CASE REPORT

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A new form of neurotherapy for a patient with anxiety disorder and anomic aphasia after neurosurgery for a ruptured brain aneurysm post-COVID-19

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Abstract

Introduction and Objective. The aim of this study is to evaluate the effectiveness of a new, neuromarker-based form of neurotherapy for a patient with anxiety disorders and anomic aphasia after a neurosurgical operation for a ruptured brain aneurysm of the left middle cerebral artery (MCA), detected after COVID-19.

Case Report. A 78-year-old right-handed patient, not previously treated for any chronic diseases except stage II hypertension, contracted COVID-19, confirmed by real time RT- PCR. He was treated on an outpatient basis. Two months later, he developed an unusually severe headache and disorientation. A ruptured brain aneurysm of the left MCA was diagnosed. The patient underwent a neurosurgical operation – clipping– very well, with no neurological or neuropsychiatric disorders, except for mild aphasia and occasional anxiety attacks. Four weeks after surgery, anxiety disorder and mild aphasia worsened. High levels of anxiety on the Hospital Anxiety and Depression (HAD) Scale, and mild anomic aphasia in the Boston Naming Test (BNT) was found. A functional neuromarker of anxiety in comparision to a normative database (Human Brain Index, HBI) was detected. The patient was offered a new, neuromarker-based form of neurotherapy, which proved effective in reducing the disorders. The patient improved in social communication and is gradually returning to social activities.

Conclusion. In patients with anxiety disorders, anomic aphasia and related difficulties in social functioning after aSAH, especially after COVID-19, multidimensional diagnosis and therapy, preferably based on functional neuromarkers, is needed. HBI methodology can be successfully used in the neurodiagnosis and implementation of individualized neurotherapy for such patients.

Key words

clipping of cerebral aneurysm, aSAH, anxiety, HBI methodology

INTRODUCTION

On the last day of 2019 a novel severe acute respiratory syndrome associated with the coronavirus 2 (SARS-CoV-2), causing a highly transmissible and sometimes lethal pneumonia (COVID-19), was first reported in Wuhan, Hubei Province, in Central China [1, 2]. Global statistics show that, as of 31 May of 2023, there have been 767,364,883 confirmed cases of COVID-19, including 6,938,353 deaths, reported to WHO; and a total of 13,355,264 024 vaccine doses have been administered [3]. Although world statistics no longer indicate the number of people cured, an easy calculation shows that 760,426,530 people have survived. It should be stressed that despite the administration of a huge number of vaccines, many people who contracted COVID-19 and survived are still struggling with the various sequelae of the disease [4–7].

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Studies indicate that individuals who had been infected with SARS-CoV-2 and survived COVID-19 may suffer from long-term neurological and psychiatric disorders, in addition to respiratory and other organ deficiencies [6]. Specifically, early evidence suggests that COVID-19 has some **mild outcomes**, which feature:

- loss of smell (anosmia);
- loss of taste (ageusia);
- latent blinks (heterophila);
- headaches;
- dizziness;
- confusion.

There are also **more severe outcomes**, such as:

- cognitive impairments;
- emotional impairments (anxiety disorders, depression);
- seizures;
- delirium;
- psychosis;
- strokes.

The latter list also include only recently discovered and described complications that may be associated with the formation and rupture of brain aneurysms [9, 10]. Research interest has been focused particularly on the epidemiology, mechanism and consequences of such ruptures, which cause a form of haemorrhagic stroke referred to in the literature as aneurysmal subarachnoid haemorrhage (aSAH) [9–11].

The epidemiology of aSAH is difficult to assess; however, as noted by Zacharia et al. [11], it affects up to 30,000 people per year in the United States alone. Etminan et al. [12] conducted a systematic review and large meta-analysis of 75 studies from 32 countries involving 8176 patients, which shows that the global incidence of aSAH decreased from 10.2 per 100,000 person-years in 1980 to 6.1 in 2010, but there are large differences by region, age and gender. The global incidence of SAH decreased by 7.1% with every millimeter of decrease in systolic blood pressure, 11.5% for every millimeter of decrease in diastolic blood pressure, and 2.4% for every percentage decrease in habitual smoking. This global incidence rate has been increased, however, by the possibility of formation, change in morphology, and rupture of an aneurysm after COVID-19 [4]. The prevalence of aSAH is associated with numerous risk factors, both **unmodifiable** (age, gender, ethnicity, family history, location and size of the aneurysm), and **modifiable** (uncontrolled hypertension, BMI, tobacco smoking and use of illegal drugs, hormone replacement therapy) [12].

Cumulative evidence suggests that in COVID-19 there is inflammation and hypercytokinaemia, which can cause degenerative vascular changes predisposing to the formation, change in size or morphology, and ultimately to the rupture of a brain aneurysm, causing aneurysmatic subarachnoid hemorrhage (aSAH) [4, 6, 9, 10]. The authors pointed out that aSAH is a relatively serious complication, with a mortality rate of 45% to 65%. Approximately 12% of deaths occur immediately after aSAH [9]. Approximately 55% of patients survive and regain independent functioning, while 20% require assistance from others in performing activities of daily living [10–13]. More recent studies of large numbers of aSAH survivors show that they may have long-term neurological and neuropsychiatric deficits [14, 15].

In the case of patients who contracted COVID-19, the situation is even more complex, because this disease can cause other diseases or co-occur with them, both during the acute period and long after its end, in the so-called 'post-COVID syndrome' or 'long COVID' [6]. Particular attention should be paid to those patients who after COVID-19 underwent neurosurgery due to a ruptured brain aneurysm. Despite the possibility of early intervention for ruptured brain aneurysms and aggressive post-operative management, which improves overall outcomes, the disease is debilitating, with a mortality rate of up to 50%, and less than 60% of survivors can return to functional independence [13]. The remaining 40% may be functionally dependent due to the overlapping of neurological and neuropsychiatric disorders caused by COVID-19 [6] and disorders related to aSAH, which causes brain damage. The functioning of the patient will be related to the depth and type of symptoms occurring before, during and after the acute period of COVID-19 [6], the aSAH itself (size and location of bleeding into the brain) [9, 10, 14], and the course of the neurosurgical operation, which is always a serious operation on the brain, as the nervous tissue and various patterns of neuronal connections are damaged [15–17]. The patient's health may also be complicated by the occurrence of post-COVID syndrome [18].

