

A clinicopathological study of autism

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ABSTRACT

Brain tissue from six autistic mentally challenged individuals was studied as part of a neuropathological investigation of autism. In cases where the parents had not been seen prior to the patient's passing, diagnostic interviews using standardised questions were done.

Areas of cortical abnormalities were found in four of the six brains, four of which were megalencephalic. The brainstem, especially the

inferior olives, had developmental anomalies as well. In all of the adult instances, there was a decrease in the number of purkinje cells, which was occasionally accompanied by gliosis. The results refute earlier assertions of localised neurodevelopmental problems. They do indicate that the cerebral cortex is probably involved in autism.

Keywords: Autism; Neuropathology; Megalencephaly; Cortical dysgenesis; Brainstem

INTRODUCTION

Autism is a severe developmental disease that typically manifests before the age of three. It is marked by deficits in reciprocal social interaction and communication, confined and stereotyped patterns of behaviour, and specialised interests. About four out of every 10,000 children are affected by the core disease, which is significantly more prevalent in men (4:1) than females. Leo Kanner was the first to describe the syndrome; he considered that children with the condition had normal intelligence, and for many years the ailment was believed to be psychogenic. The discovery that three-quarters of patients are intellectually challenged and that at least one-quarter develop epilepsy first pointed to an organic foundation. As a result, it was frequently believed that autism was an unexpected side effect of brain damage brought on either by medical conditions or obstetric risks. Only a small percentage of instances of autism are linked to medical conditions that cause mental impairment, most frequently tuberous sclerosis or Fragile X. This has only recently come to light. The results of investigations involving twins and families indicate that the great majority of idiopathic cases result from strong unique hereditary effects. As a result, autism typically seems to be a severe form of a certain illness process. Although numerous brain areas have been linked to the development of autism, its neurological underpinnings are still unknown. Only a few post-mortem

investigations have been done on autism, a rare illness that was only recently described. Darby examined 33 different cases but didn't discover any persistent problems. Williams et al. described four cases, two of which had associated disorders (phenylketonuria and likely Rett's syndrome); the other two were idiopathic. In one of the idiopathic cases, there was a reduction in the density of pyramidal cell dendritic spines in the mid-frontal gyrus and a decrease in the number of cerebellar Purkinje cells. Coleman et al. performed cell counts on samples from one case and two control patients in various cortical areas. Although the glia: neuron ratio was lower in the autistic brain than in the two control participants, consistent abnormalities were not discovered.

DESCRIPTION

A recent analysis of this case's brainstem found a superior olive and hypoplastic facial nucleus. Purkinje cell density was measured in the brains of four autistic and four control subjects by Ritvo et al., who found that the counts of Purkinje cells were significantly lower in the autistic subjects' brains (the histopathological results in the cerebral hemispheres and brainstem were not reported). There have been two reports of people who were thought to have autism but were actually quite retarded. The most thorough post-mortem investigation of autism to date was conducted by Bauman and Kemper on six brains. There was a decrease in Purkinje cell density in each case, however the degree of the decrease varied. Three of the four patients had a

