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Case of study of a patient with ischemic stroke

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Dedication

I dedicate this bachelor thesis to my family in Saudi Arabia my father, my mother, my brothers and my sisters who were supporting me throughout my educational course. I dedicate it to my baby boy who gave me the motivation and brought a brightness to my life and for my lovely wife for her incredible support throughout my course in Czech Republic and the great support that she gave me in many ways more than anyone else. It is also dedicated to university staff who were cooperating with student in a nice manner and specially to my professors for all what they gave me during my study period.

Abstract

Title: Case study of physiotherapy treatment of patient with Ischemic stroke and hemiplegia affected the left side of his body.

Goals:

To show the full kinesiological assessment, special therapy approaches and therapy effect during 7 sessions. The theoretical part explains the clinical picture, physiological, pathophysiology, aetiology, categories, pharmacotherapy and surgical intervention of the stroke. And the main goal of the practical part is to present the examinations, the therapeutic approaches and the conclusion that were made after the rehabilitation process with the patient.

Methods:

During my clinical practice, we used the main structure for the Kinesiological assessment according to the Prague School approaches. Manual muscle testing according to Kendall (2005) and Janda (2013) were also used (under the instructions and the protocols from the Kladno hospital). Finally, we applied final kinesiological assessment to compare the patient situation before and after the therapy and to see the effect of our work.

Results:

As a result of our work we found that the therapy course shows some noticeable results in increasing the ROM and the function of the affected upper extremities. However, significant improvement in patient stability and in overall condition appeared, which influence the improvement in ADL and increase the self-reliance on the patient.

Keywords: Stroke, Ischemic, Approaches, ROM, PNF, DNS, Physiotherapy, Rehabilitation, Treatment.

Declaration

I declare that I wrote my graduation dissertation (bachelor/graduate) independently, and that I have stated all the information sources and literature I used. Neither this thesis nor any substantial part of it have been submitted for the acquisition of another or the same academic degree.

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1. Introduction

This work, based on a case study of an ischemic stroke patient combined with hemiplegia of his left body side which I selected this case to be my study case for the bachelor thesis due to its high frequency to have this diagnosis in my country (Saudi Arabia) and after my father was diagnosed with ischemic stroke almost one year ago, I decided to have this diagnose as my final project. The main parts of my thesis are Theoretical part and practical part .

The theoretical part consists of a general explanation of the brain parts and its Anatomy, Neurophysiology of the brain, Stroke categories, types and their Aetiology, Epidemiology, pathophysiology, symptoms after having a stroke, Patients clinical picture, view in post-stroke stages and Physiotherapeutic approaches.

The practical part is counted as the main part of my work where it includes a kinesiological assessment (initial and final examination), Day to Day therapy using some Physiotherapeutic approaches which I've been taught from the faculty during my study and under the instruction of Kladno hospital, Conclusions of both initial and final examinations and the effect of the therapy.

The main goal of the therapy was to restore functions that have been lost and to increase levels of patient independence during his activity of daily living especially on the left hand.

My work was held at the Kladno hospital for two weeks from Monday the 30th of Jan 2017 to Friday the 10th of Feb 2017. I had 7 sessions with the patient who had an ischemic stroke. All sessions were supervised by Bc. Tomáš Modlinger.

2. Background

2.1. Epidemiology of Stroke

Acute ischemic stroke (AIS) is also referred to as stroke in the US and characterized by acute loss of blood supply in specific areas of the brain. In most cases, AIS occurs due to poor blood supply in the vascular territory of the brain which results in temporary or permanent and irreversible neurological function. AIS was also referred to as stroke syndrome or cerebrovascular accident (CVA) leading to loss of neurological function. Stroke is considered as a non-specific state of brain injury that is caused by several pathophysiologic pathways which leads to neuronal dysfunction. Based on current evidence, stroke is classified into 2 broad categories, i.e. acute ischemic or haemorrhagic stroke while AIS is primarily caused due to embolic or thrombotic occlusion of a cerebral artery.

Stroke is the 3rd leading cause of death in the US while it is the leading cause of long-term and serious disability. In the US, 85% of strokes are ischemic while 15% are haemorrhagic. In the case of non-haemorrhagic stroke, only 25% are due to embolism from the heart (thromboembolic strokes), 25% of strokes are due to small vessel disease or lacunar strokes while the remaining are large vessel disease (Morris D & Schroeder E n/d). In terms of mortality, stroke occurrence has been closely associated with death rates in patients, wherein an estimated 158,448 deaths were reported in 1998 due to stroke in the US. Based on current evidence, stroke is the 2nd leading cause of dementia and leading cause of chronic disability in the US (Ovbiagele, & Nguyen-Huynh 2011). The impact of stroke worldwide is estimated to be even more worse but no defined prevalence or incidence rates have been published. As per recent evidence, stroke affects early 7% of the adult population in the US which translates to about 7 million people. (Ovbiagele, & Nguyen-Huynh 2011). The prevalence and incidence rate of stroke is estimated to increase by 10 to 15% every year increasing the burden of the health disorder in the healthcare sector (Morris D & Schroeder E

n/d). Based on recent evidence, 3% are subarachnoid haemorrhage, 10% are primary haemorrhages, while 87% are ischemic infarctions (Ovbiagele, & Nguyen-Huynh 2011).

An estimated 800,000 first-time (primary) or recurrent (secondary) stroke cases occur in the US every year wherein over 600,000 cases are first-time or new cases of stroke. In respect to the global context, the primary haemorrhages are the highest and constitute for about 10% to 25% of all stroke cases (Morris D & Schroeder E n/d). In context to ethnicity, Latin Americans, Africans, and Asians then to have a higher rate of primary haemorrhages compared to their European counterparts. The prevalence of ischemic stroke is common in Europeans which is considered as a major healthcare burden. However, in Asians, an estimated 25% of cases are primary haemorrhages while it accounts for 10% to 17% of primary haemorrhages in Western countries (Ovbiagele, & Nguyen-Huynh 2011).

The risk of ischemic stroke doubles for every decade after the age of 55 years, wherein the incidence rate of stroke between the age of 35 to 44 is approximately 30 to 120 of 100,000 per year while for people between 65 to 74 years, it is approximately 670 to 970 of 100,000 per year. Based on current evidence, stroke does occur among children but the prevalence rate is very low compared to adults (approx. 1 to 2.5 of 100,000 per year). In context to ischemic stroke cases, the incidence rate is only 15 to 30% for children while it is about 50 to 70% for primary haemorrhages. The primary cause of stroke in children is sickle cell disease wherein the incidence rate is highest between 2 to 5 years (Ovbiagele, & Nguyen-Huynh 2011). The total annual cost for stroke is estimated to be \$72 billion while the projected cost is estimated to triple between 2012 and 2030 to about \$184 billion for individuals between 65 and 79 years. However, in the case of ischemic and haemorrhagic stroke, clinical examinations cannot be solely used to differentiate the two. It is

recommended to review computed tomography (CT) scanning or magnetic resonance imaging (MRI) (Morris D & Schroeder E n/d).

2.2. Stroke Categories

It is important to understand the different categories of stroke itself prior to understanding the various forms of ischemic stroke and the underlying biological mechanisms. Based on current evidence there are 3 types of stroke which were formally derived from the multi-centre Trial of ORG 10172 in Acute Stroke Treatment (TOAST): (a) Large-artery (b) small-vessel, or lacunar and (c) cardioembolic infarction (Kuklina, et al. 2012).

Most clinicians and healthcare providers (HCPs) are involved in the identification of the stroke type prior to initiating any medical or surgical intervention. In the case of a larger-artery infarction, a thrombotic in situ occlusions takes place on the atherosclerotic lesions in the carotid, vertebrobasilar, and cerebral arteries. In most cases, patients with large-artery occlusion may suffer from these lesions that is proximal to major branches in these areas of the brain. In some cases, large artery infarctions could also be of cardioembolic origin (Chen, , Zeng, & Hu 2014). Many advanced diagnostic imaging techniques can help identify patients with large artery infarctions. The second type of stroke, i.e. cardiogenic emboli is reported to be one of the most common sources of stroke. In nearly 90% of patients, cardiogenic emboli is a common but recurrent for of stroke. Based on current evidence. Cariogenic emboli accounts for over 20% of all forms of acute stroke while it has the highest rating for a 1-month mortality. Most researchers claim that cardiogenic emboli is highly complex, complicated to treat, and the survival rate for recurrent cardiogenic emboli is the lowest among all types of stroke.

The third type of stroke, i.e. small vessel or lacunar strokes is characterized by the presence of a small focal area of ischemia. In this case, patients are observed to have a minor

obstruction to a single small vessel while it penetrates deeply into the arteries. This is a rare form of stroke which has a distinct and specific form of vascular pathology that can be identified through advanced brain imaging diagnostic modalities (Tsai, Thomas, & Sudlow, 2013).

2.2.1. Large-artery infarctions

In the case of large-artery occlusion, patients often suffer from high risk of mortality and that most clinicians need to assess the underlying pathological features within the shortest framework. Large-artery infarction is characterized by embolization of atherosclerotic debris which may have a cardiac origin or typically from a common or internal debris within the carotid arteries. In a few cases, large-artery occlusion is formed due to the plaque ulceration and in situ thrombosis which is easily identifiable through advanced Non-contrast computed tomography (CT) or Magnetic resonance angiogram (MRA). It is common for most clinicians to identify that large-vessel or artery ischemic strokes are found in the MCA territory while the ACA territory is affected at a lesser degree (Xu. 2014).



Fig 1. Left anterior and middle cerebral artery territories consistent with acute infarction (Jauch, 2015).

In most cases, clinicians would assess a patient using non-contrast computed tomography. In figure 1, a CT of a 52-year old depicts a history of worsening right-sided weakness and aphasia (Jauch, 2015). It is indicative of left anterior and middle cerebral artery territories with acute infarction that reflects diffuse hypodensity and sulcal effacement with mass effect. It is clearly evidence that the patient has a developing petechial hemorrhage the large area of infarction which is characterized by the presence of scattered curvilinear areas of hyperdensity (González, 2012).

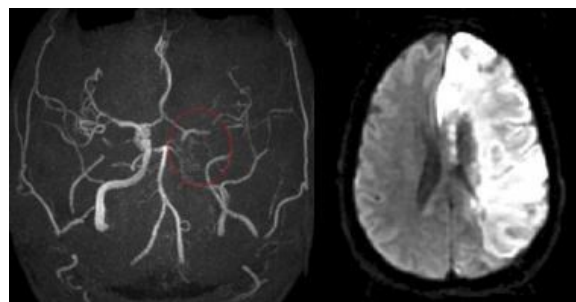


Fig 2. Occlusion or high-grade stenosis of the distal middle cerebral artery (MCA) trunk (Jauch, 2015).

In some cases, clinicians would identify large-artery occlusions through magnetic resonance angiogram (MRA). In figure 2, the MRA reflects an occlusion of the left precavernous supraclinoid internal carotid artery (ICA, red circle). The right image indicates the presence of true restricted diffusion on the apparent diffusion coefficient (ADC) map based on diffusion-weighted image, a key feature of consistent acute infarction (González, 2012).

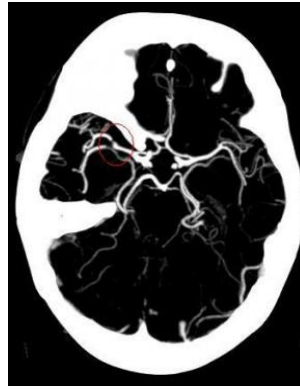


Fig 3. High-grade stenosis at the right middle cerebral artery (MCA) trunk (red circle) (Jauch, 2015).

In some cases, clinicians would use CT combined with maximum intensity projection (MIP) to identify high-grade stenosis at the branching point of the right middle cerebral artery (MCA) trunk (Fig 3). In most cases, high-grade stenosis at the MCA is indicative or suspected for embolus or thrombus. CTA is the most valuable imaging diagnostics since it helps detect large- vessel stenosis and occlusions accurately that accounts for 1/3rd of ischemic strokes.

2.2.2. Small vessel or lacunar strokes

Based on current evidence, lacunar strokes are the most common forms of stroke affecting the elderly population. Small vessel or lacunar strokes is often a cause of major health complications including hypertension and cardiometabolic health disorders. Lacunar strokes accounts for about 15-20% of ischemic strokes. Clinicians indicate that patients who suffer from lacunar strokes often have a better mortality and morbidity rate compared to other forms of stroke if diagnosed and treated early. Based on the pathophysiology of the patient, clinicians indicate that lacunar strokes form as a result of occlusion of the penetrating branches of the circle of Willis, the lenticulostriate arteries, the penetrating branches of the

MCA, basilar artery, or even the vertebral artery. Based on current evidence, hypertension among adults or elderly patients is a common cause of lacunar or small-vessel strokes.

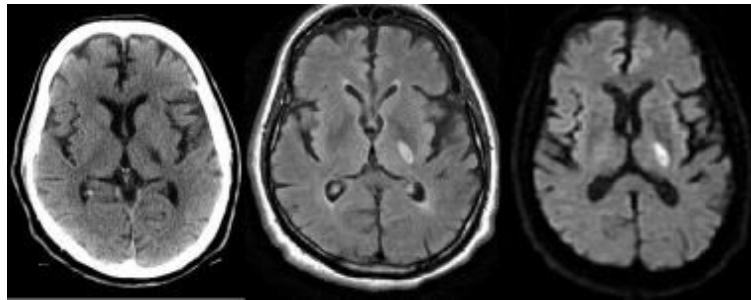


Fig 4. Lacunar stroke CT and MR images (Jauch, 2015).

In most cases, a patient may reflect hypodensity in the left posterior limb of the internal capsule in an Axial noncontrast computed tomography (CT) (Left image), indicating right-sided weakness (Fig 4). It is also indicative of high signal on the fluid-attenuated inversion recovery (FLAIR) sequence on the lesion (middle image) and apparent diffusion coefficient (ADC) maps which is indicative of an acute lacunar infarction (right image). In most cases, lacunar infarcts occur in the deep grey matter-like structures and are no more than 1.5cm in size. They also appear in the brainstem, corona radiata, and cerebellum (Fig 4). It is important for clinicians to identify the underlying pathophysiology in the case of small-vessel strokes.

