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INTRACRANIAL ARACHNOID CYST ASSOCIATED WITH AUTISM SPECTRUM DISORDER: A CASE REPORT

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ABSTRACT Autism is defined by the association, beginning before the age of three, of a qualitative impairment in reciprocal social interactions, and an impairment in verbal and non-verbal communication. Neuropediatric investigation of autism focuses on the search for an underlying organic cause. In this case report, we describe a child being followed for a cluster of signs consistent with autism spectrum disorder, with minor neurological signs. During his neuropediatric evaluation, a subarachnoid cyst was discovered in the left temporal intracranial, exerting a mass effect on cerebral structures that could explain some of the symptoms. Our patient underwent surgery, which resulted in a significant improvement in the mainly minor neurological signs reported on examination. Arachnoid cysts are developmental anomalies diagnosed in childhood. They are discovered incidentally during radiological examination and are responsible for a variety of symptoms, particularly neurological. Surgical intervention in these cases can improve neurological symptoms, especially those linked to compression of cerebral structures.

KEYWORDS : Autism spectrum disorders, Minor neurological signs, Temporal arachnoid cyst, neuropediatric evaluation, microsurgical fenestration

INTRODUCTION:

Autism Spectrum Disorder (ASD), considered a severe, global disorder of psychic development of multi-factorial origin **(1),** is defined by a set of behavioral disorders that manifest qualitative alterations in the development of reciprocal social interactions and verbal and non-verbal communication with a restricted repertoire of activities. These behavioral disorders are early-onset and should appear before the age of three.

According to the DSM-5 **(2),** autism spectrum disorder is defined by the presence of persistent deficits in communication and social interaction in a variety of contexts, associated with restricted and repetitive behaviors, interests or activities. These symptoms must be present from the earliest stages of development, be responsible for significant clinical repercussions (social, academic or professional), and the disorders must not be explained by an intellectual handicap or overall developmental delay.

The approach to autism needs to be two-pronged: early detection of autism, justified by the mobilizing nature of autistic disorders in their early stages; and the search for an underlying organic cause through a systematic neuropediatric work-up **(3).** The child psychiatrist must confirm the diagnosis of autism spectrum disorder and ensure its therapeutic management **(4).**

METHODS:

This is a case study with iconographic presentation and literature review.

CLINICAL CASE:

K.R. born on 10/07/2014, youngest of two siblings. Followed since the age of six for language delay, significant psychomotor delay associated with difficulties in interaction and communication with behavioral disorders.

K. was born at term by vaginal delivery, with a weight of 3.90 kg, a height of 50 cm and a head circumference of 32 cm. The pregnancy was well monitored and unremarkable. Delivery and postpartum were unremarkable.

The developmental trajectory was marked by early language acquisition, with the appearance of the first words at 1 year and word phrases at 1 year and 3 months. The first motor acquisitions were marked by the onset of walking at 15 months.

From the 18th month, parents noted the appearance of emotional withdrawal and repetitive behavior (opening and closing drawers, a tendency to line up toys, occasional rocking in low moments).

At the age of 3 and a half, the school psychologist suspected an autism spectrum disorder, but the diagnosis was questioned by the pediatrician and child psychiatrist.

Between the ages of 4 and 6, he showed distress towards learning, refusing to go to school. In class, he appeared to be the scapegoat of some of his classmates. According to his teachers, he was described as an endearing but "difficult" child, curious and uncontrollable when frustrated.

For all these reasons, K.'s family consulted the pediatrician again, who confirmed the existence of a disorder but did not give a definitive diagnosis, and K. was then referred to our establishment for further treatment.

The initial child psychiatric examination revealed a child with significant language and psychomotor difficulties, as well as difficulties with interaction, communication and behavioral problems. In addition, K. showed sleep disturbances, separation anxiety, intolerance to frustration, food selectivity, numerous gestural stereotypies and psychomotor instability.

Neuropsychomotor and neuropsychological assessments revealed a number of neurodevelopmental dysfunctions. K. had no voluntary motor disorders of pyramidal origin, but showed minor neuromotor signs as evidenced by synkinesias and hypotonia of the lower limbs. He also had a coordination acquisition disorder of mixed type (ideomotor, visuo-spatial, visuo-constructive) developmental dyspraxia, with impaired inter-hemispheric coordination, global coordination and postural adaptation, against a hypotonic background in the lower limbs. This picture was accompanied by difficulties in motor planning and programming, with a comorbidity of dysexecutive (planning, working memory and inhibition) and auditory and visual attentional disorders.

