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Hearing Loss in Stroke Cases: A Literature Review

Arun Banik[1], Vivek Kumar Jha[2*], Pooja Yadav[3], Chandan Kumar Dubey[4]

[1] Professor (Speech Pathology and Audiology), Department of Audiology and Speech-Language Pathology, Vinayaka Mission's Research Foundation, deemed to be University, Puducherry, India.
[2] Professor (Speech Pathology and Audiology), Department of Audiology and Speech-Language Pathology, Faculty of Allied Health Sciences, Shree Guru Gobind Singh Tricentenary University, Gurugram, Haryana, India.
[3] Clinical Supervisor (Speech Pathology), Department of Audiology and Speech-Language Pathology, Faculty of Allied Health Sciences, Shree Guru Gobind Singh Tricentenary University, Gurugram, Haryana, India.
[4] Assistant Professor (Special education,), Department of Special Education (HI), Faculty of Education, Shree Guru Gobind Singh Tricentenary University, Gurugram, Haryana, India.

**Corresponding author's E-mail: jhavivek98@yahoo.com*

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Abstract

Stroke is the most common cause of neurological disability (MacDonald, Cockerell, Sander, & Shorvon, 2000) and about 1 in 3 stroke life survivors are functionally reliant on it after one year (Murray and Lopez 1996). The majority of stroke survivors need restoration (MacDonald et al., 2000), requiring them to be adequately informed of the prognosis, nature, and proposed treatment of their illness. Hearing plays an important role in effective communication between healthcare professionals and patients (Bensing, 2000), therefore hearing loss may restrict contribution in recuperation programs, leading to an inferior level of bodily performance (Landi et al., 2006). Both hemorrhagic and ischemic strokes may interrupt all stages of the hearing path and lead to hearing shortages that start acutely previously, throughout, or shortly after the occurrence of the stroke. Yet, hearing shortfalls after stroke have not been as lengthily investigated as visual shortfalls, possibly due to the potentially "invisible" nature of this loss compared to more noticeable symptoms (e.g., dysphasia or motor loss). Hearing impairment after stroke may be a vital unmet need for stroke patients and additional research into patterns, detection, prevalence, and treatment are compulsory. In SCA infarction, the ischemic lesion happens in the area where threads from the nucleus have already traversed, and therefore sensory hearing impairment is noticed in the contralateral sideways. There is clear evidence that PICA and AICA territory strokes may result in mixed cochlear/retro cochlear, and less frequently retro cochlear-only patterns of hearing impairment (H. Lee et al., 2002). Hearing impairment for both AICA and PICA infarcts is mostly one-sided. Stroke may affect all levels of the auditory pathway and lead to hearing reception and/or perception deficits. Sudden-onset hearing loss after stroke of the vertebra-basilar territory and/or low brainstem is one of the less frequent neurologic impairments, while cortical or central deafness is even rarer. However, studies of populations with stroke indicate that hearing loss is very common, while in the general population, a past history of stroke increases the likelihood of having hearing loss. Auditory-processing deficits after stroke are less well studied than hearing loss and possibly under documented. Auditory dysfunction may impact on patient communication and may even predict long-term patient outcome after stroke. Despite this, clinical guidelines for auditory assessments after stroke are rudimentary. This study reviews the available information of auditory function in patients with stroke. On the basis of the information available, it is suggested that screening the patient's hearing before the patient leaves the stroke ward with a short test and a minimum set of hearing-related questions and subsequently screening the patient's hearing needs with targeted questions at the chronic stage of stroke may be a cost-effective bare minimum assessment approach to addressing the hearing needs of this complex population.

1. Introduction

A person who is not able to hear as well as someone with normal hearing – hearing thresholds of 20 dB or better in both ears is said to have hearing loss. Hearing loss may be mild, moderate, severe, or profound. It can affect one ear or both ears and leads to difficulty in hearing conversational speech or loud sounds. (WHO,2021).

Hearing loss can be described by variations in type, degree, and configuration. The three basic types of hearing loss are sensorineural, conductive, and mixed.

Sensorineural hearing loss: It is due to cochlear (sensory) or vestibulocochlear nerve/CN VIII (neural) auditory dysfunction.

Conductive hearing loss: It is due to a problem conducting sound waves through the outer ear canal, tympanic membrane, or middle ear ossicles.

