Subcortical crossed aphasia- A clinical case report.

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Abstract

A 42- year old woman, right hander, underwent flow diverter placement surgery for right ophthalmic ICA aneurysm cure following which she suffered with acute subcortical crossed aphasia, left upper and lower limb paralysis. There was an intra-parenchymal hemorrhage with hematoma in right external capsule, putamen, coronaradiata, a part of the centrum semiovale and perilisional edema in the posterior internal capsule post surgery. The patient's language profile was characterized by marked word finding difficulty. Spontaneous speech had perseverative and paraphasic errors, phonemic substitutions, hesitations and groping. Auditory comprehension of casual conversation was functional. Based on the lesion site and speech-language profile she was diagnosed as having subcortical crossed aphasia.Within three months, the patient showed rapid spontaneous improvement in the domains of both comprehension and expression. Though there was a rapid recovery of language functions in the patient, some of the language deficits still persisted for which she received language intervention. With intensive language intervention, she showed significant improvement in object naming, word fluency, phoneme fluency and repetition. Profound improvement was noticed in auditory comprehension as well. As crossed aphasia is a rare phenomenon, understanding of this clinical existence depends entirely on the analysis of published case studies. Additional reports on crossed aphasia may provide more information on its characteristics, prognosis and response to treatment. Further, evidence based studies are required to shed light on the efficacy of language intervention in individuals with crossed aphasia.

Introduction

Aphasia is an acquired neurogenic language disorder caused due to an injury to the brain that affects different modalities of language. The most common cause of aphasia is stroke. However, brain damage resulting from tumors, trauma, and infections can also result in aphasia (Damasio, 1992).

Aphasia is not a single problem, rather it is best thought of as a group of different disorders. Each individual with aphasia will present a heterogeneous profile of language strengths and weaknesses. The various symptoms of aphasia are divided into broad classes by most of the classification systems. A common approach is to differentiate aphasia based on characteristics of verbal expression as the fluent aphasia and the non-fluent aphasia (Goodglass & Kaplan, 1972). Subcortical aphasia and Crossed aphasia are the other variants of aphasia also called as exceptional aphasia as they do not fit neatly within the common classification systems.

Subcortical aphasia is a form of aphasia which is a result of damage to subcortical regions such as the thalamus, striatum, internal capsule, and the basal ganglia. The clinical picture can be similar to those occurring from cortical lesions. Also, the subcortical damage can co-exist with the cortical lesions. The recovery from the deficits is usually early in subcortical aphasias when compared to cortical aphasias. The aphasic symptoms can arise when the inputs from the subcortex to the frontal lobe is altered, or may directly occur from damage to subcortical areas that support language processing (Boissezon, Demonet, Marie, Raboyeau, Albucher, & Chollet, 2005). Thalamic aphasias are usually characterized with fluent output, impaired comprehension and naming with predominant verbal paraphasias. But, striato-capsular aphasia is phonetic impairment of language, opposite to thalamic aphasia where lexical-sematic processing seems to be affected.

Crossed aphasia is a type of aphasia that occurs when a person's language centers are not in the expected hemisphere. In most right-handed individuals, language centers are located in the left hemisphere. This is also true for a majority of left-handed people, although there are exceptions for both groups. An example of crossed aphasia would

be a right-handed person who has a right hemisphere stroke that results in aphasia. The occurrence of Crossed aphasia is very rare. The prevalence of Crossed aphasia in right-handed patients is found to be between 1% and 3% of all stroke survivors (Coppens, Hungerford, Yamaguchi & Yamadori, 2002). The characteristics of Crossed Aphasia include non-fluent language output with agrammatism regardless of lesion site, initial mutism and relatively preserved auditory comprehension skills (Brown & Hecaen, 1976; Kim, Shin & Kim, 2011).

Crossed aphasia resulting from a right hemispheric lesion is rare because usually aphasia in right-handers are caused by stoke in the left hemisphere. The diagnosis of Crossed aphasia is usually made when there is the presence of aphasia resulting from a right hemisphere lesion in a person who has strongly preferred right-hand use. Also, there should not be any alterations in the structural integrity of the left hemisphere, no familial history of left-handedness along with no history of brain damage in childhood (Shizaki, Ueyama, Nishida, Imamura, Hirano & Uchino, 2012). Many cases have been reported in the past, but the exact mechanisms underlying language disorders of crossed aphasia is yet not traced.

