

NEUROLEPTIC - INDUCED ACUTE DYSTONIA IN SCHIZOPHRENIA AND MANIA¹

RAVI S. PANDEY² K.N. SREENIVAS² AND D.K. SUBBAKRISHNA³

Out of 17 schizophrenic and 14 manic patients diagnosed according to ICD-10, 11 and 9 patients respectively developed acute dystonia following a single intravenous injection of 40mg haloperidol. The results are not statistically significant ($p > 0.05$) suggesting that manics and schizophrenics are equally vulnerable to develop neuroleptic - induced acute dystonia.

Nasrallah *et al.* (1988) have reported that manics more often suffer from neuroleptics-induced acute dystonic reactions than schizophrenics whereas other workers (Remington *et al.* 1990; Keepers and Cassey, 1987) have found that manics and schizophrenics are equally susceptible to develop this side effect. This controversy prompted us to conduct this study.

MATERIAL AND METHOD

The patient population of this study was drawn from the acutely excited psychotic patients needing immediate neuroleptization, who attended the outpatient psychiatric facility of our hospital. Each patient was given 40mg. of haloperidol intravenously very slowly and watched for the development of acute dystonic reaction for three hours. We used this dose and three hours of observation because our initial observation (unpublished) has demonstrated 40mg of haloperidol to be a safe and adequate dose for rapid neuroleptization and all patients who had to develop acute dystonic reaction with this dose, developed it within 102 minutes of the injection. During the process of injecting the

drug, if a patient calmed down, the remaining amount of drug was rejected. Each patient was hospitalized after the observation period. In the wards the patients were diagnostically evaluated by psychiatrists using the ICD-9 criteria. The evaluating psychiatrist did not know the dystonic and nondystonic status of patients i.e. whether a given patient had developed acute dystonia with haloperidol injection or not.

RESULTS

Out of 58 patients, we had to exclude 27 patients from the final analysis. We excluded patients who had taken oral neuroleptics in the past 7 days of fluphenazine deconoate injection in the past one month ($n = 12$), patients who received a diagnosis other than schizophrenia, manic depressive psychosis-manic phase, or manic depressive psychosis, circular currently manic ($n = 9$), patients who slept during observation period ($n = 7$) and patients who required less than 40mg of haloperidol for tranquillization ($n = 4$). Some patients had more than one reason for their exclusion. After excluding the above patients, there were 17 and 14 patients in

schizophrenic and manic group respectively. Both the groups were found to be comparable in age

and sex. Table I shows the number of dystonic and non-dystonic patients in the two groups.

Table-I : Distribution of dystonic and nondystonic response in schizophrenic and manic group

Response to halo peridol injection	Schizophrenic group (n = 17)	Manic group (n = 14)	Statistical significance*
Dystonic	11	9	p = 0.64**
Nondystonic	6	5	

* Fisher's Exact Probability

** Not Significant

DISCUSSION

Ours was a prospective study unlike the one by Nasrallah *et al.* (1988). We gave 4mg of haloperidol intravenously which shortened the observation period to 3 hours. Remington *et al.* (1990) used multiple, hourly injections of haloperidol intramuscularly (upto 70mg. in seven hours) because of which their observation period exceed two days. We believe that occurrence of transient dystonic reactions can be detected better during a shorter observation period. In addition to this, we are able to exclude all the patients who slept during observation period (because dystonia does not occur during sleep) whereas the same would not have been possible in the study by Remington *et al.* (1990) because all patients are likely to fall asleep during such a long observation period. However, exclusion of a very large number of patients from the study, makes our study population unrepresentative of the usual manic and schizophrenic patients attending a psychiatric clinic.

We also could not exclude patients who had a family history of Parkinson's disease or other diseases with extrapyramidal manifestations because adequate and reliable family history was not available in majority of the cases.

Since tardive dyskinesia is commoner in manics than schizophrenics (Hamra *et al.* 1983; Yassa *et al.* 1983) one might be tempted to extrapolate that acute dystonia also will be more common in manics. One has to remember that the underlying biochemical mechanisms producing tardive dyskinesia and acute dystonic reactions are different (Marsden & Jenner, 1980). It is, therefore, not necessary that both tardive dyskinesia and acute dystonia should have similar prevalence in manics and schizophrenics.

REFERENCES

- Hamra, B.J.; Nasrallah, H.A.; Clancy, J. and Finn, R. (1983) Psychiatric diagnosis and risk for tardive dyskinesia. *Arch. Gen. Psychiatry*, 40; 346-347.

NEUROLEPTIC-INDUCED ACUTE DYSTONIA

Keppeler, G.A. and Cassey, D.E. (1987) Prediction of neuroleptic induced dystonia. *J. Clin. Psychopharmacol.*, 7, 342-345.

Marsden, C.D. and Jenner, P. (1980) The pathophysiology of extrapyramidal side-effects of neuroleptics drugs. *Psychol. Med.*, 10; 55-72.

Nasrallah, H.A; Churchill, C.M. and Hamdan Allan, G.A. (1988) Higher frequency of neuroleptic-induced dystonia

in mania than in schizophrenia. *Am. J. Psychiatry.* 145; 1455-1456.

Remington, G.J.; Volneskos, G.; Pollock, B.; Reed, K. and Coulter, K. (1990) Prevalence of neuroleptic - induced dystonia in mania schizophrenia. *Am. J. Psychiatry.* 147; 1231-1233.

Yassa, R.; Gharirian, A. M. and Schwartz, G. (1983) Prevalence of tardive dyskinesia patients. *J. Clin. Psychiatry.* 44; 410-412.