Many scientists have suggested that anxiety disorders, depression and fatigue are particularly frequent and debilitating for the patient [19], as they occur in many patients after COVID-19; they have also been reported in almost half of those patients who have survived aSAH. These disorders tend to have a long-term course [20], and may intensify over time [21, 22]. However, they are rarely studied, and there is an urgent need for treatment alternatives, since they significantly reduce the quality of life of patients [21]. This constitutes a serious challenge for modern medicine [6], and requires modern neurodiagnosis and neurotherapy [23].

The aim of our research was to assess the effectiveness of a new form of neurotherapy based on a functional neuromarker of anxiety for a patient with anxiety disorders and anomic aphasia after neurosurgery for an aSAH, detected after recovery from COVID-19.

CASE REPORT

A 78-year-old right-handed patient, not previously treated for any chronic diseases, except stage II hypertension, with no genetic burden and no addictions, was infected with SARS-CoV-2. He presented respiratory symptoms pathognomonic for COVID-19 and a high fever of 39.8 °C. An Abbot RT-PCR control antigen test was positive for COVID-19. He was treated on an outpatient basis. Two months after the infection, the patient developed a violent, very severe headache, which he describes as a 'shot'. He does not remember the events of that day and had difficulty naming many objects. After 5 days, when the headache persisted and nausea appeared, followed by disorientation, the patient was referred by the family doctor to the emergency department (ED) due to the suspicion of a stroke. A CT of the head showed a massive subarachnoid haemorrhage, grade III according to Fisher's scale, in the basal cistern of the brain (Fig. 1).

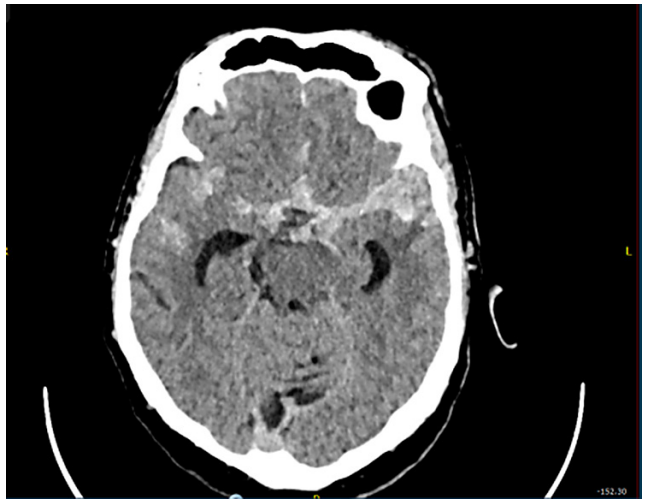

Figure 1. CT scan: massive subarachnoid haemorrhage, grade III according to Fisher's scale, in the basal cisterne of the brain.
Source: own clinical material

The patient was admitted to the Department of Neurosurgery and Neurotraumatology at the Jagiellonian University Medical College in Kraków, for further

treatment, with a diagnosis of aneurysmatic subarachnoid haemorrhage (aSAH). On admission to the Department, clinical examination revealed that the general condition of the patient was not severe: he was conscious, but showed mild disorientation and features of anomic aphasia, he had a stiff neck, but there was no paresis of the cranial nerves or limbs. Laboratory tests showed leukocytosis of 12.17 x10^3/uL. Other blood tests, including a blood coagulation test, were within normal limits. An angio-CT of the cerebral vessels revealed a brain aneurysm at the bifurcation of the left middle cerebral artery (MCA) (Fig. 2).

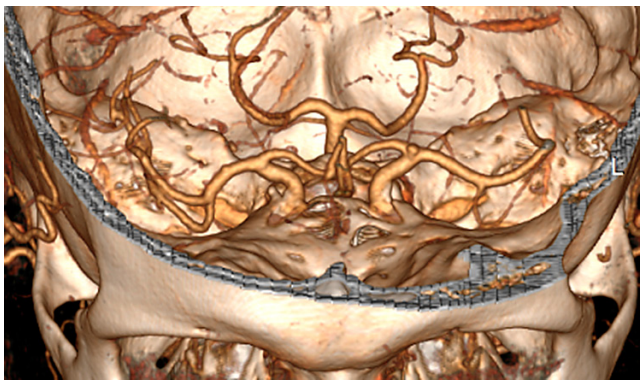

Figure 2. Angio-CT of the head, 3D reconstruction: aneurysm of the bifurcation of the left middle cerebral artery (MCA)

The patient underwent a pterional craniotomy on the left side. The dura mater was opened, and post-haemorrhagic cerebral oedema was visible. The lateral sulcus of the brain was dissected, and under the operating microscope revealed numerous blood clots. After removal of the the clots, a brain aneurysym located at the bifurcation of the left middle cerebral artery (MCA) was visualized. The aneurysm was dissected from the adhesions. A Yasargil titanium clip was placed on the aneurysym neck. The post-operative period was uneventful. The patient was discharged home in good general condition on the 7th day after surgery; at that time he was cardiorespiratory efficient, with signs of mild aphasia.

Four weeks after the surgery, the patient's anxiety disorder and difficulties in communication in social situations worsened. He was referred for further diagnostics and therapy to the Reintegration and Training Centre of the Polish Neuropsychological Society. On admission, the patient complained of severe anxiety, especially in social situations while performing more difficult obligations, mainly due to the need to search for the names of objects. Because he lives alone, this causes him a problem (e.g. when shopping) and therefore results in a depressed mood. The patient reports that the anxiety increasese even more when he tries to re-engage in social activities.

NEUROPHYSIOLOGICAL TESTING

In connection with the patient's medical history presented above, and the results of tests suggesting that the patient additionally had mild anomic aphasia, a high level of anxiety, and a depressed mood, neurophysiological testing was conducted.

