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Brief Communications

NEUROLEPTIC MALIGNANT SYNDROME – A CASE REPORT

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Introduction

Neuroleptic malignant syndrome is recognised as a rare and idiosyncratic side effect of almost all antipsychotic drugs (Vinken and Bruyn 1968). This disorder receives little or no attention in most of the psychiatric and psychopharmacologic text books. Long acting phenothiazines as well as other neuroleptics have been incriminated the most, but other drugs such as Tiaprides, MAO-I, Tricyclic combinations, Lithium – neuroleptic combinations have also been known to cause this catatonic syndrome. It is described that organic brain disease, exhaustion and dehydration may be additional factors (Itoh, Ohtsuka & Ogita 1977, Meltzer 1973). Even short duration of exposure to neuroleptics can bring about the syndrome which is characterised by generalised rigidity, akinesia and mutism, hyperpyrexia, autonomic dysfunction and fluctuating levels of consciousness. Among the sixty cases in literature, 20 per cent mortality rate has been reported by Caroff (1980), usually due to cardiovascular collapse, cardiac arrhythmias, renal or respiratory failure (Morrant 1984).

We present a case report of a patient who had received Haloperidol 300 mg. and Chlorpromazine 1000 mg over 10 days in the past for control of symptoms of mania, but who developed neuroleptic malignant syndrome within 72 hours of hospitalization and administration of these drugs.

Case Report

Mrs. 'A', a 45 year old female, whose diagnosis according to DSM III criteria was Affective Disorder, Bipolar, Manic type, was hospitalised for symptoms of marked aggressiveness, abusiveness, overtalkativeness and sleeplessness. During the next 3 days she received a total of 60 mg. Parenteral Haloperidol (I. M.) and Chlorpromazine 300 mg. in divided doses. Three days after the initiation of haloperidol and Chlorpromazine she was noted to be mute with marked generalised rigidity and akinesia. Her pulse, B. P., and temperature were normal. At that time we felt she was having E. P. S., so all neuroleptic medication was stopped and she received only antiparkinsonian agents, but the akinesia and rigidity worsened. Soon, she developed pyrexia (105°F), profuse diaphoresis, urinary incontinence and refused to take oral feeds. There was clouding of her sensorium but she would respond only to painful stimuli. The B. P. recorded was 160/116 mm. Hg., Pulse 140/mt., and tachypnoea was present. In addition to the generalised hypertonus she also had marked tremors. No pyramidal signs were detected. The fundus examination, apart from arteriosclerotic changes Gr. II, was grossly normal.

There were no significant laboratory finding. Blood counts, chest X ray, kidney function test, blood glucose and serum electrolytes did not show any abnormalities.

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Apart from increased heart rate, the ECG was within normal limits.

The consulting Physician opined encephalitis and later septicaemic shock, even though, at this stage, a doubt of NMS was kept in mind. Parenteral antibiotics, I. V. fluids, and steroids were started.

However, her general condition continued to deteriorate over the next 24 hours. Her body temperature varied from 102°-106°F., never touching baseline. There were wide fluctuations of her B. P., lowest recording being 50 mm. Hg., systolic (diastolic not recordable). Her pupils were dilated and sluggishly reacting to light, and she was not responding to even painful stimuli. Her respiration became stertorous and she died on the 5th day of becoming mute and rigid.

Blood and urine cultures which were sent at the time of admission were reported sterile. Post mortem C. S. F. examination did not reveal any pathological findings.

Discussion

Mrs. 'A', whose past history revealed several episodes of mania, developed NMS after the administration of chlorpromazine and haloperidol. The diagnosis of NMS was made after excluding all organic diseases that can cause catatonic stupor as described by Gelenberg (1976), such as encephalitis, meningitis, malignant hyperthermia, etc. The patient had shown a good response to the same drugs in the past attacks. This idiosyncratic reaction with such a combination has not been commonly reported. In fact, most of the reports have primarily implicated long acting fluphenazine derivatives as the causative agents (Caroff 1980). The patient developed classical symptoms as described in the literature. Delay and Deniker (1968) suggest that respiratory insufficiency is a cardinal feature of NMS as observed in this patient who also had marked dyspnoea not secondary to any lung pathology although Caroff (1980) remarked that the dyspnoea observed in the 16 cases reported in the literature was the result of pneumonia or pulmonary emboli following prolonged immobilisation. The development of the symptoms had been rather explosive leading to death due to cardio respiratory failure, although it has been reported that NMS lasts 5-10 days after cessation of oral neuroleptics.

References

CAROFF, S. N. (1980), The Neuroleptic Malignant Syndrome. *Journal of Clinical Psychiatry*, 41, 79-83.

DELAY & DENIKER (1968), Drug induced Extra Pyramidal Syndromes In: Handbook of Clinical Neurology Vol. 6, 248-266.

GELENBERG, A. J. (1976), The Catatonic Syndrome, *The Lancet* 1339-1341.

ITOH, H., OHTSUKA, N. & OGITA, K. (1977), Malignant Neuroleptic Syndrome. *Folia Psychiatry Neurology Journal*, 31, 565-576.

MELTZER, H. V. (1973), Rigidity, Hyperpyrexia and Coma following fluphenazine Enanthate. *Psychopharmacologia* 29, 337-346.

MORRANT, J. C. A., (1984), A Catatonic Syndrome Resulting in Death. *Canadian Journal of Psychiatry*, 29, 147-150.

VINKEN, P. J. & BRUYN, G. W. (1968), The Neuroleptic Malignant Syndrome. Diseases of the Basal Ganglia, Vol. 6, 258-262