2.2.3. Cardioembolic infarction

Based on current evidence, cardiogenic emboli comprise of about 20% of total acute stroke cases and is known to have a higher mortality and morbidity rate compared to lacunar strokes. In the case of cardioembolic infarction, clinicians state that emboli could be a result of debris or clogs from the heart or even the extracranial arteries such as the aortic arch. In some cases, the right-sided circulation (paradoxical emboli) could also cause cardiometabolic infarction which relates to the obstruction of the passage through a patent foramen ovale.

Based on current evidence, some of the most common sources of cardiogenic emboli include:

(a) Mural thrombi which may include severe congestive heart failure, dilated cardiomyopathy, myocardial infarction, or atrial fibrillation. (b) Valvular thrombi which may include endocarditis, mitral stenosis, or the use of prosthetic valve and (c) Atrial myxoma (Jang, & Chang, 2013).

Based on current evidence, about 2-3% of incidence of stroke are associated with an acute history of myocardial infarction. (Arboix, & Alió, 2010). In most cases of bilateral and multiple infarcts, the underlying pathophysiology could be a result of recurrent emboli or embolic showers (Fig 5). (Jauch, 2015).

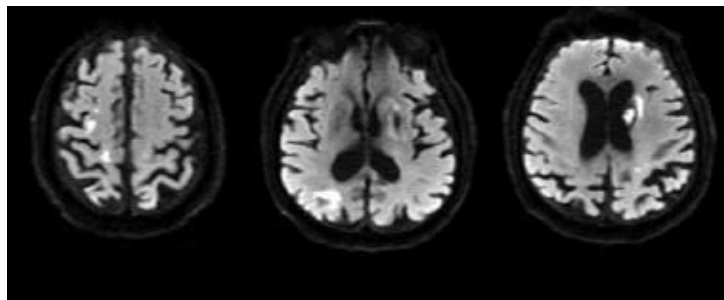


Fig 5. Cardiometabolic stroke (Jauch, 2015).

2.2.4. Thrombotic Strokes

Although large artery, lacunar, and cardioembolic strokes are most common, researchers have also identified key forms of stroke, i.e. thrombogenic which occurs due to the loss of endothelial cells from an injury. The loss is associated with a cascade of reactions and activation of clotting cascade due to the exposure of the sub-endothelium. It also leads to the inhibition of fibrinolysis, and blood stasis. It is common to observed ruptured atherosclerotic plaques in the case of thrombotic strokes. Researchers state that thrombotic stroke is associated with arterial stenosis that leads to turbulent blood flow. The cascade of reactions leads to the formation of thrombus, platelet adherence, and atherosclerosis (ie, ulcerated

plaques). These reactions often lead to the formation of blood clots which are the primary source for embolization and occlusion of arteries. In patients with widespread atherosclerosis, intracranial atherosclerosis could also cause thrombotic stroke. In rare cases, younger patients may also suffer from thrombotic stroke due to the following: (a) Fibromuscular dysplasia (b) Hypercoagulable states which include protein C deficiency, antiphospholipid antibodies, and pregnancy (c) Vasoconstriction associated with substance abuse such as amphetamines or cocaine (d) Arterial dissections and (e) Sickle cell disease (Arboix, & Alió, 2012).

2.2.5. Watershed Infarcts

Another form of stroke is the vascular watershed which is often a cause of infarctions that take place between the distal areas of the arterial territories (Juergenson, Mazzucco, & Tinazzi, 2011). In most cases, they occur due to severe hypo-perfusion or are secondary to embolic phenomena such as carotid occlusion or prolonged hypotension. (Fig 6). (Jauch, 2015).

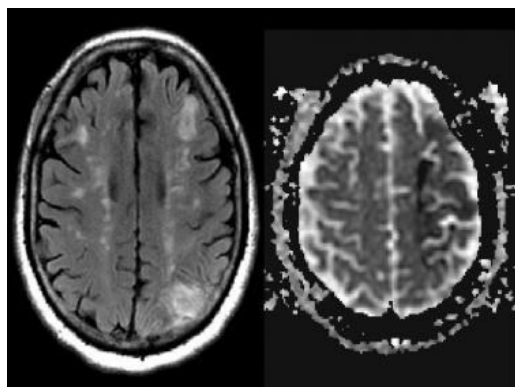


Fig 6. Watershed infarcts (Carotid occlusion or prolonged hypotension) (Jauch, 2015).

In a case study, a 62-year old man with diabetes and hypertension with a history of aphasia and transient episodes of right-sided weakness, a Magnetic resonance imaging (MRI) scan was obtained (Fig 6). It is clearly evidence from the left image that the man suffered from watershed stroke which is characteristic of high signal arranged in a bilateral and linear

fashion in the deep white matter (Left image, fluid-attenuated inversion recovery (FLAIR). It is clear from the image that the anterior and posterior middle cerebral artery (MCA) watershed areas which is typical of such cases, specifically in the deep border-zone or infarction. On the right image, clinicians also indicate an old left posterior parietal infarct while there is clear view of left-sided infarcts characteristic of low signal on the apparent diffusion coefficient (ADC) map.

2.3. Type of Ischemic Stroke

2.3.1. Posterior Circulation Infarction

In the past decade, researchers have stipulated that posterior cerebral artery (PCA) is less common than anterior cerebral artery stroke. Clinicians and researchers involved in understanding the various mechanisms in PCA require in-depth knowledge of the various structure-function relationships of this region of the brain and the neurovascular anatomy. It is important for clinicians and neurologist to identify key mechanisms which may help them identify key preventive therapies and medications to improve the quality of life of patients (Nouh, Remke, & Ruland, 2014). In the case of ischemic stroke, there is limited or obstructed blood flow to specific cerebral structures. In most cases, an interrupted oxygen and glucose delivery can be tolerated by the neuron metabolism. However, after 6 minutes, cell death is evident. In such cases, the large cortical neurons are affected and often leads to ischemia in patients. Neuronal damage is often permanent and irreversible but can be prevented in some cases. Patients with PCA have a lower risk of developing chronic disability compared to patients with anterior middle cerebral, cerebral, or basilar artery infarctions (Mehndiratta, Pandey, Nayak, & Alam, 2012).



Fig 7. Unenhanced head computed tomography (CT) scan demonstrating a subacute L posterior cerebral artery (PCA) infarct (Jauch, 2015).



Fig 8. Computed tomography (CT) scan of the brain showing hypodense areas in the right occipital lobe consistent with a recent posterior cerebral artery (PCA) ischemic infarct (Jauch, 2015).

In the case of patients with cerebrovascular accident, active neurorehabilitation is highly recommended since prolonged neural plasticity is certain after stroke. Based on current evidence, it is empirical for patients to receive active intervention by a team of rehabilitation experts to increase the probability of the patient for maximal independence in activities of daily living (ADL). (Mehndiratta, Pandey, Nayak, & Alam, 2012). In such cases the role of the physiatric clinician is essential since he/she prevents medical and surgical complications

while and also facilitates the integration of the various therapeutic services that would increase health outcomes and reduce risk of complications (Nouh, Remke, & Ruland, 2014).

2.3.2. Anterior Circulation Infarction

2.3.2.1. Origins and Sites of Occlusion

Based on current evidence, ischemic stroke, i.e. anterior circulation infarction is the most common form of its type and accounts for over 70% of all cases (Tariq, et al. 2011). Anterior circulation infarction is by far the most common form of ischemic stroke which is caused due to occlusion of small single perforator (penetrator) arteries or one of the major intracranial arteries. In context of anterior circulation infarction (ACI), the most common causes of arterial occlusion are as follows: (a) A emboli, that is primarily caused by the atherosclerotic arterial narrowing which is at the juncture of the common carotid artery or the atheroma in the aortic arch from various cardiac sources (b) The combination of superimposed thrombosis and atherosclerotic stenosis. In most cases of lacunar stroke, clinicians have reported that lipohyalinotic intrinsic disease, specifically of the small vessels are primary causes. In most cases, the most common site of occlusion includes the carotid siphon (intracranially) and proximal 2 cm of the origin of the artery (Boone, et al. 2015 and Dharmasaroja, et al. 2015).

2.3.2.2. Circulatory Anatomy

It is important to understand the anterior circulation of the brain, wherein it reflects the areas of the brain that are supplied by the right and left internal carotid arteries and their branches. The arterial circulation is involved in supplying to both the cerebral hemispheres through the internal carotid arteries. However, the arterial circulation does not involve supply to the occipital and medial temporal lobes. (Jichici, 2015).The posterior circulation, is involved in the supply to these lobes (Fig 9).

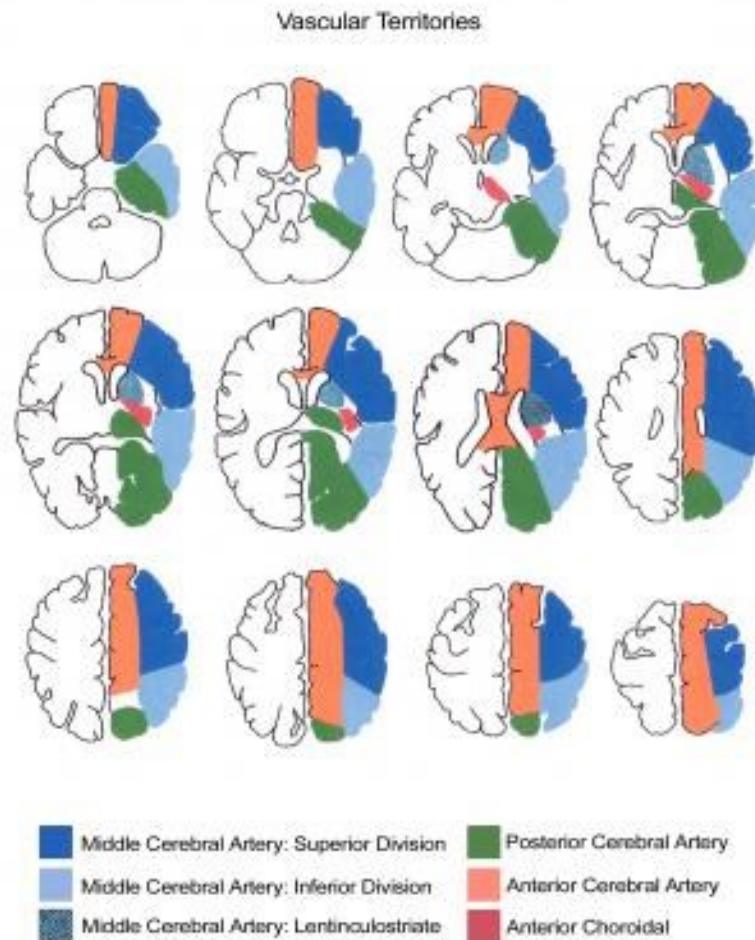


Fig 9. Anterior circulation stroke (Vascular territories) (Jichici, 2015).

At the level of the thyroid cartilage in the neck, the internal carotid artery is known to originate at the juncture of the common carotid artery. Based on advanced imaging modalities, it has been observed that the extracranial portion of the artery does not have any specific branches and it passes through the carotid canal. The intracranial portion of the artery comprises of supraclinoid portions, cavernous (an S-shaped carotid syphon), and the petrosal.

In context to the major intracranial branches, they appear from the supraclinoid portion. The first of the branches include the ophthalmic artery entering the orbit via the optic foramen to supply the optic nerve and the retina. On the contrary, the posterior communication artery appears to be on the distal side of the ophthalmic artery which meets

the posterior cerebral artery. Before the internal carotid terminates (the bifurcation end), the anterior choroidal artery appears which then enters the anterior and middle cerebral arteries. At the point of the anterior clinoid process, the ACA appears to branch medially while the MCA appears to be a direct continuation of the artery. The circle of Willis comprises of various major vessels of the anterior and posterior circulations which is based at the base of the brain. It is deemed to comprise the major vascular communication of blood vessels (Jichici, 2015).

In the case of internal carotid artery occlusive disease, collateral circulation plays a key role for the source of blood and oxygen supply. It is at the Willis circle, where the 2 primary sources of collateral flow are provided, i.e. the anterior and the posterior communicating arteries. In such cases, there are various mechanisms through which the body can adapt to acute occlusions. For example, the blood could flow from the from the contralateral ICA via the A1 segment of the contralateral anterior cerebral artery. This is followed by the flow of blood through the anterior communicating artery to the ipsilateral ACA which is also termed as reversal of flow. Most clinicians state that there is clear evidence that blood supply takes place via the posterior communicating artery from the posterior circulation (reversal flow). However, there is a high degree of variance with respect to the normal vascular anatomy with respect to the Willis circle. For example, the posterior cerebral arteries may appear to be at the internal carotid artery in about 20% of patients as compared to normal vascular variants. Thus, researchers state that the probability of variance could exists in specific parts of the brain with respect to anterior circulation. This is highly observed in foetal variants of posterior cerebral arteries. However, the human body is developed to counter-attack most acute occlusions. Thus, in the event that the primary collateral circulation fails, the presence of secondary collaterals appears in the form of branches, specifically the ipsilateral external carotid artery. Other forms of secondary

collaterals include the branches of the maxillary artery anastomose along with the ophthalmic artery that help in the reversal of flow via the ophthalmic artery itself through the occluded ICA. There is a possibility of reversal of flow in the MCA via the distal MCA branches with the help of leptomeningeal collaterals. Thus, the circulatory anatomy with respect to anterior and posterior circulation is complex and variable. However, the reversal of flow mechanisms is often perceived to be life savers in most cases (Jichici, 2015).