Many research articles in the past have attempted to compile the cases with crossed aphasia with the aim of highlighting its frequency of occurrence, lesion site, specific symptom manifestations, and recovery patterns. Most of the understanding, in regard to crossed aphasia, is through the case studies. Habib, Joanette, Ali-Cherif, and Poncet, (1983) noted that almost all patients with vascular crossed aphasia had a lesion situated in the deep structures of the right hemisphere.

Sapir, Kokmen, and Rogers, (1986) reported a 74-year-old woman, a right-hander, who suffered from subcortical crossed aphasia secondary to a vascular lesion of the right cerebral hemisphere. The case exhibited severe anomia and paraphasia but had relatively preserved auditory comprehension. Based on the detailed profiling of the language deficits of the case, it was concluded that the patient's aphasia symptoms were not so different from those associated with left cortical lesions.

Alexander and Annett in 1996 described a few of the cases with crossed aphasia. One among those is a 66-year-old man, right-hander, who had an infarct in the superior division of the right middle cerebral artery (MCA). Posterior frontal rolandic, post rolandic and anterior insular cortex were also involved. Also, there was an infarct in the right lenticular-striatal arteries including the putamen, lateral caudate and frontal subcortical white matter. Initially, he had mild agrammatism with reduced phrase length, word finding difficulties and phonemic paraphasias. His language became fluent and paragrammatic by 8 weeks post onset. But, errors were ample in, spontaneous speech, repetition, reading, and writing. Authors suggested that such findings can be interpreted as the evidence for the presence of crossed cerebral dominance in right-handed persons.

In the same study, he reported of crossed aphasia in a 77-year-old woman, a right-hander, who suffered from a large infarct in the cortical and subcortical regions exclusively in the left middle cerebral artery. She exhibited moderate deficits in confrontation naming and had perceptual errors. Another case was reported with crossed aphasia was a 39-year-old right handed man had an infarct in the putamen, lateral head of caudate, anterior paraventricular white matter, and frontal subcortical white matter. He exhibited mild dysarthria; had fluent language output with mild word-finding deficits and phonemic paraphasias. Phonemic errors were also observed in repetition, oral reading, and writing. Based on the language deficits observed secondary to subcortical damage the author concluded that even subcortical structures have an important role to play in language functioning.

Coppens, Hungerford, Yamaguchi, amd Yamadori, (2002) presented a thorough analysis of published crossed aphasia cases. It was reported that patients with crossed aphasia were comparable to persons with aphasia secondary to left hemisphere lesions in terms of age, gender distribution and aphasia type distribution. Hence it was concluded that crossed aphasia due to a damage only to the right subcortical structures or damage to both right cortical and subcortical structures have characteristics similar to those of aphasia associated with left cortical areas.

Coppens and Hungerford, (2011) reported a few cases with crossed aphasia with the aim of discussing the major issues related to symptomatology and diagnosis. One of those was a 77-year-old man, right-hander, who suffered from a thromboembolic infarction in the right hemisphere involving the frontal as well as parietal areas. The verbal output was non-fluent and had poorly articulated and perseverated utterances. The automatic speech was considerably easier to produce and auditory comprehension was unaffected. He also exhibited major word finding and repetition difficulties. With an intensive speech-language therapy the case showed significant improvement in all the abilities and was left with mild residual aphasia. The authors conclude that the anatomical, as well as clinical correlations, appear similar to uncrossed aphasia and the rate and extent of recovery also appear in par with typical aphasia following left-hemisphere damage.

The lesion distribution and patterns of recovery in crossed aphasia are reported to resemble those of aphasia caused due to left hemisphere lesions (De-

warrat, Annoni, Fornari, Carota, Bogousslavsky, & Maeder, 2009). Crossed aphasia has often been said to be mild in severity and transient in nature (Brown & Hacaen, 1976; Sapir, Kokman & Rogers, 1986; Kim, Shin & Kim, 2011). However, Marien, Paghera, De Deyn, and Vignolo, (2004) reported that patients with crossed aphasia were still aphasic in the chronic stage and suggested that crossed aphasia is not a transient disorder. However, only a few cases had long-term follow-up, and the precise underlying mechanism of recovery is not yet known.

