case report 90

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A case is presented of the treatment of a unipolar delusional depression with electroconvulsive therapy. During the course of the treatments, the patient suddenly became extremely confused, bizarre, and agitated. The decision as to whether to continue the ECT rested on understanding this response. The decision-making process, possible explanations, and preventive measures are discussed.

an organic psychosis associated with electroconvulsive therapy

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Transient organic brain syndromes are commonly seen in patients undergoing electronconvulsive therapy. Usually the symptoms are minor, clear rapidly, and do not present difficult management problems. Occasionally the severity of these symptoms presents very difficult dilemmas for the treatment team.

The patient, a 54-year-old, married, insurance salesman and father of two children had been hospitalized five years earlier with a diagnosis of "psychotic depression and mild organic brain damage." He had been treated successfully with a course of eight electroconvulsive treatments, and only mild confusion was noted after each of these treatments. Subsequently, he was maintained on amitriptyline HCl and chlorpromazine HCl (doses unknown) for several months and returned to work.

He remained well until approximately one year prior to this admission, when he began to experience difficulties at work. Four months prior to admission he had been fired because of poor performance. His depression worsened, and his alcohol intake increased substantially, to three to four martinis and six beers per day. He denied a history of blackout spells, previous withdrawal symptoms, or social sequelae of alcoholism. There was no history of manic episodes nor family history of any psychiatric illness. He and his wife described some marital difficulties during the previous six years, but there were no other social or psychological stresses which we could identify as possibly precipitating the present episode.

Two months earlier he was hospitalized for the surgical removal of renal stones. During his convalescence at home, he became increasingly depressed, with frequent crying spells, obsessive thought and ruminations, insomnia, anorexia (with a 10-pound weight loss), and agitation. At that point he was admitted to another hospital.

Their records revealed that upon admission the patient was having "paranoid ideation, self-depreciating verbalization, impaired reality testing, and auditory hallucinations." He was begun on trifluoperazine HCl 30 mg per day with minimal response after 10 days. Chlorpromazine was begun and eight days later amitriptyline HCl added. His thought disorder cleared on these medications but depressive features remained prominent. Upon transfer (for financial reasons) the diagnosis was "recurrent psychotic depression." Medications consisted of amitriptyline HCl 100 mg b.i.d., chlorpromazine HCl 150 mg t.i.d., and triamterene 50 mg/hydrochlorothiazide 25 mg (for prevention of renal calculi).

Upon his admission to our hospital, the patient was described as a middle-aged, greying man, unshaven but neatly dressed. Though cooperative, he exhibited psychomotor retardation and slow, soft speech. He also seemed sedated. Thought processes were rational, coherent, and goal-directed, though occasionally he was circumstantial and tangential. There was no evidence of loosened associations, delusions, or hallucinations.

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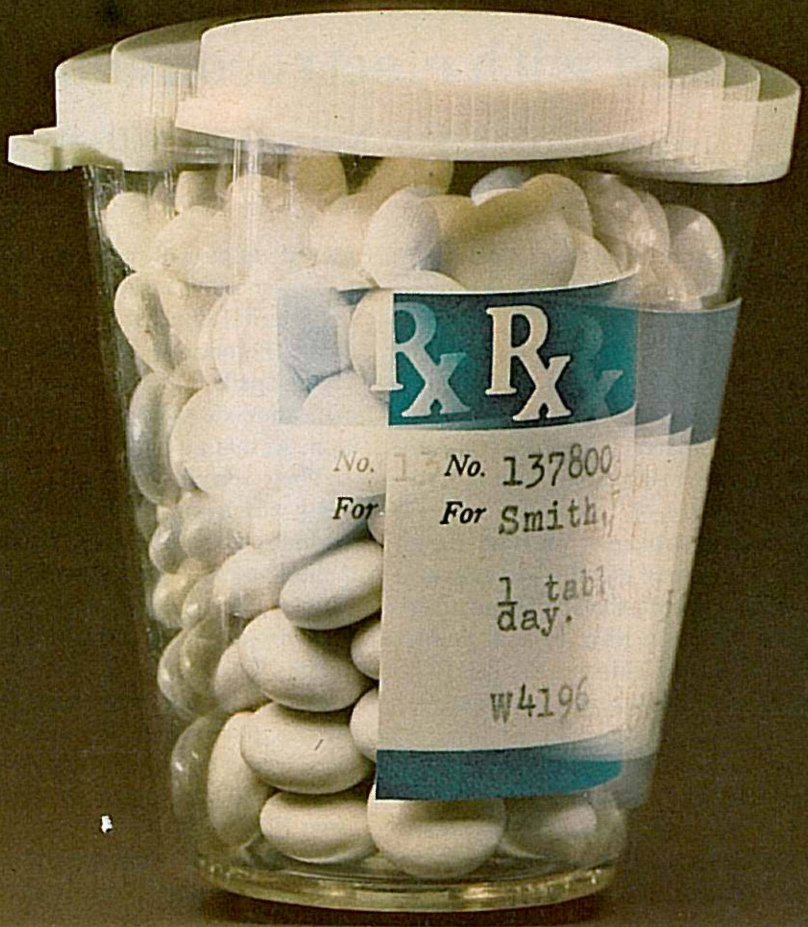

