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Chapter 19

Recovery from Diffuse Brain Injuries: Two Case Studies

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Additional information is available at the end of the chapter

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Abstract

Subarachnoid hemorrhages (SAHs) are grave medical emergencies, whereas 30–50% of all SAHs may ultimately result in death. Subarachnoid hemorrhages share many resemblances with other neurological traumas such as a cerebral vascular accident, meningitis, and/or traumatic brain injury. Autoimmune encephalopathies (AE) occur when human antibodies assault the body's cell surfaces and/or synaptic proteins. Consequently, widespread nervous system and diffuse brain involvement may occur. With subarachnoid hemorrhages and autoimmune encephalopathies, multiple areas of cognition and language can be impaired. Case studies in communication sciences and disorders are underutilized, yet are important in evidenced-based practice. Speech-language pathologists in medical settings have worked with patients and families with similar types of disorders. Therefore, speech-language pathologists should be well equipped to provide therapy with these types of injuries. This chapter presents two case studies and cognitive language rehabilitation strategies following diffuse brain injuries.

Keywords: brain injuries, diffuse injuries, subarachnoid hemorrhage, autoimmune encephalopathy, cognitive language rehabilitation

1. Introduction

1.1. Brain injuries

Brain injuries are a major cause of death and disability worldwide [1–3]. It is well documented that brain injuries exhibit immediate and long-term consequences on language abilities [4]. Language abilities in adolescent individuals with brain injury have been noted to be worse than matched controls (matched for age, gender, general ability, and interests) in syntax comprehension, particularly in the areas of listening and grammar [5]. Inflammation and neurodegeneration are known to occur after initial traumas as secondary injuries [6].

Brain injuries can be classified according to several dichotomous taxonomies, that is, primary vs. secondary injuries, focal vs. diffuse pathologies, and inflammatory vs. non-inflammatory diseases [7–9]. Traumatic brain injuries (TBIs) s can be classified into two categories of primary and secondary injuries [1]. The primary injury consists of the actual physical trauma, while the secondary injury results from "biomolecular and physiological changes that follow these insults" [1, p. 97]. Secondary injuries are slow to change and may be a major factor in the patient's recovery [1]. Primary damage to the brain and central nervous system occurs with the initial insult (lacerations, fracture, contusions, axonal injury, intracranial hemorrhage), whereas a secondary injury is the resultant of such factors as increased intracranial pressure, interruption in the brain blood barrier flow, brain inflammation, disruptions in cerebral blood flow, ischemia, hypoxia, reperfusion, and/or reoxygenation injury [8, 9]. In addition, a series of events can lead to an accumulation of white blood cells in the injured brain area can lead to inflammation and secondary brain damage.

Graham et al. [9] stated that "Neuroimaging as a means of identifying intracranial pathology after head injury has allowed the clinician adoption of an alternative classification of focal and diffuse damage" (p. 641). Focal damage can be determined with certainty; however, the diffuse damage is much more difficult to ascertain regions of damage and consequently what cognitive and language areas are affected. Diffuse brain injury refers to damage to white and/or gray brain matter and not to a focal or localized injury [9]. However, several categories of diffuse brain injuries share similar pathophysiologies [9]. The diffuse brain injuries range in severity from concussion syndromes to diffuse axonal injury (DAI) to comatose. Currently, the term of traumatic axonal injury (TAI) is used to describe axonal injury after the initial trauma, that is, secondary axotomy (secondary severing or denervation of an axon) [9]. Secondary injuries can occur over a time span of hours, days, or weeks after the initial trauma. Consequently, secondary traumas may exert neurochemical changes in the brain, disrupt cerebral blood flow, disrupt the brain blood barrier function, and/or have neurotoxic effects on glial cells [10].