Mixed hearing loss: It is the result of damage to conductive pathways of the outer and/or middle ear and to the nerves or sensory hair cells of the inner ear.

Causes of conductive hearing loss:

Absence or malformation of the outer ear, ear canal, or middle ear, benign tumors, fluid in the middle ear space (e.g., from upper respiratory infection or middle ear infection), head trauma (e.g., skull fracture, damage to the tympanic membrane or middle ear structures), impacted cerumen, infection of the ear canal (e.g., external otitis, swimmer's ear), otitis media (i.e., infection of the middle ear), otosclerosis (i.e., reduced vibration of the middle ear bones), perforated tympanic membrane, poor Eustachian tube function; and presence of a foreign body.

Causes of sensorineural hearing loss:

Autoimmune inner ear disease, benign tumors, **enlarged vestibular aqueduct syndrome**, genetic causes, both syndromic (e.g., Charge syndrome, Pendred syndrome, Waardenburg syndrome) and non-syndromic (e.g., genetic mutation), head trauma (e.g., inner ear damage, TBI), infections (e.g., bacterial, viral, parasitic), **Meniere's disease,** noise exposure, ototoxic medications and chemicals, presbycusis, vascular deficits (ASHA).

Mixed hearing loss: It occurs when there is a combination of one or more causes of conductive hearing loss and one or more causes of sensorineural hearing loss.

Stroke is rapidly developing clinical signs of focal (or global) disturbance of cerebral function, with symptoms lasting 24 hours or longer, or leading to death, with no apparent cause other than of vascular origin. (WHO,1970). Strokes can basically be classified into two major categories, ischaemic stroke and haemorrhagic stroke.

Ischaemic stroke is caused by interruption of the blood supply to a part of the brain resulting in sudden loss of function, while **haemorrhagic stroke** is attributed to rupture of a blood vessel or an abnormal vascular structure.

In general, 80% of stroke cases are seen to be ischemic, rest 20% are seen to be haemorrhagic, nonetheless, types of strokes still depend on the respective population

Stroke remains a disease of immense public health significance in the 21st century despite the advances in our understanding of several important areas of the disease such as the epidemiology, quality of life, and pathophysiology.

In both the developed and developing world, ischaemic stroke is currently the predominant stroke subtype.

Hypertension remains the leading risk factor of stroke in both developed and developing countries despite the racial differences in the risk factors of stroke.

Since the burden of stroke is expected to increase significantly in future, there is the need for a better understanding of the factors associated with high blood pressure, especially in countries with a high risk of stroke.

Pathophysiology of Stroke:

Generally, there is some alteration in brain metabolism if blood flow is interrupted for 30 seconds. In ischaemic stroke, disruption of blood flow to the brain for a few minutes' causes hypoxia and hypoglycemia, which leads to infarction of brain tissues. A vicious cycle (ischaemic cascade) ensues due to the accumulation of sodium, calcium, and water in the injured brain cells, which leads to the release of excitatory neurotransmitters causing further cell injury.

The brain, which is the main organ affected by stroke, is metabolically active and needs about 50ml/100g/min blood flow with an oxygen metabolic rate of 3.5cc/100g/min.

If the blood flow drops below 10ml/100g/min, brain cell functions are severely affected, while neurons are unable to survive long at levels below 5ml/100g/min.

In haemorrhagic stroke, the hematoma causes compression of tissue resulting in tissue injury, the brain's regulatory mechanism attempts to maintain equilibrium by increasing blood pressure but the increased intracranial pressure forces out cerebrospinal fluid causing damage to circulation.The blood from brain haemorrhage exerts some direct toxic effects on brain tissue and vasculature. Mass effect ensues with neuronal damage resulting from excitotoxicity, free radicals, apoptosis, ischemia, diaschisis, neuropathic products, and pressure necrosis.

Correlation between Hearing Loss and Stroke:

Hearing loss is common after a stroke. This is because the auditory pathways can be affected at all levels, which can lead to perception and recognition deficits. Individuals with a history of stroke may be more likely to suffer from hearing loss during subsequent strokes. Strokes that affect the outer part of the brain stem are more likely to affect hearing and lead to sudden hearing loss. A stroke occurs when the blood supply to the brain is blocked and the brain is deprived of oxygen. If this damage occurs in the part of the brain responsible for hearing and balance, it can cause lasting damage, including hearing impairment, dizziness and vestibular issues.