2.3.2.3. Ischemic Patterns

In most cases, the process of acute ischemia is highly variable across patients. Although many patients may have similar clinical syndromes, the underlying pathophysiological profiles are completely different in most of the cases. In the past decade, researchers have identified many variations in the pathophysiology of acute ischemia with the help of various advanced brain imaging modalities such as the single-photon emission computed tomography [SPECT] scanning, positron emission tomography [PET] scanning, and magnetic resonance imaging [MRI] (Tshikwela, et al. 2015). Most clinicians used these advanced imaging parameters to identify the various pathophysiologic ischemic stroke syndromes including any injury or perfusion which may help them to use active treatment. Based on current evidence, researchers have identified 2 key patterns with respect to the new MRI methods:

- The first pattern is characteristic of perfusion-diffusion mismatch, wherein researchers state that the possibility of viable ischemic tissue is high and that salvaging such tissues is possible through immediate and timely reperfusion. Based on diffusion-weighted imaging, it is clearly evidence that a larger portion of hypo-perfusion forms depicting the zone of ischemic injury in this pattern. Based on current evidence, an estimated 70% of patients may have similar perfusion-diffusion mismatch patterns in

the first 24 hours of ischemic attack. Clinicians can use MR angiography (MRA) to identify arterial occlusion in many patients (Jichici, 2015).

- Based on current evidence, the second pattern represents complete ischemia wherein radiologist state that the diffusion and perfusion lesions are the same size. In most patients, complete ischemia is characteristic of equivalent sizes of perfusion and diffusion lesions. Clinicians, radiologist, and researchers refer to the equivalent size of both perfusion and diffusion as a complete infarct. However, this form of pattern is only observed in about 10-20% of patients who arrive in the first 24 hours of ischemia stroke. Similar to the identification of perfusion-diffusion mismatch, clinicians and researchers can use MRA to identify an arterial occlusion in complete ischemia (Staykov, & Schwab, 2014).
- In contrast to the previous 2 patterns, researchers and clinicians have identified a third pattern which is characterized by reperfusion. In this case, clinicians and radiologists would observe no abnormal findings in the MRA since the perfusion deficit no longer exists in the patient. It is often a rare finding and observed in about 10-15% of patients for the first 24 hours of ischemic stroke. However, many researchers consider this finding critical and essential since it helps them develop or recommend individualized therapies for better outcomes (Jichici, 2015).
- Researchers state that reperfusion is an important and critical component of the ischemic process, specifically for the first 24 hours. Based on current evidence, about 20-40% of arterial occlusions are reported to have cleared in the first 24 hours of most patients while the recanalization rates it estimated to be as high as 90% in 21 days and about 70% in 7 days.
- Lastly, researchers report that an early reperfusion of less than 24 hours is indicative of profound prognostic benefits. It is associated with a smaller infarct size and faster

and improved outcomes among patients. However, later perfusions may not alter prognostic outcomes and is also related to a higher risk for oedema formation or haemorrhagic conversion of the infarct (Staykov, & Schwab, 2014).

2.4. Anatomy

The brain is considered as the most metabolically active organ of the human body due to the various pathophysiological mechanisms that take place not only for survival but to protect the body. However, it represents only 2% of the total body mass while it requires about 20% of the total resting cardiac output. It helps in regulating necessary glucose and oxygen supply to other parts of the body including for its own metabolism (Jichici, 2015).

2.4.1. Arterial distributions

Based on current evidence and simplified anatomical modelling of arterial distributions in the brain, it can be concluded that the anterior, middle, and posterior cerebral arteries are the 3 major arteries that supply blood and oxygen to the cerebral hemispheres. It is clearly evidence that the anterior circulation is carried out with the help of the anterior and middle and cerebral arteries that appear or arise from the supraclinoid internal carotid arteries. Clinicians and radiologists have also reported that the medial portion of the frontal and parietal lobes are supplied by the anterior cerebral artery (ACA). It also supplies to the anterior portions of basal ganglia and anterior internal capsule (Fig 10) (Jichici, 2015).

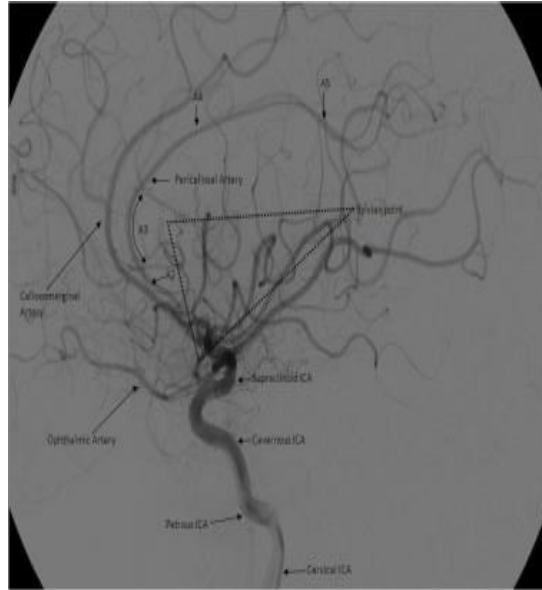


Fig 10. Lateral view of a cerebral angiogram (Jichici, 2015).

In the above image, the lateral view of a cerebral angiogram provides detailed information on the various branches of the Sylvian triangle and the anterior cerebral artery (ACA). In the above diagram, it is evident that the pericallosal artery appears to either arise from the distal to the origin of the callosomarginal branch of the ACA or the anterior communicating artery. As per cerebral angiogram (Fig 10), the segmental anatomy of the ACA can be defined as follows: (a) The A1 segment terminates at the anterior communicating artery while it extends from the internal carotid artery (ICA) juncture (b) the A2 segment is an extension of the genu of the corpus callosum and the junction to the rostrum (c) The A3 is an extension of the bend of the genu of the corpus callosum and (c) The A4 and A5 segment are extensions that are present above the callosal body (posteriorly) but it is located on the superior portion of the splenium. The apex of the Sylvian triangle is referred to as the Sylvian point while the rest of it appears on the opercular branches of the middle cerebral artery (MCA).

The middle cerebral artery (MCA) is known to directly supply blood to the parietal and frontal lobes and to some extent the lateral and anterior portion of the temporal lobes which can be observed via the branches that perpetuate to the internal capsule, putamen, and the globus pallidus (Fig 11). It is evident, that the MCA is the primary or dominant source of blood and oxygen (vascular) supply to about 80% of the hemispheres (Fig. 11).

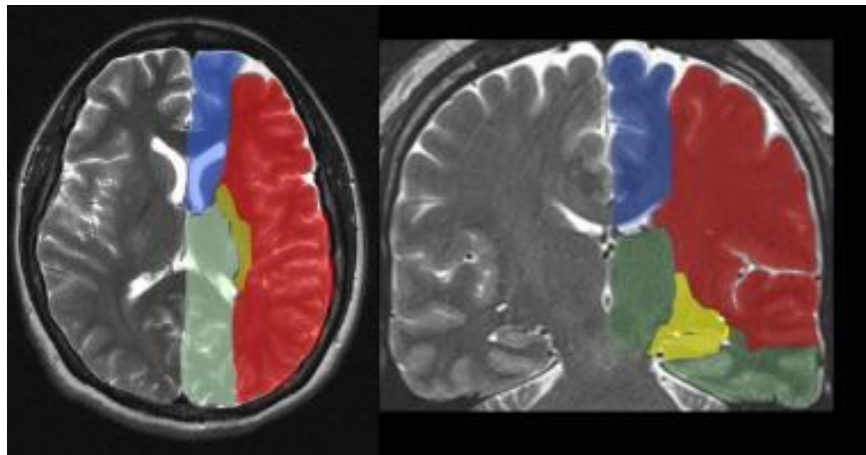


Fig 11. The supratentorial vascular territories of the major cerebral arteries (Jichici, 2015).

The middle cerebral artery (MCA; red) is primarily responsible for vascular supply to the lateral hemispheres including the basal ganglia, lateral frontal, parietal, and anterior temporal lobes. (Fig 11). The anterior cerebral artery (ACA; blue) is responsible for the vascular supply of parietal and frontal lobes while the posterior cerebral artery (PCA; green) is responsible for the vascular supply to the inferior temporal and occipital lobes. Lastly, the anterior choroidal artery (yellow) is responsible for the vascular supply to the part of the hippocampus that extends to the superior and anterior surface of the visible occipital horn (lateral ventricle) and the posterior limb of the internal capsule (Fig 11). (Jichici, 2015).

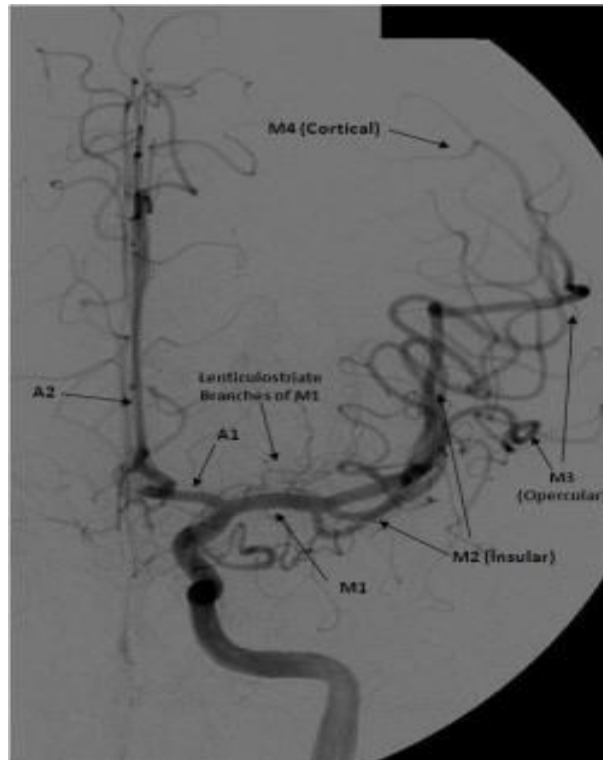


Fig 12. Frontal view of a cerebral angiogram (Jichici, 2015).

The above diagram provides an illustration of the anterior circulation which is based from the frontal view of a cerebral angiogram. It can be visualized with the help of a selective injection to the left of the internal carotid artery also referred as the ICA. The A1 segment and the A2 segment make up the anterior cerebral artery (ACA) which are placed proximal to the anterior of the communicating artery and distal to the same respectively. In the above diagram the 4-distinct segment of the middle cerebral artery (MCA) can be visualized which are as follows: (a) The M1 or the horizontal segment that is extending towards the anterior basal portion with respect to the limen insuale or the insula cortex (b) The M2 or the insular segment (c) M3 or the opercular branches and (d) M4 segment which lies on the lateral hemispheric convexities on the distal cortical branches (Fig 12). (Jichici, 2015).

It is also important to understand the various posterior cerebral arteries which appear on the basilar artery which are primarily responsible for the posterior circulation. The posterior

cerebral artery (PCA) comprises of various perforating branches that are responsible for vascular supply to the posterior and medial temporal lobes and occipital lobes as well as the thalami and brainstem. Based on current evidence, the cerebellar hemispheres receive blood and oxygen from the following: (a) The posterior inferior cerebellar artery (PICA) that provides vascular supply inferiorly. It appears from the vertebral artery (b) The superior cerebellar artery provides vascular supply superiorly and (c) The anterior inferior cerebellar artery (AICA) that arises from the basilar artery provides supply anterolaterally (Fig 13). (Jichici, 2015).

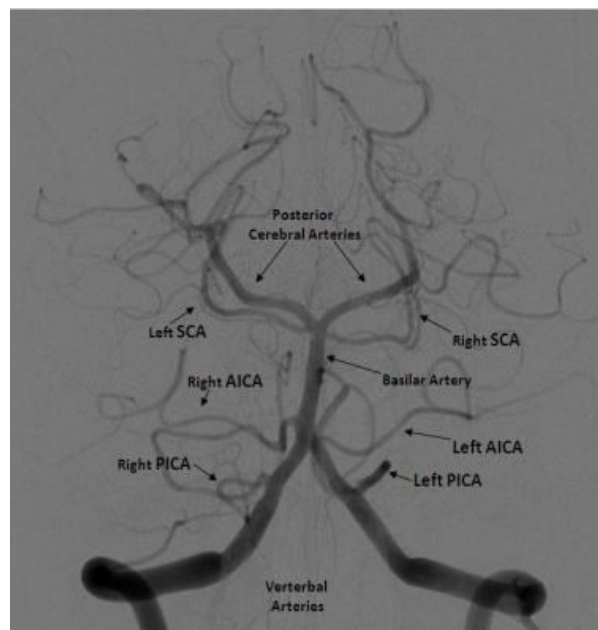


Fig 13. Frontal projection from a right vertebral artery angiogram (Jichici, 2015).

The above diagram clearly indicates posterior circulation which is characterized by the frontal projection with the help of a right vertebral artery angiogram. (Fig 13). (Jichici, 2015).

2.5. Pathophysiology

The clinical or biological pathophysiology of acute ischemic stroke has been widely examined and reviewed over the past 50 years. Researchers have identified various pathways

and biological mechanisms or cascade of reactions that lead to either temporary or permanent/irreversible changes in the brain of a patient with ischemic stroke. These underlying pathways often relate to the clinical syndromes of each patient but vary among patients with respect to extrinsic and intrinsic pathways. Vascular occlusion which is secondary to thromboembolic disease is the most common cause of acute ischemic strokes. Some of the major consequences of ischemia include depletion of cellular adenosine triphosphate (ATP) and cell hypoxia. Due to the sudden and rapid depletion of ATP, there is insufficient energy to maintain ionic gradients for cell depolarization and cell membrane. In most cases, clinicians would observe a rapid influx of calcium and sodium ions along with a passive inflow of water into the cell. Which in turn leads to the formation of cytotoxic oedema (Woodruff, et al. 2011).