EEG recording. The electroencephalogram (EEG) was recorded with the Mitsar 21-channel EEG system (Mitsar Ltd., St. Petersburg, Russia), with a 19-channel electrode cap with tin electrodes that included Fz, Cz, Pz, Fp1/2, F3/4, F7/8, T3/4, T5/6, C3/4, P3/4, O1/2 [24]. The cap (Electro-cap) was placed on the patient scalp according to the standard 10–20 system. Electrodes were referenced to linked ear lobes (off-line) and the input signals sampled at a rate of 250 Hz (bandpass 0.5–30 Hz). The ground electrode was placed on the forehead. Impedance was kept below 5 kΩ. The patient sat upright in a comfortable chair looking at a computer screen (17 inches) 1.5 meters in front of him. All recordings were made by the last author of this article. The ERP wave forms were averaged and computed off-line, and trials with omission and commission errors were automatically excluded.

Behavioural task. The task consisted of 400 trials sequentially presented to the patient every 3 seconds. Three categories of visual stimuli were used:
1) 20 different images of animals – referred to later as **A**;
2) 20 different images of plants – **P**;
3) 20 different images of people of different professions (presented together with an artificial 'novel' sound) referred to as **H**.

The trials consisted of presentations of pairs of stimuli with inter-stimulus intervals of 1 s. The duration of the stimuli presentation was 100 ms. Four categories of trials were used: **A-A**, **A-P**, **P-P**, and **P-H**. In the trails with A-A and P-P pairs, the first and the second stimuli were identical (physically the same). The trials were grouped into 4 sessions with 100 hundred trials in each session. In each session a unique set of 5 **A** stimuli, 5 **P** and 5 **H** stimuli was selected. Each session consisted of a pseudo-random presentation of 100 pairs of stimuli with equal probability for each category and each trial category [23, 24]. The task of the patient was to press a button with the right hand to all **A-A** pairs as fast as possible, and to refrain from pressing in response to other pairs. The patient performed 10 trials without recording to see if he understood the instruction, and recognized the objects. He rested for a few minutes after completing 100 trials. Stimuli occupied about 3.8° of the visual field around the centre of the screen. Visual stimuli were selected to have similar 2D sizes and luminosities.

Artefact correction procedures. Eye blink artefacts were corrected by zeroing the activation curves of individual independent components corresponding to eye blinks. These components were obtained by the application of Independent Component Analysis (ICA) to the raw EEG fragments [23, 24]. 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:
1). 100 μV for non-filtered EEG;
2). 50 μV for slow waves in 0–1 Hz band;
3). 35 μV for fast waves filtered in the band 20–35 Hz.

In addition, the recordings were visually inspected and the remaining artefacts excluded.

Hypothesis. Because the patient complained not only of anxiety, but also of a depressed mood, 2 working hypotheses were tested to find a neuromarker of anxiety and depression.

1) *Hypothesis of anxiety.* In ERPs of patients with general anxiety, an increase of P1 wave in response to visual stimuli in general and to emotionally meaningful stimuli in particular, will be observed. Enhancement of the visual P1 wave will be interpreted as the effect of activated amygdala at early stages of visual processing [23].
2) *Hypothesis of depression.* According to this hypothesis, some patients with depression show excessive frontal alpha at the left side, and consequently reveal a frontal alpha asymmetry [24].

For testing these hypotheses, EEG spectra of the patient were computed and compared for all conditions to 100 healthy subjects taken from the normative database of the Human Brain Institute (HBI) in Chur, Switzerland, age-appropriate for the patient. EEG spectra were separately computed for Eyes Open, Eyes Closed and the GO/NOGO task conditions. The artefact free fragments of EEG were divided into 4 sec epochs with a 50% overlap. The Hanning time window was used. The EEG spectra were computed for each epoch and averaged, and the mean value and standard deviations for each 0.25 Hz bin were computed. For comparison of the EEG spectra before and after intervention, the t-test was used [24, 25].

EEG SPECTRA

In the clinical EEG no paroxysms were found in the patient. However, clear spectra asymmetry was observed, indicating lesions of the left side of the brain, that is, **excess of low alpha at C3** (Fig. 3). EEG was characterized by posterior alpha rhythms enhanced in the eyes closed condition: the frequency of the dominant rhythm 9.8 Hz was within normal limits (8–12 Hz) (L>R). EEG spectra deviations from the reference indicate idling of the left Rolandic fissure. The ERPs deviations from the reference indicate **hypersensitivity of neurons in the C3 area.**

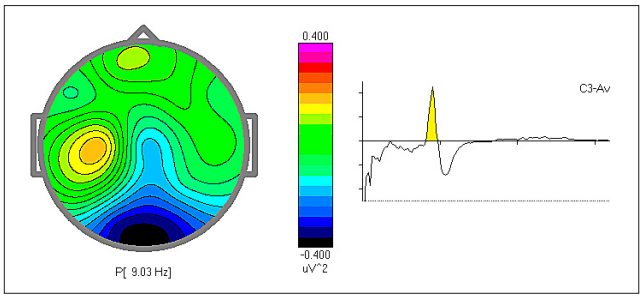

Figure 3. Spectra differences and maps for the largest deviation from normality. The difference with p-values is shown in vertical bars

It was also found in the results that the spectra of the independent component and sLORETA image (Fig. 4) corresponding to the deviation from norms.

EVENT RELATED POTENTIALS (ERPS)

Event Related Potentials (ERPs) maps and time courses of the largest deviations from normality are presented in Fig. 5. Analysis of the data showed that there was a significant difference in the performance of the PsyTask with a GO/NO GO stimuli paradigm, compared to the norm. This applied to both omission and commission errors. Task execution time was slightly longer than the norm and amounted to 446 ms.