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history of epilepsy, which may have been significant. In four of the cases, Purkinje cell density had dropped by 50-95% in some regions. Additionally, there was a qualitative decline in cerebellar granule cell density in two brains. The cerebella nuclei's neurons were larger in the brains of two youngsters and smaller and fewer in number in the brains of three adults. In one brain, the dentate nucleus was misaligned. Although inferior olivary neurons were still present, they were relatively tiny in adults and expanded in younger people. The inferior olivary neurons tended to collect along the convolutional edges in five brains. In the forebrain, the hippocampus, subiculum, mamillary body, septal nuclei, and amygdala were all found to have abnormally tiny, closely packed neurons. Only the first of these six patients' hippocampal neuronal counts has been published. The cerebellum and medial temporal regions have primarily been the focus of neuropathological disorders, therefore speculation about their potential role in autism has been intense. Reduced Purkinje cell density, in the absence of either retrograde olivary cell loss or glial cell hyperplasia, according to Bauman and Kemper, suggested that the cerebellar anomaly emerged at or before 30 weeks of gestation. Vermal lobules I-V of the cerebellum did not show hypoplasia in an MRI investigation of autistic people and control controls. Courchesne's team has claimed that developmental cerebellar abnormalities are the most consistent neuroanatomical lesion in autism and that these abnormalities can cause the defining symptoms in a variety of ways based on the results of post-mortem and neuroimaging investigations. However, using comparable imaging procedures, the localised vermal anomaly has not been duplicated. The idea that medial temporal anomalies are what cause the autistic spectrum has been supported by the observation of temporal horn dilatation on pneumoencephalography. The temporal horn dilatation finding has not been confirmed, and there were no changes between autistic and control participants in the only quantitative MRI examination of the posterior hippocampus. The focus on the medial temporal lobe and cerebellar features emerged mostly as a result of the scant post-mortem evidence of abnormalities in other regions. Nevertheless, the idea of neocortical involvement in autism has been raised because it is linked to epilepsy, abnormal EEG, and mental disability. The primary aims of this investigation were to ascertain whether more severe neuropathological deviations than formerly hypothesised, and to assess the earlier observations. The first four cases in this study's first four cases' brain weights have been reported previously.

CONCLUSION

This series has already revealed more neuropathology than has been previously reported. Increased brain size, anomalies in the development of the cerebral cortex, brainstem, and cerebellum, and in some cases secondary disease were among the findings. There has been no verification of claims of regularly increased neuronal density in the hippocampus. It is doubtful that any discrepancies with earlier findings are the result of a misdiagnosis. Despite the fact that only one person was seen in real life, all of the instances had been given a clinical autism diagnosis. Additionally, after the death, case notes were examined, and the parents were ADI interviewed. The application of the findings to those who are more able is unclear, though, because all of the subjects in this study were mentally disabled. The severity of the early motor impairments, the severity of

the abnormalities, and the presence of Purkinje cell inclusions in Case 1 highlight the variety of autism. Four big brains showed negligible signs of severe oedema. In two cases, childhood macrocephaly was also discovered. There were no gyral abnormalities, although the temporal lobes were enlarged and hyperconvoluted. The cerebral hemispheres, cortex, and cerebellum all had microscopic disease. Individual cases of abnormal cortical development included areas of increased cortical thickness, high neuronal density, neurons in the molecular layer, neuronal disorganization, inadequate differentiation of the grey-white matter boundary, neuronal heterotopias, and focally increased numbers of single white matter neurons. Three brains in the brainstem had faulty inferior olives, and two more had ectopic neurons connected to the olivary complex. In two cases, larger arcuate nuclei were related to ovarian abnormalities. A slight locus coeruleus anomaly was also present in one brain. Inclusions were visible, however Purkinje cell density was decreased in every adult case. There were slight developing cerebellar anomalies in two individuals. Was there any indication of increased density in any of the CA subfields? The hippocampal neuronal density appeared to be very high. In contrast to control patients, there was no evidence of a statistically significant increase in cell density in the cases handled by the Institute of Psychiatry; nevertheless, sampling and the number of cases were small. In any case, there was no hippocampal sclerosis or other abnormalities. Tissue sampling for neurochemistry has restricted the examination of the amygdala, but except from the most recent example, no abnormalities have been found. There was also evidence of acquired disease in other brains. No empty baskets were visible, despite the fact that there were more Bergmann glia and more GFAP staining in three of the cases (the presence of groups of basket cell axons without the Purkinje cell perikaryon that they normally ensheath is typically interpreted as evidence for acquired Purkinje cell loss). In two cases, cerebral subpial gliosis was seen. In the insular cortex (and, in the most recent case, in the molecular layer of the cerebellum), there were more corpora amylacea. The two areas of cortical gliosis were most likely brought on by the childhood head injury.

Investigate the impact of threat, power, and group status in various circumstances. Out group prosocial behaviour toward conflict opponents, particularly in conflict circumstances, has ramifications not only for children's personal development but also for wider structural and cultural change.