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Chapter

Autism: A Neurodevelopmental Disorder and a Stratum for Comorbidities

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Abstract

Autism is a neurodevelopmental disorder which is more common in males than females. It is characterized by social communication disorders and restricted repetitive behaviors. There is wide heterogeneity in its etiology, clinical presentations, management and consequently prognosis. Although the etiology of autism remains unclear, the most currently proven theory is that it is a complex neurodevelopmental disorder that displays "brain network abnormalities". fMRI studies have shown decreased brain connectivity or functional synchronization between frontal and more posterior cortical regions. Dynamic brain activity through high resolution electroencephalograghy (EEG) has revealed local overconnectivity and long-range underconnectivity. This disrupted connectivity pattern would involve connectivity between hemispheres (corpus callosum), together with axonal and synaptic connectivity within each hemisphere. Inconsistent morphometric changes involving both gray and white matter structure also exist. Clinically, autism is associated with multiple comorbidities (somatic, neurologic and psychiatric); some of which are attention deficit hyperactivity disorder, dyspraxia, and sensory processing disorders.

Keywords: autism, MRI findings, comorbidities

1. Introduction

According to the American Psychiatric Association's Diagnostic and Statistical Manual of Mental Disorders (DSM-5), autism spectrum disorders (ASDs) are characterized by social communication impairment and repetitive restricted behaviors. Autism is the commonest neurodevelopmental disorder in the scope of ASD. The social impairment affects both verbal and nonverbal communication [1].

1.1 Social deficits

There is lack of social attention and attention shifting in the autistic children in parallel with lack of development of joint attention skills [2]. The affected children display emotional reactions that do not associate with the surrounding events. They show negative emotions more frequently than positive emotions, without justifiable cause for inducing either response. Their play patterns are solitary, and they do not develop typical interactive social play with other children [3].

Abnormality in face perception is a core feature in autism. Face processing includes unchangeable facial features as those relating to gender and identity and changeable facial features such as emotional expression and gaze direction. Autistic children ignore looking at faces of others and are unable to understand facial expressions. Fixation time on the eye area of the face is reduced in ASD individuals. Opposite to what occurs in typically developing individuals, processing of gaze direction in autism experimentally produced more activation in fusiform gyrus for averted than for direct look. This was termed "covert attention," as autistic individuals are visually attentive and perceptive, but in an atypical manner.

During recognition of neutral faces, the autistic children exhibit a reduced activation of fusiform gyrus, superior temporal sulcus, amygdala, and occipital lobes, the primary areas for face recognition. In spite of this fact, autistic children showed typical activation when looking at familiar faces like that of a mother. Inferior temporal, middle, and inferior frontal gyri are also involved in face processing. It is important to note that reduced connectivity in brain networks between areas of face processing emerged as a holistic approach to explain the atypical face perception in autistic children [4].

The social processing involves social cognition and social motivation. Social cognition involves processes like attention, memory, and theory of mind, by which the person infers the internal state of others. Social motivation resembles directing attention to socially relevant stimuli and enjoying social activities. Both activities depend on the function of face processing. So it is related to the areas of face processing in addition to striatum (social interaction) and orbitofrontal cortex (social motivation) [5]. Impaired connectivity in social executive functions is present in ASD children [6].

1.2 Restricted repetitive behavior

Autistic individuals resist change in their daily routine or the familiar surroundings. They do not explore while playing, and the toys are manipulated with little creativity or symbolic function. They are cognitively inflexible, as they may be preoccupied with parts of objects, or attached to unusual objects or movements, as watching the rotatory activity of fans. They could show stereotypic repetitive behavior that may be injurious to self or others. They tend to have a repetitive sensory motor behavior, insistence on sameness, and sometimes self-injurious acts [7].

2. Body

2.1 Etiologies

Autism has a strong complex genetic basis. Abnormalities in gene expression affect the molecular, synaptic, cellular, and brain network levels. There is variability in results of brain imaging studies [magnetic resonance imaging (MRI) and diffusion tensor imaging (DTI)] in autism, which report structural cerebral changes and functional connectivity disruptions. Alterations in overall gray and white matter volume and in regional lobes and gyri were witnessed.

