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E.C.T. INDUCED CATATONIA: A CASE REPORT

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For a long time catatonic signs were considered to be the specific features of schizophrenia (Vogdt 1958). It soon became clear that such features may occur in functional psychoses other than schizophrenia and in fact, may even be more common in affective psychosis than schizophrenia (Abram & Taylor 1976). Apart from functional disorders like schizophrenia, affective psychosis and hysteria, a catatonic syndrome may be caused by a large number of neurologic, metabolic and toxic conditions (Galenberg 1976). There have been reports of catatonic features occuring in encephalitis lethargica (Schilder 1969), uraemia (Steinman & Yager 1978) systemic lupus erythematosus (Kronofol et al. 1977), subdural haematoma (Woods 1980), malaria (Durrant 1977), thrombocytopenic purpura (Read 1983) and during drug therapy such as with neuropleptics (Galenberg & Mandels 1977), disulfiram (Weddington et al. 1980) steroids (Sullivan & Dickerman 1979) or even with epidural morphine (Engquist et al. 1980) or during glutethimide withdrawal (Campbell et al. 1983). However, we have not come across any case of catatonia occurring during electro–convulsive therapy (E.C.T). We report such a case.

Case Report

Ms. A, a twenty six year old woman who met the DSM III criteria for the diagnosis of Bipolar Affective Disorder (American Psychiatric Association 1980) had been admitted thrice in the past two years and was being treated with lithium on outpatient basis at NIMHANS, Bangalore and was well controlled. She discontinued lithium and was readmitted during an episode of depression. At the time of admission, physical examination was normal and the patient had weeping spells, psychomotor retardation, delusion of guilt and suicidal ideas. She was started on amitriptyline 100 mgm / day and was administered modified E.C.T. a day after admission. Following ECT, she developed echolalia, posturing and waxy flexibility for eight hours during which physical examination was normal but cognitive functions could not be tested. The next morning, the patient had no catatonic features or cognitive deficits but depressive features persisted and she was given the second modified E.C.T. after which she again developed exactly similar catatonic features which persisted for about six hours. Suspecting an association between ECT and catatonic features, ECT was stopped and the patient was treated with amitriptyline 125 mgs per day. With this, she recovered completely in 18 days. Throughout this period, physical examination was normal. A CAT scan done three days after the first ECT was also normal. After complete recovery, with informed consent, she was given one direct ECT and

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she again developed exactly the same catatonic features lasting for eleven hours.

Discussion

To our knowledge, this is the first case report of catatonic features developing after electro–convulsive therapy. Since drugs are known to produce catatonic features (Galenberg & Mandels 1977) one may argue that the catatonic features in our patient might have been related to the premedication given for modified ECT (intravenous thiopentone sodium and succinyl choline) and not to ECT per se. However, the patient developed catatonic features even after direct ECT where no premedication was given, suggesting that the catatonic features were due to ECT and not due to premedication. Since ECT is frequently used for the management of catatonic (Wells 1973) we feel that clinicians should be aware of the possibility of development of catatonic features due to ECT, as ignorance of such an association may lead to further administration of ECT which might be causing the catatonic state. Such an association also envisages an attractive theoretical possibility. Occurrence of catatonic features is well recorded in patients with temporal lobe epilepsy (Taylor 1972). It is possible that catatonic features following ECT are mediated through the effects of ECT on temporal lobes. If it is so, it will be desirable to use a brief stimulus technique (B.S.T) to induce seizure activity in these patients as B.S.T. delivers less electrical energy to brain in general and temporal lobes in particular (Maxwell 1968 ; Valentine et al. 1968, Weaver et al. 1974 ; 1977). It is also to be remembered that EEG abnormalities are often found in catatonic stupor (Plum & Posner 1972) suggesting its neurophysiological similarity with epilepsy. One is also reminded of Symond's (1962) hypothesis that epilepsy itself may be a continuous rather than an episodic process and the schizophreniform psychosis may be the manifestation of underlying continuous process whereas fits and EEG changes may be merely epiphenomena.

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