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Catatonia in Autistic Disorder: A Sign of Comorbidity or Variable Expression?[1]

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Catatonia, once solely attributed to schizophrenia, is now thought to be associated with many disorders. Autistic disorder shares some symptoms with catatonia, namely, mutism, echopraxia/echolalia, and stereotypies. Catatonia in autism may therefore be a variant of the autistic condition. However, organic deficits and psychiatric disorders, such as bipolar disorder, have also been linked with the manifestation of catatonia. Individuals with autism presenting with these comorbid conditions may therefore be at increased risk for catatonia. Little is written of the association of autism and catatonia to clarify the possibility of catatonia as a variant or a sign of a comorbid condition. The authors discuss three autistic patients and suggest specific etiologies for the symptoms of catatonia which presented in these cases. The therapeutic and diagnostic importance of comorbid disorders in autism is stressed.

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INTRODUCTION

Autistic disorder is a neurobiological condition with deviance of social relatedness, communication, stereotypies, and an insistence on environmental sameness (American Psychiatric Association [APA], 1987). The core symptoms of autism do not adequately address a group of problems found in this population which are important contributors to morbidity. Hyperactivity (Campbell, Fish, Korein, Shapiro, & Collins, 1972), aggression (Bemporad, 1979), self-injurious behavior (Coleman & Gillberg, 1985), affective disturbance (Komoto, Usu, & Hirata, 1984), and catatonia (Wing & Attwood, 1987) are a few examples of special management problems for which the autistic population may be at greater risk. Each of these problems may further contribute to the already severe dysfunction of the autistic condition. Of these associated problems, little has been written of the relationship of catatonia and autism (Wing & Attwood, 1987).

Catatonia was first described by Kahlbaum in 1873 as a distinct disease with a cyclic course composed of melancholia, mania, stupor, confusion, and dementia (Kahlbaum, 1974). Catatonia defines a constellation of signs and symptoms characterized by alterations in mental status and motor signs. Abrams and Taylor's criteria described eight motor signs of catatonia including the symptoms of mutism, echopraxia/echolalia, stereotypy, catalepsy, automatic obedience, negativism, posturing, and stupor in their studies of catatonia (Abrams & Taylor, 1977; Abrams, Taylor, & Stolurow, 1979; Taylor & Abrams, 1977). Modern descriptive psychiatry appropriated the term "catatonia" to a subtype of schizophrenia, where it remained until very recently (American Psychiatric Association, 1968; Bleuler, 1908; Kraepelin, 1905). Revisionist thinking in the 1980s attributed the "symptom cluster" of catatonia to multiple disorders (Galenberg, 1976). Recent reports attest to its multiple etiologies. Catatonia has been associated with many psychiatric conditions: bipolar disorder (Ries, 1985) and conversion reactions and associated states (Altshuler, Cummings, & Mills, 1986). Several studies report the association of catatonia with neurologic conditions: epilepsy (Shah & Kaplan, 1980), viral encephalopathy (Penn, Rocy, & Lapham, 1970; Raskin & Frank, 1974), cerebral arterial malformations (Reimer & Nagaswami, 1974), cerebral infarctions (Tippin & Dunner, 1981), posterior fossa atrophy (Joseph, Anderson, & O'Leary, 1985), midbrain abnormalities (Sternbach & Yager, 1981) and Wernicke's encephalopathy (Nielson, 1953). Medical conditions, including subdural hematomas (Woods, 1980), hyperparathyroidism (Hockaday, Keynes, & McKenzie, 1966), myxedema and Addison's disease (Akthar & Buckman, 1977), have also reported catatonic features. Some medications often used to treat catatonia have also been implicated in its causation (Johnson & Manning,

1983). Catatonia therefore appears to be a final common behavioral expression with numerous etiologies.

Catatonia is not part of the diagnostic description of autism, but has been observed in autistic adolescents. Odd hand posture, interruption and freezing of ongoing movements, difficulty in completing action, and catatonic excitement have been seen in minor or marked forms in association with the autistic spectrum (Wing & Attwood, 1987). Mutism is one feature of catatonia which is included in the diagnostic criteria for autism (APA, 1987). Echopraxia/echolalia and stereotypy are others. Because autistic disorder shares some features with catatonia, would catatonia in an autistic individual demonstrate a variant expression of the disorder? Or, since catatonia is associated with multiple disorders, would catatonia in autism suggest the possibility of a separate comorbid condition? The authors collected three cases of catatonia in adolescents with autistic disorder whose description and follow-up many clarify this issue.

CASE REPORTS

Three cases of autistic disorder seen in the Division of Child and Adolescent Psychiatry at the University of Minnesota Hospital and Clinic in the past 10 years are reported here.

