Rev Bras Psiquiatr. 2011;33:410-411

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RBP Psiquiatria
Revista Brasileira de Psiquiatria
Volume 33 • Número 4 • Dezembro/2011
Official Journal of the Brazilian Psychiatric Association



Letter to the editors

Organic mental disorder after pneumococcal meningoencephalitis with autism-like symptoms

Transtorno mental orgânico após meningoencefalite pneumocócica com sintomas semelhantes aos do autismo

Dear Editor,

There is evidence to support the view that the medial temporal lobe and perhaps, more specifically, the amygdala is likely to be a neural substrate underlying social deficits in autism.[1,2] We would like to report a case of a 24-year-old right-handed woman, with a history of loss of eye contact and facial and nonverbal expression, loss of family relationships, and an absence of social and emotional reciprocity after an episode of pneumococcal meningitis 11 years earlier. Until 11 years of age, she had normal neurological and behavior development. She attended school until seventh grade and had normal social interaction and language development. At the age of 10, her total IQ was 108 (data from school records). At 11 years of age, she received the diagnosis of meningitis. Cerebrospinal fluid (CSF) culture demonstrated the presence of gram-positive diplococci identified as *Streptococcus pneumoniae*. At the age of 24, in the first assessment the patient presented with absence of spoken language, stereotypical behavior, and loss of the ability to self-care. She also had inability to recognize objects and faces. A clinical and neuroimaging evaluation was performed at the age of 24. Klüver-Bucy syndrome signs were negative. The Wechsler Adult Intelligence Scale IQ score was 52 (total), 48 (verbal) and 40 (nonverbal). Magnetic resonance imaging showed bilateral enlargement of the ventricles and temporal lobe atrophy, including reduced volume in the amygdala-hippocampal complex. Using the Structured Clinical Interview for DSM Disorders, it was concluded that the diagnosis was organic mental disorder with autism-like behavioral changes after meningoencephalitis.

Further investigation of the underlying neural substrate within behavioral disorders resulting from early medial temporal lobe damage in monkeys revealed that the extent of the medial temporal lobe involved in the disease may also be of great significance.[3] For example, early damage to the amygdaloid complex yielded a pattern of socioemotional disturbances similar to that described for the combined amygdala-hippocampal lesions, although the magnitude of these disturbances was smaller.[2,3] So far, the experimental results indicate that early damage to the amygdaloid complex appears to be more closely related to the emergence of autistic-like behavior in monkeys than to early damage to the hippocampal formation.[1,2,3]

In this case, we believe that meningoencephalitis produced brain lesions in neural circuits involved in global development disorders (those for which pathogenic mechanisms are established during development) and thus mimicked a picture of autism. An autistic syndrome has been reported after herpes virus infections in teenagers,[4] and there have been reported relationships between autism and congenital infections (rubella, toxoplasmosis, and cytomegalovirus).[5] To date, there was no case report on atypical autism after pneumococcal meningoencephalitis. One limitation of this report is the lack of information on the first years of the patient's life from her main caregiver, her mother, who died two years before this report.

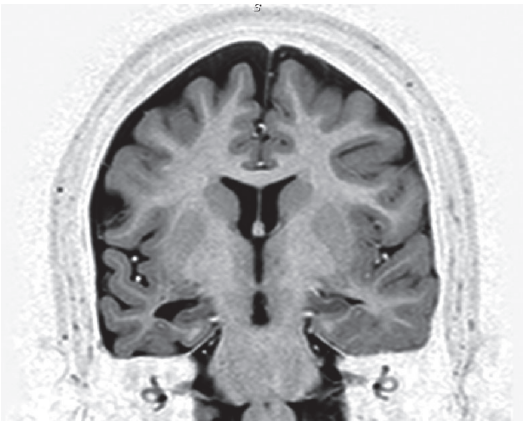


Figure 1 Coronal MRI showing global cortical atrophy, more evident in right temporal lobe. There is right amygdala-hippocampal atrophy.

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In conclusion, we believe that autism-like symptoms must be remembered as possible future consequences of nervous system infections. In addition, this case illustrates the importance of the medial temporal lobe in social development.

Leonardo Baldaçara[1], Thaynne Diniz[2], Bruna Parreira[2], Jaqueline Milhomem[2], Raquel Baldaçara[2]
[1] Universidade Federal de São Paulo (UNIFESP), Universidade Federal do Tocantins
[2] Universidade Federal do Tocantins (UFT)

Disclosures

Leonardo Baldaçara
Employment: *Universidade Federal do Tocantins (UFT); Universidade Federal de São Paulo (UNIFESP), Brazil.*
Thaynne Diniz
Employment: *Universidade Federal do Tocantins (UFT)*, Brazil.*
Bruna Parreira
Employment: *Universidade Federal do Tocantins (UFT)*, Brazil.*
Jaqueline Milhomem
Employment: *Universidade Federal do Tocantins (UFT)*, Brazil.*
Raquel Baldaçara
Employment: *Universidade Federal do Tocantins (UFT)**, Brazil.*
* Modest
** Significant
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