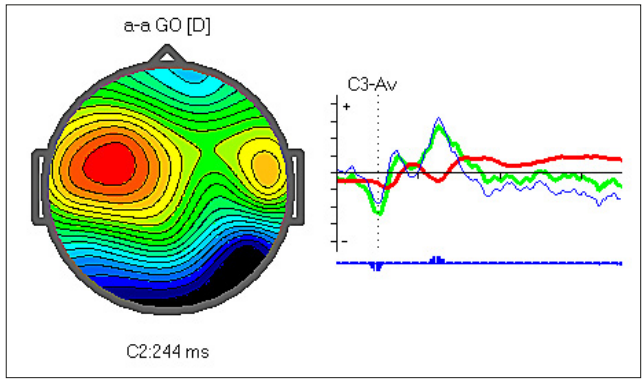

Figure 5. Event Related Potentials (ERPs) maps and time courses of the largest deviations from normality.
Green – client; Red – normative data; Blue – the difference with p-values.

The results presented above allow confirmation of hypothesis 1: **the presence of an anxiety neuromarker**, and refutation of hypothesis 2: **the absence of a depression neuromarker**. The detection of the neuromarker of anxiety motivated the attending physician to refer the patient for neurotherapy.

NEUROTHERAPY – AUTHORIAL COGNITIVE NEUROFEEDBACK

The patient was offered the authors' own programme of cognitive neurofeedback (EEG Biofeedback), also known as Neurotherapy. In accordance with the global standards in this field proposed by Kropotov[24], the data obtained in qEEG and ERs studies, including a functional neuromarker of anxiety, were used to prepare the neurotherapy protocol. The patient received 20 sessions of Neurofeedback: electrodes bipolar at C3 and Cz, training of alpha DOWN (normalization of activity to 4–12 Hz). Kropotov [24] pointed out that this is an activation protocol intended to de-activate the left Rolandic fissure [25]. The therapeutic videos developed for neurotherapy containing various social situations and gentle background music adapted to the patient's needs. The patient was placed in front of a computer screen and asked to relax and watch a therapeutic video. When abnormal brain work decreased slightly, and the

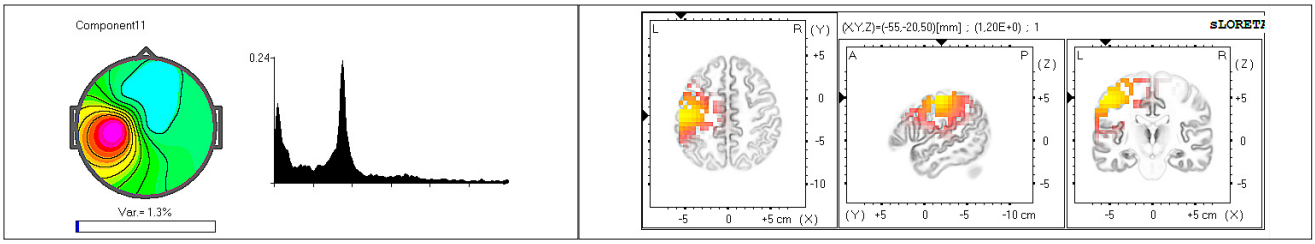

Figure 4. Spectra: Spectra of Independent component and sLORETA image corresponding to the deviation from norms.

normal brain work increased, the image was clear, no objects were blocking the screen (e.g. people at the bottom of the screen), and the pleasant sound of music was heard [25, 27]. The training procedure was modified: during one therapeutic meeting, the number of therapeutic videos shown was adjusted to the patient's abilities (1 – 5 videos). Five of the videos had a programme in which there were various objects, such as animals, plants, and people; after its completion, the patient was asked to identify which objects he remembered or which he liked. It should be noted here that a similar procedure, but not combined with neurofeedback, is used in Visual Action Therapy (VAT) to treat anomic aphasia [26]. The next session was started by asking the patient which objects he remembered from the previous session. Neurofeedback equipment by Elmico was used in the training [27].

NEUROPSYCHOLOGICAL TESTING

The Polish version of the Boston Naming Test (BNT) [28] was used to study confrontational word retrieval. This is an adaptation of the test introduced in 1983 by Edith Kaplan, Harold Goodglass and Sandra Weintraub [29], a widely-used neuropsychological assessment tool for measuring confrontational word retrieval in people with aphasia, or other language disorders caused by a variety of brain injuries [30–32]. The BNT contains 60 drawings graded in difficulty [29–32], because patients with anomia often have greater difficulties with naming

In examination 1, before neurotherapy, the patient named 51 of 60 drawings, which means mild anomic aphasia. In examination 2, after neurotherapy, the patient named 59 of 60 drawings, which is the norm for his age, indicating no anomic aphasia.

Anxiety symptoms were assessed with the Polish version of the Hospital Anxiety and Depression Scale (HADS) [33],which shows good validity and reliability [34, 35]. This screening instrument contains 7 symptoms for anxiety and 7 symptoms for depression. The maximum score for each subscale is 21. The cut-off score was set at 8 for both the Anxiety and Depression subscales of HAD, as this value reflects symptomatology indicative of clinical depression or anxiety disorders [36–38]. The results obtained are illustrated in Figure 6. In examination 1, the patient scored 17 points for the anxiety subscale, which placed him at the lower limit of severe anxiety, and 7 points for the depression subscale, which indicated no depression. In examination 2, after neurotherapy, the patient obtained a score of 11 points on the anxiety subscale, which placed him at the lower limit of mild anxiety, and 3 points for the depression subscale, indicating a complete lack of depressive symptoms.

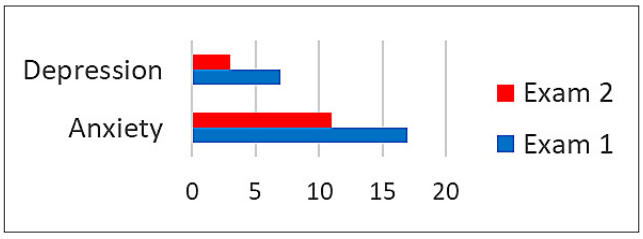

Figure 6. Profile of Anxiety and Depression symptoms subscales in the examined patient.
Score: 0–7 points (Normal); 8–10 points (Mild); 11–15 points (Moderate); 16–21 points (Severe)

To sum up the obtained results of the neurophysiological and neuropsychological tests, it would appear that the neurotherapy applied contributed to the improvement of brain function in the patient. It is not surprising, therefore, that a reduction of anxiety and the disappearance of the symptoms of mild anomic aphasia were obtained, resulting in the marked improvement in the patient's functioning in social life.

