Do You Know of Cases of Wernicke’s Aphasia Post Herpes Simplex Viral Encephalitis?

Betty L. McMicken1, Andrew Kunihiro2 and Long Wang1

1California State University, Long Beach, California, USA
2University of Arizona, Tucson, Arizona, USA

Corresponding author: Betty L. McMicken, PhD, Associate Professor, California State University, Long Beach, California, USA, Tel: (949) 500-1868; E-mail: Betty.McMicken@csulb.edu

Received: October 31, 2014; Accepted: November 01, 2014; Published: November 08, 2014

Copyright: © 2014 McMicken BL, et al. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

Editorial

This editorial is a request for clinician researchers to report on speech recovery in cases of Wernicke’s Aphasia post Herpes Simplex Viral Encephalitis. The literature, while it contains detailed case histories of Wernicke’s cases post stroke, does not contain extensive material on speech recovery post encephalitis. A case report is now in progress, but there is little current and detailed literature in which to compare the clients progress. What is clear in the literature is the etiology of the disorder Herpes Simplex Virus Type 1 (HSV-1), along with HSV-2, are neurotropic members of the Herpesviridae family. HSV-1 most commonly causes cold sores but can also lead to genital herpes infections, while HSV-2 predominately causes genital herpes. Transmission occurs via mucosal surfaces including the oral and respiratory surfaces or through compromised skin, such as by sharing drinking vessels or utensils, kissing, and other high-risk skin-to-skin contact. HSV-1 is usually contracted during infancy or childhood through exposure to an infected adult. Transmission does not require an active infection or visible sores and can be spread through asymptomatic viral shedding [1,2]. There is a high seroprevalence in the general population, with an estimated 70–90% of asymptomatic individuals harboring the HSV-1 [3,4]. There is currently no cure for HSV but symptoms can be managed with antiviral medications [5].

Exactly how HSV-1 infiltrates the central nervous system to cause to HSE is greatly debated, with the olfactory bulb and trigeminal ganglia implicated in mice models [6]. The olfactory pathway is the most likely avenue of infection, as recurrent herpes labialis, which occurs in the trigeminal ganglia, rarely leads to HSE [7]. HSE normally affects the temporal lobe, which is responsible for retention of visual memory, language comprehension, processing of sensory input, and emotion. Therefore, symptoms of HSE include aphasia, confusion, and behavioral changes there can also be extratemporal involvement, including the frontal and parietal lobes, with an estimated 16% of patients with HSE having extratemporal infections [8]. This localization of infection is thought to be caused by the proximity of the temporal lobe to the olfactory bulb or trigeminal nerve [9] or preference of HSV for limbic cortices. Briefly, HSV-1 causes degeneration of cell nuclei and loss of plasma membranes, leading to multi-nucleated giant cells. This in turn causes inflammation, hemorrhaging, and eventual tissue necrosis and liquefaction [10].

When damage occurs to Brodmann area 22 (Wernicke’s area), located in the superior temporal gyrus, such as by HSE, problems with auditory and written comprehension occur. This is known as Wernicke’s aphasia (e.g. fluent or receptive aphasia), and is characterized by fluent, albeit nonsensical verbal and written language that conveys little useful information. Individuals affected by Wernicke’s aphasia are usually unaware of any errors in their speech or writing. Those with Wernicke’s aphasia can string sentences together with real words paired in nonsensical combinations, neologisms (newly synthesized non-words), or both [11]. Depending on the location of brain lesion, auditory and reading comprehension can be affected to varying degrees. For example, in aphasias with agraphia, individuals cannot understand written language but have limited ability to understand words spelled out loud and can generate written output [12].

The literature describes a variety of rehabilitation methods that have helped to improve recovery in activities of daily living and semantic processing, such as positive reinforcements or extinction techniques [13], associations (motor imagery strategy) [14], and community-based programs [15]. According to Altschuler et al. [16], no proven method exists for rehabilitation of the Wernicke’s aphasia, and there is little direction in the literature for speech-language pathology treatment of Wernicke’s aphasia patients. In this case, it was decided to follow the suggestions of Marshall using Context-Based Therapy [17,18]. It was recommended that the patient participate in a language treatment program on an intensive schedule, with emphasis on combining elements of the PICA multidimensional scoring system scoring [19], and Marshall’s description of Context-Based Therapy [18], an offshoot of the Context Centered Therapy concept of Wepman [20]. Emphasis was on the auditory system, after Schuell’s treatment philosophy [21,22], thus attempting to increase the functional nature of communication across all language modalities. Also, exploration of all patient’s language abilities using her previous writings, hobbies and editorial work as stimuli were used to assist with both receptive and expressive language modalities. Any communication means possible was explored, from pointing to a picture of a food on a menu, to use of a clock or calendar, or facial expressions, and also capitalizing on residual words to achieve meaning in a message [16].

The Primary Investigators (PI) client, which is in the process of recovery, has been in speech-language treatment for 17 months. While initially demonstrating severe impairment in visual and auditory processing in addition to verbal and written expression, the client is now functional in all language modalities with some residual auditory memory and verbal errors if not focused on the task. She continues to have difficulty with complex problems involving time presented visually and auditorily, and with unstructured language tasks. Learning is apparent and measurable on a weekly basis.

Is this a typical recovery pattern for Wernicke’s Aphasia post herpes simplex viral encephalitis?
References