Central nervous system inflammatory diseases continue to be investigated, defined, and demarcated [7]. However, the pathological processes linking brain injury to neurodegenerative diseases still remain unclear. There is mounting evidence that neuroinflammatory processes may play a significant role in Alzheimer's disease, other dementias, and many neurodegenerative diseases [6, 11]. It is also known that traumatic brain injury also begins long-term neurological degenerative brain processes, for example, microglia activity and inflammation [11]. Brain inflammation is known to be a chronic hallmark of neurodegenerative diseases. Johnson et al. [11] stated that, "thus, persistent neuroinflammation following injury may prove mechanistically important in the link between TBI and Alzheimer's disease" (p. 29). Moderate and severe TBI leads to increased risk of later developing dementia [6, 12, 13]. Tau and amyloid beta pathologies are well-known sequelae in Alzheimer's disease [14]. Tau is proteins abundant in the neurons of the central nervous system and is known to become defective in the brain leading to dementias [15]. Even one traumatic brain injury may lead to tau and amyloid beta brain pathologies [16]. Repeated concussions may also lead to progressive brain abnormalities [6]. Chronic neuroinflammation as seen in chronic traumatic encephalopathies (CTEs) contributes to neurodegeneration. Autopsy studies have demonstrated the presence of reactive microglia even months or years after a single TBI [6].

1.2. Diffuse brain injury

According to Ewing-Cobbs and Barnes [17], "the diffuse injury consists of the cumulative Effects of diffuse axonal injury, hypoperfusion, excitotoxic cascades of neurotransmitters, and chronic alteration in neurotransmitter functions" (p. 210). Diffuse brain damage is likely to affect white matter tracts resulting from diffuse axonal injury affecting memory and executive function [18]. In addition, secondary inflammation resulting from a brain trauma may induce microglial activity which leads to further cognitive decline [2].

Ewing-Cobbs and Barnes [17] found that language outcomes after a traumatic brain injury are dependent upon several factors including age at the time of trauma, focal vs. diffuse brain injuries, and developmental acquisition of language functions at the time of the insult. Furthermore, they state that cognitive and language outcomes are more promising after perinatal and early focal damage. Diffuse brain injuries sustained in early childhood seem to be less promising, thus, indicating lessened behavioral and neural plasticity. Language and cognitive abilities that are in process of being acquired appear to be more vulnerable to disturbance than abilities that are better established [17]. Focal lesions can result in substantial reorganization of function; however, diffuse neuronal lesions do not result in reorganization and consequently poorer outcomes may result.

The sequelae of and pathological mechanisms resulting from a traumatic brain injury and consequent cognitive and language impairments are unclear. The resulting damage may lead to loss of neurons, axonal injuries, microbleeds, and disruptions to the blood-brain barrier (BBB) [3].

Diffuse brain injury has been associated with vascular and axonal impairments [19]. Initial responses include inflammation and disruption in the blood-brain barrier (BBB), and initiation of the body's systemic immune system, for example, glial cells reaction to the inflammation [19]. Changes in the blood-brain barrier at a microvascular level following a brain injury can last for months or years after the trauma. The interruption to the functioning of the BBB has been associated with the negative effects of a brain trauma. Diffuse brain injury occurs in the absence of a cerebral contusion (i.e., bruising of the brain) and does not lead to gross neuronal damage. However, diffuse axonal injury in the cortex, hippocampus, and dorsolateral thalamus may occur in the absence of a contusion and yet lead to cognition, attention, language, and memory impairments [2]. Damage to the hippocampus is associated with memory difficulties [20]. In conclusion, cognitive communication disorders seen in diffuse brain injuries may affect such abilities as memory, word retrieval, attention, an organization of information, and problem-solving [2, 18, 21]. Therapy concerns regarding cognitive communication disorders will be addressed in the following section.

1.3. Subarachnoid hemorrhages and autoimmune encephalopathies

Subarachnoid hemorrhages (SAH) and TBIs are major contributors to neurological cases and demonstrate similar problems in sub-acute care [22]. TBI incidence in industrialized and non-industrialized nations is estimated to be between 150 and 250 cases per 100,000 population, while the incidence of subarachnoid hemorrhages is estimated to be between 10 and 25 cases per 10,000 population [23].

