0022-3018/78/1662-0139$02.00/0
THE JOURNAL OF NERVOUS AND MENTAL DISEASE
Vol. 166, No. 2
Printed in U.S.A.

SINGLE CASE STUDY

GOOD OUTCOME IN A CATATONIC PATIENT WITH ENLARGED VENTRICLES

HOYLE LEIGH, M.D.,[1] WILLIAM ANTHONY CALLAHAN, M.D.,[1] AND DANIEL EINHORN, M.D.[2]

In the differential diagnosis of the catatonic syndrome, the demonstration of an intracranial anomaly is often taken as evidence of irreversibility. We present the case of a 27-year-old white female with catatonia who was found to have enlarged ventricles on automatic computerized tomographic axial scan. She had a complete resolution of the catatonia and psychotic symptoms without any change in the size of the ventricles. This resolution occurred when the patient was treated for her ulcerative colitis with colectomy and the steroids she received for the colitis were gradually withdrawn. The discovery of a structural anomaly of the brain per se should not discourage the clinician from identifying and treating all other factors that might contribute to the catatonic syndrome.

Catatonic syndrome is now recognized to be related to a number of disorders such as lesions in the basal ganglia and limbic system, viral encephalitis, metabolic derangements, and other diverse processes (1–6). Automatic Computerized Tomographic Axial Scan (ACTA) is a noninvasive tool which may screen for possible intracranial lesions in catatonic patients. The presence of an intracranial anomaly per se, however, should not be taken as conclusive proof that it alone is responsible for the catatonic syndrome, nor should it be taken, necessarily, as a grave prognostic sign. We present a case of a young woman who was hospitalized with a catatonic syndrome superimposed on chronic ulcerative colitis. ACTA Scan after admission revealed markedly enlarged third, fourth, and lateral ventricles. In spite of this intracranial abnormality, she recovered fully from the catatonic syndrome after colectomy and withdrawal from the steroids which had been administered to control her ulcerative colitis.

CASE REPORT

A 27-year-old white married female was admitted to the medical service for treatment of severe ulcerative colitis. She was a high school graduate and the mother of two children. She had delivered her second child approximately 2 weeks prior to admission, the delivery being complicated by ABO incompatibility in the fetus. Upon arrival at the hospital, she was mute and immobile. Her family stated that the patient had complained of "racing thoughts" and a feeling that she was going "crazy" beginning several weeks prior to admission. In fact, the patient had stopped taking prednisone for a while on her own because she felt it was making her "crazy". For 3 weeks prior to hospitalization, she received 60 mg of prednisone p.o. per day. She had no previous psychiatric history. On the 2nd hospital day, the psychiatric consultant noted her mutism, unresponsiveness, and waxy flexibility. Her eyes were open, and she appeared vigilant, occasionally grimacing at people in her room. The physical examination on admission revealed an emaciated and diaphoretic young woman with labile hypertension (170–130/120–90 mmHg), tachycardia (90–125/min), and fever (rectal temperature 100–101 F.). Laboratory findings showed anemia (hematocrit 29–27%), hypokalemia (2.2 mEq/l), and hypomagnesemia (1.6 mEq/l.). All other values including thyroid studies were within normal limits. Upon admission, she was placed on a regimen of hydrocortisone 100

[1] Department of Psychiatry, Yale University School of Medicine, 333 Cedar Street, New Haven, Connecticut 06519. Send reprint requests to Dr. Leigh.
[2] Tufts University School of Medicine, Boston.

mg i.v. four times a day. Serum potassium and magnesium were corrected without visible improvement in the patient's mental status. An ACTA Scan performed on the 2nd day of admission revealed "grossly enlarged lateral, third, and fourth ventricles with no evidence of cortical atrophy." A repeat ACTA Scan 3 days later confirmed enlarged ventricles with no evidence of a posterior fossa mass. An EEG revealed moderate generalized bilateral rhythmic slow wave activity with no focal or epileptic features. A pneumoencephalogram revealed free flow of air into the ventricles and over the convexities, confirming the existence of markedly enlarged ventricles. There was questionable bilateral cerebral atrophy. The most likely diagnosis was considered subacute sclerosing panencephalitis.