Some of the studies have tried documenting improvement in language abilities with intensive language intervention. One such study is by Robin and Sheinberg, (1990) who supplied data from a longitudinal study on the effects of subcortical lesions on speech and language abilities. In the study, they reported about four individuals who had lesions in the right subcortical structures. All these individuals received language intervention and were monitored over periods for two years. It was revealed that there was a significant improvement noticed in naming, repetition and spontaneous speech but the comprehension remained mild-moderately impaired.

The efficacy of language intervention in persons with crossed aphasia has been reported by Bhatnagar, Buckingham, Creegan, and Bey in 2011 through a unique case, who exhibited a congenital lesion in the right hemisphere. She exhibited aphasia following a ruptured right subcortical arteriovenous malformation. The patient received language treatment for impaired verbal output which eventually improved the quantum of verbal output.

Kim, An, Shin and Kim in 2017 documented language recovery and metabolism of the brain in a patient with crossed aphasia who was provided an intensive language treatment by a specialized speech-language therapist. Conventional speech-language therapy, 40-min sessions three times a week was provided to the patient. At 3 weeks and 24 months after onset, the serial F-18 fluorodeoxyglucose positron emission tomography (FDG PET) scan was performed. Between the baseline and follow-up evaluation, subtraction imaging was conducted. The patient was diagnosed as Broca's aphasia in the initial evaluation. Four months later, there was a significant improvement in spontaneous speech, repetition, naming, and reading, with a relatively moderate progress of comprehension. At 2 year followup, the patient showed improvement significant improvement in expression domains but there was no much progress seen for comprehension. Also, subtraction PET scan revealed that there was 20% increase area of F-18 FDG uptake corresponded to increase in metabolism in the areas such as the right basal ganglia, the right medial temporal lobe, occipital lobes, and the left cerebellum.

There are umpteen numbers of questions still to be answered regarding the crossed aphasia population. It appears that careful examination and report of the symptoms displayed by the crossed aphasia patients can resolve some of these questions. This paper is an attempt to profile a case who exhibited subcortical crossed aphasia following a surgery for aneurism cure. The patient's aphasia symptoms did not seem to differ from those associated with left cortical lesions. Though there was a spontaneous recovery of language functions, some of the expressive and comprehension deficits still persisted. The patient received intensive language intervention for the same and showed significant improvement in both receptive skills and verbal output.

Method

Participant details

A 42- year old woman, a right-hander, suffered from acute aphasia, left upper and lower limb paralysis following a surgery is done for right ophthalmic ICA aneurysm cure. She had 17 years of formal education and was a native speaker of Kannada language.

Initially, the patient had recurrent seizure attacks with associated symptoms like blurring of vision, giddiness, and severe localized headache. The patient was seen by the Neurologist and had prescribed medication for seizures. Though the patient was under medication, the seizure symptoms did not subside and she continued to have yearly once seizure episode for five to six years. Following this, she was recommended to undergo Computed Tomography Angiography (CTA). The CTA report revealed a fairly well defined saccular aneurysm arising from Supraclinoid segment of Right ICA Aneurysm with its tip directed Antero-supero medially (see figure 1). The patient had to undergo Flow diverter placement surgery for the same. Series of CTA scans were taken in order to monitor the patient's condition. The CTA took soon after the surgery revealed no infarct or hemorrhage. But two days post-surgery, the patient developed sudden weakness in left upper and lower limbs, complete loss of speech along with deteriorated visual acuity and drowsiness. CTA scan taken was repeated and it revealed Intraparenchymal hemorrhage with a hematoma in right external capsule, putamen, corona radiate, a part of the centrum semiovale and perilesional edema in the posterior internal capsule. Expansion of hematoma on post-operation day three and no further expansion of hematoma on post-operation day four was noticed on subsequent CTA scans. After confirming the complete collapse of an aneurysm, the patient was discharged. The deficits such as left hemiplegia and speaking difficulty that arose following the surgery persisted even after the patient was discharged from the hospital.