Subarachnoid hemorrhages (SAHs) are grave medical emergencies, whereas 30–50% of all SAHs may ultimately result in death [24]. SAHs occur as a result of ruptured aneurysms or from a TBI. The subarachnoid hemorrhage is when blood accumulates below the arachnoid mater space on the brain's surface between the dura mater on top and the pia mater beneath (all part of the brain's meninges). Individuals who have suffered an SAH experience meningeal inflammation as a result of blood seeping into the cerebral spinal fluid (CSF). Subarachnoid hemorrhages share many resemblances with other neurological traumas such as a cerebral vascular accident, meningitis, and/or traumatic brain injury. Individuals with bacterial or viral meningitis have been reported to have difficulties with short-term memory, working memory, attention, and/or cognitive speed [25–27].

Autoimmune encephalopathies (AE) occur when human antibodies assault the body's cell surfaces and/or synaptic proteins. As a result, AE consist of a larger spectrum of inflammatory nervous system disorders including limbic encephalitis which is noted by subacute onset, memory loss, seizures, possible personality and mood changes, and alteration in one's senses [28]. In essence, encephalopathies can impact all areas of cognition [29]. In summary, subarachnoid hemorrhages and autoimmune encephalopathies both result in meningeal inflammation and diffuse brain injuries [29, 30].

1.4. Clinical history of cases

Two case studies involving patients with a subarachnoid hemorrhage (BA) and autoimmune encephalopathy (SJ) are presented. Both patients were healthy adults with no prior histories of speech, language, cognitive, or serious health conditions when the onset of the traumas occurred. The SAH typically results from a ruptured aneurysm or from a traumatic brain injury and consequential meningeal inflammation occurs. Autoimmune encephalopathies consist of a spectrum of inflammatory central nervous system disorders which may also include brain edema, and limbic encephalitis [29]. Consequentially, cerebral inflammation is a common outcome from both of these disorders.

BA was 52 years old when he suffered a subarachnoid hemorrhage (SAH). Prior to this incident, he was healthy with no history of cognitive or neurological issues. He exhibited slightly above average blood pressure and was taking anti-cholesterol medication prior to the trauma. BA experienced an aneurysm and hemorrhage to his right vertebral artery. This was surgically repaired 2 days after the SAH using two stents and a coil.

Immediately after the SAH, BA was screened by the hospital-based speech-language pathologist. The SLP used the Western Aphasia Battery-Revised [31] picture description task. BA's wife is a licensed and certified speech-language pathologist. The hospital-based SLP and the wife made the determination that BA was functioning "within normal limits" based on these results. The neurosurgeon and primary care physician also concurred with this assessment. However, this screening masked more pronounced difficulties that appeared later such as short-term and working memory difficulties, attention and cognitive speed processing, coping issues, and disruption in daily living skills [25–27, 32–35]. BA also continued to experience difficulties with word anomias, semantic paraphasias, phonemic and phonological paraphasias, and occasional errors in order of syntactical elements up to 2 years post-trauma.

SJ was a healthy 34-year old when she was admitted to the hospital suffering from confusion, disorientation, short-term memory loss, slowed mental processing and an inability to express herself. Over a period of a few months, she was assessed by a clinical psychologist with the Rey Auditory Verbal Learning Test (RAVLT) [36] and the Boston Naming Test [37]. According to the psychology report, SJ presented with low to average verbal immediate memory and low to average verbal delayed memory from the RAVLT. Recognition cues did not improve verbal recall. In addition, the Boston Naming Test indicated confrontation naming to be within normal limits.

SJ was later assessed by a neurologist using the Mini-Mental State Examination (MMSE) [38] and the Montreal Cognitive Assessment (MoCA) [39]. She scored 25/30 on both the MMSE and the MoCA. Specifically, SJ scored poor on attention, low to average on sustained attention, and low to average on delayed memory scores. SJ answered quickly, but incorrectly on tasks of sustained attention. SJ was diagnosed with autoimmune encephalopathy (AE). 4 months afterward, SJ reported that her episodes of confusion and cognitive issues ceased. At this time, she also received a 3-day regime and a dose of Solumedrol (a corticosteroid as SJ also suffered from myasthenia gravis); whereupon, she reported that she sustained attention and recall improved. The anti-inflammatory could have reduced any brain inflammation that could have occurred.

