Industrial Psychiatry Journal 2003 [illegible]: 65-66

INFANTILE AUTISM

R. SAINI, A.GUPTA, A.M. CHOUDHARY

Childhood autism is one of the most disabling illnesses of neurologic, emotional and intellectual development. It afflicts about one in every 2,000 children. Boys make up about 75 percent of all autistic individuals. About 80 percent of autistic children develop signs of the disorder in the first year of life. By three years, the full-blown syndrome is usually present. Autistic children are generally normal in appearance, healthy and attractive; their life expectancy is normal. Here we present such a case of autism with onset in early childhood.

CASE REPORT

A 4½-year-old boy was brought by his parents with complaints of not speaking, not maintaining eye contact and a tendency to play alone since 2½-years of age. He was the only child born to middle class parents after normal pregnancy, labour and delivery. His early childhood till about 21 months of age was normal in that he achieved adequate level of motor, social and linguistic abilities in which he learnt to walk unassisted, attained adequate bowel control, reciprocated social gestures and had a vocabulary of about 50 word which he used appropriately. However he appeared to be relatively undemanding and placid as compared to other children. He did not cry when he was left unattended while his mother did household chores or when strangers picked him up. Between 21- 24 months, his mother noticed that he was using lesser and lesser words for communication and stopped learning new words. From two-word sentence his language reduced to single word holo phrases followed by even monosyllables and with diminished use of appropriate gestures. By 24 months his speech was further reduced to occasional production of elementary noises, which he made at play, use of gestures and facial expressions to communicate had disappeared and he did not appear to understood simple instructions against an earlier pattern in which he could do that. There was a gradual change in his behavior during this period in that he turned from a convivial, warm and affectionate child into a recluse who preferred to play alone, would be oblivious to the presence of others in the vicinity, would not respond when his name was called and appeared passive when he was held close by his parents. There was a gradual decline in his interests in the diversity of his toys and sources of enjoyment. Earlier he enjoyed playing with toy-car, tricycle, stuffed toys, playing with toolkit of his father's motor cycle and watching cartoons on TV. However, with onset of illness his interests reduced to playing only with toys having wheels or rotating objects like ceiling fan to which he developed unusual fascination. He would rotate incessantly along with the fan without showing any distress and would seem to enjoy that experience. He developed a new fad for eating toothpaste as well as smelling washing power and bathing soap.

The childs other areas of development viz. Motor sermons and adaptive functions developed normally in that he learnt to run around comb furniture items achieved complete bowel and bladder control dressing with minor assistance, taking bath on its own drying himself with a towel after bath, feeding himself using spoon. His sleeping and eating habits and activity levels remained within normal limits. There was spontaneous improvement in child's condition in 6 months before referral in that the repetitive spinning behavior gradually disappeared, he showed some emotional warmth when his father returned from works in the evening and picked him up, he again sorted vocalizing few words like mama and papa spontaneously through not intended at moving communications. However he continued to indulge in solitary play with wheeled toys did not maintain eye contacts, did not respond when called and id not mix with other children or adults. There was no evidence to suggest seizure or hallucinatory behavior, child abuse or neglect.

Physical examinations did not reveal any stigmata of neurological illness. Mental state examination revealed a well kempt individual with placed facial expression who showed no signs of concern or joy when exposed to OPD environment child guidance clinic, did not make eye contact did not respond to verbal commands and co-operated poorly for assessments. He uttered a few elementary noises, during the evaluation, without any intent for meaningful communication. He showed restricted interests in wheeled toys. Though formal testing was not feasible but he appeared oriented had no evidence of hallucinations or delusions. Intelligence as judged from his acquisition of motor and adaptive skills as for his age appeared to be in normal range. Biodrives were stable.

DISCUSSION

The typical signs of autism include: withdrawal, isolation and aloofness; failure to develop language; preoccupation with inanimate objects, such as a spinning top or a light switch; ritualistic behaviors, such as endlessly arranging toys or objects by size, color or shape; repetitive behaviors performed without interruption for extended periods of time, such as hand-flapping

or flicking the fingers in front of the eyes; and an intense aversion to the slightest change, so that even the most trivial disruption of an established routine can cause extreme anxiety and emotional turmoil. All these were seen in the present case.

There is now quite convincing evidence that genetically regulated disturbances in brain development underlie some, perhaps most, of the cases of autism. Autism occurs 50 to 150 times more frequently in families where there was already an autistic member. In monozygotic twins, when one is autistic, the other is also autistic more than 90 percent of the time. Estimates of the heritability of autism derived from both family and twin studies have ranged from 80 to 100 percent. Taken together, these studies provide substantive evidence that a genetic defect in brain development underlies childhood autism (Cook, 2000). Our present case apparently did not have any genetic loading.

One of the most pressing questions scientists have been trying to answer is what part or parts of the brain are damaged in autism? The areas that appear to be prime candidates are the structures of the limbic system and the cerebellum. The limbic system acts as both an augmentation and a switching center, relaying information between brain areas while simultaneously adding emotional coloration to it. In monkeys, destruction of the amygdala, a part of the limbic system, causes a series of behaviors that closely resemble those of autistic children. Another part of the limbic system, the hippocampus, is known to be involved in the acquisition of recent memory. There may be a defect in the normal developmental migration of these neurons, so that they do not reach their proper destinations, and so cannot establish their normal functions. Some investigators have also shown that there is a selective loss of Purkinje cells in the cerebellum of autistic individuals. This result in a loss of the neurons that make synaptic contacts with the Purkinje cells, ultimately involving many centers in the cerebellum. The significance of this observation is not clear, because the cerebellum's principal role is to aid in coordination of motor function and the position of the limbs and body in space. Cerebellum receives extensive connections from the limbic structures and itself sends connections to the cerebral cortex, the major cognitive and information-processing center for the brain. So it is possible that a defect in Purkinje cell development could impede the function of the cerebellum (Bauman, 1909; Baren-Cohen et al, 2000).

Autism represents a severe developmental disturbance of the brain that most likely occurs at the late or end stages of brain development, during which the final connections between brain cells are established that will define the communication network of the mature brain. It is believed that these developmental disturbances giving rise to autism are the result of genetic mutations, occur during the end stages of brain differentiation, and take place in circuits affecting the limbic structures, the temporal cortex and possibly the cerebellum that are responsible for language and information processing, and the emotional coloration that accompanies it (Joseph, 1999).

REFERENCES

Bauman, ML. (1999). Autism: Clinical features and neurobiological observations. In: Tager-Flusberg H. Neurodevelopmental Disorders. Cambridge, MA: MIT Press; 383-399.

Baren-Cohen S, Ring HA, Bullmore ET, Wheelwright S, Ashwin C, Williams SCR. (2000). The amygdala theory of autism. Neuroscience and Biobehavioral Reviews, 24: 355-364.

Cook EH. (2000). Genetics of autism. Mental Retardation & Developmental Disabilities Research Reviews, 4(2): 113-120.

Joseph RM. (1999). Neuropsychological frameworks for understanding autism. International Review of Psychiatry, 11(4): 309-325.

Maj R Saini, Graded Spc. (Psy), CH (WC), Chandigarh; Sqn Ldr A Gupta, Resident, Dept of Psy. AFMC, Pune-411 040.; Dr. AM Choudhary, MD, DNB, Consultant Psychiatrist Pune.