CASE STUDY

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COGNITIVE DYSFUNCTIONS IN A MINIMALLY VERBAL (MV) PATIENT WITH AUTISM SPECTRUM DISORDER (ASD)

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SUMMARY

Background: The aim of this study was to examine the neurophysiological correlates of cognitive dysfunctions in a patient with the minimally verbal variant of Autism Spectrum Disorder (ASD + MV), who after reaching adulthood showed progressive deterioration of his cognitive skills.

Case study: The patient was a 25-year-old male, diagnosed with ASD. He never developed spoken language, and communicated only by gesturing or writing on a computer.

Conclusions: Our findings confirmed comorbidity of ASD and epilepsy, accompanied by dysfunction of cognitive control. We also found that spontaneous EEG and event-related potentials (ERPs) in a cued GO/NOGO task can be used to assess functional brain changes concomitant with ASD.

Key words: intellectual disability; epilepsy, event related potentials; EEG; QEEG

INTRODUCTION

Since the publication of DSM5 in 2013, individuals previously diagnosed with autism, Asperger's syndrome, or a range of otherwise unclassified developmental psychosocial disorders are now diagnosed with Autism Spectrum Disorders (ASDs) (Khetrapal 2010; Close, Lee, Kaufmann et al. 2012; Woolfenden, Sarkozy, Ridley et al. 2012; Kropotov 2016; Jack & Pelphrey 2017). Some variants of ASD are characterized by the predominance of particular symptoms, including autism with intellectual disability (ASD + ID), autism with a history of developmental regression (ASD + R), and minimally verbal autism (ASD + MV). Due to the novelty of this approach to diagnosis, there has not been as much research yet on ASD as was done previously on the separate categories of autism, such as Asperger's syndrome, especially in respect to neuroimaging (Kropotov 2016). Estimates of the proportion of children with ASD who are minimally verbal vary from 25% to 35%. However, there is a lack of consensus in defining "minimally verbal," and there are few detailed reports of communication outcomes for these children following intervention (Rose, Trembath, Keen et al. 2016).

A literature search will reveal relatively few studies on ASD + ID, ASD + MV, or ASD + R, and the results published to date have not yet given a clear picture of the correlation between the clinical picture and the neuroimaging results in any of these syndromes (Jack & Pelphrey 2017). While it is certainly true that more studies with larger sample sizes will be necessary, it seems to us more important to examine more than one imaging modality, including studies of event-related potentials (ERPs), which by their nature would enable researchers to look for consistent patterns within clinically diverse samples. We believe that one of the reasons why so little is known about this phenomenon is the lack of interdisciplinary work involving both researchers in neuroimaging and clinicians in neuropsychology.

CASE STUDY

The patient, a 25-year-old right-handed male, was diagnosed with the ASD + MV according to the DSM5 diagnostic criteria. The history derived from clinical interviews revealed that he had never developed spoken language, and communicated only by gesturing or writing on a computer. In childhood he exhibited a particular set symptoms, such as:

- persistent deficits in social interaction and communication;
- deficits in social-emotional reciprocity;
- reduced sharing of interests and emotions;

failure to initiate or respond to social interactions;
deficits in nonverbal communicative behaviors;
abnormalities in eye contact and body language.

It is also characteristic of ASD generally that symptoms were present from early childhood, leading to significant impairment in his social functioning. At the same time, no intellectual disability was diagnosed.

From the age of three to the age of eighteen he was under psychological care. From the very beginning his most pronounced difficulty was inability to communicate. In psychological testing he presented difficulties with processing semantic information. For example, it was manifested in arranging simple patterns in puzzles, because he paid attention only to the shape of individual elements, and was not able to establish relationships between particular elements due to inability to see the semantic meaning of the whole puzzle. It was also observed in the inability to draw objects: his drawings were limited to perseverative lines. He has never developed spoken language, and communicates by gesture or by typing on a computer. The patient functioned well during his school years, when he was undergoing psychological therapy. He could make basic social contacts and communicate using a computer. The communication procedure thus involved two steps:

- His father or his teacher asked questions (he would not acknowledge questions addressed to him by strangers).
- He gave his answers by computer or gesture.
- His written messages were to some extent comprehensible, despite many agrammatisms, lack of punctuation and frequent spelling mistakes (which are relatively rare in Polish, due to a much simpler letter-sound correspondence than in English). He had acquired and developed written language to a level that he was able to finish secondary school, but did not graduate. His visual and auditory abilities were within applicable norms, but some motor and behavioral problems were observed from early childhood. He learned to walk with difficulty, and had problems with balance and coordination. He also exhibited bizarre behavior. From the age of 18 he attended an Educational Center 3 times a week. He successfully communicated with the psychologist at the Center, and for about a year he expressed his joy that he was able to communicate (see Table 1).