As a consequence of intraparenchymal hemor-

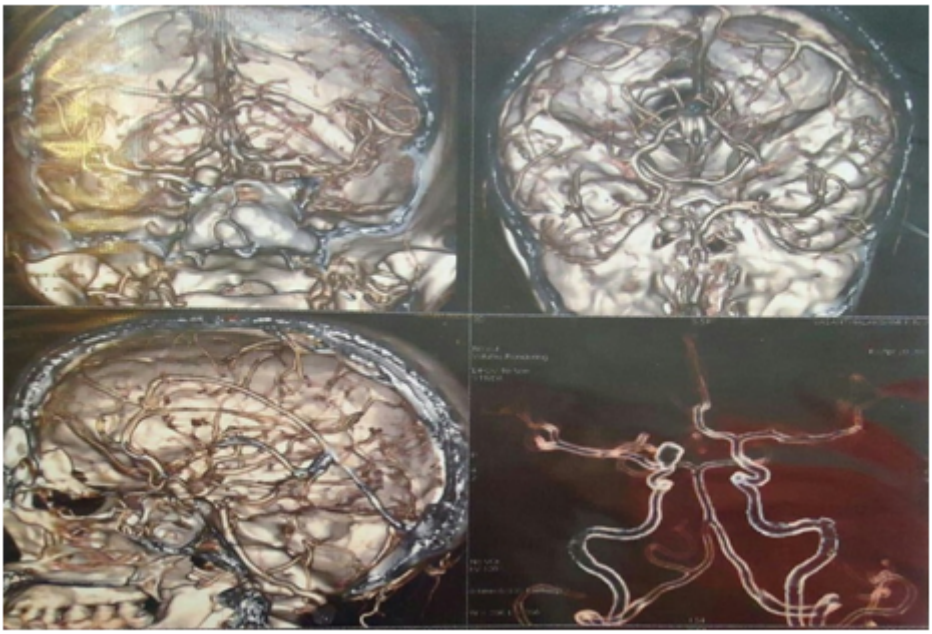

Figure 1: Computed Tomography Angiography (CTA) revealing a fairly well defined saccular aneurysm arising from Supraclinoid segment of Right ICA.

rhage with hematoma, the patient had complete loss of speech, associated weakness in the oro-motor structures, deteriorated visual acuity and weakness of both left upper and lower limbs. She was unable to recognize her family members and was able to communicate only through simple gestures.

Within three months, the patient showed spontaneous improvement in the domains of both comprehension and expression. She was able to recognize her family members and friends and could recall past events. Speech became fluent but had perseverative errors, phonemic paraphasias, hesitations and groping. Auditory comprehension of casual conversation was functional. Though there was a spontaneous recovery of language functions in the patient, significant naming deficits, difficulty in comprehending complex commands, and difficulty in repeating complex sentences, oro-motor weakness on left, difficulty in reading, calculations and writing still persisted. Hence the patient had to seek clinical help and consulted a speech-language pathologist four months post-surgery.

Materials

Edinburgh Handedness Inventory, Pen shifting test and Western Aphasia Battery- Kannada and simple pictures for narrative discourse assessments were used.

Procedure

Detailed profiling of patient's baseline speech-language skills was carried out after taking an informed consent from the patient by an experienced speech-language pathologist. She did not have any history of developmental delay or childhood brain damage. She was alert and cooperative during testing and responded appropriately to all tasks. Initially, the patient's handedness was assessed by using the Edinburgh Handedness Inventory (Oldfield, 1971). Edinburgh Handedness Inventory provides handedness score based on the hand preferred for performing certain basic daily routine activities. In order to check for weakness in the upper limbs, pen shifting test was done. Western aphasia battery- Kannada (WAB-K) developed by Shyamala & Ravikumar in 2008 was administered to profile the language abilities. Also, reading, writing, calculation abilities, right hemisphere functions and narrative discourse abilities were assessed informally.

Test Results-Baseline assessment

The patient obtained a handedness score of +1.0 on Edinburgh handedness scales which clearly indicated that she was a pure right-hander. In pen shifting test, she was unable to shift the pen from left hand to the right but could shift from the right hand to the left. This clearly indicated the left upper limb weakness in the patient. On informal speech mechanism examination, the patient exhibited poor intraoral breath pressure, reduced movement of lips to the right side on retraction, slighter deviation of the jaw to the right side, reduced velar movement on right and slower diadochokinetic rate.

