Indian J. Psychiat. (1984), **26**(1), 95—96

CATATONIA ASSOCIATED WITH URAEMIC ENCEPHALOPATHY

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SUMMARY

Catatonia is only a clinical diagnosis with a variety of possible aetiological conditions. Among the many neuropsychiatric disorders in renal failure, catatonia is one which has not been established well. The authors report two cases in which despite strong evidence for primary psychiatric disorder, on investigation the catatonic state was found to be associated with ureaemic encephalopathy.

Since the description of Catatonia by Kahlbaum a century ago, there has been overwhelming evidence for non-specificity of catatonic signs (Abrams & Taylor, 1976), with seemingly innumerable causes being cited as being responsible. Uraemia is one of them (Gelenberg, 1976). During the years that followed a few more conditions have been reported to present as catatonia viz. systemic lupus erythematosus (Kronfol *et al.*, 1977), disulfiram encephalopathy (Weddington *et al.*, 1980), malarial fever (Durrant, 1977), glutethimide withdrawal (Campbell *et al.*, 1983) etc. We report two cases of catatonia associated with uraemia.

CASES

1. Mr. A, a 60 years old married male who had been on out patient treatment for 8 years for recurrent depressive episodes responding well every time to tricyclic antidepressants, was referred to us by a family physician. The history for the current episode was of grimacing, making some gurgling sounds and disturbed sleep for two weeks with the patient not taking any food and being totally uncommunicative for 3 days. There was no history of fever, headache, vomiting, convulsions of any cranial trauma. Examination revealed the patient to be stuporose but afebrile with the vitals within normal limits. There was no clouding of consciousness. The systemic examination was normal. X-ray chest PA view and X-ray skull revealed no radiological abnormality. CSF and EKG were found to be normal. Random blood sugar was 65 mgs% and blood urea 79 mgs%. The patient had meanwhile been febrile at 101°F, with no other physical signs, oliguria having been noticed in the ward. The clinical impression at this stage was of catatonia due to metabolic encephalopathy resulting from renal shutdown possibly prerenal in origin. The patient was maintained on intravenous fluids and started on antibiotics. Over the next 24 hours the patient continued to be febrile with a regular pulse of 68/min, blood pressure of 140/100 mms of Hg, with no other physisical signs. Blood chemistry revealed a blood urea of 118 mgs% and serum creatinine of 2.5 mgs%. The catatonic syndrome appeared to be symptomatic of acute tubular necrosis as evidenced by a rising blood urea level (despite intravenous fluids) and elevated serum creatinine value. The patient was transferred to the Nephrology unit of a general hos-

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pital and after 4 days the patient expired despite supportive measures for renal failure.

2. Mr. B., a 45 years old married male, was brought with a 9 day history strongly indicative of depressive disorder, with no feature suggestive of organic illness except for intermittent high grade fever. The patient was stuporose with complete mutism and automatic obedience. Physical examination revealed no abnormality whatsoever. A clinical diagnosis of catatonia, depressive in origin, was made and the patient was to be treated accordingly after excluding any potentially hazardous organic condition. The X-ray chest PA view, X-ray skull and EKG were normal. Random blood sugar estimation was 50 mgs%, blood urea 85 mgs% and serum creatinine 2.8 mgs%. During these hours it was observed in the ward that the patient had been febrile, had passed minimal amount of urine and continued to exhibit mutism and automatic obedience. There was no evidence of clouding of consciousness at any time. The renal dysfunction appeared to be the cause for presenting as catatonia. The patient was transferred to the Nephrology unit of a general hospital, was treated for acute tubular necrosis and discharged after improvement.

COMMENTS :

The essential feature of catatonia is a marked abnormality of motor function. In uraemia, usually the muscle tone is heightened, otherwise, in general, the neurological consequences, of uraemia are similar to the effects on the central nervous system of other metabolic and toxic disorders. A clinical diagnosis of catatonia is not an end in itself but only the beginning of an ardous process of differential diagnosis from amongst a multitude of variable clinical conditions. In every catatonic patient, not only should "non schizophrenic" psychiatric diagnosis be considered, but "nonfunctional" or "organic" conditions should always be looked for.

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