2.6. Aetiology

Acute ischemic stroke is associated with a limited or restricted blood supply in specific parts of the brain. The primary cause of ischemic stroke can be attributed to relative hypoperfusion (rarely), thrombosis in situ, or extracranial or intracranial thrombotic embolism (highly prevalent). Acute ischemic stroke is characterized with complex pathophysiological processes which leads to neuronal damage and neuronal functional decline due to sudden and rapid reduction in blood flow. Many researchers and clinicians have determined the range of thresholds that describe acute ischemia. However, irreversible neuronal ischemia and injury is associated with a blood flow rate of less than 18 mL/100 g of tissue/min while a blood flow rate below 10 mL/100 g of tissue/min is associated with rapid cell death. In the US, 85% of strokes are ischemic while 15% are haemorrhagic. In the case of non-haemorrhagic stroke, only 25% are due to embolism from the heart (thromboembolic strokes), 25% of strokes are due to small vessel disease or lacunar strokes while the remaining are large vessel disease (Morris D & Schroeder E n/d). Based on a review of literature, some of the key risk factors

for ischemic stroke which include both modifiable and nonmodifiable conditions. It is important for clinicians to identify key risk factors that may help reveal potential therapeutic strategies for the patient and understand the underlying pathophysiology. The risk factors that help clinicians determine an appropriate treatment and secondary prevention plan. Some of the non-modifiable risks factors include (a) age (b) race (c) ethnicity (d) gender (e) fibromuscular dysplasia (f) history of migraine headaches and (g) heredity: family history of stroke or transient ischemic attacks (TIAs). Some of the modifiable risk factors include: (a) hypertension (b) Diabetes mellitus (c) Cardiac disease (d) Hypercholesterolemia (e) Carotid stenosis (f) Hyperhomocystinemia (g) Lifestyle issues: Excessive alcohol intake, tobacco use, illicit drug use, physical inactivity (h) Transient ischemic attacks (TIA) (i) Obesity (j) Oral contraceptive use/postmenopausal hormone use and (k) Sickle cell disease (Iadecola, & Anrather, 2011)..

2.6.1.3. CADASIL

A mutation in the NOTCH3 gene can cause Cerebral arteriopathy, autosomal dominant, with subcortical infarcts and leukoencephalopathy (CADASIL) (Gordhan, & Hudson, 2013). This is a rare genetic disorder that is known to affect the small arteries of the brain. In patients with NOTCH3 gene mutations, the risk of stroke is high including stroke mortality. In most patients, stroke-like symptoms or episodes occur before 46 years while the disease reaches its peak by the 65 years. NOTCH3 gene mutation can affect people between 19 to 67 years. In young patients, there could be gradual changes in the white matter of the brain which progresses by adulthood or by the 3rd or 4th decade of life. In patients with CADASIL, migraines are common in about 30-40% of patients. The risk of cognitive deficits is highest in patients with CADASIL, wherein about 60% of patients have symptomatic cognitive deficits. The risk of developing multi-infarct dementia is also high in such patients while symptoms can be observed as early as 35 years (Gordhan, & Hudson, 2013).

2.6.1.4. Other mutations

In the past decade, there has been extensive genomic-based research and evaluation of patients at risk or those who have suffered stroke. Based on the evaluation, researchers have stated that specific loci that are additional are more common among patients who have had ischemic stroke. A team of experts reported that 2 single-nucleotide polymorphisms on 2q23.3, have been associated with early onset ischemic stroke. Loci-based mutations have been confirmed to be associated with the risk of developing stroke. For example, PITX2 and ZFHX3 located on 9p21 and with variations in HDAC9, PITX2, ZFHX3, and HDAC9 located on 7p21.1 have a strong association with ischemic stroke. A team of experts have also identified specific loci-based mutations, wherein large-artery stroke has been associated with variations in PITX2 and ZFHX3 that are located on 9p21 or variation in HDAC9, PITX2, and ZFHX3. HDAC9 that are located on 7p21.1. Cardiovascular disease has been attributed to mutations in the 9p21 locus, thus making it a key marker for genetic research for ischemic stroke. Mutations on the locus, 12p13 have also been identified to be associated with ischemic stroke by a team of researchers while polymorphism at 2q36.3 by another team of experts have revealed that adenosine substitution related to a lower risk of ischemic stroke. An additional study suggested an association between ischemic stroke and a locus on 12p13 (Matarin, et al, 2010).

2.7. Treatment and management of stroke

In the past decade, researchers have always focused on preserving the tissues in the ischemic penumbra as the central goal of therapy. Clinicians should consider immediate and active intervention to decrease perfusion with an aim to prevent the formation of an infarct. In most acute ischemic cases, clinicians aim to restore blood flow to the affected tissue areas by optimizing collateral flow (Jaffer, et al. 2010). Some of the common recanalization strategies that can be included in the early approach strategies include: (a) The administration of

intravenous (IV) recombinant tissue-type plasminogen activator (rt-PA) combined with intra-arterial approaches. This step helps to establish revascularization. Once revascularization takes place, most of the cells in the penumbra are rescued prior to the development of irreversible injuries. However, the restoration of blood flow can help in mitigating the risk of irreversible damage to the neuronal sites but should be done quickly by highly experienced and trained professionals. Although the mortality rate of ischemic stroke patients is high, immediate and active intervention may prevent permanent or irreversible damages to the neuronal sites. Clinicians should provide comprehensive care for patients who may have survived new onset stroke but suffer from chronic disabilities. It is empirical to provide short and long-term needs of patients with chronic disability. A combination of medications, lifestyle modifications, and the inclusion of a multidisciplinary team of experts in the comprehensive care plan for acute ischemic stroke patients is highly recommended (Jaffer, et al. 2010).

2.7.1. Pharmacotherapy

Till date, clinicians have preferred the use of alteplase (rt-PA) as the only fibrinolytic agent for the acute treatment and management of acute ischaemic stroke in patients. In most cases, streptokinase has benefited patients with acute myocardial infarction. However, in patients with acute ischemic stroke streptokinase has been shown to increase the risk of intracranial hemorrhage and death. Based on current evidence, fibrinolytics (rt-PA) have been helpful in restoring cerebral blood flow in some patients with acute ischemic stroke. In most patients, this approach has led to the improvement or resolution of neurologic deficits. However, rt-PA has key side-effects such as increased risk of extracranial haemorrhage, angioedema or allergic reactions. As per the AHA/ASA guidelines, patients with new onset ischemic stroke are recommended to take aspirin 325 mg orally, within 24-48 hours. Although the benefit of aspirin is moderate in most clinical cases of stroke it reduces the risk

of recurrent stroke. The use of anti-hypertensives is recommended for new onset acute ischemic stroke for improved outcomes in the first 24 to 48 hours. The use of neuroprotective agents for early onset acute ischemic stroke is under clinical investigation. Since there are no randomized control trials for the same, it cannot be recommended. However, new researcher studies may provide insights on the clinical benefits of new neuroprotective agents (Prasad, et. al. 2011)

2.7.2. Surgical Interventions

In the past decade, many researchers have widely accepted surgical interventions for patients with acute ischemic stroke. There is limited evidence on the use carotid endarterectomy but many clinicians have provided key insights on its success in the treatment and management of internal carotid artery occlusions. Alternative strategies have been explored to limit the severity of ischemic injury by limiting the duration of ischemia in the form of neuronal protection. However, there is no clear evidence either on such strategies or any neuroprotective agents that have helped patients recover actively (Giles, et al. 2010).

2.7.3. Occupational therapy and physiotherapy

Based on current evidence, clinicians have included occupational therapy and physiotherapy as part of the comprehensive care plan for patients with acute ischemic stroke. Occupational therapy (OT) is implemented as part of the core plan to help acute ischemic stroke patients regain from their abilities, cope with existing or new disabilities, and learn new skills. OT is a form of rehabilitation which has been recommended by the Stroke Association. OT and other forms of therapy are part of the normal process of recovery in the form of rehabilitation therapy. It focuses on relearning and adapting everyday activities based on one's post-stroke condition. It is aimed to help the patient learn new skills and adapt to new skills and practices that in turn would help the patient live a full and independent life.

Some tasks such as getting out of bed, making a cup of tea, brushing, or even walking around the house are part of the learning and development stages of OT (Veerbeek, et al. 2014).

An occupational therapist is often referred to as OT, a highly skilled and experienced professional in the field of occupational rehabilitative therapy. The role of the OT is highly complex, challenging, and diverse in the healthcare sector. However, in post-ischemic stroke care, the stroke association has confined specific roles, tasks, duties, and responsibilities of an OT for efficient care of the patient. An OT would coordinate and work with other stroke care team members who would provide valuable information on patient's overall health, post-stroke care needs, and medication regimen and mandatory rehabilitative care. The multidisciplinary team of post-stroke care may include a physiotherapist, nurse, speech and language therapist, physician, social worker, and allied doctors/healthcare professionals (Merchant, et al. 2016).

The role of the OT is dependent on the patient's needs. For example, the therapist may require to assist the patient for physical inculpabilities such as walking, sitting, or even moving arms/legs. In some cases, the OT may also help the patient who may face difficulties to support carers. The OT would also examine and assist the patient in memory/attention, vision issues, sensation, and/or even emotional issues such as depression, anxiety, and stress. The OT is often seen as a point of contact for the patient for all non-medical but essential requirements for the patient. Patients who suffer from anxiety or depression require special care and attention. In some cases, the OT may require to coordinate with the physician and nurse to include any pharmacological regimens. However, the OT may help the patient through non-pharmacological approaches to seek better and faster recovery and achieve enhanced health outcomes. Post-stroke care by the OT would include exercise regimen and other health-related activities that promote quality of life and make the patient independent

and confident. The rehabilitative therapy would often include a set of goals that would be broken down into segments which gradually increase over time. For example, the goal is to walk till the nearest grocery shop. However, it would start by assisting the patient to walk till the door, then till the path, which is then followed by the main goal, till the grocery shop.

2.7.3.1. Key responsibilities and roles of occupational therapist

The occupational therapist has a diverse and complex role in assisting and caring for the acute ischemic stroke patient. The assessment, identification, evaluation, and implementation of key tasks or interventions is a key role of the OT, specifically with an aim to improve post-stroke care, quality of life, and overall health outcomes (Stroke Association 2012).

Assessment of difficulties and family guidance

The occupational therapist or OT would primarily assess and guide the family members/caregiver on how to help/assist the patient. For example, the OT may instruct a family member to help the patient to walk towards the toilet or use of hand rails or other supportive objects in the house to prevent falls. The family member would be trained and educated to assist the patient while moving from one place to another including specific exercise and medication regimens. The OT would assure the family member or caregiver to help the patient in difficult tasks such as moving in the bed, maintaining the right posture, or even walking and moving in the right direction/form. The key objective is to help the patient overcome existing difficulties and lead a quality of life (Stroke Association 2012).

Activities that improve patient abilities and independence

The OT would primarily focus on including activities that would gradually improve the patient's abilities and increase overall independence (Merchant, et al. 2016). For example, the OT would train and encourage the patient to walk for a short distance, increase the

distance every week, and increase until the primary goal is achieved. For example, the patient is required to take her medications that are placed in the lower drawer of the kitchen cabinet. On the first attempt, the patient would be encouraged to move till her bedroom door, then near the alley, and at a later stage she would walk till the kitchen door unassisted or independently (Stroke Association 2012).

Everyday activities as part of core rehabilitative process

The patient in the post-acute care rehabilitative process would be trained to perform everyday activities with the help of a highly experienced and skilled OT. It is important for the OT to encourage and motivate the patient to perform key tasks (Merchant, et al. 2016). Some of the common activities that the OT may assist, guide, and train the patient include, stretching (exercise) while at bed prior to beginning morning routine, arm/wrist movement exercise, brushing, holding a cup/glass, making a hot cup of coffee, waking around the house using walking stick/support, using television remote, moving a light-weight chair, and/or accessing the washroom/toilet unassisted or with the help of railing/support (Stroke Association 2012).

Inclusion of strategies and techniques to overcome difficulties

It is important for the OT to include techniques and strategies that would help the patient overcome physical and other difficulties. Some common examples include helping the patient to move and exercise regularly, encouraging the patient to move around the house to facilitate physical activity, and decrease the use of aids and other equipment while moving or walking. In case the patient has difficulty walking, the OT can include specific morning and evening exercises that would increase the patient's ability to walk and move around the house.

Provision of aid and equipment

In the acute stage of post-stroke care, the OT may provide walking aids and other equipment to help the patient move around the house (Merchant, et al. 2016). For example, hand railings can be attached to the corridors and toilet to help the patient walk and move around without the risk of falls. In some cases, the OT may also provide the patient with a walking stick to facilitate movement in and around the house. A walking stick or walker can help patients with limited physical capabilities. Eyeglasses to improve vision is also highly recommended for patients with vision problems (Stroke Association 2012).

Regular patient visits at home

The occupational therapist plays a key role in regularly monitoring and evaluating the patient's overall health and well-being (Merchant, et al. 2016). The core objective is to increase physical capabilities and decrease overall dependence on aid/equipment and physical assistance. Post-stroke care and management of the patient, the OT would regularly visit the patient at home to assess and review overall progress (Merchant, et al. 2016). In some cases, the OT may recommend downgrading exercises, modifying specific exercise regimens, and encourage the patient to move independently without physical aid/equipment. The OT would also review the patient's mental and physical capabilities to ensure that there is progress in quality of life and that most of the implemented techniques and strategies are effective and feasible (Stroke Association 2012).

Evaluation of home environment

The patient's home environment plays a pivotal role in the overall development and well-being of patient (Merchant, et al. 2016). The occupational therapist would assess for specific components such as lighting, wet floors, presence of equipment or aids,

washroom/toilet lighting/structure, bed size/height, presence of stairs or blind corners, obstacles or household items that may pose a threat or increase risk of falls, cord lines/wiring, location of telephone/mobile to access in case of emergency, location of the hallway and main door, and the presence of any pet/animals. The occupational therapist would also review the external environment of the house in terms of noise, humidity, and temperature (Stroke Association 2012).

Advise to return work/lead a normal life

Post-acute care management of the patient, the OT would advise the patient to resume work and perform certain activities at the workplace. It is important that patients with acute ischemic stroke lead a normal life and continue to do the same daily tasks/activities to prevent any psychological or emotional problems such as depression or anxiety (Stroke Association 2012).

2.7.4. Physiotherapy

In most acute ischemic stroke patients, weakness and paralysis is a common healthcare issue and disability. In severe cases, patients suffer from physical disability and restricted movement which has a negative impact on the overall quality of life. Based on current evidence, physiotherapy has been associated with improved quality of life and health outcomes among patients with acute ischemic stroke (Carter, et al. 2010).