On WAB- Kannada, the patient had poor scores on comprehension of sequential commands, object naming, word fluency, and repetition tasks (see Table 1). The spontaneous speech had adequate information content. She was fluent with some persever-

ative errors, phonemic paraphasias, hesitations and groping. Auditory comprehension of casual conversation was functional. Auditory word recognition, comprehension of yes/no questions and comprehension of simple auditory commands were preserved. But the patient exhibited significant difficulty in comprehending and following complex sequential commands.

Table 1: Results on the Western Aphasia Battery-Kannada at 3 months post surgery

Subtest	Raw score
Spontaneous speech	
Information content	8/10
Fluency	8/10
Auditory comprehension	
Yes/No questions	54/60
Auditory word recognition	59/60
Sequential commands	37/80
Repetition	77/100
Naming	
Object Naming	37/60
Word fluency	4/20
Sentence completion	8/10
Responsive naming	9/10
Aphasia Quotient	74

Performance of repetition task was flawless except that she was unable to repeat two of the complex sentences. Performance on object naming was with errors primarily represented by phonemic paraphasia and perseveration. She was unable to complete word fluency task and had an expression of frustration or perplexity. But she could perform well on sentence completion and responsive naming tasks. Reading and writing skills were informally checked. Reading comprehension was intact but in reading aloud phonemic paralexic errors and perseverations were observed. In writing, the patient exhibited patterns which were similar to the speaking impairment. Paragraphic errors such as phoneme substitutions, neologisms, and omissions were present.

No evidence of non-verbal apraxia of speech and oral apraxia were observed; she had no difficulty imitating or following directions for oral volitional movements. Right hemisphere functions were informally assessed. She did not exhibit visual neglect, spatial disorientation or defective emotion and affect processing.

The informal assessment was done to check the calculation abilities. In mental arithmetic, she was able to perform rote memory multiplication and simple addition, but not simple subtractions or more complex multiplications. In written arithmetic, she was able to perform addition and subtraction not involving carrying but had difficulty in computing the carried number/borrowed numbers in addition and subtraction. Also, had difficulty to perform complex multiplication and division.

In addition, a narrative discourse for picture description and conversation was assessed informally. For narrative discourse, simple pictures were provided which had three to four narrative events. In both the tasks, most of the narrative elements were mentioned but had frequent word finding difficulties, pauses, repetitions, and phonemic paraphasias. Discourse structure was unaffected. The patient had communication intent. She initiated describing pictures without hesitations and asked for assistance in the understanding picture when needed. Coherence was maintained. Frequent topic shifts or perseveration in the topic were not observed. Information content was meaningful and could provide adequate information but linguistic fluency was disturbed. Also, revision behaviors and repair strategies were noticed.

Details of therapeutic intervention

After detailed profiling of the language abilities, the therapeutic intervention was provided by an experienced speech-language pathologist. Manual for adult aphasia treatment in Kannada (MAAT-K) developed by Goswami and Shanbal in 2011, was used to provide language intervention. Three sessions of language therapy per week were provided and the duration of each session was about 50 minutes to one hour. MAAT-K consists of five main domains: Functional communication, Comprehension, and Expression, Repetition, Naming, Reading, and Writing. The subsections of the different domains cover a series of activities which can be carried out by the clinician to elicit a wide range of responses from individuals with aphasia.

The therapy goals taken up for the patient were to improve confrontation naming, generative naming, responsive naming and repetition skills so as to reduce her word finding difficulty thereby improving fluency during the conversation and narrative discourse. The treatment technique, Semantic feature analysis was incorporated for the same. Also, frequent feedback was provided regarding the error response. To improve comprehension of two-three step commands, semantic and syntactic judgment, sufficient cueing (semantic, graphical and pantomime) and repeated trials were given. To improve semantic association semantic mapping was used. With 25 sessions of intensive language therapy, the patient showed significant improvement.

Test results-Post therapy

Western Aphasia Battery-Kannada was readministered after 25 sessions of language therapy. The frequency of therapy was three sessions per week and each session ranged from 50 minutes to one hour. Scores on fluency, comprehension of sequential com-

mands, object naming, and repetition improved (see table 2) when compared to the baseline scores on WAB-K. Noticeable improvement was noticed in object naming and word fluency. Repetition improved and the patient could follow complex commands effortlessly. The profound improvement was noticed in fluency for conversation and narrative discourse as well. Nevertheless, the patient had residual deficits in naming.