This sample shows that language was not fully mastered by the patient. First, many agrammatisms can be noted (more so in Polish than can adequately be represented in English translation); secondly, he does not use punctuation. There are also many paraphasias. For example, he wrote the neologism *lokomonie* instead of the word *kolonie* (“summer camp”), probably referring to the center, though later he seems to mean writing on the computer. He tries hard to find the right form of the word. He also attempts to say that he likes to come to the center because he can communicate with others and be active. At home he feels unhappy due to the lack of activity. He constantly repeats *lokomonie* and “tomorrow” meaning that he would like to come there. His desire to come to the center is so great that he is also ready to accept the suggestion of the psychologist to play with lego blocks. Perseverations are another significant feature, associated with dysfunction of the prefrontal area. Dysfunction in the prefrontal cortex would also explain the tendency to manifest aggressive behaviors.

Deterioration

At the age of 19 his ability to communicate with the use of written language deteriorated. He started cursing and using words expressing aggression. At such

Table 1. Samples of computer communication with center psychologists

Psychologist	**Patient**
Do you want to play computer games?	No
And what do you want to do?	I can write like.
Good. I thought you want to play today. But if you want to write it's O.K.	I enjoy writing with you I feel fine tomorrow tomorrow tomorrow I will already tomorrow tomorrow when I will be I hope to write
And did you find the cabbage soup tasty?	The cabbage soup was sour and I do not like and tomorrow tomorrow do not cook tomorrow tomorrow I count and I do not know tomorrow when *lokomonie* tomorrow no when when me *kolonie* (in Polish *kolonie* means "summer camp"; *lokomonie* is a neologism)
And what will you do when you get back home?	Me is no me when computer *lokomonie* not to have.
Can you write another word explaining what "lokomonie" means?	Tomorrow *lokomonie* I count count when *lokomonie* when
Does it mean that you are watching something on the computer screen?	No *lokomonie* when when cinema poorly me tomorrow when I will not completely when *loko monie*
You are sad that you will not come tomorrow?	Surely [expletive] when me is *lokomonie* when others *lokomonie* when me is and write and when no when *loko onie*
Use another word for lokomonie	*lokomonie* when me is when me is write
Do you mean blocks, those small ones, lego? Do you have them at home?	Surely [expletive] yes I arrange blocks *lokomonie* I may tomorrow when lego blocks
We have no lego blocks, so bring them, we may also buy some.	O.K. I will bring them.

times, his mother noted a specific sort of episode, with rolling eyeballs and loss of any logical contact. An increase in the duration and number of such episodes was observed. In addition, aggressive and auto-aggressive behaviors progressed. The patient was knocking against the door and breaking his glasses, and banging his head against the wall. He was admitted to the neurology department, but clinical EEG did not reveal any paroxysmal activity, and so he was treated with anti-aggressive medication. He was discharged from the hospital without signs of aggression, but was now completely passive. Over time his behavior deteriorated dramatically. He would lie in bed almost all day, and get up only to use the toilet; he did not wash himself and refused to eat, which resulted in the appearance of extreme neglect. His ADL scale went down to 2 points, and he developed cachexia.

Due to these problems, in 2018 his family approached the Reintegration and Training Center of the Polish Neuropsychological Society to ask for help in diagnosing him. He was then tested with HBI methodology, using both clinical EEG and QEEG/ERP, which consisted in recording 19-channel EEG in resting state

conditions (eyes open and eyes closed), and a cued GO/NOGO task, and comparing the parameters of EEG spectra and ERPs with normative and patient databases. Severe cognitive dysfunction was found.

Cued GO/NOGO task

To test the brain correlates of cognitive control, we used a specific variant of the cued GO/NOGO task (Close, Lee, Kaufmann, & Zimmerman, 2012; Kropotov, 2016, 2018; Jack & Pelphrey, 2017). In this task, assorted images from the categories "animal" (*a*) and "plant" (*p*) served as relevant stimuli. The trials consisted in presenting paired stimuli (*s1-s2*) with inter-stimulus intervals of 1000 ms and inter-trial intervals of 3000 ms. Four categories of trials were used: *a-a, a-p, p-p and p-h+novel sound,* where *h* was an image of a human being. The duration of stimuli was 100 ms. The subject's task was to respond by pressing a button with the right hand in response to GO trials (a-a) and to withhold a response in NOGO trials (a-p).