Early brain overgrowth, especially in an early age of 2–3 years old toddlers remains, however, one of the most replicated findings. Compared with typically developing children, global gray matter (GM) and white matter (WM) volumes were significantly increased and also right superior temporal gyrus regional GM and WM volumes. Higher fractional anisotropy value was also observed in the corpus callosum, posterior cingulate cortex, and limbic lobes of autistic children

[8]. The converging findings of structural and white matter abnormalities in autism suggest that alterations in neural anatomy of different brain regions may be involved in the associated behavioral and cognitive deficits in this disorder.

Nevertheless, recent neural models of autism spectrum disorders have moved the focus from a lesion model to connectivity disorder model. Aberrant conductivity between different brain regions in autism is currently the most frequently addressed neurodevelopmental model. Minshew and Williams [9] have implicated intra-hemispheric connectivity to be mainly involved in the disorder. Vissers et al. [10] have studied functional and structural brain connectivity in individuals with high functioning individuals with ASD. They supported the findings that long-range cortico-cortical functional and structural pathways displayed weaker connectivity in people with ASD than in controls, but with less evidence for local overconnectivity. Other researchers have supported a local overconnectivity and long-range underconnectivity pattern of brain functioning in autism through the use of high-resolution electroencephalography (EEG) [11]. Cortical underconnectivity between brain regions, especially the frontal cortex and more posterior areas, is relatively well established in autism. This supports the view that there is weaker coordination between different parts of the brain that should be working together to accomplish complex social and language tasks. This is opposite to what is occurring during normal development [12].

In this cerebral connectivity disorder of autism, the cerebellum has also been strongly implicated. Although the role of cerebellum as error detector and coordinator of movement and balance was the typical portrait of cerebellar function, yet recognition of nonmotor functions of the cerebellum has recently come into view. While some parts of the cerebellum are predominantly connected to sensorimotor cortex, other connections project to cognitive and affective regions and comprise a large fraction of cerebellar connectivity [13]. Impairment of these connections was also reported in autism.

As a model for aberrant conductivity in autism, we could consider the reported comments about deviations in corpus callosum, white matter, and neurotransmitters. So, brain connectivity includes connectivity between the two hemispheres done mainly by the corpus callosum (CC), or between multiple areas in the brain accomplished by tracts in white matter and by synapses and neurotransmitters. People need this connectivity as different regions of the brain need to communicate in order to identify a face, understand, and respond to others and to different social situations. Disruption of white matter tracts in regions related to social functioning is implicated in autism [14]. In autism, defective joint attention was related to decreased connectivity and synchronization between posterior involuntary attention related to responding to joint attention (RJA) and anterior volitional joint attention related to initiating joint attention (IJA) [15].

The corpus callosum (CC) constitutes the main commissural tract between the two hemispheres (more than 200 million axons). A study by Hardan et al. [16] that investigated the corpus callosum by MRI-based morphometry has identified decreased total volume of CC and several of its seven subdivisions. This was found in other studies and could reflect in the form of social deficits, repetitive behavior, and sensory processing abnormalities.

There are neural circuits for social cognition, which involves attention, memory, motivation, and emotion. Abnormalities in social brain structures and circuitry that are modulated by several neurotransmitters and neuromodulators have been linked, through human fMRI and animal research, to disorders of social functioning as in autism [17]. Neurotransmitter systems involved in autism spectrum disorders have been identified as GABA, glutamate, serotonin, catecholamines, and acetyl choline [18, 19].

In the area of communication abilities, the gifts of memory, understanding, emotional expression, and learning are used on a daily basis. Sometimes, these abilities are disrupted due to deviant central nervous system development (neurodevelopmental disease), which includes long-range underconnectivity and local overconnectivity. Conditions like autism spectrum disorders (ASDs), and attention-deficit hyperactivity disorder (ADHD), can emerge secondary to these disruptions.

2.2 Comorbidities

Autism is in comorbidity relationship with many disorders as epilepsy [20], with intellectual disability and with attention deficit hyperactivity disorder (ADHD) [21]. Other disorders such as fragile X, Rett syndrome, and tuberous sclerosis are also described. Intellectual disability, epilepsy, and ADHD can share a common neurobiological basis and are factors of poor prognosis of autism [22]. Comorbidities are the main reasons for referral to outpatient clinics and admission to hospitals. Among the most challenging co-existing dysfunctions are cognitive impairment, hyperactivity, sensory processing disorders, and dyspraxia. They mask and hinder proper diagnosis and are the cause of inadequate management [23, 24]. That is why Gadow et al. [25] strongly recommend looking at the presence of comorbidities before starting any treatment for autism.