Table 2: Results on the Western Aphasia battery-Kannada post therapy

Subtest	Raw score
Spontaneous speech	
Information content	8/10
Fluency	9/10
Auditory comprehension	
Yes/No questions	57/60
Auditory word recognition	59/60
Sequential commands	60/80
Repetition	92/100
Naming	
Object Naming	54/60
Word fluency	14/20
Sentence completion	9/10
Responsive naming	9/10
Aphasia Quotient	87.2

Discussion

The case had Right Supra Ophthalmic Segment ICA Aneurysm and underwent Flow diverter placement surgery for the same. As a consequence of complication, she developed Intraparenchymal hemorrhage with a hematoma in right external capsule region. The clinical profiling of the case showed that the crossed aphasia was developed after an Intraparenchymal hemorrhage with a hematoma in the right external capsule region. These findings concur with previous observations that the crossed aphasia in right-handed persons is associated with lesions of the basal ganglia along with or without the cortical lesions in the right hemisphere (Habib et al., 1983; Coppens, Hungerford, Yamaguchi, & Yamadori, 2002; Kim, Shin, & Kim, 2011).

In the present case and in others (Habib et al.,, 1983; Coppens, Hungerford, Yamaguchi, Yamadori, 2002; Kim, Shin, & Kim, 2011), the language disturbance is a result of right hemisphere lesion. One possible explanation for this may be that the fibers communicating between the cortical and subcortical structures are interrupted due to subcortical infarct causing altered inhibitory-disinhibitory mechanism of the subcortex. Also, cortical hypo-perfusion may occur leading to disturbed language functioning (Kasyapa, 2016).

Discussion with the patient and her family indicated that she did not have any history of developmental delay or brain damage at childhood. She was never been forced to shift from left to right-handedness. The Eidenberg Handedness inventory revealed that she was a pure right-hander. She had a formal education of 15 years and was a native speaker of Kannada .

As a consequence of hemorrhage, she was unable to recognize family members, had reduced verbal output, deteriorated visual acuity and weakness in both left upper and lower limbs. The case showed impairment in all channels of communication such as auditory comprehension, naming, spontaneous speech, and repetition. Her initial verbal language was characterized by significant word finding difficulty, fragmented utterances, and unintelligible speech. These findings, as well as findings by others (Brown & Hecaen, 1976; Alexander & Annett, 1996; Kim, Shin & Kim, 2011), suggest that language deficits as a consequence of crossed aphasia are reported to resemble those of aphasia resulting from left hemisphere lesions.

Within three months she showed spontaneous recovery. Speech became fluent but had perseverative errors phonemic paraphasias, hesitations and groping. Auditory comprehension of casual conversation was functional. These findings are in line with previous studies which stated that the crossed aphasia is usually mild in severity and transient in nature (Brown & Hacaen, 1976; Sapir, Kokman & Rogers, 1986; Kim, Shin & Kim, 2011).

Though there was a spontaneous recovery of language functions in the patient, some of the language deficits still persisted. When the case reported to a speech-language pathologist, she exhibited significant naming deficits, hesitation, groping and mild-moderate deficits in auditory comprehension. With an intensive language intervention, she showed significant improvement in object naming, word fluency, and phoneme fluency. Repetition was flawless and the patient could follow complex commands effortlessly. The profound improvement was noticed in fluency for conversation and narrative discourse as well. These findings are in coherence with the previous studies by Robin and Schienberg, (1990); Bhatnagar, Buckingham, Creegan, and Bey, (2011); Kim, An, Shin, and Kim, (2017) where they reported significant improvement in language abilities following an intensive language intervention.

Our case showed ample recovery of language skills with language intervention, with residual deficits in naming. It is difficult to attribute the recovery to language treatment alone. Some evidence suggests that the prognosis and response to treatment in crossed aphasia are not different from those in left corti-

cal aphasia (Robin & Scheinberg, 1990; Kim et al., 2017). Additional reports on crossed aphasia may shed more light on its prognosis and response to treatment.

Conclusions

Crossed aphasia is a rare phenomenon for which we do not yet have a satisfactory explanation. Understanding its clinical existence depends entirely on the analysis of published case studies. Hence, case studies need to be reported systematically and in as much detail as possible. Further, evidence-based studies are required to shed light on the efficacy of language intervention in individuals with crossed aphasia. Positron emission tomography and cerebral blood flow studies and pre and post language intervention reports are necessary to document improvement in language functioning in individuals with crossed aphasia.