DISCUSSION

Both the SARS-CoV-2 coronavirus infection itself, causing the symptoms of COVID-19, and the sequelae of this disease, are pressing problems for public health. Although most cases do not result in severe illness requiring hospitalization, there is growing evidence that SARS-CoV-2-induced inflammation can exacerbate pre-existing diseases or cause new ones [6]. As relatively recent reports have shown, it can also lead to the formation, altered morphology and rupture of aneurysms [39].

To the best of our knowledge, the description presented here of a patient with neurosurgically-treated aSAH associated with COVID-19, who was offered a new form of neurotherapy, based on the functional neuromarker of anxiety, is the first worldwide. This description is extremely important for modern medicine, mainly because it allows us to discuss the interrelationships between:

- the history of SARS-CoV-2 infection and COVID-19, as a possible cause of the onset of aSAH;
- mild anomic aphasia and the onset of aSAH;
- the cause of anxiety disorders;
- the link between anxiety disorders and social communication difficulties;
- the introduction of new forms of neurotherapy based on functional neuromarkers; and
- the improvement observed in the presented patient.

THE RELATIONSHIP BETWEEN COVID-19 AND ASAH

Although COVID-19 is a primary respiratory disease, complications related to systemic vascular involvement are common [37]. Cerebrovascular haemorrhagic events, both stroke [8] and aSAH [11, 12], are not uncommon in COVID-19. Several mechanisms may play an important role in the pathogenesis of aSAH in COVID-19, such as:

- viral endotheliopathy, i.e., damage to the vascular endothelium following viral infection;
- a cytokine storm following inflammation;
- coagulopathy associated with abnormalities in the coagulation cascade;
- unregulated hypertension, and
- immunologic abnormalities associated with inadequate immune system reactivity, such as inadequate production of autoantibodies [38–41].

Clinical trials and studies in the last 2 years have introduced the terms 'post-COVID-19' or 'long-COVID' syndrome, which is a consequence of an excessive, prolonged immune response [18, 33]. Symtomatology in this syndrome can involve many different systems and organs. If the blood-brain barrier (BBB) is breached, the impact affects the nervous system, including various areas of the brain [6, 18, 41]. Complex

neurological, neuropsychiatric and neurocognitive problems will then arise, the nature of which is still under research. These are referred to as 'NeuroCOVID' [18, 40–42]. The literature and data on other similar inflammatory conditions in viral diseases (SARS and MERS) and concomitant vascular damage have illuminated several possible explanations for the changes that may explain the pathogenesis of brain aneurysm in or after COVID-19 [2]. Although the direct effect of COVID-19 on the formation and rupture of brain aneurysms is still not fully explained, an indirect link appears to be the inflammatory processes discussed above [39]. It seems clear that COVID-19-induced hypercytokinemia can be considered as a factor degrading the integrity of cerebral vessels, which predisposes to the formation, malformation or rupture of cerebral aneurysms. Case reports of patients who developed aSAH after COVID-19 infection have also provide much information in this regard [4]. Such reports have revealed that aSAH can occur in patients both after a mild course of COVID-19 with flu-like symptoms, and after a severe course with pneumonia, acute respiratory distress syndrome (ARDS) and sepsis [39–41].

In the search for the causes of aneurysm formation and rupture, some authors have attempted to correlate the age of onset and the type of aneurysm. Dodd et al. [39] identified a total of 10 patients – 5 women (50%) and 5 men (50%), median age – 38.5 years. Four of the 10 patients (40%), despite being infected with SARS-CoV-2, had no symptoms related to the infection, 3 patients (30%) had mild to moderate symptoms, and 3 patients (30%) had a severe course of COVID-19, with pneumonia, ARDS and sepsis. Four of 10 patients developed dissecting pseudoaneurysms (40%), 3 in the posterior circulation and 1 in the anterior circulation. Among the 6 saccular/pseudoaneurysms, 4 (67%) were ≤4 mm in diameter.

The authors concluded that COVID-19 may be associated with aneurysm formation, but its morphology, compared with traditional aSAH, shows a higher incidence of small aneurysms and dissecting pseudoaneurysms, especially in young patients. However, the age of patients who developed aSAH varies. Studies of the natural history of aSAH show that 8 of 10 patients (80%) were under the age of 50. However, in the International Subarachnoid Aneurysm Trial (ISAT), only about 40% of patients were younger than 50, while 60% of patients were older [43]. The authors conclude that, with the current state of knowledge, it is impossible to conclude unequivocally that a history of COVID-2 disease is directly associated with aSAH. It is also difficult to determine the effect of age on the appearance of aSAH after this disease. This is because there are no neuroimaging studies of individual patients prior to SARS-CoV-2 infection to conclusively demonstrate or preclude the presence of a pre-existing aneurysm.

It is also worth noting that there are many factors responsible for the formation and rupture of cerebral aneurysms, e.g. both genetic and vascular, including uncontrolled hypertension, traumatic brain injury, hormone replacement therapy, and nicotine addiction, which are not relevant in the case presented here [3]. Since the patient we studied did not have a family history of aneurysms, as well as no significant risk factors other than grade II hypertension, we can speculate that aSAH may be related to the recent history of SARS-CoV-2 infection and COVID-19 disease. Dodd et al. [39] have argued that not only active, but also recent COVID-19 disease may promote the formation and rupture of cerebral aneurysms. However, we cannot be absolutely certain about this. Therefore, we should agree with Fiani et al. [4] that great caution is needed when demonstrating a relationship between COVID-19 and brain aneurysm formation, malformations or aSAH: further differentiated, long-term retrospective studies on a larger patient population are needed to confirm the existence and nature of any such relationship.