In the past decade, physiotherapy has gained significant importance due to its cost-effectiveness, efficacy, clinical benefits, and feasibility. Some of the most common techniques involved in physiotherapy include manipulation, exercise, electrical treatment, skills training, massage, and allied rehabilitation processes. The core objective of physiotherapy is to regain strength and movement of a patient as fast as possible. In the case of a patient with acute ischemic stroke, neuro-physiotherapy is an effective and safe

intervention. It focuses on treating problems/issues that originate from the brain. However, neuro-physiotherapy should be applied in conjunction with other alternative therapies for better health outcomes. A neuro-physiotherapist is highly skilled, trained, and experienced to care and manage stroke patients with complex needs, specifically to treat the various changes that occurred due to underlying neurological conditions (those affecting the nervous system) (Stroke Association 2012).

In the case of acute ischemic stroke, specifically blockage of the internal carotid artery, there is irreversible or permanent damage to the nerve cells in the brain. In such cases, there is no or limited scope for the damaged cells to grow or regenerate back. Thus, the scope of neuro-physiology is to train and reorganize the brain the undamaged part of the brain. A neuro-physiotherapist plays a crucial role in assessing the functions of the brain and the patient's ability to cope with existing physical limitations and difficulties. The process of reorganizing the undamaged part of the brain for normal functioning is referred to as neuroplasticity.

Physiotherapist work with other members of the stroke care team similar to that of the occupational therapist. The physiotherapist would play a key role in assessing and revisiting the patient's post-stroke care. The physiotherapist would coordinate with a multidisciplinary team of experts such as speech and language therapists, social workers, care giver, doctors, nurses, occupational therapists, primary physician, surgeon, and allied healthcare professionals. It is important for the physiotherapist to coordinate with each member, specifically guide and assist family members and caregiver for proper care transition at home.

In the early stages of post-stroke care, the physiotherapist would focus on preventing complications and restore the ability of the patient to move and lead a normal life. However, in severe cases where irreversible damage has already been done, the physiotherapist would

focus on helping the patient to become independent rather than focusing on full recovery (Carter, et al. 2010). For example, the physiotherapist may recommend the patient to use a walking aid or a walker-like equipment to enable movement within the healthcare setting or home. In most cases, the transition of a patient from a stroke care unit to a rehabilitation unit is common, wherein the physiotherapist would play a lead role in assisting the patient in basic physical movement and rehabilitation (Stroke Association 2012).

The location for physiotherapist is dependent on the severity and complexity of the patient, wherein a rehabilitation unit is the most preferred choice for a physiotherapist. However, in some cases, patients are referred to a specialist community rehabilitation centre. In case the patient cannot be transferred to his/her home, it is important to provide a cost-effective and safe environment for steady recovery. In such cases, a patient is transferred to an intermediate care centre (which is a transition between a home and hospital). The physiotherapist would play a key role in developing and implementing a comprehensive plan for the patient, specifically exercise and body positioning while sleeping, walking, and sitting.

2.7.4.1. Key responsibilities and roles of physiotherapist

The physiotherapist plays a critical, complex, and challenging role in the care and management of patients with acute ischemic stroke. The core objective of the physiotherapist is to motivate, encourage, and support the patient to lead a quality life and engage him/her in various exercise regimen and plans. Based on current evidence, the physiotherapist would have the following key roles and responsibilities:

Assist stroke nurses

The physiotherapist would play a key role in coordinating with the stroke care nurse, specifically in developing and implementing a comprehensive care plan. The physiotherapist

would also communicate with the stroke nurse to identify and assess any complications, prevent the same, and enable therapies that would fasten the recovery process.

Advise on patient position

Physiotherapist recommend and advise the patient on sitting, sleeping, and walking position. It is important for the patient to sleep in an appropriate position to avoid discomfort and anxiety. Encouraging the patient to walk and perform exercises would help in preventing complications and improve quality of life. Sitting posture of the patient should be appropriate.

Decision on care and management of the patient

The physiotherapist would focus on the providing daily exercise instructions to the patient, starting from easy to moderate exercise programs. For example, the patient would be instructed to make circular leg movements while sitting followed by mild walking at home and brisk walking at a later stage. The physiotherapist would recommend specific exercises to prevent muscle stiffness, spasm, tightness, or even joint stiffness. The physiotherapist would also advise the patient to perform breathing exercises and chest physiotherapy to prevent lung infections, improve breathing, and prevent blood clot formation (Carter, et al. 2010).

Motivate and encourage the patient

The physiotherapist would play a diversified role in encouraging and motivating the patient. It is important to assist and motivate the patient to exercise regularly, take medications on time, and be mobile as frequent as possible. The more the patient moves about, lower the risk of complications. Physiotherapy helps in preventing relapses of stroke (Carter, et al. 2010).

Offer therapy to the patient

The physiotherapist would assist the patient in performing key tasks and a minimum of 45-minute physiotherapy session every day. In patients with acute ischemic stroke, muscle weakness and fatigue are a common problem. The physiotherapist would play a key role in implementing strength exercises combined with treadmill training and movement therapy. It is important for each patient to receive individualized care, specifically the type of exercise and management process (Carter, et al. 2010).

Collaborative care and approach with rehabilitation team

It is important for the physiotherapist to coordinate and communicate with allied healthcare members such as the language/speech therapist, occupational therapist, dietician (if any), physician, and nurse to understand and evaluate the progress of the patient. Collaborative care and approach has been associated with improved health outcomes (Carter, et al. 2010).

Advise to family and caregivers

Proper training and education of family and caregivers regarding post-stroke care and rehabilitation is critical and important. Physiotherapist would help play a critical role guiding family members on various exercise regimens and approaches for enhanced outcomes. Both alternative therapies are aimed to reduce stress and anxiety among patients, improve the overall quality of life, and enhance overall health outcomes (Prasad, et. al. 2011).

2.7.5. Prognosis of stroke patients

Based on recent clinical studies, researchers have stated that the mortality of patients with stroke is about 77% for 1 year while the mortality rate after 30 days and within 30 days was 20% and 19% respectively. However, in most patients, the severity of stroke, the patient's comorbid condition, and the overall post-stroke complications determine the overall

prognosis of the patient. Based on current evidence, the overall prognosis is highly variable in patients and is dependent on individual factors. In a recent study a team of researchers have also reported that the baseline National Institutes of Health Stroke Scale (NIHSS) score can be considered as a key predictor for early mortality risk. It is better than the currently used mortality prediction models or score cards. In context to the NIHSS score, patients with cardiogenic emboli have the highest 1-month mortality rate with respect to acute stroke. A team of researchers have also indicated that the use of computed tomography (CT) scan evidence for infarction in the initial or early presentation has been associated with poor outcomes while it also increases the for haemorrhagic transformation post-fibrinolytic therapy. Based on a review of recent literature, a team of experts have reported that acute cardiac dysfunction and arrhythmia are closely associated with acute ischemic stroke. Furthermore, these clinical syndromes often lead to worsening functional outcomes and increased morbidity at 3 months. In patients with hyperglycaemia, there is an increased risk of extension of the infarcted territory followed by reduced reperfusion in fibrinolysis and poor outcomes. Lastly, an estimated 31% of patients have reported to need help while caring for themselves, over 20% require help while walking, and nearly 71% if patients had impaired vocational capacity. Most patients require assistance with respect to self-help or chronic disability in long-term follow-up (Jichici, 2015).

3. Special Part (Case Study)

3.1. Work methodology

I have done my bachelor's practice in neurological department at regional hospital Kladno, from 30. 1. 2017 to 10. 2. 2017 my work was guided and supervised by Bc. Tomáš Modlinger.

My thesis patient is a man who had an ischemic stroke with left side hemiparesis. The patient has been informed about my thesis practice so we could cooperate and that his personal information, anamnesis and his present situation will be used

7 therapeutic sessions I spent with the patient every morning and afternoon in the training room. Initial and final examinations were included in beginning of first session and at the end of last sessions respectively.

I used a measuring tape, plastic goniometry and neurological hammer during both initial and final examinations. Rope ball, blue half ball, balance board, spongy balls, toys and coins were used during the therapy sessions.

However, according to the instructions from the supervisor, therapy procedure should mainly focus on upper extremities, so we were concentrating on it by using PIR according to Karel Lewit and PNF Kabat were also included in the therapy sessions.

3.2. Anamnesis

Examined person: N. V

gender: Male

Year of birth: 1955

Diagnosis: Ischemic stroke in ICA leads to hemi-paresis on the left side of the body on

24-1-2017

STATUS PRAESENS:

Subjective:

The patient does not feel any pain but he is complaining about the weakness and the decrease of his fine motor function on the left upper extremities

Objectively:

The patient was one week after the day he had the stroke. He was normal in the communication, he did not have any speech disorders, Cognition and orientation of time and place was normal and he does not need any support while walking

Assistive devices: Does not to use any

Dominant limb: Right part

Glasses: He does not need

Height: 177cm

weight: 85 kg

BMI : 27.1

Chief complaint:

Ischemic stroke affected ICA on the right part of the brain leads to hemi- paresis of the left side of his body 1 week ago

History of the problem:

On 24-1-2017 the patient woke up at 5 in the morning and he could not move his upper and lower extremities on the left side, he was transferred to the hospital by his brother then the medical staff took immediate action and gave him the proper medication.

He also said that he felt numbness on his left hand 3 months before that day so he went to the hospital and had an appointment with a neurologist but one day before the appointment he was diagnosed with the ischemic stroke

AA: (allergic anamnesis): non

Diet: no diet

Functional anamnesis/history:

The patient struggles the most when he wears or takes off his clothes, the decreased of his left hand's function limits some activities such as eating, driving, fixing things etc.

FA (family anamnesis):

The patient's brother was affected by ischemic stroke and he dead at the age of 62 and the rest of his family were healthy.

OA (occupation anamnesis, vocation):

He used to be a carpenter before he got affected by stroke and he does not work so far.

Social anamnesis:

He is divorced man and he has a daughter, he is living with his brother.

Sport, regular physical activity:

In general, the patient is active but he does not attempt to sport.

Pharmacological anamnesis:

The patient starts to take these medication tablets after he was diagnosed with the ischemic (as prescriptions from the doctor) Trombex tbl 75 mg, Anopyrin tbl 100 mg, Apo-panto tbl 40 mg, Rosucard tbl 40 mg

Abuses: The patient is heavy smoker (40 cigarette a day).

3.3. Initial Kinesiological Examinations By Physiotherapist:**3.3.1. Postural Examination-Aspection-Plumb Line Test:**
o back view:

- The base of support: Slight narrow base of support
- Shape and position of the ankle joints: On the right side, it is laterally rotated and on the left side there no rotation
- Shape and thickness of the Achilles tendon: Symmetrical.
- Contour of the calf muscles (medial and lateral part): The prominence of soleus and medial part of the calf (Gastrocnemius medial head) is in the right calf more than the left.
- Popliteal line - symmetry, the slope: Both are in the same height
- Contour of the thigh muscles - especially the adductors: both are not prominence in both sides in general the shape is symmetrical in both
- Subgluteal line - height, length, symmetry: Higher in the right side
- Gluteal muscles - symmetry, shape, tonus: Symmetrical and both has the same shape.
- Symmetry of thoracobrachial triangles - the shape and size of triangles: Wider on the right side
- Position of pelvis: Pelvis is shifted to left side
- Curvature of the spine in the frontal plane (scoliosis ...): L, THL spine is slightly shifted to the left side.

- Position of the scapula - (angulus infrascapularis, margo medialis): On the left is slightly higher than the right side
- Position of the head: In neutral position

o front view:

- The base of support: slight narrow
- The position of the feet, examination of the arches (vault): lateral rotation in the right foot
- The position and shape of the toes: the patient has relaxed toes
- Weight distribution (medial X lateral edge): The patient is depending on his right side more
- Shape and position of the knee joints (varus X valgus) and patella: Slight lateral rotation in the right knee
- Shape of the thigh muscles (the quadriceps femoris): Not clear
- Position of the pelvis: The pelvis is shifted to the left side
- Symmetry of the muscle tone of abdominal muscles: Not clear
- Position of the navel (belly muscle symmetry): Physiological position
- Symmetry of thoracobrachial triangles - the shape and size of triangles: Wider on the right side
- Position and symmetry of the chest (position of nipples): Slight higher on the right side
- Position of the shoulder girdle (contour, tonus, protraction, ...): The patient has protracted shoulder
- Position of the head (symmetry, position of ears, face symmetry, ...): Head is neutral position

o side view (right):

- Shape and position of the ankle joints (plantarflexion X dorsiflexion) : in a balanced position
- Shape and contour of the shin (symmetry, ...): physiological position
- Position of the knee joints (hyperextension X semiflexion): neutral position, no hyperextended, no semi flexion
- Position of the pelvis - anteversion (increased anteversion X retroversion): slight posterior tilt of the pelvis
- Position and curvature of the L, L/Th, Th and C spine: decreased lordosis in L spine
- Shape of the abdominal muscles (prominence): not clear
- Position of the shoulder girdle (protraction ...): protracted shoulder
- Flattening; and tops of the curves): non
- Position of the head (forward holding ...): slight protraction of the head

o side view (left):

- Shape and position of the ankle joints (plantarflexion X dorsiflexion): In a balanced position
- Shape and contour of the shin (symmetry, ...): Physiological position
- Position of the knee joints (hyperextension X semiflexion): Neutral position, no hyperextended, no semi flexion
- Position of the pelvis - anteversion (increased anteversion X retroversion): slight posterior tilt of the pelvis
- Position and curvature of the L, L/Th, Th and C spine: decreased lordosis in L spine
- Shape of the abdominal muscles (prominence): not clear
- Position of the shoulder girdle (protraction ...): protracted shoulder
- Flattening; and tops of the curves): non
- Position of the head (forward holding ...): slight protraction of the head

3.3.2. Palpation: pelvis (conclusion):

- Iliac crest: Left side is lower than right side.
- PSIS: Left side is lower than right side.
- ASIS: Left side is lower than right side.