Among the several comorbidities associated with autism, this chapter is going to focus on three commonly encountered conditions: ADHD, dyspraxia, and sensory processing disorders. ADHD, characterized by symptoms of inattention and hyperactivity/impulsivity [1], is frequently associated with autism. The diagnosis of this disorder is difficult to make when present concomitant with autism. In fact, in previous versions of DSM, ASD and ADHD were regarded as distinct disorders. The child was either diagnosed as ASD or ADHD, with a common negative impact mainly on semantics and pragmatics in both of their language profiles. A diagnosis of ASD was considered an exclusion criterion for the diagnosis of ADHD. However, recent research recognizes considerable clinical, genetic, and neuropsychological overlap between ASD and ADHD and within the DSM-5, and ADHD can now be diagnosed in conjunction with ASD.

Both disorders share a portion of their heritable etiology. About 50–72% of the contributing genetic factors overlap between ASD and ADHD. Furthermore, similar deficits in executive function, social cognition, and motor speed have been linked to both ASD and ADHD [26]. Both diseases have similar neuropathology and also share similar symptomatology with considerable overlap in their core and associated symptoms and a frequent overlap in their comorbid conditions. Consequently, it is apparent that ASD and ADHD diagnoses belong to a broader spectrum of neurodevelopmental disorders, an abnormal connectivity spectrum disorder, which results from neural long-range underconnectivity and short-range overconnectivity. Many psychopathological, neuropsychological, brain imaging, genetic, and medical findings have suggested that these disorders are part of a continuum [27].

There are some recorded similarities between these two disorders. First, males are more commonly affected as having ASD or ADHD than females. A review of automated medical records of children revealed the percentages of males evaluated in ASD and ADHD groups were 80.4 and 77.7%, respectively [28]. Second, both disorders are often diagnosed later during childhood. The respective median ages for a diagnosis of ASD or ADHD were 4.7 and 6.4 years [29]. Third, these disorders share symptomatology, showing considerable overlap in the core and associated symptoms, that is, issues with attention, impulsivity, repetitive behaviors, impairments

in socialization and communication, anxiety, sensory processing abnormalities, and ritualistic behaviors, such as counting, ordering, repeating or arranging [30]. Fourth, these disorders share neuropathology.

In clinical practice, the often reported co-occurrence of ASD and ADHD might link them in several pathways: inattention/impulsivity and social ineptness; hyperactivity and stereotypic, repetitive behaviors; and the semantic pragmatic language deficit. The clinical links between ASD and ADHD are variable and strong, as well as the neurodevelopmental basis [31].

Another comorbid disorder that occurs frequently with autism is dyspraxia. Praxis is the ability to conceptualize, plan, and successfully complete motor actions in novel situations. It is a naturally emerging skill that develops as the child interacts successfully with people and objects in the environment and enables the child to learn new skills by watching, imitating, and exploring.

Developmental dyspraxia is the failure to acquire the ability to perform appropriate complex motor actions. It is related to problems of transitive gestures (pantomimed tool use), intransitive actions (symbolic gestures such as waving goodbye), imitative actions (such as imitating meaningless hand or body postures), motor planning, and difficulty conceptualizing novel ways to interact with objects [32]. Many researches have illustrated that children with autism have difficulties in all categorizations of developmental dyspraxia [33].

Autistic children have impaired motor function, including clumsy gait, impairments in coordination, balance, and posture, and abnormal performance of skilled gestures [34]. The deficient performance of skilled motor gestures secondary to command, imitation, or tool use is actually one of the most consistent motor signs in autism [35], which is also consistent with "developmental dyspraxia" [36].

Motor praxis concerns have been reported for children with ASD based on scores from a variety of motor tests and movement observations. Autistic children have been reported to show deficits in their ability to produce meaningful and meaningless gestures on command, imitate demonstrated gestures without objects, and imitate gestures involving real or imaginary tool use. These praxis abilities require the child to interpret sensory information and then formulate internal action models. That is why some researchers suggested that impairments in dyspraxia may contribute to the primary features of the disorder, including impaired social interaction and communication skills [37].