MILD ANOMIC APHASIA AND THE ONSET OF ASAH

Anomic aphasia, which was found in the patient we studied, is one type of fluent aphasia, along with Wernicke's aphasia, transcortical sensory aphasia and conductive aphasia [23, 28–30]. It primarily consists in difficulty naming objects and finding words in real-time communication, i.e. in social situations [44]. It can occur as a consequence of various injuries to the brain or the neuronal connections used in the naming process [28, 45]. Our patient suffered from mild anomic aphasia detected by clinical examination before neurosurgery. This diagnosis was confirmed by the Boston Naming Test (BNT), in which the patient, prior to therapy, had mild naming problems and relatively well preserved speech fluency, repetition and comprehension. These disorders can be explained by the damage to the brain tissue and neuronal connections associated with the location of the ruptured brain aneurysm in the blood supply area of the left cerebral artery [37], and in consequences the hemorrhage of a large area of the diffuse network of neuronal regions in the left frontal, temporal and parietal cortex.

Neuroimaging studies conducted by other researchers have found that proper naming requires efficient, distributed neuronal networks mainly in the left frontal, temporal and parietal cortex [36–38]. Other studies have shown that one of the correlated areas of neuronal networks related to naming is the anterior cingulate or frontal lobe [46]. It has also been suggested that anomic aphasia may be associated with damage to the arcuate fasicle (AF) of the left cerebral hemisphere (in right-handed individuals) following aSAH [47]. However, since frontal lobe damage is associated with Broca's aphasia, a non-fluent form of asphasia, further evaluation and reinterpretation [48–50] of the cause of anomic aphasia is required. However, it would be necessary to use a methodology that images neuronal networks of the brain, such as tractography [45].

THE CAUSE OF ANXIETY DISORDERS IN OUR PATIENT

Anxiety is a reflection of everyone's experiences as, since any serious illness or injury is associated with an increase in anxiety in the patient [51]. However, people with anxiety disorders have intense, excessive and persistent worries and concerns about coping with everyday life, especially social situations. Pąchalska et al. [27] report that anxiety disorders often involve repeated episodes of sudden feelings of intense anxiety and fear or dread that culminate in brief or prolonged panic attacks. These symptoms interfere with daily activities; they are difficult to control, are disproportionate to the actual threat, and may appear over time following the illness. Anxiety disorders can take the form of generalized or social anxiety (social phobia), or other specific phobias and anxieties, including separation anxiety. A patient may also present with more than one anxiety disorder. Anxiety

disorders are often associated with somatoform disorders, such as in COVID-19 [51–55]. The causes of the anxiety disorder in the patient in the presented Case Report are not simple to describe or obvious to explain. After all, his anxiety disorder may be related to his COVID-19 experience, and fit into the symtomatology of the post-COVID-19 syndrome, or it could have been associated with aSAH formation, or with the neurosurgery itself. These issues will be discussed below.

The genesis of anxiety disorders in the aftermath of COVID-19 is most clearly indicated by data from the Office for National Statistics, which reported a sharp increase in anxiety/depression symptoms in the general population (regardless of infection status) compared to pre-pandemic data, after adjusting for socio-economic factors [51]. It should be added that there are more than a dozen reports showing that COVID-19 survivors are at increased risk of developing mood and anxiety disorders at 3 months and even later [42–44]. Klaser et al. [54], in a carefully designed study, assessed symptoms of anxiety and depression using 2 validated questionnaires in a large cohort of 413,148 people between February – April 2021. In this group, 26,998 tested positive for SARS-CoV-2, while the rest of the individuals tested negative. The authors adjusted for physical and mental prepandemic correlates, body mass index (BMI), age and gender. The results revealed that anxiety and depression were more prevalent in SARS-CoV-2-positive individuals (30.4%) than in SARS-CoV-2-negative individuals (26.1%). With this study, the authors show an increased correlation between SARS-CoV-2 infection and symptoms of anxiety and depression. This allows us to assume that the anxiety disorders present in the Case Report patient could be related to the SARS-CoV-2 infection itself and the COVID-19 experience.

Another reason that may have caused the onset (or exacerbation) of anxiety in the presented patient could have been his condition after aSAH. Tang et al. [19] found that symptoms of anxiety and depression occur in almost half of the patients who survived aSAH. Moreover, these disorders tend to have a long-term course. These authors used standardized test to study persons receiving outpatient counseling 3 months after aSAH and living independently in the community, including the Beck Depression Inventory-II-NL and the State Anxiety Inventory, adminstrated after 3 months (T1), one year (T2) and 2–5 years (T3). It turned out that depressive symptoms were present in 39% (T1), 41% (T2) and 54% (T3) of patients, while anxiety symptoms were present in 52% (T1), 48% (T2) and 53% (T3). Of the patients with depressive symptoms at T1, 72% still had symptoms at T3, compared to 67% for anxiety. Depressive symptoms and anxiety at T1 were predictor variables for anxiety at T2 (variance explained by 43%). The authors concluded that the prevalence of depressive symptoms and anxiety remains high during the first 2–5 years after aSAH.

The existence of a bi-directional association between anxiety and aSAH survival was also demonstrated by Moris et al [46]. These authors found anxiety and depression levels twice as high among 70 patients who survived aSAH. In a study 16 months after the second haemorrhage, moderate or high levels of anxiety were found in about 40% of patients, and about 20% experienced moderate or severe levels of depression, compared to the first study on admission to hospital shortly after aSAH. Standardized measures of anxiety and depression were not associated with haemorrhage severity, but were significantly associated with a return to normal pre-surgical functioning (return to work and involvement in social activities). The results of this study are consistent with the anxiety of the patient in the the Casre Report, which appeared 2 months after aSAH and worsened further several weeks later, especially when he tried to re-engage in social activities.

It should also be mentioned that anxiety can co-occur with depression and even be an axial symptom of it [16, 56]. Depression was common even several years after aSAH, and was associated with female gender, pre-disease depressive symptoms, anxiety, psychoactive substance use and any psychiatric disorders and coping styles. Co-occurring cognitive impairment, fatigue and physical disability also increased the risk of depression. Depression reduced quality of life in patients after aSAH. However, this topic will not be elaborated here, because the patient in the Case Report did not have this disorder on the HAD depression subscale.