The pictures were selected from textbooks for children in such a way that the overall luminance and the image sizes of animals and plants were approximately equal. To avoid habituation to repetitive stimuli, 20 different images of animals, plants and humans were randomly presented in various combinations. To maintain a certain level of alertness, novel sounds were occasionally presented in "ignore" trials simultaneously with images of a human. They produced an orientation reaction, confirmed by the elicitation of the P3 novelty ERP wave.

The trials were grouped into four blocks of 100 trials each. In each block a unique set of five *a*, five *p*, and five *h* stimuli were selected. Each block consisted of a pseudo-random presentation (requiring an equal number of trials in four categories) of 400 trials, with 100 trials within each trial category. The patient practiced the task before the recording started, and rested for a few minutes after each 200 trials. During the actual trial, the patient sat upright in a comfortable chair looking at a computer screen. Stimuli were presented on a 17-inch CRT computer screen, which was positioned 1.5 meters in front of him and occupied 3.8° of the visual field.

Data recording

The patient's responses were recorded in a separate channel. The average for response latency was calculated, as well as the standard deviation. Omission errors (failure to respond in GO trials) and commission errors (failure to suppress a response to NOGO trials) were also computed. The EEG was recorded referentially to linked ears, allowing computational re-referencing of the data (re-montaging). The EEG was re-referenced computationally to the common average montage. The recording was performed according to the 10-20-system, band-pass-filtered between 0.3 and 50 Hz, and digitized at a rate of 250 samples per second per channel with a 19-channel electroencephalographic PC-controlled system, the "Mitsar-201" (CE 0537) manufactured by Mitsar Co., Ltd. Electrodes were applied using caps manufactured by Electro-Cap International, Inc. that

enables recording from 19 scalp sites. The tin recessed electrodes contacted the scalp using ECI ELECTRO-GEL. Quantitative data were obtained using WinEEG software (Kropotov 2016)

Artifact correction

Eye blink artifacts were corrected by zeroing the activation curves of individual independent components corresponding to eye blinks. These components were obtained by application of Independent Component Analysis (ICA) to the raw EEG fragments. The method is described in Makeig et al. (cited by Percy 2011; see also Anney, Klei, Pinto et al., 2010). Comparison of the method applied in our study with an electrooculogram (EOG) regression technique is described in Tereshchenko et al. (cited by Woolfenden, Sarkozy, Ridley et al. 2012; see also Kropotov 2016). In addition, epochs with excessive amplitude of filtered EEG and/or excessive faster and/or slower frequency activity were automatically marked and excluded from further analysis. The exclusion thresholds were set as follows:

- 100 μV for non-filtered EEG;
- 50 μV for slow waves in 0-1 Hz band;
- 35 μV for fast waves filtered in the band 20-35 Hz (Jack & Pelphrey 2017).

RESULTS

Raw EEG

Several episodes of 3-Hz paroxysms were found in the 40 min EEG recording in the eyes open, eyes closed, and task conditions. Fig. 1 (left) depicts one of these episodes. After application of Independent Component Analysis (ICA) to the EEG fragment, four main components were extracted (Fig. 1, middle). One of those components (Delta) corresponds to the 3Hz paroxysm and is generated (according to LORETA) in the prefrontal and temporal areas (Fig. 1, right).

Behavioral data in VCPT

Table 2 shows some behavioral parameters of the patient in the task (omission errors, commission errors, reaction time, and errors in the variance of response) in comparison to average data from a control group of 53 healthy subjects of the same age (Table 2). Considerable differences in reaction times can be seen between the examined patient and the mean reaction times in the control group. This reflects the substantial difficulties encountered by the patient in performing the task.