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1. Byck R: Drugs and the treatment of psychiatric disorders, in Goodman LS, Gilman A (eds): *The Pharmacological Basis of Therapeutics*, ed 5. New York, Macmillan Publishing Co, Inc, 1975, pp 170–171.

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kind to many patients with psychotic symptoms

For Brief Summary, please see following page.

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His mood was profoundly depressed, and his affect appropriate for thought content. He denied suicidal ideation, although he admitted to such in the past. He was oriented to person, place, and year but not to day or date. He appeared to concentrate adequately, performing serial sevens slowly but without error. Intelligence was judged to be normal. Short-term and remote memory were intact, except for difficulty in recalling the events of the first three weeks of hospitalization. Judgement seemed intact.

Physicial examination, including neurologic exam, was entirely within normal limits. Routine laboratory studies including electrolytes were unremarkable.

Treatment

Our initial impression was that this patient was suffering from a recurrent unipolar depression, the delusional portion of which had resolved. There was also a history of significant alcohol abuse. We felt that minor cognitive difficulties noted in the mental status exam probably resulted from his extremely depressed state, rather than from an organic brain syndrome.

Because of excessive sedation and some orthostatic hypotension, the chlorpromazine HCl was reduced while levels of amitriptyline HCl were continued. On the third hospital day (chlorpromazine HCl 150 mg per day) the patient's condition changed rather suddenly. He began ruminating, became quite agitated, and was unable to tell whether it was morning or afternoon. He reported auditory hallucinations commanding him to hurt himself. He banged his head on the floor, attempted to pull his hair out, and talked of poking his eyes out. He required intermittent seclusion and restraints for self-protection. When not restrained, he curled in a fetal position on the floor for long periods of time.

At this time rapid tranquilization with thiothixene was begun for behavioral control. Over the next few days there was a moderate decrease in his auditory hallucinations and in his agitated and self-destructive behaviors, but his guilty ruminations, delusions, and regressed behavior seemed unaffected. It was not possible to further increase his thiothixene (45 mg per day) or amitriptyline because of the symptomatic orthostatic hypotension. Because of this, nondominant unilateral electroconvulsive therapy was begun on the 11th hospital day and planned for alternate days three times a week. His amitriptyline HCl was reduced and discontinued, but the thiothixene was continued for behavioral control.

During the routine workup for the ECT, skull films had revealed a double floor to the sella turcica. Tomograms tended to confirm this interpretation. In the absence of symptoms of pituitary disease, we elected to proceed.

After each of the first three treatments the patient was so agitated and belligerent that he required physical restraint and intravenous diazepam 5-10 mg for sedation. Upon awakening minutes to several hours later, the patient showed no signs of agitation and confusion. How-

ever, later in the day, after the third treatment, he again became agitated and confused. He was disoriented to time and place. His speech was rambling, incoherent, and he demonstrated echolalia. His behavior was bizarre and inappropriate. For example, he put his cigarette out on the television, chewed on a toy, and ate from other patient's trays. This clinical picture differed from that preceding ECT in that the affect was labile and non-depressed — he often acted silly. Because of the severity of this clinical state and its persistence over the next few days, we became concerned, wondering if this represented something other than those changes usually associated with electroshock therapy.

A neurology consultant felt that a focal brain lesion was unlikely but suggested computerized tomography. Thiothixene was also stopped in order to determine whether drug toxicity might be a contributing factor. The scan was read as "minimal cerebral atrophy consistent with the patient's age and history of alcohol intake." On the basis of these findings the ECT treatments were continued. As the treatment progressed, the profoundly bizarre and agitated behavior diminished slightly but the cognitive defects remained. Evaluation of his depressive signs and symptoms was impossible; nonetheless, the original plan of eight ECT treatments was completed.