Both BA and SJ received self-administered, clinician-guided therapy. BA is a certified and licensed speech-language pathologist and also a university professor in speech-language pathology; in addition, BA's wife is a certified and licensed speech-language pathologist. SJ's sister is a certified and licensed speech-language pathologist. Consequentially, both BA and SJ did not receive traditional therapy, but self-administered therapy as guided by the SLP family member.

Self-administered, clinician-guided therapy has been noted in treating verbal anomias with individuals with aphasia [40, 41], cued naming therapy in an individual with aphasia [42], and a parent-administered program for teaching gestures to an individual with Angelman syndrome [43]. The objectives were selected by the clinicians; however, all treatments resulted in increased communication abilities in these individuals with severe and varied communication disorders (i.e., aphasia, word anomia, and Angelman syndrome).

2. Research methods

Both BA and SJ experienced diffuse brain injuries as a result from the subarachnoid hemorrhage (SAH) and autoimmune encephalopathy (AE). Encephalopathies affect all, areas of cognition and language with memory loss occurring in half of all patients with this disorder [29]. Consequentially, BA's and SJ's diffuse brain injury symptoms included: disorientation, memory loss, word retrieval difficulties, psychological tiredness, fatigue, concentration difficulties with coping, symptoms of post-traumatic stress disorder, and dependence on others [22, 29, 30, 44–49].

Therapy for diffuse brain injuries addresses issues of memory loss and retrieval difficulties, attention issues, organizational difficulties, problem-solving, and executive functioning [21].

Speech-language and cognitive rehabilitation therapy provided to both patients revolved around functional outcomes (i.e., return to work and life goals). Cognitive remediation was adapted from work with individuals with traumatic brain injury and stroke [50, 51]. Therapy techniques specifically focused on memory deficits, concentration issues, difficulty with daily living skills, irritability, impatience, dependence on others including use of: (a) cueing; (b) fading; (c) use of hierarchical targets; (d) anchoring; (e) repeated practice; and/or, (f) use of strategies [50]. Both patients have returned to work and have not received therapy after a period of 5 years.

2.1. Therapy approaches for subarachnoid hemorrhage and autoimmune encephalopathies

Cognitive and language rehabilitation approaches for both BA (i.e., subarachnoid hemorrhage) and SJ (i.e., autoimmune encephalopathy) were self-administered, clinician-guided since both patients were able to return to work within 4 months after both brain traumas had occurred, and the deficits were not noted to be severe enough to warrant direct speech-language therapy. However, it should be noted that although the deficits may be viewed as minor by others (e.g., family members, therapists, medical professionals), the patient may view the deficits as being major [32].

BA's wife was a certified and licensed speech-language pathologist, while SJ's sister was a certified and licensed speech-language pathologist. Both the wife and sister assisted in providing self-administered, clinician-guided therapy to the patients.

BA began reading and writing on a daily basis after his release from the hospital and return home, approximately one hour per day for both activities. He then began writing one page one hour each day and also reading an additional hour per day for weeks two to three. Afterward, he increased these activities to three hours per day for weeks 4–12. BA returned to work on week 13 post-release from the hospital. BA wrote a brief newsletter article, completed a research article, and peer reviewed refereed one journal articles 3 months following the trauma. Specific self-administered language techniques included semantic cueing, phonemic cuing, semantic feature analysis [52] and practicing strong relationships between noun and verbs; verb and agents (e.g., walk/sidewalk; sleep/bed). As a reminder, BA is also a speech-language pathologist.

BA estimated his attention levels post-trauma as follows: year 1, 85%; year 2, 90%; year 3, 95%; year 4, 96%; year 5, 98–100%.