Despite the difficulties described above, the number of trials during presentation of the first stimulus was sufficient to calculate reliable ERPs. The ERP of the patient at the Pz electrode (green line) in comparison to the ERP of the healthy control group (red line) are shown in Figure 3 (left). The difference wave of “patient minus healthy controls” (blue line) shows statistically significant fluc-

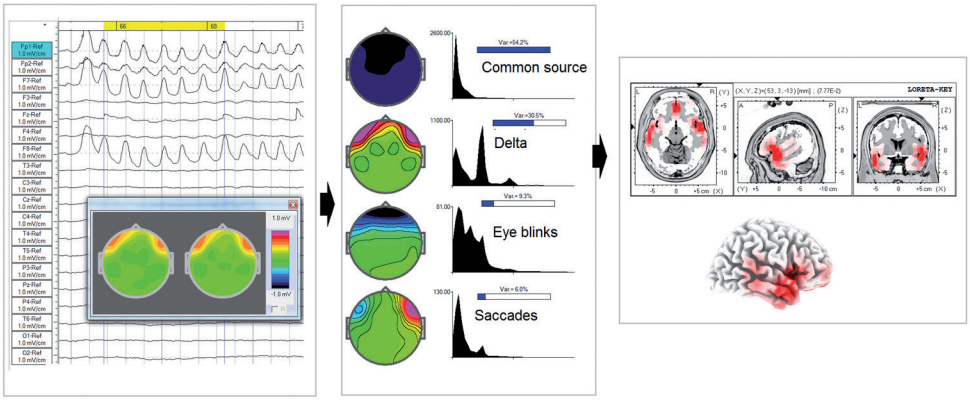

Fig. 1. Burst of 3 Hz paroxysmal activity in the patient. Left: Raw EEG fragment in Eyes open condition. Middle – main independent components extracted from the fragment. Right – LORETA images of the current source densities of the independent component corresponding to the paroxysm.

Table 2. Behavioral data in the VCPT

Data	Omission	Commision	RT	Var (RT)
Patient	80%	13%	558ms	49.8ms
Norms	2.5%	0.7%	420ms	9.8ms
P value	0.00	0.00	0.11	0.00

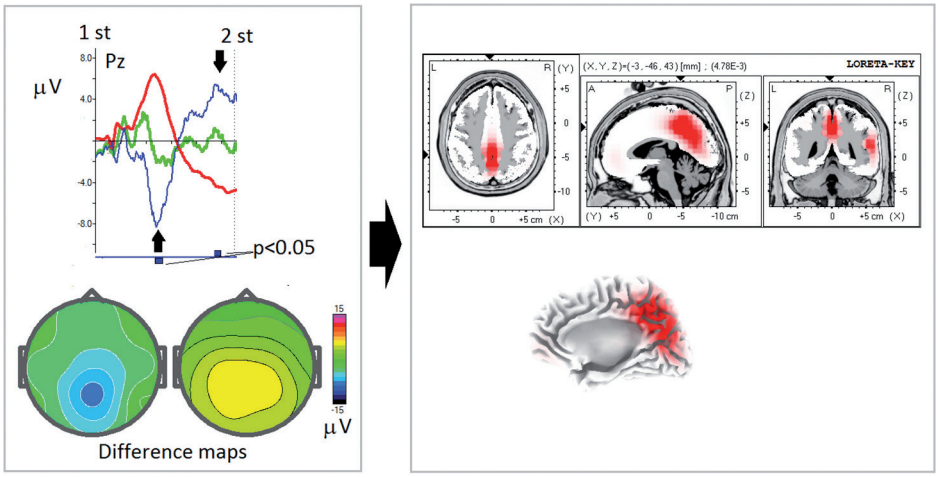

Fig. 3. Event-related potentials (ERPs) of the patient in comparison to healthy controls. Left: ERP to the first stimulus (cue - an image of an animal in the trial) at Pz electrode in the patient (green line), in the group of healthy controls of the same age (N=53), and the difference wave as "Patient-HC" (blue line) with marks of statistically significant difference, and maps of those differences. Right: LORETA images of the difference wave at P3 cue latency.

tuations. One can see that the patient's brain does not produce either the late positive potential (P3 cue) generated in the medial parietal cortex (see LORETA image at the right) or the contingent negative potential (CNV) that in healthy controls is developed before the second stimulus presentation.