References

Alexander, M. P., & Annett, M. (1996). Crossed aphasia and related anomalies of cerebral organization: case reports and a genetic hypothesis. *Brain and Language, 55*(2), 213-239.

Bartha, L., Marien, P., Poewe, W., & Benke, T. (2004). Linguistic and neuropsychological deficits in crossed conduction aphasia. Report of three cases. *Brain and Language, 88*(1), 83-95.

Bhatnagar, S. C., Buckingham, H. W., Puglisi-Creegan, S., & Hacein-Bey, L. (2011). Crossed aphasia in a patient with congenital lesion in the right hemisphere. *Aphasiology, 25*(1), 27-42.

Boissezon, X., Démonet, J. F., Puel, M., Marie, N., Raboyeau, G., Albucher, J. F., & Cardebat, D. (2005). Subcortical aphasia: a longitudinal PET study. *Stroke, 36*(7), 1467- 1473.

Brown, J. W., & Hecaen, H. (1976). Lateralization and language representation Observations on aphasia in children, left-handers, and "anomalous" dextrals. *Neurology, 26*(2), 183.

Chengappa, S.K., & Ravikumar. (2008). Normative and clinical data on the Kannada version of Western Aphasia Battery (WAB-K). *Language in India, 8*:6 June.

Coppens, P., Hungerford, S., Yamaguchi, S., & Yamadori, A. (2002). Crossed aphasia: an analysis of the symptoms, their frequency, and a comparison with left-hemisphere aphasia symptomatology. *Brain and Language, 83*(3), 425-463.

Coppens, P. (2011). Crossed Aphasia. In Encyclopedia of Clinical Neuropsychology (pp. 747 747). Springer New York. Damasio, A. R. (1992). *Aphasia. New England Journal of Medicine, 326*(8), 531-539.

Davis, G. A. (2007). Aphasiology: Disorders and clinical practice. Pearson College Division.

Dewarrat, G. M., Annoni, J. M., Fornari, E., Carota, A., Bogousslavsky, J., & Maeder, P. (2009). Acute aphasia after right hemisphere stroke. *Journal of Neurology, 256*(9), 1461-1467.

Goodglass, H. K., & Kaplan, A. E. (1972). *The assessment of aphasia and related disorders.* Philadelphia: Lea & Febiger.

Goswami, S.P., & Shanbal, J.C. (2011).Manual for Adult Aphasia Therapy in Kannada. Independent project at All India Institute of Speech and Hearing, University of Mysore, Mysore, India.

Habib, M., Joanette, Y., Ali-Cherif, A., & Poncet, M. (1983).Crossed aphasia in dextrals: A case report with special reference to site of lesion. *Neuropsychologia, 21*(4), 413-418.

Kasyapa, V. (2016). *Aphasia from Harrison's Principles of Medicine.* Health and Medicine. Dejongs.

Kim, H. S., Shin, J. B., & Kim, J. M. (2011). Crossed transcortical motor aphasia, left spatial neglect, and limb and magnetic apraxia due to right anterior cerebral artery infarction. *Annals of Rehabilitation Medicine, 35*(6), 949-953.

Kim, N. Y., An, Y. S., Shin, J. C., & Kim, Y. W. (2017). Recovery of language functions and brain metabolism in a patient with crossed aphasia: 2-year follow-up of brain FDG PET images. *Aphasiology,* 1-9.

MariĂT̆n, P., Paghera, B., De Deyn, P. P., & Vignolo, L. A. (2004). Adult crossed aphasia in dextrals revisited. *Cortex, 40*(1), 41-74.

Oldfield, R. C. (1971). The assessment and analysis of handedness: The Edinburgh inventory. *Neuropsychologia, 9*(1), 97-113.

Robin, D. A., & Schienberg, S. (1990). Subcortical lesions and aphasia. *Journal of Speech and Hearing Disorders, 55*(1), 90-100.

Sapir, S., Kokmen, E., & Rogers, P. J. (1986). Subcortical crossed aphasia: A case report. *Journal of Speech and Hearing Disorders, 51*(2), 172-176.