3.3.3. Anthropometric Measurement (length, circumferences)

UPPER EXTREMITIES	RIGHT (cm)	LEFT (cm)
Length of upper limbs	70	69.5
Length of humerus	27.5	28
Length of forearm	26	26
Length of hands	17	17
Circumference of upper limb	31	30.5
Circumference of forearm	28	27.5

Table 1: Initial Anthropometric measurement (upper extremities)

LOWER EXTREMITIES	RIGHT (cm)	LEFT(cm)
Anatomical length		
Functional length	83	82.5
Length of thigh	41	41
Length of middle leg	42	41.5
Circumference of thigh	10cm above knees 45	46
Circumference of knee	39	39.5
Circumference of calf	38	38
Circumference of ankle	26	25
Circumference of foot	23.5	23.5

Table 2: Initial Anthropometric measurement (lower extremities)

3.3.4. Gait Analysis:

Patient has in general stability. He uses one crutch by his left hand which has four points in contact with the ground. And he needs therapist support too.

- Width of the base of support: Slight narrow.
- Walking rhythm: periodic walking.
- Walking speed: Slow.
- Stride length: Asymmetric. Left step length is slightly shorter than the right step.

- Movement of the foot: Ideal in right foot and decreased dorsal flexion on the left foot.
- Movement of knees joints: decreased flexion in both legs.
- Movement of hip joints: decreased extension in both legs, the extension is not pure (shifting)
- Movement of the trunk: the trunk is shifted left and right with every step with no rotation.
- Activity of back muscles: more activated in low thoracic and lumbar area in both sides.
- Activity of abdomen muscles: Low activity in both sides and more in left side.
- Position and movement of head: Tilted to the right side.
- Stability of walking: in general, the patient was stable in walking.
- Walk with eyes closed: Patient could perform it but not in straight line (the body was shifting to the right side).

3.3.5. Specific Testing

- **Romberg Test (I-III):** the patient was able to do the test I- II correctly without losing his balance but he lost his balance while closing his eyes Romberg III.

Romberg I – normal standing

Romberg II – feet together

Romberg III – closed eyes

- **Single-leg Stance Test:** The patient was losing his balance within few seconds (3-5) and he was able to stand more on his affected leg (left).

Modification of Standing:

- Standing on tiptoes: the patient could perform it but felt tired due to weakness of the muscles (the right side was slightly higher)
- Standing on heels: the patient could do it properly
- The patient was able to perform both of without correction.

3.3.6. Goniometry: Classic (two-arms)

ACTIVE	PASSIVE
Shoulder: S: R: 30-0-150 L: 20-0-140 F: R: 140-0-0 L: 110-0-0 T: R: 120-0-30 L: 110-0-10 R: R: 75-0-65 L: 55-0-65	Shoulder: S: R: 45-0- 160 L: 35-0-150 F: R: 160-0-0 L: 140-0-0 T: R: 90-0-30 L: 120-0-15 R: R: 80-0-70 L: 65-0-65
Elbow: S: R: 0-0-150 L: 0-15-130	S: R: 0-0-150 L: 0-0-145
Forearm: R: R: 80-0-80 L: 30-0-50	R: R: 80-0-80 L: 35-0-55
Wrist: S: R: 65-0-70 L: 30-0-40 F: R: 20-0-30 L: 5-0-25	S: R: 70-0-80 L: 30-0-45 F: R: 25-0-35 L: 10-0-30
Distal Interphalangeal joint 1 st finger: S: R: 0-0-80 L: 0-0-60 Distal Interphalangeal joint 2 nd finger: S: R: 0-0-80 L: 0-0-70 Distal Interphalangeal joint 3 rd finger: S: R: 0-0-80 L: 0-0-70 Distal Interphalangeal joint 4 th finger: S: R: 0-0-80 L: 0-0-70	Distal Interphalangeal joint 1 st finger: S: R: 0-0-85 L: 0-0-80 Distal Interphalangeal joint 2 nd finger: S: R: 0-0-85 L: 0-0-80 Distal Interphalangeal joint 3 rd finger: S: R: 0-0-85 L: 0-0-80 Distal Interphalangeal joint 4 th finger: S: R: 0-0-85 L: 0-0-80
Proximal Interphalangeal joint 1 st finger: S: R: 10-0-100 L: 0-0-80	Proximal Interphalangeal joint 1 st finger: S: R: 10-0-100 L: 0-0-80

Proximal Interphalangeal joint 2 nd finger:	Proximal Interphalangeal joint 2 nd finger:
S: R: 0-0-100 L: 0-0-90	S: R: 0-0-100 L: 0-0-90
Proximal Interphalangeal joint 3 rd finger:	Proximal Interphalangeal joint 3 rd finger:
S: R: 0-0-100 L: 0-0-90	S: R: 0-0-100 L: 0-0-90
Proximal Interphalangeal joint 4 th finger:	Proximal Interphalangeal joint 4 th finger:
S: R: 0-0-100 L: 0-0-90	S: R: 0-0-100 L: 0-0-90
Metacarpophalangeal joint 2 nd finger:	Metacarpophalangeal joint 2 nd finger:
S: R: 0-0-90 L: 0-5-90	S: R: 0-0-90 L: 0-5-90
Metacarpophalangeal joint 3 rd finger:	Metacarpophalangeal joint 3 rd finger:
S: R: 0-0-90 L: 0-10-90	S: R: 0-0-90 L: 0-10-90
Metacarpophalangeal joint 4 th finger:	Metacarpophalangeal joint 4 th finger:
S: R: 0-0-90 L: 0-10-90	S: R: 0-0-90 L: 0-10-90
Metacarpophalangeal joint 5 th finger:	Metacarpophalangeal joint 5 th finger:
S: R: 0-0-90 L: 0-10-90	S: R: 0-0-90 L: 0-10-90
Hip:	Hip:
S: R: 10-0-130 L: 5-0-110	S: R: 15-0-130 L: 10-0-120
F: R: 30-0-10 L: 25-0-10	F: R: 30-0-15 L: 30-0-10
R: R: 35-0-35 L: 20-0-25	R: R: 45-0-45 L: 25-0-30
Knee:	
S: R: 0-0-140 L: 0-0-130	S: R: 0-0-150 L: 0-0-135
Ankle:	
S: R: 20-0-45 L: 15-0-35	S: R: 20-0-45 L: 15-0-30
R: R: 20-0-40 L: 15-0-35	R: R: 20-0-40 L: 10-0-30

3.3.7. Muscle Length Test (according to Janda or Kendall):

- **length of ankle flexor muscles (gastrocnemius- soleus):** (Kendall 2005)
gastrocnemius:
R: normal length
L: slight shortness we could reach 10 degrees with max pressure
Soleus:
R: normal length
L: slight shortness we could reach 20 degrees with max pressure
- **length of hip flexor muscles:** (Kendall 2005)
R: shortness in both one and two joints hip flexor muscles
L: shortness in both one and two joints hip flexor muscles
- **length of hip adductor muscles:** (Janda 2013)
R: grade 0
L: grade 1 ABD 30 deg
- **length of hamstring muscles:** (Kendall 2005)
R: slight shortness
L: marked shortness
- **length of piriformis muscles:** (Janda 2013)
R: grade1 limited ADD
L: grade1 limited ADD
- **length of pectoralis major** (upper and lower part) (Kendall 2005)
R: normal length
L: normal length
- **length of pectoralis minor:** (Kendall 2005)
R: moderate shortness
L: moderate shortness
- **length test for teres major, latissimus dorsi, rhomboid major and minor muscles:**
(Kendall 2005)
R: slight shortness
L: slight shortness
- **length of medial and lateral shoulder rotators:** (Kendall 2005)
R: moderate shortness in both

L: moderate shortness in both

- **length of carnial part of trapezius:** (Janda 2013)

R: grade 2 hard barriers on the shoulder

L: grade 1 slight resistance on the shoulder

3.3.8. Examination Of Muscles Tonicity (palpation):

Name	Result
TRAPIZEUS	R: Slight hyper tonic, slight pain L: Normal tonic, no pain
DELTOID	R: slight hyper tonic, no pain L: Normal tonic, no pain
SUPRASPINATUS	R: slight hyper tonic, no pain L: Normal tonic, no pain
INFRASPINATUS	R: slight hyper tonic, no pain L: Normal tonic, no pain
BICEPS	R: slight hyper tonic, no pain L: Normal tonic, no pain
TRICEPS	L: slight hyper tonic, no pain R: Normal tonic, no pain
E CARPI RAD	L: slight hyper tonic, no pain R: Normal tonic, no pain
E CARPI ULNA	L: slight hyper tonic, no pain R: Normal tonic, no pain
F CARPI RAD	L: slight hyper tonic, no pain R: Normal tonic, no pain
F CARPI ULNA	R: slight hyper tonic, no pain L: Normal tonic, no pain
PECTORALIS MAJ	R: Normal tonic, no pain L: Normal tonic, no pain
PECTORALIS MIN	R: Normal tonic, no pain L: Normal tonic, no pain
RECTUS ABD	Normal tonic, no pain

QUADRATUS LUMB	Hypertonic, slightly painful
GLUTEAUS MAX	R: normal tone, Slightly painful L: hypotonic, no pain
PIRIFORMIS	R: Hyper tonic, painful L: Hyper tonic, painful
ADDUCTORS	R: Hyper tonic, Slightly painful L: Normal tonic, no pain
TIBIALIS ANTERIOR	R: slight hyper tonic, no pain L: Normal tonic, no pain

Table 4: Initial Examination of muscles tonicity

3.3.9. Joint Play Examination:

We examined the mobility of the upper and lower extremities joints in all 3 positions mainly in supine and prone positions to find the restricted joint.

Examination Of Upper Extremities Joints:

Joint name	Left	Right
Shoulder joint ventrodorsal-and caudal direction.	No Restriction	No Restriction
Shoulder blade against thoracic wall	Restricted in caudal direction	No Restriction
Acromioclavicular Joint ventrodorsal-craniocaudal direction	No Restriction	No Restriction
Sternoclavicular Joint ventrodorsal-craniocaudal direction	No Restriction	No Restriction
Elbow Joint medial-lateral direction	No Restriction	No Restriction
Radiocarpal Joint	No Restriction	No Restriction
Carpometacarpal Joint	No Restriction	No Restriction
Carpal Bones (distal/proximal rows of carpal bones)	Restricted in the direction of palmar/dorsi flexion	No Restriction

Carpometacarpal Joint of the thumb	No Restriction	No Restriction
Metacarpophalangeal Joints	No Restriction	No Restriction

Table 5: Initial examination of joint play upper extremities.

Examination Of Lower Extremities Joints:

Lower Extremity		
Sacroiliac joint	No Restriction	No Restriction
Patella (all directions)	No Restriction	No Restriction
Knee joint (medial \ lateral direction)	No Restriction	No Restriction
Tibiofibular joint (dorsal \ventral direction)	Restriction in ventral direction	No Restriction
Talocrural joint (dorsal \ventral direction)	No Restriction	No Restriction
Subtalar joint (dorsal \ventral direction)	No Restriction	No Restriction
Lisfranc's joint (dorsal \ ventral direction)	Restriction in dorsal \ventral directions	No Restriction
Chopart's joint (dorsal \ ventral direction)	No Restriction	No Restriction
Metatarsophalangeal Joints	No Restriction	No Restriction
Interphalangeal Joints	No Restriction	No Restriction

Table 6: Initial examination of joint play lower extremities.

3.3.10. Neurological examination:

Deep tendon reflexes:

- **Knee jerk reflex:**
Physiological, in both sides.
- **Achilles tendon reflex:**
Right foot physiological
Left foot physiological
- **Biceps tendon reflex:**
In both biceps tendons, physiological.
- **Triceps tendon reflex:**
Right triceps tendon, physiological.
Left triceps tendon, physiological.
- **Wrist reflex:**
Physiological in both sides

Specific neurological test:

o Paretic test:

- **Mingazzini test upper extremities:** patient could hold the upper extremities in a position but the affected leg was dropping down slowly (1cm every 5 second)
- **Mingazzini test lower extremities:** patient was able to hold his legs in position but the left leg was dropping down slowly (1cm every 3 second)

o Pyramidal test: ☐

- **Trömner signs:** Negative in both hands. ☐
- **Hoffman test:** Negative in both hands. ☐
- **Oppenheim test:** Negative in both legs. ☐
- **Chaddock's Sign:** Negative in both legs. ☐
- **Babinski test:** Negative in both legs.

3.3.11. Examination Of Cranial Nerves:

Nerve No.	Nerve Name	Result
I	Ophthalmic	Physiological
II	Optic	Physiological
III	Oculomotors	Physiological
IV	Trochlearis	Physiological
V	Trigeminus	Physiological
VI	Abdducens	Physiological
VII	Facialis	Physiological
VIII	Vestibulocochlearis	Physiological
IX	Glossopharyngeal	Physiological
X	Vagus	Physiological
XI	Accesorius	Physiological
XII	Hypoglossal	Physiological

Table 7: Initial Examination of cranial nerves

-Note: there was no impairment of the cranial nerves as the result of the cranial nerves examination showed.

Sensation Examinations (Superficial sensation):

Dermatomes	Result
C5	Physiological
C6	Decreased
C7	Decreased
C8	Decreased
T1	Physiological
T2	Physiological
T3	Physiological

Table 8: initial superficial sensation examination

Note: we examined the deep sensation of the lower and upper extremities of the patient in both sides and we had Physiological results in all of them

3.3.12. ADL Evaluation

The patient could transfer himself alone without any external support. He takes more than the usual time to wear his T-shirt and his slippers due to the difficulties to abduct and flex the whole arm also the disability of fine motor function. During eating he is using his right hand but he is using his effected hand to hold for example the meat, not using the knife with it. In general, he focusses on his right side while doing any activity during the day.

Barthel Test For ADL Evaluation:

Activity	Result
Stool incontinence	10\10
Urinary incontinence	10\10
Grooming	10\10
Toilet use	5\5

Feeding	5\10
Transfers	15\15
Walking	15\15
Dressing	5\10
Climbing stairs	10\10
Bathing	5\5
Sum of results: 90 /100	

Table 9: initial Barthel test.

