CATATONIC SYNDROME : TREATMENT RESPONSE TO LORAZEPAM

Sir,

We read with interest the study by Payee et al. (1999). Authors remark that no study on the treatment of catatonia with lorazepam has been carried out in India. In this context, we would like to mention that a similar but more exhaustive study has been conducted by us on a larger patient population at Central Institute of Psychiatry, Ranchi. We share a few findings from our study, which is currently in the process of publication.

Our study had 60 patients who were divided into four treatment groups - low dose (4 mg/day) and high dose (8 mg/day) oral lorazepam and low dose (4 mg/day) and high dose (8 mg/day) intravenous lorazepam. Besides using the Bush-Francis Catatonia Screening Instrument and the Bush-Francis Catatonia Rating Scale (BFCRS), Brief Psychiatric Rating Scale (BPRS) was also used to assess the psychopathology. Some of our findings concurred with the findings of the aforementioned study. For instance, we found more patients with schizophrenia as well as acute and transient psychotic disorders than affective disorders presenting with catatonic syndrome. Moreover, severity and duration of catatonia did not predict the response to treatment with lorazepam. However, there were other results, which were at variance with the authors' study. We observed that treatment response after 72 hours of treatment with lorazepam was the best predictor of the final treatment outcome.

Furthermore, although response to lorazepam did not vary significantly across the different psychiatric diagnoses, patients with acute and transient psychotic disorders did show greater improvement.

A new finding was the positive correlation between psychopathology based on BPRS scores before treatment and the severity of catatonia. However, there was no correlation between BPRS scores before treatment and the scores of catatonia on BFCRS following treatment. This indicated that the severity of psychopathology did not have any predictive value in the therapeutic response of lorazepam in catatonia. Other novel findings not alluded to before in the available literature included the faster onset of action of intravenous lorazepam in comparison to oral lorazepam. There was a significant difference between the oral and parenteral groups after half an hour of treatment. This may be attributed to difference in pharmacokinetics between the two routes of lorazepam administration. However, the treatment response did not differ between the two groups after 96 hours of treatment. Moreover, response of catatonic signs to low dose and high dose lorazepam did not differ significantly in either the oral or the parenteral group.

Although our study is of open-label design, nevertheless it allows a few tentative conclusions. First, schizophrenia along with acute and transient psychotic disorders remain a dominant cause of catatonic syndrome in India, which differs from western studies implicating affective disorders as the main cause (Rosebush et al., 1990; Bush et al., 1996). Second, an initial dramatic improvement in catatonic symptomatology on parenteral lorazepam has no predictive value in ascertaining future response to it. Third, over a period of four days, there is no significant difference in response of catatonic signs to oral or parenteral lorazepam. Finally, no additional benefit is accrued by giving higher doses of lorazepam in the treatment of catatonic syndrome.

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