DISCUSSION

It is not clear whether the limited linguistic ability seen in children with ASD+ MV relates to deficits in cortical representation of an object and/or in linking an object to the appropriate semantic information (Ortiz-Mantilla, Cantiani, Shafer et al., 2019). Some authors assume that children with ASD frequently present overlapping clinical symptoms that embrace seizures, intellectual disability, developmental delays, cognitive dysfunction, and behavioral impairments, including hyperactivity and impulsivity (Jack, Pelphrey 2017; Close, Lee, Kaufmann et al. 2012; Khetrapal 2010; Woolfenden, Sarkozy, Ridley et al. 2012). While only a handful of studies have yet investigated this issue, current estimates of co-diagnoses in children with epilepsy and ASD stand at approximately 20–25% (Kropotov 2016; Pąchalska, Kaczmarek & Kropotov, 2019) but rise to nearly 40% within the ASD population ((Danielsson, Gillberg, Billstedt et al., 2005; Spence & Schneider, 2009; Gillberg, Billstedt, Sundh et al., 2010; Mouridsen, Rich & Isager, 2011; Woolfenden, Sarkozy, Ridley et al., 2012). A considerable number of studies have suggested that persons with ASD are at higher risk for epileptic seizures. Tuchman, Moshe, and Rapin (2019) argue that autism is more likely to develop in infants with epileptic encephalopathy (Amiet, Gourfinkel-An, Bouzamondo et al., 2008), particularly when infantile spasms are present. Abnormal epileptiform activity on electroencephalography (EEG) has also been documented in approximately 60% of ASD patients who have not had a seizure (Ekinci, Arman, Isik et al., 2010), signifying that those patients are also at high risk for developing epilepsy (Spence & Schneider, 2009; Chez, Chang, Krasne et al., 2006; Kim, Donnelly, Tournay et al., 2006; Hara, 2007; Saemundsen, Ludvigsson, Hilmarsdottir et al., 2007; Anney, Klei, Pinto et al., 2010).

Similarly, mounting evidence has indicated that children newly diagnosed with various epilepsies often exhibit comorbid ASD-like behavioral and neuropsychiatric problems (Kanner 2010), and that early-onset seizures (before 2 years of age) may predict that infants are at high risk for developing ASD (Mouridsen, Rich, & Isager, 2011). Still, establishing a valid estimation of the association between these disorders is difficult, and complicated by the fact that both the diagnostic criteria for ASD and the classification of epilepsy syndromes continue to evolve. Indeed, while Rett syndrome (RTT) was classified as an ASD in DSM4, the recent discovery of its genetic underpinning has caused it to be excluded from DSM-5 in 2013 (Kropotov, 2016). Approximately 70% of RTT patients exhibit seizures, and nearly all show evidence of abnormal EEGs (Anney, Klei, Pinto et al., 2010; Glaze, Schultz & Frost, 1998; Berg & Plioplys 2012). Furthermore, Asperger syndrome (Loscher, Cramer, Ebert 1998) has been renamed as Social (Pragmatic) Communication Disorder (SCD; see Siedler, Gałkowski, Pąchalska 2019). This indicates that SCD is linked to an individual's use of verbal and nonverbal social communication in everyday life. Patients with SCD show little intellectual impairment compared to other patients within the ASD spectrum, are at lower risk for epilepsy, and are usually among the highest functioning (Amiet, Gourfinkel-An & Bouzamondo, 2008).

The changes in ASD diagnosis are likely to affect the degree of overlap between ASD and epilepsy, but there is little doubt that significant comorbidities between these two conditions will continue to exist. It is also generally accepted that children with ASD are at higher risk for seizures if they have an intellectual disability, or when specific neurologic conditions are present, such as tuberous sclerosis or neurofibromatosis. Reverse predictions can also be made in patients with epilepsy, where autism is more likely to develop in infants with epileptic encephalopathy, particularly when infantile spasms are present (Saemundsen, Ludvigsson, Hilmarsdottir et al., 2007). The awareness of the possibility of co-occurrence of these disorders will facilitate their prevention or modification (Pachalska, Kaczmarek & Kropotov, 2019). It is also of vital importance for creating hypotheses, animal model selection, and the experimental design used in basic scientific research. Together, epilepsy and ASD are now estimated to affect more than 10% of the population worldwide, and continue to be among the costliest to society since symptoms generally present in early childhood persist throughout adulthood, and are associated with considerable psychosocial disability.

CONCLUSION

Our findings confirmed the comorbidity of ASD with epilepsy in the patient we studied. At the same time, some other disorders that are commonly observed in clinical practice were confirmed. The most pronounced of these were impairments of executive functions. It is important to point out that the confirmation of these disorders was obtained with the use of hard experimental data: namely, EEG and QEEG/ERP.

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