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WILSON'S DISEASE PRESENTING WITH SCHIZOPHRENIA LIKE PSYCHOSIS : A CASE REPORT

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Though neuropsychiatric symptoms are well known in Wilson's disease, purely psychiatric presentation mimicking a schizophrenic psychosis for a number of years, without other hepatic or neurological symptoms, is relatively rare.

There are occasional reports in the world literature regarding association of Wilson's disease and schizophrenia, however, it is difficult to conclude from these reports any specific relationship between the two (Breard- 1959, Franklin and Bouman, 1953, Gysin and Cooke, 1950; Jervis *et al.*,1942; Pierson, 1952; Wilson, 1912). Some authors have doubted the diagnosis of schizophrenia in Wilson's disease (Beard, 1959). However, it cannot be denied that Wilson's disease may manifest with schizophrenia like psychosis which on many occasions mis lead a psychiatrist or neurologist towards incorrect diagnosis. This becomes particularly relevant when we realize that Wilson's Disease is, in present times, largely treatable.

CASE REPORT

S. N. 18 years old boy originally from Tamil Nadu was born of a consanguinous marriage. There was no family history of mental illness or retardation. His birth, developmental milestones and early childhood were reported to be normal. On detailed questioning, however, the family felt that during late childhood and adolescence he was more docile, dependent and childish. His father noticed that compared to other siblings, he was very slow in writing and perhaps in other motor activities also. However, history did not suggest any evidence of mental retardation or intellectual decline during this period.

His psychiatric illness began in September 1982 with gradual social withdrawal (e.g., avoiding contact with friends and relations), muttering to self, sleeplessness and fear of people watching him and planning to harm him and his family members. The paranoid idea gradually progressed to such an extent that he started reacting to it with marked terror by saying that explosives are being kept behind the furniture. There were no associated hallucinatory experiences or any Schneiderian first rank symptoms. History revealed one episode of 'seizure'-a tonic state lasting for 15 minutes during which he was totally unresponsive, about 2 years prior to onset of psychiatric symptoms, for which he was treated with phenobarbitone 60 mg daily. When patient developed psychiatric symptoms he was shown to a neurologist first, who changed his anti-epileptic treatment to carbamazepine 900 mg per day. Patient showed improvement in his excited behaviour but paranoid ideas and occasional sleep disturbance persisted and the patient did not return

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to his premorbid level of functioning. The patient was brought to the O. P. D. services of A. I. I. M. S., New Delhi during the last week of December 1982 and on evaluation he was diagnosed as a case of schizophrenia with mild mental retardation. He was treated with trifluoperazine 15-20 mg per day and carbamazepine 600 mg per day in divided dosage. His paranoid ideas, sleeplessness and social withdrawal improved greatly. However, he was noticed to have very marked extrapyramidal symptoms in the form of tremors, increased muscle tone and dysarthria even with small dosage of trifluoperazine. These side effects persisted for a long time even on withdrawal of trifluoperazine. At this stage of remission, I. Q. assessment was tried on WAIS but patient did not cooperate on verbal scale. On performance scale his I. Q. was 65 without any subtest scatter.

In the month of January 1984, the patient's condition worsened. This coincided with the change of his residence He started showing unprovoked violent and destructive behaviour, screaming and shouting without any apparent reason, echolalia and verbal stereotypy. He was uncooperative on the mental status examination, however, there were no delusion or hallucination. After hospitalisation the patient was treated with chlorpromazine upto 1000mg per day, which could not control his excitement. Instead chlorpromazine produced progressively severe extrapyramidal symptoms in the form of tremors at rest, stiffness of muscles, rigidity, dysphagia and dysarthria, which persisted even after withdrawal of chlorpromazine. In view of the past history of epilepsy and atypical psychotic presentation, an EEG was done which was essentially normal. CAT scan, however, showed mild dilatation of ventricles, subarachnoid cisterns and sylvian fissure, suggesting generalised cortical atrophy. A detailed physical examination at this stage revealed a prominent K. F. ring which was probably missed in earlier examination by different observers. It was later confirmed by the slit lamp examination. Physical examination also showed an enlarged in liver, 2 cm below the costal margin.

His haemogram, routine investigations, X-ray skull, X-ray chest, did not show any abnormality. Blood V. D. R. L. was negative. Liver function tests showed normal serum bilirubin and protein, and mildly elevated level of SGOT (40 KU), SGPT (42 KU) and serum alkaline phosphatase (16 KAU). The value of Urinery Copper (in 24 hours urine) before d-penicillamine therapy was 300 microgram (normal value 0- 25 microgram). There was an elevation of urinary copper (430 microgram) after administration of 1 gm of d-penicillamine.

After establishing the diagnosis of Wilson's disease the patient was treated with d-pencillamine in the dosage of one gram per day. Within three weeks, the extrapyramidal side effects improved considerably. The psychotic symptoms also improved greatly with this treatment without any use of antipsychotic drugs.

COMMENTS

The main interest of this case is, of course, the unusual presentation of Wilson's disease. After one episode of probably an epileptic fit, at the age of fourteen, psychiatric symptoms remained the prominent feature for next two years and also responded to anti-psychotic drugs for more than a year. Due to predominant schizophrenia like picture diagnosis was missed by both neurologists and psychiatrists for a long time. Early recognition is of importance as many cases of Wilson's disease, if detected early, respond well to treatment.

REFERENCES

BEARD, A. W. (1959). The association of Hepatolenticular degeneration with Schizophrenia : A review of the literature and case report. Acta Psychiat. Scand., 34, 411.

FRANKLIN, E. C. AND BOUMAN, A. (1953). Liver dysfunction in hepatolenticular degeneration : A review of 11 cases. Am. J. Med., 15, 450.

GYSIN, W. M. AND COOKE, E. T. (1950). Unusual mental symptoms in a case of hepatolenticular degeneration. Dis. Nerv. Syst., 11, 305.

JERVIS, G. A., NOTKIN, J., FREIMAN, I. S. AND MOORE, T. (1942). Progressive lenticular degeneration (Wilson's disease). Psychiat. Quart., 16, 357.

PIERSON, H. (1932). Two types of Wilson's disease; Hepato lenticular degeneration. J. Neuropath. Exp. Neurol., 11, 19.

WILSON, S. A. K. (1912). Progressive lenticular degeneration : A familial nervous disease associated with cirrhosis of liver. Brain, 34, 295.