Case 1

Developmental History

Pregnancy was complicated by the mother's use of phenytoin secondary to a seizure disorder from head trauma, and delivery was complicated by toxemia, placenta previa, and Cesarean section. Remarkable at delivery of this firstborn, white male infant was bilateral cleft lip, which was repaired uneventfully during his first year. Developmental delays were noted early. The patient walked at 19 months and began speaking at 2 years. He did not notice or respond to people, or imitate the usual social conventions (did not wave by-bye). At 3, he showed only fleeting eye contact and perseverated several words. He had variable reactions to stimuli, at times crying when he heard a light switch or a vacuum cleaner; at other times, he appeared not to notice. At age 6, he demonstrated finger flicking, head banging, and preoccupation with spinning wheels. He was upset, displaying temper tantrums, over change in his daily routine, or if objects were moved in the home. In middle childhood, he regularly insisted on talking about

the activity of birds leading to upset, regardless of whether or not the conversation continued or was terminated.

Psychometric testing at 3 years put him in the borderline to dull normal range of intelligence (Stanford-Binet IQ score of 79). He was diagnosed as autistic at age 6, at which time he had taught himself to read; however, comprehension was very poor.

Treatment

Treatments have included special education since the age of 4. Medications, such as valproic acid and diazepam, were not helpful in modifying temper tantrums. Behavior modification and individual psychotherapy were tried with modest success.

Family History

The mother's family history is unremarkable for psychiatric disorder or epilepsy. The father is currently a very successful businessman, with prodigious musical talent. His parents and siblings are also very successful. There is no family history of depression, mania, suicide, schizophrenia, or retardation.

Presentation of Catatonia

At the age of 16 years 1 month, he presented to the clinic with complaints of auditory and visual hallucinations which were threatening and could result in agitated and aggressive behavior. He withdrew from physical contact and was unable to maintain eye contact; speech was minimal, but when spoken, was very pressured. He was started on haloperidol (1 mg bid) but was admitted to the hospital 4 days later with decreased activity, anorexia, posturing (including outstretching of his arms for upwards of 20 minutes), catalepsy, noncompliance, and muteness. He had previously described to his parents that the hallucinatory experiences prohibited him from eating. He satisfies Joseph et al.'s (1985) criteria for catatonia with mutism, akinesia, catalepsy plus negativism, and posturing.

Course and Laboratory Studies

The patient was hospitalized for 4 days. Haloperidol was increased to 2 mg tid. He was noted on admission to refuse to eat, drink, or void;

he was negativistic, mute with posturing of his arms. A 5-mg dose of haloperidol was given, which resulted in increased eye contact and eating, and decreased posturing and negativism. Routine laboratory studies, including thyroid and vitamin levels, were normal. A magnetic resonance image of the head showed borderline prominent lateral and third ventricles, but was otherwise negative.

Follow-Up

Over the next 4 years, the patient moved to a community living situation. Perseverative and obsessive preoccupation with weather patterns has resulted in episodes of aggressive and self-injurious behavior. He was treated for aggressive and self-injurious behavior with thioridazine (as high as 600 mg/day) with haloperidol (15 mg/day) during an evaluation at another institution. Slowly, his medication was reduced. Three years later while on 400 mg of thioridazine, he had one seizure during a clinic visit. A complete workup, including EEG and electrolytes, was within normal limits. A neurological exam performed 1 hour after the seizure was completely normal. An EEG performed 1 year later showed mild posterior slowing. A computerized tomographic X-ray scan of the head showed the lateral ventricles to be within the upper limits of normal, and the third ventricle to be of normal size.

The patient ($4^1/_2$ years post hospitalization) was on 320 mg of thioridazine, as well as 100 mg of imipramine for anxiety and irritability, with no further seizures. With decreasing medication dosage, he showed increasing agitation, perseverative thoughts, aggression, and self-injury. No further catatonia was observed. Hallucinations, similar to those described above, were reported infrequently.

Case 2

Developmental History

Pregnancy was complicated by vaginal spotting in the third trimester and toxemia, resulting in premature delivery by Cesarean section of dizygotic twins. This firstborn male weighed 4 pounds 13 ounces.

The child did not reach out in preparation to be held within the first year. Motor milestones were reached on time. First words were spoken at 22 months of age and have never included eye contact. At about 33 months of age, speech ceased but later returned with echolalia. He also began to repetitively tap his eyes. He smiled little in response to social interaction

and interest in people including peers was minimal. He withdrew, started spinning the wheels of toys, and imitated the sound of a vacuum cleaner. In middle childhood, he frequently repeated the swishing sounds of windshield wipers. As a youngster, he insisted on eating the same food prepared in the same way. Changes in this or other routines resulted in extreme temper tantrums.