Besides motor praxis dysfunction, speech-language pathologists have observed co-occurrence of childhood apraxia of speech (CAS) with autism. CAS is difficulty in coordinating volitional motor movements that are required for clear and intelligible speech. It can be witnessed in verbal and nonverbal autistic children in the form of defective vowel production, prosody, and difficulty in imitation of speech sounds. This definitely augments the problem of social and language delay in autism and presents a big obstacle in the pathway of verbal language. The possible presence of this obstacle might to be considered and evaluated before the start of therapy because comorbidity between autism and CAS is still vague, and verbal language remains the ultimate goal of success of therapy from the parents' perspective.

A recent research, however, has found autism and apraxia of speech to be highly comorbid. A 3-year study on 30 children with communication delay has shown that 63.6% of children originally diagnosed as having autism had speech apraxia and 36.8% of children originally diagnosed with speech apraxia had autism. The drawn conclusion from this study was that two-thirds of the children initially diagnosed with autism also had apraxia [38]. It is advisable that children with autism are observed for signs of apraxia and children with apraxia are observed for signs of autism. This observation in clinical practice translates to the fact that language delay in autistic children may not be purely of an "autistic" origin.

A third comorbidity commonly occurring with autism is sensory processing disorders (SPDs). Sensory processing means the brain's ability to register, organize, and make sense of the information received through one's senses. SPDs are commonly encountered with autism and have recently been included among the diagnostic criteria in DSM-5. They might even be encountered in children with other developmental disabilities and in typically developing children as well. When sensory processing is dysfunctional, the individual's ability to cope with the demands of the environment would be disrupted [39].

Suarez [39] have drawn a hierarchical classification of SPD, dividing it into three main categories: sensory-based motor disorder (poor motor planning and/or postural instability resulting from improper processing of information from the senses), sensory discrimination disorder (inability to perceive differences and similarities in data received from the senses which can make reading very challenging), and sensory modulation disorder (impairment in intensity and nature of behavior in response to sensory information). The latter subtype is the one commonly encountered in autism, and it has three subcategories: sensory hyperresponsiveness, sensory hyporesponsiveness, and sensory seeking.

Consequently, autistic individuals with SPD can be categorized into hyporesponsive, hyperresponsive, or sensory seekers. The hyperresponsiveness means overreaction to sensations that are typically harmless or not even perceived by others. Inappropriate behavior outbursts may be triggered by feeling textures on the skin (clothes and food), movement activities (swinging), or hearing sudden noises (doorbells). They are overcautious and resist changes in daily routine. The hyporesponsiveness requires intense sensory input to attract the attention of the child as sustained loud sound. Symptoms include not responding to name, or even to painful stimuli. Sensory seeking is characterized by excessive drive for certain sensory stimuli, as putting things in the mouth or touching people to the point of annoying them. Sensory seeking may be injurious or disrupting to the development of meaningful social relationships.

The proposed division of sensory modulation disorder into distinct subcategories serves theoretical understanding of the problem. Clinically, however, the autistic children show a mixture of symptoms that belong to more than one subcategory. They might be annoyed by ordinary sounds to the degree that they cover their ears, and they might be attracted to very fine sounds as the sound of turning of a page, or they might ignore a very loud sound [40]. Some researchers have reported positive associations between hyporeactivity and social communication symptom severity, whereas others have found that child hyperreactivity is likely to negatively affect family life and social adaptive behaviors of school-age children [41].

Questions have arisen regarding the relation of restricted repetitive behavior and sensory processing disorders (SPDs) in autism. Gabriels et al. [42] have suggested the presence of a subgroup with frequent restricted repetitive behavior and multiple abnormal sensory responses due to significant relationship between both. Hyper- or hyporeactivity to sensory stimuli have actually been included in DSM-5 as one of the forms that exist under the title of "Restricted Repetitive patterns of Behavior."

3. Conclusion

Autism is a diverse manifold neurodevelopmental disorder affecting many of the child's abilities. Some disabilities are core features, while others are comorbidities. The clinical picture therefore differs from one child to another. The main deficit in neurodevelopment is that of aberrant connectivity.

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