The assessment also revealed clinical signs such as a tendency to logorrhea, a lack of relational and emotional adjustment but empathy,

and the presence of hand stereotypies.

Following this initial assessment, further investigations were carried out to clarify the diagnostic hypotheses and adapt the therapeutic management.

A psychometric assessment using the WISC-5 concluded that the cognitive performance profile was extremely disharmonious, with deficits in verbal comprehension, perceptual reasoning, working memory and processing speed.

Orthoptic, neurovisual and neurophysiological assessments revealed no abnormalities.

The speech therapy work-up confirmed difficulties with written language, and highlighted problems with syntactic comprehension and a tendency to disorganize free narrative.

Neurological examination revealed no EEG abnormalities, but brain MRI revealed the presence of a left temporal arachnoid cyst exerting a significant mass effect on the temporal lobe (without involvement), with no other structural abnormalities.

The cyst was decompressed by microsurgical fenestration. The hypothesis that this cyst could play a non-harmless role, given its compression on the left temporal and prefrontal structures, may explain at least part of the symptomatology.

Further assessments revealed that, one year after the operation, K. had made significant progress in all the neuro-psychomotor functions reassessed, with disappearance of hypotonia in the lower limbs and better overall coordination, as well as a favourable evolution in praxis despite psychomotor rehabilitation that could not be organized, suggesting at first sight that there was a direct benefit from the surgery. Mixed dyspraxia persisted as a result of attention and oculomotricity disorders, as well as executive function disorders.

Deficits in planning and attention with impulsivity persisted, but hand stereotypies improved.

With regard to language, there was an improvement in evocation difficulties and lexical access, with good verbal comprehension. Exploration of social cognition showed progress in theory of mind and affect recognition.

DISCUSSION:

Through this case report, we underline the importance of a global evaluation of patients presenting with an autism spectrum disorder. It is recommended that, in the presence of multiple neurodevelopmental symptoms, associated with minor neurological signs objectified by a neuropsychomotor work-up, an MRI investigation should be proposed. Returning to the literature, heterogeneous cerebral anatomical anomalies are often reported in patients presenting with autism spectrum disorders (49%, including 8.5% cysts) **(5).**

In this clinical case, we find that part of K.'s symptomatology appears to be linked to the compressive cyst, and the implementation of neurosurgical intervention led to progressive improvement of certain specific disorders linked to the cerebral areas involved, which were expressed neurologically and psychopathologically.

We can still expect a positive future evolution of child K., due to the significant reduction of the cyst after the microsurgical intervention, which could allow a reorganization of the cerebrospinal fluid circulation and of the cerebral structures which will no longer undergo compression. This could lead us to underline the reversibility of certain disorders after decompression as found in certain studies **(6),** or even an improvement in his intellectual efficiency which would be interesting to re-evaluate.

First described in 1831 **(7),** the arachnoid cyst is an abnormal formation of arachnoid tissue during embryonic development of congenital origin **(8).** Forming a kind of pocket with purely arachnoid walls, it can develop wherever arachnoid tissue exists. The arachnoid membrane is one of the three membranes covering the brain. The arachnoid cyst contains cerebrospinal fluid developed within the arachnoid. It's a rare condition, most often discovered by chance in children and adults alike. Its pathogenesis remains poorly understood. It is usually asymptomatic. The clinical symptomatology of arachnoid cysts will depend on the location and size of the cyst, but will always combine signs of intracranial hypertension with those associated with the anatomical location of the cyst. The cyst then becomes asymptomatic, compressing neighbouring cerebral formations. It is often recurrent headaches that are the first warning signs, prompting the prescription of a cerebral MRI or CT scan.

CONCLUSION:

The management of autistic syndrome is the responsibility of child psychiatry. The neuropediatric contribution is highly complementary to the diagnostic approach, in that it seeks to identify an organic disorder at the origin of the autism. The decisive factor in the etiological approach remains clinical analysis, which may find arguments in favor of secondary autism: ante- or perinatal history, dysmorphic signs, skin spots, abnormalities on neuropediatric examination compatible with an underlying central nervous system disorder. In such cases, a neuropediatrics-oriented paraclinical work-up is fully justified.

Such an approach may have specific consequences, linked to the cause of the autism: possible indications for genetic counseling, optimization of the child's care if the cause of the autism leads to a specific therapeutic measure, and ultimately, on the family's experience of the child's illness through the identification of an underlying cause of the autism, which often has a guilt-relieving effect on the parents.

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