During the week following treatment, the patient's mood brightened and his irritability, confusion, and disorientation subsided, although memory difficulties remained prominent. At discharge, two weeks later, there were no signs of depression, he was nondelusional, and the mental status examination revealed no evidence of organic dysfunction. Neuropsychological testing demonstrated "bright normal to superior functioning on psychometric, memory, attentional, and motor measures." The patient was discharged on imipramine HCl 100 mg at bedtime. Follow-up at three months revealed that he was doing well.

Discussion

This was a patient with depressive and psychotic symptoms, who was resistant to psychotropic medication (or could not be given adequate amounts) and for whom electroconvulsive treatment was instituted. Our diagnosis was recurrent unipolar depression of psychotic proportions. Soon after ECT was begun, the patient became agitated, disorganized, and demonstrated extreme intellectual deterioration — confronting us with important treatment decisions.

There were a number of possible explanatory considerations. One was that the renewed agitation and confused behavior were related to a psychotropic drug the patient was receiving — i.e. that the agitated behavior may have reflected an idiosyncratic response to the thiothixene. However, we felt that this was unlikely, and that the thiothixene was needed (45 mg per day) to control the patient's self-destructive tendencies and lessen the need for constant restraint. There has been concern expressed over the combination of ECT and neuroleptic drugs (also antidepressants), but this is generally related to hypotensive propensities and adverse interactions with the barbiturates during the treatments, rather than to an intrinsic adverse interaction between ECT and a specific drug.[1] The likelihood of an anticholinergic psychosis was entertained but no signs of anticholinergic toxicity were found.

We were concerned about whether an undiagnosed cerebral lesion was being aggravated by the ECT. This phenomena has been reported by others, and a brain

SELF-ASSESSMENT *(answers on page 40)*

Choose the correct response(s) to each of the following:

1. Possible explanations for agitation, disorganization, and cognitive deterioration during a course of ECT are:
   a. uncovering an underlying schizophrenia.
   b. organic-psychotic syndrome secondary to ECT.
   c. precipitation of a manic episode.
   d. underlying organic central nervous system pathology.
2. Regressive ECT:
   a. involves giving the patient more than one treatment per day.
   b. is used to treat severely regressed hysterical patients.
   c. is more beneficial than antipsychotic agents for schizophrenic patients.
   d. can produce a severe dementia.
3. Methods that may help minimize confusion and amnesia during a course of ECT include:
   a. unilateral dominant placement of electrodes.
   b. unilateral nondominant placement of electrodes.
   c. supplemental vitamin B_{12}.
   d. I. V. drip method for the administration of drugs for anesthesia and relaxation.
4. Moderate cognitive and orientation disturbances are always indicative of an organic brain syndrome.
   a. True
   b. False
5. Post-ECT excitement states are best treated with:
   a. oral barbiturate.
   b. oral diazepam.
   c. physical restraint and patience.
   d. I. V. diazepam.
   e. I. V. barbiturate.
6. Which of the following statements about the use of psychotropic drugs with ECT are accurate?
   a. Use of tricyclic antidepressants during a course of ECT increases the likelihood for positive response.
   b. Antipsychotic agents should always be discontinued during a course of ECT.
   c. Psychotropic drugs that can cause orthostatic hypotension may synergize with the I.V. barbiturates in this manner.
   d. The use of tricyclic antidepressants after a course of ECT may prove to be prophylactic for the recurrence of an affective disorder.

tumor is, of course, a definite contraindication to ECT, with risk of fatality.[2] The nonlocalized scan findings and discussions with our neurologic consultants helped allay these fears.

It was also possible that the ECT had activated an underlying psychosis (perhaps schizophrenia). This is described as occurring most often in quiet, blocked, or depressed schizophrenics, in whom an acute delusional hallucinatory syndrome emerges after the first few treatments.[3] These patients have often been considered depressed, and following ECT an unsuspected schizophrenic psychosis becomes overt. Halpern stated that in this manner ECT can serve as a diagnostic aid in schizophrenia.[4] Slater and Roth, discussing depressed patients, also noted that concealed symptoms may emerge from ECT, revealing the patient as "an unmistakable schizophrenic."[5] However, considering our patient's age, premorbid personality, and the course of the illness, this did not seem to be the case.