The results of the studies presented above do not allow for an unambiguous explanation of the cause of the anxiety disorder in the presented patient. It is possible that all the factors mentioned contributed to the formation and development of these disorders. There is a possibility that it was a post-COVID anxiety, but it is more likely that it was associated with having undergone aSAH and neurosurgery. However, it should be borne in mind that the cerebral incident and neurosurgery may both have triggered and exacerbated the already existing anxiety disorder, insofar as it can be linked to the post-COVID-19 syndrome.

LINKING ANXIETY DISORDERS AND SOCIAL COMMUNICATION DIFFICULTIES

The literature has shown links between anxiety and word-finding skills in real-time communication [57], especially when it is necessary to name objects or remember names [58]. The lack of such an important social skill as naming can lead to negative reactions from others who may expect clear and legible messages, especially in difficult social situations [59, 60]. These relationships can be illustrated by the reciprocal correlations between:

1) increasing levels of anxiety disorders;
2) difficulties in finding names;
3) difficulties in social communication, and ultimately
4) difficulties in social functioning [57].

The association of anxiety disorders and social communication difficulties can be linked to anomic aphasia, which involves impaired naming ability [45]. Patients with this type of aphasia have difficulty naming not only difficult and low-frequency objects, but also easy and high-frequency objects[25, 26]. Of course, there may also be a tip-of-the-tongue phenomenon here, which is related to working memory, and becomes more prominent with age [61]. However, this phenomenon, as studies of aSAH patients have shown, occurs far less frequently in anomic aphasia than in other types of aphasia [62]. It is emphasized that anxiety disorders can cause anomic aphasia symptoms to be exacerbated in difficult social situations requiring so-called functional communication [63]. The authors also emphasize that the reciprocal correlation between anxiety and communication intensifies when individuals are anxious, depressed or sleep deprived [57].

The patient described here suffered from anomic aphasia and difficulties in social communication, which is consistent with published reports of inter-relationships between these disorders. However, future research would benefit from the development and introduction into clinical practice of objective tests of social communication deficits to verify the current findings, and potentially help identify the specific processes underlying the disorders discussed here and their interrelationships.

When discussing such a complex picture of overlapping disorders, it is difficult not to mention one more factor: aSAH, which is caused by extravasation of blood in the subarachnoid space due to aneurysm rupture [64]. It is not only life-threatening in the acute phase, but can also cause secondary brain damage due to delayed cerebral ischaemia (DCI) and other complications that are difficult to treat, not only in the days but also weeks after the initial bleeding [39]. It is worth noting here that the prognosis of patients with aSAH is strongly influenced by the development of delayed cerebral ischemia (DCI) and the difficulty of using appropriate methods for prevention, diagnosis and treatment of DCI [65]. DCI could also partly explain the occurrence in our patient of the worsening of anxiety disorders and difficulties in social communication and coping with life over time after the cerebral incident.

NEW FORMS OF NEUROTHERAPY BASED ON FUNCTIONAL NEUROMARKERS

Today, the entire premise of mental health is shifting, presenting a remarkable moment in the history of science during which the discipline of psychiatry of the 20th century, is becoming the discipline of clinical neuroscience of the 21st century [27]. The introduction of new forms of neuro-imaging, especially functional neuro-imaging, has become a revolution in the understanding of mental illness. It has allowed us to understand that mental illness is:

1) a disruption of a set of electrical circuits rather than a chemical imbalance;
2) a developmental disorder for which early intervention is necessary;
3) the result of a complex interaction between genetic and environmental factors [66–73].

Intensive research conducted worldwide has made it possible to diagnose diseases and syndromes on the basis of functional neuromarkers, which have been available for nearly 2 decades. Such a functional neuromarker is calculated based on quantitative EEG (qEEG) studies and the temporal pattern of cortical electrical activation in event-related potentials (ERPs), where the EEG spectra and ERPs of the subject are computed and compared with the normative data base from the Human Brain Index (HBI), located in Chur, Switzerland. Functional neuromarkers (Fig. 7) have so far been used not only to diagnose brain dysfunction, but also to select and profile drug treatment (pharmacotherapy), to construct neurotherapy protocols for neurostimulation with Neurofeedback, Transcranial Direct Current Stimulation (tDCS) and Transcranial Magnetic Stimulation (TMS) [24, 27, 66–74]. Thus, there are tremendous opportunities to use this methodology in the diagnosis and treatment of neurological and psychiatric diseases, including anxiety disorders. The neurofeedback training provided to the Case Report patient consisted in brain wave training with a qEEG assessment. This provided operant conditioning of neural oscillations, in which the brain is trained to gain control over specific EEG parameters through real-time visual or auditory feedback. Desirable brain activity is amplified and undesirable activity is inhibited.

There is a vast amount of research on abnormal EEG and qEEG patterns associated with various medical and psychiatric disorders [27, 68–75]. Strong research evidence also indicates that there are functional brain abnormalities associated with anxiety and panic disorder [76–78] and post-traumatic stress disorder (PTSD) [69, 71, 72, 79]. A