A stroke in the temporal lobe of the brain may result in long-term changes in hearing, including a person's ability to recognize sounds or spoken words. While rare, spoken hallucinations can also occur. Sudden hearing loss or sudden sensorineural hearing loss (SSNHL) is just as the name suggests – hearing loss that occurs all of a sudden or over a few days. SSNHL usually only affects one ear. Those who develop this condition have an increased risk of suffering a stroke within the next few years caused by a disruption in blood supply to the ears and brain. (Bamiou D. E., 2015).

Review Of Literature:

Sensorineural hearing loss (SNHL) may be highly predominant in stroke survivors (Edwards et al., 2006; Formby, Phillips, & Thomas, 1987; O'Halloran, Worrall, & Hickson, 2009) ; this may be due to the pathology of the internal ear (H. Lee, 2012), cochlear nuclei, or auditory nerve, i.e., the part of the central auditory pathway before the passage of the auditory fibers at the superior olivary complex brainstem level (Luxon, 1980). SNHL is distinctly dissimilar from "Central Deafness", i.e., the rare and histrionic occurrence of "deafness" with an extra attentional impairment component that is attributed to two-sided cortical damage, in the incidence of comparatively preserved cochlear and neural purpose (Timothy D. Griffiths, 2010). The observed association between stroke and hearing loss could be attributed to age-connected changes of the auditory nerve and inner ear, as the mainstream of stroke sufferers are usually over the age of 60 (Jacquin et al., 2012), while risk factors for stroke such as atherosclerosis, cigarette smoking, and others have also been associated with a more insidious start of hearing impairment with advancing age (Yamasoba et al., 2013), or otherwise, to the hearing pathways being directly affected by the stroke (H. Lee et al., 2006). Furthermore, if the stroke includes the central auditory path in the brain, patients may suffer from additional auditory processing deficits that are not reflected by the hearing verges (D. E. Bamiou et al., 2012; D. E. Bamiou et al., 2006).

This review aims to regulate the type of hearing recognized on Pure Tone Audiometry, after infarction of different cerebral artery areas. Sudden hearing loss after stroke is less common than other neurological damages, but it may be the initial exhibition of vertebrobasilar ischemia, which is more public in the presence of vascular risk aspects (Kim & Lee, 2009; H. Lee & Baloh, 2005; H. Lee & Cho, 2003; H. Lee et al., 2002; H. Lee, Whitman, Lim, Lee, & Park, 2001). Anterior Inferior Cerebellar Artery territory stroke is the foremost cause, and is reported to be the reason for 83% of cases, while Posterior Inferior Cerebellar Artery stroke books for 12% (H. Lee et al., 2002). The kind of hearing damage in both AICA and PICA is stated to be mainly cochlear (H. Lee et al., 2002; H. Lee & Yi, 2008) but can also be mixed cochlear/retro cochlear, and less frequently retro cochlear only (H. Lee et al.,

2002). However, it should be distinguished that with the exception of tests such as auditory brainstem evoked responses and acoustic reflexes, very few studies have conducted any psychoacoustic tests, such as localization and chronological resolution, that may help resolve if the hearing impairment is central or peripheral (Ulbricht, 2003). Additionally, the occurrence of severe to profound hearing impairment confounds the interpretation of auditory brainstem-evoked acoustic reflexes and responses. Despite these limitations, there is clear evidence that PICA and AICA territory strokes may result in mixed cochlear/retro cochlear, and less frequently retro cochlear-only patterns of hearing impairment (H. Lee et al., 2002). Hearing impairment for both AICA and PICA infarcts is mostly one-sided. However, two-sided hearing impairment is also likely when there is more widespread damage (Chang et al., 2013; C. C. Lee et al., 2011).