The most likely explanation of this behavior was that it was an exaggeration of effects normally seen with electroconvulsive therapy. We postulate that in view of the patient's history of alcoholism and the "minimal cerebral atrophy" demonstrated on the scan, there may have been a reduced threshold or increased sensitivity to those effects normally seen after treatments. These effects — some immediate confusion, and an amnesia which usually clears up in one to two hours — may become more severe as treatment progresses. There is often mild emotional disturbance and the patient may appear dull or silly. This induction of organic impairment has been utilized by some to treat schizophrenic patients. The technique, "regressive ECT," requires multiple daily treatments to induce an organic psychosis.[6,7] Particularly beneficial clinical responses to this form of treatment, compared with usual methods, have not been substantiated, and prolonged severe dementia has been reported.[8-9]

It is not unusual for individual treatments to be followed by periods of postconvulsive excitement occasionally leading to dangerous aggressiveness. This kind of circumscribed agitation was seen following our patient's first three treatments. As in cases described by others, it was brief, being successfully treated with I.V. diazepam.[3]

In 1945, Kalinowsky reported an organic psychotic syndrome occurring during electroconvulsive therapy,[10] and the phenomena has been accepted in the literature. This organic-psychotic reaction takes a variety of forms, with Korsakoff-like pictures being most frequent. Patients are described as bewildered, often showing severe psychomotor excitement with delusional fears and hallucinations. These organic reactions seem to be differentiated from other psychotic syndromes in that there is more organic blurring of consciousness, greater emotional lability, more flight of delusions, and more vividness to the hallucinations. In general, the patients seem to be less coherent, and there is severe impairment of intellectual function with a tendency to perseveration and circumstantiality. Organic signs dominate the picture. Though the presence of delusions or hallucinations in our patient was difficult to ascertain, the extreme cognitive dysfunction was remarkable. Being reassured by the description of these organic psychoses, we were able to complete eight ECT treatments, the agreed upon goal. Kalinowsky and Hippius warned that a frequent mistake is the premature discontinuation of treatment if the organic reaction is mistaken for an aggravation of the patient's original psychosis.[2] Conversely, overtreatment may occur if the reaction is felt to be part of the patient's primary illness. By disappearing spontaneously within a week to 10 days following the last treatment, the organic reaction in this case confirmed the earlier experience of Kalinowsky and Hippius.

We speculated in retrospect about whether this response could have been predicted. If so, we could have better prepared the family and our staff, and perhaps minimized the reaction. We knew the patient had exhibited mild confusion after his treatments five years ago, but this is not unusual. Documentation for the diagnosis upon transfer of "mild organic brain syndrome" was lacking, and the minor signs of initial mental status examination which might have been attributed to an organic brain syndrome are commonly seen in delusional unipolar depressions.[11] We are aware of no studies which specifically discuss organic reactions occurring more often or with greater severity in chronic alcoholics being treated with ECT. There are several papers discussing ECT in patients with various kinds of CNS disease.[12-14] None of these papers indicate an exaggerated organic response to ECT therapy is expected. Elmore and Sugarman suggest that such a prediction may be based upon a past history of prolonged schizoid or borderline adjustment, evidence in psychological testing, or poorly organized ego structure, such as faulty contact with reality or loosening of associations.[15] These observations would not have been pertinent in this case.

If we had been prewarned, several measures might have been useful. Unilateral ECT over the nondominant hemisphere was used for this patient but not specifically as a prophylactic measure. Unilateral ECT has been reported to be accompanied by less amnesia and confusion than the conventional bilateral ECT.[16] In general, desirable clinical effects for the unilateral and bilateral ECT are found to be equivalent, although there is some difference of opinion regarding this.[17]

Kalinowsky described the use of an intravenous drip method of administering ultrashort-acting barbiturates prior to ECT.[18] Utilized in this manner, the amount of barbiturate required to introduce amnesia is one-half to one-third the amount necessary for induction when rapid intravenous injection is used. This method also decreases the amount of succinylcholine chloride needed to achieve maximal relaxation. When administered with continuous oxygen, this method may result in a reduced period of apnea, thus ensuring better brain oxygenation.

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At the time this report was written all three authors were associated with the Department of Psychiatry and Center for Health Sciences at the University of Wisconsin at Madison. Dr. John R. Marshall remains there as Professor of Psychiatry along with Dr. Alice M. Tariot, Resident in Psychiatry. Dr. Ned G. Kalin is now a Clinical Associate at the National Institute for Mental Health.

GREENBLATT *(continued from page 14)*

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6-month index

ANSWERS TO CASE REPORT SELF-ASSESSMENT *(page 37):* (1) a,b,d; (2) a,d; (3) b,d; (4) b; (5) d; (6) c,d.