The patient would occasionally manifest severe tremors of all of her extremities which were sometimes mistaken for convulsions by the nursing staff. At the psychiatrist's recommendations, she was placed on 2 mg of perphenazine i.m. three times a day with some improvement. The patient would on occasion "wake up" from her stuporous, immobile state, and would converse with the staff, but then would lapse back into the catatonic state. During the lucid intervals, she was apparently responding to visual and occasionally auditory hallucinations.

Throughout her hospital course, she had stools positive for blood and developed episodes of frank gastrointestinal bleeding with marked evidence of toxicity. An emergency total colectomy was performed in the 6th week of hospitalization. After the colectomy, all vital signs and laboratory findings returned to normal, and the hydrocortisone was gradually tapered. Her mental status then improved gradually such that 5 weeks after the colectomy she was completely oriented and showed good recent and remote memory. She did serial 7's well, and her calculations and abstractions were within normal limits. Her judgment was good. Her affect was normal, and she related with the staff quite well. Gone were the tremors, waxy flexibility, and any evidence of hallucinations. She was eager to go home and return to her family and stated that she had no recollection of the events that transpired during the period she was in the hospital until the time she recovered from surgery. She had no recollections of either the conversations she had with the psychiatrist during her lucid periods, nor of any hallucinations before the surgery. A repeat ACTA Scan performed at the time of discharge from the hospital 2½ months post-op still showed enlarged ventricles of the same magnitude.

DISCUSSION

This case illustrates that catatonic syndrome can indeed be a multifactorial phenomenon. This patient had a number of possible etiological factors in her catatonia, including hypokalemia, hypomagnesemia, postpartum state, and steroids, as well as the enlarged ventricles. In addition, the toxicity from ulcerative colitis and fever may have contributed to the final clinical syndrome. Even when metabolic derangements such as hypokalemia were corrected, the patient continued to exhibit catatonic syndrome. Many felt initially that this syndrome was due to an irreversible process in the brain associated with the enlarged ventricles. Such thinking led some clinicians to despair of effective therapy and even to suggest the futility of colectomy in "such an obviously brain-damaged patient." Subsequent course of this patient indicates, however, the value of attacking any treatable source of a multifactorial problem. In retrospect, the patient's own feelings of "going crazy" while on steroids could have been taken more seriously. With the treatment of the ulcerative colitis by colectomy, and tapering off of the steroid, her mental status improved to premorbid state, without any change in the enlarged ventricles. Although the structural anomaly might conceivably have been associated with the brain's vulnerability to catatonia, it could not have been the major "cause" of the patient's behavioral symptoms. Future studies are needed to collect normative data concerning the incidence of "asymptomatic" enlarged ventricles and other intracranial anomalies on ACTA Scan. This case draws our attention to the fact that

every effort should be made to identify and treat *all* possible etiological and contributing factors in a patient with catatonic syndrome. The discovery of an untreatable intracranial anomaly per se should not discourage the clinician from this endeavor.

REFERENCES

1. Abrams, R., and Taylor, M. A. Catatonia—A prospective clinical study. Arch. Gen. Psychiatry, *33:* 579–581, 1976.
2. Belfer, M. L., and D'Autremont, C. C. Catatonia-like symptomatology. An interesting case. Arch. Gen. Psychiatry, *24:* 119–120, 1971.
3. Gatewood, J. W., Organ, C. H., and Mead, B. T. Mental changes associated with hyperparathyroidism. Am. J. Psychiatry, *32:* 129–132, 1975.
4. Gelenberg, A. J. The catatonic syndrome. Lancet, *1:* 1339–1342, 1976.
5. Morrison, J. R. Catatonia—Retarded and excited types. Arch. Gen. Psychiatry, *28:* 39–41, 1973.
6. Raskin, D. E., and Frank, S. W. Herpes encephalitis with catatonic stupor. Arch. Gen. Psychiatry, *31:* 544–546, 1974.