Hearing loss due to midbrain and brainstem lesions is infrequent and has been stated in less than 1% of remote brainstem strokes (H. Lee & Yi, 2008). This is due to the fact that the rising pathway partly decussates in the brainstem, thus widespread bilateral brainstem lesion is necessary to cause hearing impairment, and such lesions are rarely well-matched with life (Timothy D. Griffiths, 2010). Hearing shortfalls after Superior Cerebellar Artery (SCA) ischemic infarction are typically contralateral (Murakami et al., 2005). The SCA branches pierce into the superior cerebellar peduncle, the dentate nucleus, and about two-thirds of the cerebellar deep white matter (Marinkovic, Kovacevic, Gibo, Milosavljevic, & Bumbasirevic, 1995). In SCA infarction, the ischemic lesion happens in the area where threads from the nucleus have already traversed, and therefore sensory hearing impairment is noticed in the contralateral sideways (Doyle, Fowler, & Starr, 1996; Murakami et al., 2005).

Hearing deficiencies after SCA infarction can also be two-sided. Cerrato et al (Cerrato et al., 2005) report a case with bilateral hearing impairment (left more than right) as one of the prevailing signs at presentation. There are case studies of ipsilateral to the stroke hearing shortfalls. Lee and Yi (H. Lee & Yi, 2008) stated two patients with upper brainstem infarction on MRI, with a hearing loss ipsilateral to the stroke. Out of these two circumstances, one had a cochlear type hearing impairment whereas the other possibly was mutual. There are reports of hearing loss after a hemorrhagic brainstem stroke. Cohen et al. (Cohen, Luxon, & Rudge, 1996) stated two-sided symmetrical hearing damage in a patient with medial brainstem participation and one-sided hearing damage in a patient with a brainstem lesion before the degustation in the pons. Hearing impairment may also exist due to a stroke of higher-range subcortical structures. Musiek and Baran (Musiek et al., 2007) stated a case of a client with a subarachnoid bleed distressing both lower colliculi. The patient grieved from total “central” deafness throughout the first week postadmission but the hearing recovered considerably two and a half weeks’ post-admission. Eventually, audiological testing showcased a progressive recovery, in which PTA ended up within normal range. Though the auditory suggested capacities improved, they did not recuperate to the usual position. The majority of patients with cortical deafness, due to functional abnormalities of the pulvinar, may suffer from some degree of attentional shortfall (Pandya, 1995). Thus, obtaining precise hearing verges may be a challenging task for clinicians, as patients are required to sustain attention for a simple task. Consequently, the degree of hearing impairment in such patients may have been overvalued. Nevertheless, audiological valuation procedures were not consistently employed, even where impartial measures would have led to a more complete meaning of the auditory shortfalls. Consequently, audiological inspections that evaluate he auditory nerve, peripheral auditory system, and brainstem are highly vital for the analysis of cortical deafness.

4. Conclusion

In assumption, peripheral hearing impairment is detected in the vast mainstream of Anterior Inferior cerebral artery infarctions but seldom in Posterior Inferior cerebral artery infarctions. Such hearing impairment may occur in seclusion and could also be a prodromal symptom that responds well to early behavior. Brainstem and advanced subcortical lesions including the arising auditory paths may also upset the hearing verges depending on the size and site of the cut. Finally, hearing impairment is also stated in patients with lesions in secondary and primary auditory cortices. However, the so-called cortical deafness may partially be due to an attentional shortfall in stroke patients. Hearing impairment is not routinely measured after a stroke. Also, because of other severe indications patient may not be aware of their hearing loss at the period of stroke. So, hearing impairment can remain unnoticed yet may have important repercussions for rehabilitation.

Hearing impairment after stroke may be a vital unmet need for stroke patients and additional research into patterns, detection, prevalence, and treatment are compulsory. Nehzat started employment as an Audiological Scientist in the Department of Neurotology at the National Hospital for Neurology and Neurosurgery in 2009. Originally, she was primarily interested in Vestibular Science and gained wide knowledge in this field working in Professor Linda Luxon’s team. Subsequently, after a time of APD

exercise provided by Dr. Bamiou in her clinics, she decided to emphasize Auditory Processing Disorders. After that registered at the University College London for a Ph.D. program and Dr. Bamiou was appointed her Ph.D. manager. Her research emphasizes hearing and auditory purpose in individuals with strokes. She examines hearing and auditory handling in this patient population, and her research query is how the disordered auditory processing in stroke patients can be remediated by the use of Personal Frequency Modulation (FM) systems. Recently, she had the exciting opportunity of visiting Professor Frank Musiek's Neuro Audiology lab at the University of Connecticut where she amended her knowledge about auditory training of the brain and auditory processing.

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