Treatment

He was hospitalized at $3^1/_2$ years of age for a short time. From $4^1/_2$ to $16^3/_4$ years of age, he was treated at the Orthogenic School in Chicago. He was then placed in a community residence for several months, then transferred to another group residence near his mother's home. He had received no medications up until his presentation with catatonia.

Family History

Family history was significant for anorexia nervosa and depression in his twin sister. The paternal great-grandfather and one of his sons were diagnosed with manic-depressive disorder. The son committed suicide. A paternal great-uncle and a paternal cousin were developmentally delayed. A paternal great-grandmother's sister was institutionalized and was paranoid. The maternal great-grandmother and her sister have experienced recurrent depression. A maternal cousin is chemically dependent and depressed; another maternal cousin was manic-depressive and committed suicide.

Presentation of Catatonia

At the age of 21 years 4 months, this young man developed daily episodes of increasing duration, lasting up to several hours of generalized stiffness, upper extremity tremor, and diaphoresis. He remained mute with fixed gaze, except for occasional groaning and rhythmic tapping of his lips with his finger. The patient was unresponsive to commands, refused meals, and lost 17 pounds. He refused to leave his residence, had difficulty initiating sleep, and refused to bathe or change his clothes. He spontaneously produced tears on several occasions. The patient satisfied Joseph et al.'s criteria for catatonia with mutism, akinesia, and catalepsy, plus mannerisms, and negativism.

Course and Laboratory Studies

The patient was hospitalized for 16 days and was noted to "freeze" in one position for several minutes with diaphoresis. A small meal took $1^1/_2$ hours to complete. He responded to haloperidol (1 mg bid) for treatment of catatonia and nortriptyline (75 mg per day) for affective symptoms with gradual improvement.

Laboratory studies, including prolactin, thyroid, and urinary-free catecholamines, were within normal limits. Neurological examination showed no focal deficit. Electroencephalogram and magnetic resonance imaging were within normal limits. Prior to the current problem, he received an IQ of 52 on the WAIS-R.

Follow-Up

After discharge, the patient continued only on nortriptyline and demonstrated only two brief cataleptic episodes of less than 15 minutes. His appetite increased dramatically. His verbalizations, activity level, mood, sleep, and compliance improved. Six months after hospitalization, he is at his usual baseline functioning and the antidepressant medication is being tapered without recurrence of affective symptoms.

Case 3

Developmental History

Pregnancy was unremarkable for this firstborn male, except for moderate edema, treated successfully with diuretics. Delivery and early neonatal course were unremarkable. Motor milestones were on time, but language was delayed and prompted evaluation at age 3. He never used gestures or the few words he acquired to communicate. He did not react to painful stimuli nor did he seek assistance. He ignored people; he looked and walked through them. He was unresponsive to either parent. He pursued the solitary investigation of objects. He avoided eye contact and reacted indifferently to being held. He had difficulty tolerating change, such that he became agitated when dressed in new clothes. Cognitive testing placed him in the moderate range of mental retardation.

Treatment

This individual has been in special education programs since preschool. In late adolescence, he was given intensive vocational training and responsibly completed janitorial tasks. At age 18, he received fenfluramine for 16 weeks with little change.

Family History

History is significant for depression in the mother who was successfully treated with nortriptyline.

Presentation of Catatonia

At the age of 20 years 2 months, he was seen for insomnia, anorexia, and rumination with a weight loss of 20 pounds. He would frequently exhale in long sighs. Vocalizations stopped. Activity level decreased. This young man became cataleptic in doorways or assumed a posture while removing his coat. He was negativistic and noncompliant. He exhibited waxy flexibility. Prior to this problem, his mother described his behavior as "the best period in his life" with heightened energy and improved functioning in his vocational program; he seemed pleased by his activities. He satisfied Joseph et al.'s criteria for catatonia with mutism, akinesia, catalepsy plus negativism, and posturing.

Course and Laboratory Studies

Laboratory tests, including thyroid, electrolytes, liver, and kidney tests, were normal. An electroencephalogram showed nonspecific diffuse cerebral dysfunction. Intelligence testing on the WAIS-R gave an IQ of 32. This individual with autism was treated for 5 months on 100 mg of nortriptyline with increased activity, and cessation of posturing and catalepsy.

Follow-Up

He was able to resume his vocational program. His hygiene and toileting improved. Medication was tapered with continued improvement. Thirteen months later, this young man returned with his mother for an evaluation. His vocalizations had become shouts. He laughed forcefully and

for prolonged periods. He exhibited binge eating and bruxism was persistent. His activity level was increased. During his evaluation, he spent much time quickly picking lint off the carpet. He was treated only with 900 mg of lithium, with a blood lithium level of 0.7 mg/liter, and improvement was noted in 3 weeks At present, he continues normoactive on lithium carbonate with mild side effects.