ADL4 0-40 points: total dependence
ADL3 45-60 points: marked dependence
ADL2 65-95 points: slight dependence
ADL1 95-100 points: independent

3.3.13. Initial Examinations Conclusion:

A 62 years old male patient is suffering from ischemic stroke in ACL with left side hemiparesis. After the examinations that we made, we found the following findings:

The patient did not have any pain and he was complaining about the disability of moving the left arm and leg. Weakness and paresis of the muscles on the left side and over activity of the extremities on the right side which slightly influences his body posture on both sides. While palpating there was hyper tonicity of forearm extensors, flexors, pectoralis minor, major, piriformis and lower extremities extensors on affected side. The patient could stand, walk in line and on stairs without any support but still needs correction and instruction as the whole body was shifted to the right side which influences the pelvis position to be laterally shifted to the left side. in general, he is in good stability. in gait analyses, we found that the decreasing of dorsal flexion in his left foot and in the knee flexion movement of the left leg as well.

ROM showed slight limitation in the ROM into extension, flexion, ABD and both sides rotation was in left side shoulder, elbow, wrist and also in distal and proximal interphalangeal joints in the direction of flexion and extension. limited flexion and extension in hip joint and knee joint has slight limitation in the flexion direction. Also, in the ankle joint we found the limitation in the direction of dorsi flexion.

While examining the joints mobility on both sides we found out that there was restrictions in the scapula in the caudal direction (depression), distal and proximal rows of the carpal bones in the direction of palmar\dorsi flexion in the upper part of his body. Also Tibiofibular restriction in ventral direction and restriction of Lisfranc's joint in ventral \ dorsal direction on the lower part of his body.

From the neurological point of view he was in a very good condition. The cranial nerves are physiological. Mingazzini test for upper and lower extremities showed that the patient Is losing the position within few seconds (3-5) on the affected side. Pyramidal signs examinations were negative. The patient has physiological reflexes in both sides of his body during the deep tendon reflexes test. The patient was able to perform the Romberg I test without any external support from the therapist. Finally, the patient is considered as a slight dependent patient according to the Barthel scale classification.

3.3.14. Goals Of Therapy

- Increase ROM in shoulder, wrist, interphalangeal, knee joints in flexion and extension directions and ADD, ABD, IR, ER in the hip joint.
- Increase muscles strength of the wrist extensors, hip, knee extensors\ flexors, ankle dorsi flexor and deep stabilizer muscles.
- Gait re-education.
- Improve the stability of the patient.
- improve fine motor function on the affected side.
- Improve body coordination.
- Improve the posture.
- Increase self-sufficiency.

- Improve overall condition.

3.4. Short-Term Plan

- Passive Stretching for shortened muscle (pectoralis major and minor, trapezius, biceps brachii, hip ADD, hamstrings, Quadratus lumborum, gastrocnemius and soleus muscles)
- Mobilization of the wrist joint (proximal, distal row of carpal, scapula, Tibiofibular, Lisfranc's joints)
- PNF strengthening techniques to strength weak muscles (triceps brachii, deltoid, infraspinatus, supraspinatus, latissimus dorsi, forearm extensors, hip flexors).
- SMT for more stability of the body.
- Fine motor training.

3.4.1. Long-Term Plan:

- Stretching exercises for Trapezius, Biceps brachii, Flexors digitorum, Flexors carpi ulnaris, Flexors carpi radialis and Triceps surae muscles on the affected side with slight isometric contraction from the patient (similar to PIR)..
- ROM exercise for shoulder joint by flexing, extending, external and internal rotating the joint passively for 12 times each direction. exercises for knee joint in flexion and extension for 12 times. and for the hip joint movement active abduction, adduction, internal, external rotation exercises 12 times. Then actively the patient will repeat all the previous exercises.
- Muscle strengthening exercises for Triceps brachii, forearm extensors. Hamstrings muscles, Tibialis anterior muscle and Deep stabilization muscles by exercises according to DNS.
- Gait training and reeducation.
- Sensomotoric exercises to improve stability in different surfaces
- Restore and improve coordination and strength of left lower and upper extremities (gripping, writing and eating)

3.5. Day To Day Therapy:

We worked with the patient 7 days, 2 hours every day divided to 2 sessions one in the morning and one in the afternoon.

- **2\2\2017**

Present State:

- **Objective:** the patient was in a good condition, no cognitive problem and no assistive devices were used.
- **Subjective:** the patient was complaining about the weakness of his left arm especially the wrist and the hand and he said that he is feeling numbness most of the time.

Goals Of Today Unit:

- To increase the ROM of the upper extremities on the affected side.
- To Strengthen the weak muscles on the affected side (serratus anterior, triceps brachii, forearm extensors, hip and knee flexors)
- Relax the hypertonic muscles especially of the forearm (flexors).

Implemented Therapy:

- Mobilization of the wrist joint (proximal, distal row of carpal) In the direction of palmar\dorsi flexion, the patient is in sitting position with his hand supported by the edge of the table. Then we mobilized the scapula while the patient in prone position and his shoulder after the edge of the table. In supine position we mobilized the Tibiofibular and the Lisfranc's joints (Karel Lewit 2010)
- Repetitive stretching of the shoulder in all directions (flexion, ABD, external rotation, horizontal ABD) 12 times from the neutral position to the direction of stretching to stretch the muscles (pectoralis major and minor, deltoid, scapular muscles) and to increase the ROM in the shoulder joint. Then we worked with elbow and forearm flexors at once by stretching the biceps brachii while dorsiflexing the wrist joint with holding and repeating the procedure 10-12 times,

so we affect the biceps brachii, Flexor digitorum, Flexor pollicis longus, Flexor carpi radialis and Flexor carpi ulnaris muscles at the same time, the same we did to the hip, knee and ankle joints muscles to stretch (hip ADD, hamstrings and calf muscles)

- PNF strengthening Repeated Contraction for the upper extremities to strength Interossi dorsalis, Extensor polices longus, Extensor digitorum, triceps brachii and latissimus dorsi muscles of the left side of the body. And the same technique for the lower extremities to affect Extensor digitorum, Extensor hallucis longus-brevis, Tibialis anterior, Interossi dorsales, Semimembranosus, Semitendinosus, Iliopsoas, Obturatorius externus, Pectineus, Gracilis, Adductor longus/brevis, Sartorius, Rectus femoris.
- Isometric strengthening exercise of left side Serratus anterior muscle by pushing the therapist hand while the patient in supine position with extended arm and to resist the PT force in different directions (extension, flexion, horizontal ADD and ABD) and to improve the stability of the shoulder.
- Gluteus maximus strengthening exercise (bridge) the patient lie on supine with the knees bent about 90 deg and then raise the hip toward the ceiling so the form a straight line.
- Strengthen the quadriceps muscles in supine we put a pillow below the patient's affected knee (flexed) and we ask him extend the leg actively against resistance of the PT's arm 10-12 times.
- Strengthen the hip flexor muscles (iliopsoas, rectus femoris, Sartorius) so the patient is setting on the edge of the table and we ask him to flex his thigh against the resistance 10-12 times

- 3\2\2017

Present State:

- **Objective:** the patient was in a good condition, no cognitive problem and no assistive devices were used.
- **Subjective:** the patient wasn't complaining about pain but the weakness of his left arm especially the wrist and the hand.

Goals Of Today Unit:

- To increase the ROM of the upper extremities on the affected side.
- To Strengthen the weak muscles on the affected side (serratus anterior, triceps brachii, forearm extensors, hip and knee flexors).
- Functional exercise for the hand.

Implemented Therapy:

- Mobilization of the wrist joint (proximal, distal row of carpal) In the direction of palmar\dorsi flexion, the patient is in sitting position with his hand supported by the edge of the table. Then we mobilized the scapula while the patient in prone position and his shoulder after the edge of the table. In supine position we mobilized the Tibiofibular and the Lisfranc's joints (Karel Lewit 2010)
- Repetitive stretching of the shoulder in directions of (flexion, ABD, external rotation, horizontal ABD) 12 times from the neutral position to the direction of stretching to stretch the muscles (pectoralis major and minor, deltoid, scapular muscles) and to increase the ROM in the shoulder joint. Then we worked with elbow and forearm flexors at once by stretching the biceps brachii while dorsiflexing the wrist joint with holding and repeating the procedure 10-12 times, so we affect the biceps brachii, Flexor digitorum, Flexor pollicis longus, Flexor carpi radialis and Flexor carpi ulnaris muscles at the same time, the same we did to the hip, knee and ankle joints muscles to stretch (hip ADD, hamstrings and calf muscles)
- PNF strengthening Repeated Contraction for the upper extremities to strengthen Interossei dorsalis, Extensor pollicis longus, Extensor digitorum, triceps brachii and latissimus dorsi muscles of the left side of the body. And the same technique for the lower extremities to affect Extensor digitorum, Extensor hallucis longus-brevis, Tibialis anterior, Interossei dorsales, Semimembranosus, Semitendinosus, Iliopsoas, Obturatorius externus, Pectineus, Gracilis, Adductor longus/brevis, Sartorius, Rectus femoris.
- Isometric strengthening exercise of left side Serratus anterior muscle by pushing the therapist hand while the patient in supine position with extended

arm and to resist the PT force in different directions (extension, flexion, horizontal ADD and ABD) and to improve the stability of the shoulder.

- Gluteus maximus strengthening exercise (bridge) the patient lie on supine with the knees bent about 90 deg and then raise the hip toward the ceiling so the form a straight line.
- Strengthen the quadriceps muscles in supine we put a pillow below the patient's affected knee (flexed) and we ask him extend the leg actively against resistance of the PT's arm 10-12 times.
- Strengthen the hip flexor muscles (iliopsoas, rectus femoris, Sartorius) so the patient is setting on the edge of the table and we ask him to flex his thigh against the resistance 10-12 times
- We trained with rope ball so the patient is asked to hold the ball by the affected hand while the rope is connected to it and throw to it to the PT, then to throw it and to hold it by the other hand lastly to throw it and to hold it by the same hand

- 6/2/2017

Present State:

- **Objective:** the patient was in a good condition, no cognitive problem and no assistive devices were used.
- **Subjective:** the patient wasn't complaining about any pain and the weakness of his left arm especially the wrist and the hand was obvious.

Goals Of Today Unit:

- To gain full ROM in the joints of the affected side of the body
- To train and improve stability.
- Functional exercise for the hand (fine motor skill).

Implemented Therapy:

- Mobilization of the wrist joint (proximal, distal row of carpal) In the direction of palmar\dorsi flexion, the patient is in setting position with his hand

supported by the edge of the table. Then we mobilized the scapula while the patient in prone position and his shoulder after the edge of the table. In supine position we mobilized the Tibiofibular and the Lisfranc's joints (Karel Lewit 2010)

- PNF Contraction-relaxation technique in 1st diagonal extension to relax Interossei palmares muscles, Flexor digitorum muscles, Flexor pollicis longus muscle, Flexor carpi radialis muscle, Flexor carpi ulnaris muscle, Biceps brachii muscles, and Pectoralis major muscle.
- Repetitive stretching of the shoulder in all directions (flexion, extension, ABD, ADD, internal and external rotation, horizontal ABD and ADD) 12 times from the neutral position to the direction of stretching to stretch the muscles (pectoralis major and minor, deltoid, scapular muscles) and to increase the ROM in the shoulder joint. Then we worked with elbow and forearm flexors at once by stretching the biceps brachii while dorsiflexing the wrist joint with holding and repeating the procedure 10-12 times, so we affect the biceps brachii, Flexor digitorum, Flexor pollicis longus, Flexor carpi radialis and Flexor carpi ulnaris muscles at the same time, the same we did to the hip, knee and ankle joints muscles to stretch (hip ADD, hamstrings and calf muscles).
- Sensomotoric training to improve the stability of the patient, we trained on different surfaces such as:
 - Balance board and we asked the patient to shift his body to the sides, forward and backward while maintaining his balance and while semi flex the knees then to do mini squat without losing the balance
 - blue half ball we asked the patient to step over it with one leg and hold the position for 4 seconds then step back and do it with other leg, also to do mini squat without losing the balance.
- We trained with rope ball so the patient is asked to hold the ball by the affected hand while the rope is connected to it and throw to it to the PT, then to throw it and to hold it by the other hand lastly to throw it and to hold it by the same hand

- for fine motor skills, we had small spongy letters and specific board is made for them so we asked the patient to pick a letter to put it inside the board to make a word
- 7/2/2017

Present State:

- **Objective:** the patient was in a good condition, no cognitive problem and no assistive devices were used.
- **Subjective:** the patient wasn't complaining about any pain.

Goals Of Today Unit:

- To Strengthen and activate the weak muscles on the affected side (serratus anterior, triceps brachii, forearm extensors, hip and knee flexors)
- To train and improve stability.
- Functional exercise for the hand (fine motor skill).

Implemented Therapy:

- PNF strengthening Repeated Contraction for the upper extremities to strengthen Interossei dorsalis, Extensor polices longus, Extensor digitorum, triceps brachii and latissimus dorsi muscles of the left side of the body. And the same technique for the lower extremities to affect Extensor digitorum, Extensor hallucis longus-brevis, Tibialis anterior, Interossei dorsales, Semimembranosus, Semitendinosus, Iliopsoas, Obturatorius externus, Pectineus, Gracilis, Adductor longus/brevis, Sartorius, Rectus femoris.
- Isometric strengthening exercise of left side Serratus anterior muscle by pushing the therapist hand while the patient in supine position with extended arm and to resist the PT force in different directions (extension, flexion, horizontal ADD and ABD) and to improve the stability of the shoulder.
- We trained the lower extremities in standing position so we asked the patient to move each leg in the direction of flexion, extension, abduction and circumduction each for 10 -12 times so we could increase the stability on one leg at the same time

- Sensomotoric training to improve the stability of the patient, we trained on different surfaces such as:
 - Balance board and we asked the patient to shift his body to the sides, forward and backward while maintaining his balance and while semi flex the knees then to do mini squat without losing the balance
 - blue half ball we asked the patient to step over it with one leg and hold the position for 4 seconds then step back and do it with other leg, also to do mini squat without losing the balance. The patient was not able to perform both exercises with closed eyes.
 - We trained with rope ball so the patient is asked to hold the ball by the affected hand while the rope is connected to it and throw to it to the PT, then to throw it and to hold it by the other hand lastly to throw it and to hold it by the same hand
 - for fine motor skills, we had small spongy letters and specific board is made for them so we asked the patient to pick a letter to put it inside the board to make a word.
- 8\2\2017

Present State:

- **Objective:** the patient was in a good condition, no cognitive problem and no assistive devices were used.
- **Subjective:** the patient wasn't complaining about any pain. And he said that he felt tired after the exercises yesterday.

