetic, biochemical and other factors (Herken et al. 2001).

Despite advances in neurotransmitter identification and the development of drugs targeting these transmitters, total remission of the disease is not always achieved and there is a frustration among clinicians over the past few decades. Potential etiologies other than neurotransmitter dysfunction merit consideration (Umadevi and Chinnaswamy 2008). One intriguing concept is the possible contribution of autoimmunity in patients with the disease. This breakdown of self-tolerance has been implicated in patients with other chronic diseases, such as type 1 diabetes mellitus and myasthenia gravis. The literature on autoimmunity as a possible mechanism in the pathogenesis of schizophrenia can be incompatible, but there is a substantial amount of circumstantial, although not conclusive, evidence of immune dysfunction in patients with schizophrenia (William et al. 2006).

Autoimmune diseases arise from an overactive immune response of the body against substances and tissues normally present in the body. In other words, the body actually attacks its own cells. The immune system mistakes some part of the body as a pathogen and attacks it (Elaine 2007). This may be restricted to certain organs (e.g. in chagas disease) or involve a particular tissue in different places (e.g. Goodpasture's disease which may affect the basement membrane in both the lung and the kidney). The treatment of autoimmune diseases is typically with immunosuppression—medication which decreases the immune response (Schizoprenia and Autoimmunity 1993).

During the last 20 years considerable data have accumulated indicating that schizophrenia may have an autoimmune immunology. Patients with schizophrenia may have autoantibodies; in addition, cellular changes may occur that are compatible with autoimmunity, e.g., an increased number of CD5+ cells. If, indeed, schizophrenia does have an autoimmune pathogenesis, we should consider an alternative therapy based on immune system-manipulating drugs in addition to antipsychotic compounds (Amital and Shoenfeld 1993). The pathophysiology of psychotic and other symptoms in schizophrenia remains a mystery despite decades of research. Even though it has been suspected for many years that autoimmune mechanisms may play a role in the pathophysiology of schizophrenia, firm evidence for this hypothesis has been lacking sofar (Ganguli et al. 1993)

In the current study, A total of 141 schizophrenic patients of age group 18-65 years of both sexes from good socio-economic background were selected from Udhayam Mananala kaapagam, a mental Health care center, Coimbatore, Tamilnadu, India. They all met DSM-IV (Diagnostic and Statistical Manual of Mental Disorders-IV) criteria (American Psychiatric Association, 2000) for schizophrenia. Informed and written consent was obtained from all subjects prior to examination. Patients with a history of drug abuse or dependence, serious medical conditions, severe head injury or seizure disorders were excluded from the study. With the help of team of psychologists, the participants were interviewed and information regarding their age, family background, family medical history and economic status were collected. Information regarding chronic illness, smoking, alcohol

consumption and drug intake was obtained by questionnaires.

Results of the current study showed that out of 141 schizophrenia patients, 4 patients have Systemic Lupus Erythmatosus (SLE), 26 patients have Type I Diabetes Mellitus, 12 patients have Grave's disease ,7 patients have psoriasis, 4 patients have celiac disease,3 patients have Sjogren's syndrome,9 patients have Intestinal malabsorption. Results suggests a strong relationship between immunological effects and the pathophysiology of schizophrenia. The cytokine profile is also altered in schizophrenics i-e there is a Th2 shift in patients with schizophrenia

Researchers found that people with a history of one or more autoimmune diseases had a higher risk of schizophrenia. In addition, patients with schizophrenia had a higher prevalence of various specific autoimmune disorders, including celiac disease, autoimmune thyroid disorders, acquired hemolytic anemia, Sjogren's syndrome, psoriasis,,polymyalgia rheumatic, myositis, and type 1 diabetes, compared to control subjects (Jones et al. 2005). Reasons for the association between autoimmune aspects and schizophrenia are focused around the notion of early infection with microorganisms that mimic the tissue antigens of the central nervous system, causing the production of antibodies (autoantibodies) against these components. Studies have also shown evidence of a genetic locus for schizophrenia in the area of the human leukocyte antigens (HLA) suggesting that schizophrenia could have an autoimmune origin (Association of Schizoprenia and Autoimmune diseases 2006). Another theory states that treatments used for autoimmune disorders could possibly trigger the onset of schizophrenia.

When most of the scientists favors the possible link between schizophrenia and autoimmunity, Schattner et al (1996) studied parameters of cellular immunity in 23 schizophrenic patients and compared them to 16 matched healthy controls and to 12 patients with rheumatoid arthritis (RA). Thus, in the group of patients studied, no substantiation for the presence of either autoimmune or occult viral cofactors in the pathogenesis of schizophrenia was noticed.

Recently, an autoimmune hypothesis has gained acceptance, which proposes that schizophrenia is one of a spectrum of neuropsychiatric diseases in which an autoimmune attack on the brain occurs. It is also possible, however, that the immunological changes seen in schizophrenic patients are secondary to the disease itself. The main evidence supporting an autoimmune hypothesis is the presence of immunological alterations in schizophrenia that also occur in other autoimmune diseases, e.g. an elevation in serum immunoglobulin levels, a decrease in mitogen responses, morphologically abnormal lymphocytes, an increase in antibrain antibodies, an increase in antibodies to nuclear factor, and a decrease in CD4+ T cells. An autoimmune etiology, if proven correct in the pathogenesis of schizophrenia, would have potential implications for the direction of future psychopharmacological therapies (Umadevi and Sneha 2008).

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