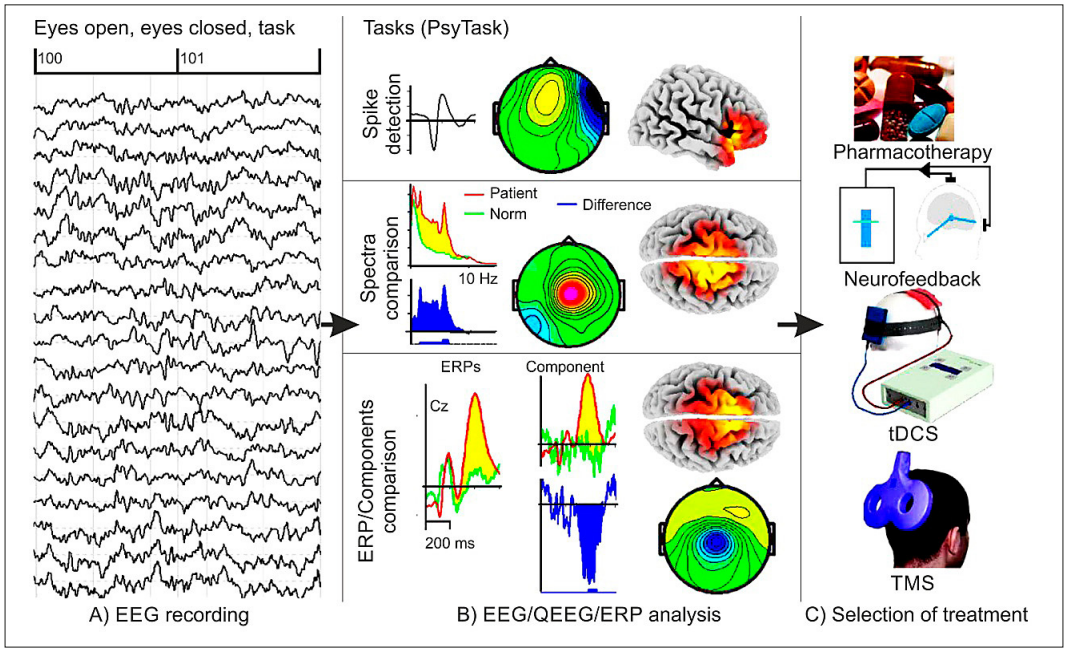

Figure 7. Possibilities for using new neurotechnologies in the diagnosis and treatment of mental and neurological diseases.
Source: Pąchalska et al [27], with modification

review of the literature on the neurofeedback treatment of anxiety disorders conducted by Moore [75] presents 8 studies of generalized anxiety disorder, 3 studies with phobic anxiety disorder, 2 studies of OCD, and 1 report of using neurofeedback with PTSD. The author concluded that neurofeedback, when used appropriately, makes it possible to reduce these disorders. Other authors, based on ongoing studies, have also concluded that neurofeedback training is an effective method for treating various mental disorders [24, 27], including anxiety [24, 27, 71, 72, 79].

Therefore, it is hardly surprising that neurofeedback training using a functional neuromarker of a given disorder – in the case of the presented patient, the functional neuromarker of anxiety – is considered the most promising method for treating various psychiatric disorders [72], including anxiety disorders, as long as the right training protocol is applied [27]. In the presented Case Report, anxiety disorders were detected on the anxiety subscale, but no depressive disorders on the depression subscale of the HAD in neuropsychological tests. These findings were consistent with neurophysiological studies, in which a functional neuromarker of anxiety was detected. No neuromarker of depression was found. Therefore, a neurotherapy protocol (electrodes bipolar at C3 and Cz), training of alpha DOWN (normalization of activity to 4–12 Hz) targeting anxiety reduction was constructed, and obtained a reduction in anxiety. This result is consistent with the findings of other researchers who have reported that such training can reduce anxiety, agitation and aggression, calm the patient and increase self-control, because this is an activation protocol intended to de-activate the left Rolandic fissure [24, 25, 27]. However, the modification of the training introduced in this case aimed at reducing naming disorders, resulted in a rapid improvement in naming ability, which is usually obtained in classical therapy methods after months of training with the so-called Visual Action Therapy, VAT, renowned as the world's best naming therapy method [26]. The patient expressed a sense of satisfaction with the effects of the therapy, as it enabled him to function better in social situations. Thus, the intended therapeutic goal. Wass achieved

To sum-up, it should be noted that the WHO officially ended the global state of emergency for COVID-19 on 5 May 2023, more than 3 years after the original declaration of the pandemic, and stated that countries should now deal with this highly contagious disease on their own. It should be remembered that COVID-19, which throughout the pandemic killed more than 6.9 million people worldwide, is still extant, and will continue to result in far-reaching consequences for individual health systems, as well as society [80]. Ongoing long-term independent epidemiological and clinical studies of the sequelae of SARS-CoV-2 infection and COVID-19 illness have shown that recovery is associated with a variety of both short-term and long-term physiological, neurological and psychiatric deficits, generally known as 'post-COVID syndrome' or 'long COVID' [6]. The abundance of symptoms that can occur in the acute phase of COVID-19 and in post-COVID syndrome is so great that multispecialty diagnosis is necessary, especially if neurological and psychiatric complications are present. One such complication is the formation and rupture of brain aneurysms with subsequent disorders requiring effective forms of diagnosis and therapy based on new neurotechnologies. Although the direct effect of COVID-19 on this condition is still under investigation, the most reliable link may well yet prove to be the complex biochemical inflammatory processes discussed above.

The presented Case Report of the 78-year-old patient – the course of the disease, its sequelae and complications, and the treatment administered – is instructive, as he suffered from both COVID-19 and aSAH. His diagnosis and treatment posed complex challenges because not all aspects of his disease are fully explicable. The most important fact, however, is the patient's recovery, which was made possible primarily by rapid neurosurgical intervention, and post-neurosurgical monitoring for aSAH, which allowed detection of his anxiety disorder, anomic aphasia, and social communication difficulties, thereby enabling specific goals to be set and the programming of specific therapies. The restoration of the patient's pre-morbid social activity was made possible through the use of a new neurotherapy, based on the functional neuromarker of anxiety. The extensive literature presented in this report also provides valuable clues as to what problems (including complex functions and social activities) should be the object of attention in efforts to help such patients recover and reduce post-morbid symptoms.

Limitations of the Report. The proposed new neurotherapy, based on the functional neuromarker of anxiety, for the reduction of anxiety, anomic aphasia and associated social communication disorders in a patient with neurosurgically-treated aSAH associated with COVID-19, is the first such programme worldwide. Although the description of this case and others can provide much information [4], in the opinion of the authors' of this Case Report, it would be necessary to conduct future studies with a larger number of patients to confirm the findings presented. This is consistent with evidence-based medicine [8, 18, 24, 66–74, 81–82] which allows interpretation of the results obtained in accordance with the microgenetic theory of symptom formation [83, 84], as demonstrated in this report.

CONCLUSION

In patients with anxiety disorders, anomic aphasia and related difficulties in social functioning after ruptured brain aneurysm, especially after COVID-19, multi-dimensional diagnosis and therapy is needed, preferably based on functional neuromarkers. Human Brain Index (HBI) methodology can be successfully used in the neurodiagnosis and implementation of individualized neurotherapy for such patients.

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