DISCUSSION

In this report we described three autistic young adults who developed catatonic features. Among the various possible collections of features that taken together constitute catatonia, we chose a definition from a study that sorted catatonic patients from a group of neurologically impaired patients. In this study, Joseph et al. (1985) operationally defined criteria for catatonia to include mutism, akinesia, and catalepsy, plus two of four of the following symptoms: negativism, stereotypy, posturing, and mannerisms. This symptom cluster is similar to the group of behaviorally defined features of catatonia used in Abrams and Taylor's series of studies (Abrams & Taylor, 1977; Abrams et al., 1979; Taylor & Abrams, 1977). Case 1 satisfies these criteria for catatonia. He had both neurological findings and a psychiatric disorder which have been linked with the presentation of catatonia. Evidence of slight structural central nervous system pathology was documented with abnormal magnetic resonance imaging and computer tomographic scanning of the head. Neurologic deficits may have arisen from perinatal problems, such as the use of phenytoin, toxemia, and placenta previa. This patient, while autistic, had characteristics of patients with organic brain dysfunction who have also developed catatonia (Joseph et al., 1985). The subject of Case 1 also had auditory command hallucinations as part of his presentation, for which the diagnosis of schizophrenia should also be considered. Autism was, until recently, thought to be related to schizophrenia (Bender, 1947; Despert, 1938; Goldfarb, 1974), but an understanding of autism as a distinct disorder became clear through differences in family histories (Kolvin, 1971), demographics (Kanner, 1943), and age of onset (Rutter, 1972). However, similarities, overlap of symptoms, and the progression of infantile autism to schizophrenia continue to be reported (Petty, Ornitz, Michelman, & Koh, 1984). This recent report described hallucinations in patients who formerly had a developmental course and symptoms consistent with autistic disorder. Catatonic features were not part of the presentation of schizophrenia from that report. Further complicating diagnostic precision are changes in diagnostic criteria. Hallucinations and delusions were exclusionary criteria for autistic disorder in

DSM-III (APA, 1980). These symptoms would not exclude a diagnosis of autistic disorder in the revised edition, DSM-III-R (APA, 1987). The evidence of neuroanatomical brain abnormalities of the kind described in the first case have also been reported in studies of patients with schizophrenia, again adding to features that overlap with autism (Johnstone, Crow, Frith, Husband, & Kreel, 1976; Pearlson, et al., 1989; Weinberger, Torrey, Neophytides, & Wyatt, 1979). One may speculate in this case that the comorbid condition of organic brain pathology, whether of neurological and/or psychiatric designation, may be responsible for the symptoms of catatonia in this young man with autistic disorder.

Cases 2 and 3 also satisfy Joseph et al.'s (1985) definition of catatonia and share numerous features with each other. Signs of depression were seen, including tears (Case 2) and spontaneous sighs (Case 3); sleep disturbance and anorexia were seen in both. Significant family history for depression, with manic-depressive disorder (Case 2) and major affective disorder (Case 3), is another similarity. Both responded to antidepressant medication. Neither had anatomical (Case 2) or electrical (Case 3) evidence of specific brain pathology. Important differences, however, include perinatal complications in Case 2, but not Case 3, and degree of retardation in Case 3 that is greater than Case 2. Striking in Case 2 was the heavy genetic loading for manic-depressive disorder. It is of note, therefore, that Case 3 developed the manic symptoms of expansive mood, increased activity, increased vocalizations, and easy distractibility, which responded to lithium carbonate about a year after the catatonic episode. A strong association of catatonia with affective disorder and mania has been reported in clinical descriptions, family history (Abrams & Taylor, 1977; Abrams et al., 1979; Halls & Ries, 1983; Taylor & Abrams, 1977), and outcome studies (Morrison, 1974). This suggests that the catatonia seen in Cases 2 and 3 may be a product of a comorbid bipolar disorder. A recent study has suggested a higher than expected association of bipolar disorder in family members of high-functioning autistic probands (Delong & Dwyer, 1988). Since bipolar disorder has been associated with catatonia, this suggests that individuals with autism with comorbid bipolar disorder may be at higher risk for catatonia.

The conclusion, based on these case reports, is that catatonia may be a sign of a comorbid condition in autistic individuals. Catatonia in an autistic individual should alert the clinician to the possibility of a comorbid condition. Autistic individuals who are also burdened with various other psychiatric, neurological, medical, and drug-related conditions may be at greater risk for catatonic episodes due to the panoply of conditions associated with catatonia. Unrecognized manic or depressive episodes with accompanying catatonia may seriously impair function but may respond to

appropriate and specific treatment. Further research into comorbid conditions in autism and response to specific treatments is necessary.

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