Also paramount to SJ was her ability to SJ return to work which she accomplished 4 months after onset of symptoms. SJ is currently enrolled in a master's degree program in speech-language pathology, and her sister is a licensed and certified speech-language pathologist. Consequently, SJ like BA was highly motivated to participate in self-administered, clinician-guided therapy. SJ continued her reading activities investigating the cause of her illness, diagnosis, prognosis, and therapeutic outcomes. SJ read approximately one hour per day increasing to two hours per day over the course of 4 months. Use of semantic cueing and semantic feature analysis was also recommended by her sister. In addition, the following approaches were utilized by the SLP family members for both patients.

The therapy approaches were adapted from several sources including non-aphasic traumatic brain injury [53, 54] neurogenic disorders [55], and other cognitive disorders (such as working memory disorders) [56]. Strategies included the following:

1. *Word association and naming tasks* – Give a word and provide other words that are associated with it (this may include nouns, verbs, and/or adjectives). For example, the therapist says "table" and the client may respond with "chairs, eating, work, dining set, coffee table, etc." Implicit and episodic memories may be triggered [54].
2. *Reading* – Provide low and high-level reading materials. For example, reading may vary from the newspaper and pleasure reading books to higher level professional articles.
3. *Writing* – Write informal, formal, low and high-level pieces. For example, the client may write notes and letters and progress to higher-level written pieces as concentration improves.
4. *Word association and memory tasks* – Separate words into categories have the client memorize words in the categories utilize mnemonics. Utilize recognition naming (the target word is given along with two foils) and/or picture description (for more spontaneous naming). For example, the therapist elicits word and category recall. The clinician may keep track of correct information units (CIUs or i.e., words that are accurate and informative to the task) [56].
5. *Auditory attention tasks* – Have the client listen to a paragraph and answer questions; restate what was said; restate the main points; and, restate main items or discussion points.
6. *Divided attention tasks* – The client should focus on specific details while dismissing erroneous detail. For example, have the client press a key when two shapes are the same color and another key when they are different colors.
7. *Working memory and attention tasks* – Divide attention between multiple tasks; the client should attend to key information; and, ignore distractions; and, notice patterns. Examples include driving a car to conducting a presentation at work.
8. *Cognitive speed* – The client should be capable of shortening response times; processing information accurately and quickly. This is similar to spaced-retrieval training [55] where the client is asked to recall information repeatedly and systematically. For example, have the client engage in conversations and increase difficulty levels (i.e., moving from personal information to higher level professional discussions).
9. Compensatory strategies – Compensatory strategies may include note taking, recording events sequentially, and systematically slowing down all tasks for increased comprehension and hence, completion rates, and regular use of calendars for scheduling events.

In addition, speech-language pathologists should target the following goals for the patient and family to facilitate an increased quality of life [57]:

1. Reduce the family burden issues;
2. Reduce the patient dependence levels;

3. Improve the patient's ability to cope to everyday and new situations;
4. Address cognitive-language impairments (e.g., concentration, memory);
5. Address mood disturbances; emotional issues (e.g., anger, irritability);
6. Address fatigue, tiredness issues (directly or indirectly);
7. Address the patient's passivity issues.

In conclusion, subarachnoid hemorrhages and autoimmune encephalopathies share many commonalities with other disorders that speech-language pathologists may have already treated such cerebral vascular accidents, traumatic brain injuries, meningeal inflammation, and memory and attention deficits. While differing etiologies may determine, to some extent, prognoses and outcomes, therapeutic approaches for many cognitive-language disorders may share similarities. Consequentially, issues of disorientation, memory loss, word retrieval difficulties, psychological tiredness, fatigue, and concentration difficulties should be targeted in rehabilitation and in the form of compensatory strategies.

Declaration of interest

The authors report no declaration of interest. The authors have no financial interest, direct or indirect, in the subject matter or materials discussed in the manuscript. This project was a non-funded study.

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