Goals Of Today Unit:

- To train and re-educate the gait and stair climbing.
- To train and improve stability.
- Functional exercise for the hand (fine motor skill).

Implemented Therapy:

- We walked with the patient in corridor of the hospital and we instructed him to correct his gait pattern, we emphasis on using heel strike (dorsi flexion) while walking and to flex his pelvis and knees more, also using upper trunk in across

with lower extremities (rotation and arm swing) the patient was good on stairs but the weakness of the knee and hip flexors was obvious on the affected leg.

- Sensomotoric training to improve the stability of the patient, we trained on different surfaces such as:
 - Balance board and we asked the patient to shift his body to the sides, forward and backward while maintaining his balance and while semi flex the knees then to do mini squat without losing the balance
 - blue half ball we asked the patient to step over it with one leg and hold the position for 4 seconds then step back and do it with other leg, also to do mini squat without losing the balance. The patient was not able to perform both exercises with closed eyes.
- We trained with rope ball so the patient is asked to hold the ball by the affected hand while the rope is connected to it and throw to it to the PT, then to throw it and to hold it by the other hand lastly to throw it and to hold it by the same hand
- for fine motor skills, we had small spongy letters and specific board is made for them so we asked the patient to pick a letter to put it inside the board to make a word.
- At the end, we applied electrotherapy (TENS) to stimulate the patient forearm extensors (extensor digitorum) for 10 mins 5 second of stimulation and 25 second rest with frequency of 50 Hz and pulse width of 300ms

- **9\2\2017**

Present State:

- **Objective:** the patient was in a good condition and so excited to exercise, no cognitive problem and no assistive devices were used.
- **Subjective:** the patient wasn't complaining about any pain. And he said that he felt tired after the exercises yesterday.

Goals Of Today Unit:

- To Strength and activate the weak muscles on the affected side (serratus anterior, triceps brachii, forearm extensors, hip and knee flexors)

- To train and improve stability.
- Functional exercise for the hand (fine motor skill).

Implemented Therapy:

- We walked with the patient in corridor of the hospital and we instructed him to correct his gait pattern, we emphasis on using heel strike (dorsi flexion) while walking and to flex his pelvis and knees more, also using upper trunk in across with lower extremities (rotation and arm swing) the patient was good on stairs but the weakness of the knee and hip flexors was obvious on the affected leg.
- Sensomotoric training to improve the stability of the patient, we trained on different surfaces such as:
 - Balance board and we asked the patient to shift his body to the sides, forward and backward while maintaining his balance and while semi flex the knees then to do mini squat without losing the balance
 - blue half ball we asked the patient to step over it with one leg and hold the position for 4 seconds then step back and do it with other leg, also to do mini squat without losing the balance. The patient was not able to perform both exercises with closed eyes.
- We trained with rope ball so the patient is asked to hold the ball by the affected hand while the rope is connected to it and throw to it to the PT, then to throw it and to hold it by the other hand lastly to throw it and to hold it by the same hand
- for fine motor skills, we had small spongy letters and specific board is made for them so we asked the patient to pick a letter to put it inside the board to make a word.
- At the end, we applied electrotherapy (TENs) to stimulate the patient forearm extensors (extensor digitorum) for 10 mins 5 second of stimulation and 25 second rest with frequency of 50 Hz and pulse width 300ms

3.6. Final kinesiological Examination By Physiotherapist:

3.6.1. Postural examination-aspection-plumb line test:

o back view:

- The base of support: Slight narrow base of support
- Shape and position of the ankle joints: On the right side, it is laterally rotated and on the left side there is no rotation
- Shape and thickness of the Achilles tendon: Symmetrical.
- Contour of the calf muscles (medial and lateral part): The prominence of soleus and medial part of the calf (Gastrocnemius medial head) is in the right calf more than the left.
- Popliteal line - symmetry, the slope: Both are in the same height
- Contour of the thigh muscles - especially the adductors: both are not prominence, in both sides the shape is symmetrical.
- Subgluteal line - height, length, symmetry: Higher in the right side
- Gluteal muscles - symmetry, shape, tonus: Symmetrical and both has the same shape.
- Symmetry of thoracobrachial triangles - the shape and size of triangles: Wider on the right side
- Position of pelvis: Pelvis is shifted to left side
- Curvature of the spine in the frontal plane (scoliosis ...): L, THL spine is slightly shifted to the left side.
- Position of the scapula - (angulus infraspinatus, margo medialis): On the left is slightly higher than the right side
- Position of the head: In neutral position

o front view:

- The base of support: slight narrow
- The position of the feet, examination of the arches (vault): lateral rotation in the right foot
- The position and shape of the toes: the patient has relaxed toes

- Weight distribution (medial X lateral edge): The patient is depending on his right side more
- Shape and position of the knee joints (varus X valgus) and patella: Slight lateral rotation in the right knee
- Shape of the thigh muscles (the quadriceps femoris): Not clear
- Position of the pelvis: The pelvis is shifted to the left side
- Symmetry of the muscle tone of abdominal muscles: Not clear
- Position of the navel (belly muscle symmetry): Physiological position
- Symmetry of thoracobrachial triangles - the shape and size of triangles: Wider on the right side
- Position and symmetry of the chest (position of nipples): Slight higher on the right side
- Position of the shoulder girdle (contour, tonus, protraction, ...): The patient has protracted shoulder
- Position of the head (symmetry, position of ears, face symmetry, ...): Head is neutral position

o side view (right):

- Shape and position of the ankle joints (plantarflexion X dorsiflexion) : in a balanced position
- Shape and contour of the shin (symmetry, ...): physiological position
- Position of the knee joints (hyperextension X semiflexion): neutral position, no hyperextended, no semi flexion
- Position of the pelvis - anteversion (increased anteversion X retroversion): slight posterior tilt of the pelvis
- Position and curvature of the L, L/Th, Th and C spine: decreased lordosis in L spine
- Shape of the abdominal muscles (prominence): not clear
- Position of the shoulder girdle (protraction ...): protracted shoulder
- Flattening; and tops of the curves): non
- Position of the head (forward holding ...): slight protraction of the head

o **side view (left):**

- Shape and position of the ankle joints (plantarflexion X dorsiflexion): In a balanced position
- Shape and contour of the shin (symmetry, ...): Physiological position
- Position of the knee joints (hyperextension X semiflexion): Neutral position, no hyperextended, no semi flexion
- Position of the pelvis - anteversion (increased anteversion X retroversion): slight posterior tilt of the pelvis
- Position and curvature of the L, L/Th, Th and C spine: decreased lordosis in L spine
- Shape of the abdominal muscles (prominence): not clear
- Position of the shoulder girdle (protraction ...): protracted shoulder
- Flattening; and tops of the curves): non
- Position of the head (forward holding ...): slight protraction of the head

3.6.2. Palpation: pelvis (conclusion)

- Iliac crest: Left side is lower than right side.
- PSIS: Left side is lower than right side.
- ASIS: Left side is lower than right side.

3.6.3. Anthropometric Measurement (length, circumferences)

UPPER EXTREMITIES	RIGHT (cm)	LEFT (cm)
Length of upper limbs	70	69.5
Length of humerus	27.5	28
Length of forearm	26	26
Length of hands	17	17
Circumference of upper limb	31	30.5
Circumference of forearm	28	27.5

Table 10: Final Anthropometric measurement (upper extremities)

LOWER EXTREMITIES	RIGHT (cm)	LEFT(cm)
Anatomical length		
Functional length	83	82.5
Length of thigh	41	41
Length of middle leg	42	41.5

Circumference of thigh (10 cm above knees)	46	46
Circumference of knee	39	39.5
Circumference of calf	38	38
Circumference of ankle	26	25
Circumference of foot	23.5	23.5

Table 11: Final Anthropometric measurement (lower extremities)

3.6.4. Gait Analysis:

Patient has in general stability. He uses one crutch by his left hand which has four points in contact with the ground. And he needs therapist support too.

- Width of the base of support: Slight narrow.
- Walking rhythm: periodic walking.
- Walking speed: Slow.
- Stride length: symmetrical.
- Movement of the foot: Ideal in right foot and decreased dorsal flexion on the left foot.
- Movement of knees joints: the flexion was increased on the left leg but slightly higher on the right .
- Movement of hip joints: decreased extension in both legs.
- Movement of the trunk: slight rotation and less shifting of the trunk.
- Activity of back muscles: more activated in low thoracic and lumbar area in both sides.
- Activity of abdomen muscles: Low activity in both sides and more in left side.
- Position and movement of head: Tilted to the right side.
- Stability of walking: more stable and faster than before.
- Walk with eyes closed: Patient could perform it correctly.

3.6.5. Specific Testing

- **Romberg Test (I-III):** He didn't lose his balance (with open and closed eyes even while feet together) we didn't get any positive result (normal)

Romberg I – normal standing

Romberg II – feet together

Romberg III – closed eyes

- **Single-leg stance test:** The patient was performing the test correctly without any correction or losing of balance.

Modification of Standing:

- **Standing on tiptoes:** the patient could perform it correctly.
- **Standing on heels:** the patient could do it properly.

the patient was able to perform both of them without correction.

3.6.6. **Goniometry: Classic (two-arms)**

ACTIVE	PASSIVE
Shoulder: S: R: 30-0-160 L: 20-0-150 F: R: 140-0-0 L: 130-0-0 T: R: 120-0-30 L: 120-0-10 R: R: 75-0-65 L: 65-0-65	Shoulder: S: R: 45-0-160 L: 35-0-160 F: R: 170-0-0 L: 140-0-0 T: R: 90-0-30 L: 120-0-15 R: R: 80-0-70 L: 70-0-70
Elbow: S: R: 0-0-150 L: 0-5-140	S: R: 0-0-150 L: 0-0-145
Forearm: R: R: 80-0-80 L: 60-0-60	R: R: 80-0-80 L: 70-0-70
Wrist:	

S: R: 65-0-70 F: R: 20-0-30	L: 50-0-55 L: 15-0-25	S: R: 70-0-80 F: R: 25-0-35	L: 55-0-55 L: 15-0-35
Distal Interphalangeal joint 1 st finger: S: R: 0-0-80 Distal Interphalangeal joint 2 nd finger: S: R: 0-0-80 Distal Interphalangeal joint 3 rd finger: S: R: 0-0-80 Distal Interphalangeal joint 4 th finger: S: R: 0-0-80	L: 0-0-65 L: 0-0-75 L: 0-0-75 L: 0-0-75	Distal Interphalangeal joint 1 st finger: S: R: 0-0-85 Distal Interphalangeal joint 2 nd finger: S: R: 0-0-85 Distal Interphalangeal joint 3 rd finger: S: R: 0-0-85 Distal Interphalangeal joint 4 th finger: S: R: 0-0-85	L: 0-0-80 L: 0-0-80 L: 0-0-80 L: 0-0-80
Proximal Interphalangeal joint 1 st finger: S: R: 10-0-100 Proximal Interphalangeal joint 2 nd finger: S: R: 0-0-100 Proximal Interphalangeal joint 3 rd finger: S: R: 0-0-100 Proximal Interphalangeal joint 4 th finger: S: R: 0-0-100	L: 0-0-80 L: 0-0-90 L: 0-0-90 L: 0-0-90	Proximal Interphalangeal joint 1 st finger: S: R: 10-0-100 Proximal Interphalangeal joint 2 nd finger: S: R: 0-0-100 Proximal Interphalangeal joint 3 rd finger: S: R: 0-0-100 Proximal Interphalangeal joint 4 th finger: S: R: 0-0-100	L: 0-0-80 L: 0-0-90 L: 0-0-90 L: 0-0-90
Metacarpophalangeal joint 2 nd finger: S: R: 0-0-90 Metacarpophalangeal joint 3 rd finger: S: R: 0-0-90 Metacarpophalangeal joint 4 th finger: S: R: 0-0-90 Metacarpophalangeal joint 5 th finger:	L: 0-0-90 L: 0-0-90 L: 0-0-90	Metacarpophalangeal joint 2 nd finger: S: R: 0-0-90 Metacarpophalangeal joint 3 rd finger: S: R: 0-0-90 Metacarpophalangeal joint 4 th finger: S: R: 0-0-90 Metacarpophalangeal joint 5 th finger:	L: 0-0-90 L: 0-0-90 L: 0-0-90

S: R: 0-0-90	L: 0-0-90	S: R: 0-0-90	L: 0-0-90
Hip:		Hip:	
S: R: 10-0-130	L: 10-0-110	S: R: 15-0-135	L: 10-0-130
F: R: 30-0-10	L: 25-0-10	F: R: 35-0-15	L: 35-0-15
R: R: 35-0-35	L: 20-0-25	R: R: 45-0-45	L: 35-0-35
Knee:		S:	
S: R: 0-0-140	L: 0-0-135	R: 0-0-150	L: 0-0-140
Ankle:			
S: R: 20-0-45	L: 15-0-35	S: R: 20-0-45	L: 20-0-40
R: R: 20-0-40	L: 15-0-35	R: R: 20-0-40	L: 15-0-35

Table 12: Final ROM examination (goniometer).

3.6.7. Muscle Length Test (according to Janda or Kendall):

- **length of ankle flexor muscles (gastrocnemius- soleus):** (Kendall 2005)
gastrocnemius:
R: normal length
L: normal length
Soleus:
R: normal length
L: normal length
- **length of hip flexor muscles:** (Kendall 2005)
R: shortness in both one and two joints hip flexor muscles
L: shortness in both one and two joints hip flexor muscles
- **length of hip adductor muscles:** (Janda 2013)
R: grade 0
L: grade 1 ABD 30 deg
- **length of hamstring muscles:** (Kendall 2005)
R: slight shortness

- L: slight shortness
- **length of piriformis muscles:** (Janda 2013)
R: grade1 limited ADD
L: grade1 limited ADD
 - **length of pectoralis major** (upper and lower part) (Kendal 2005)
R: normal length
L: normal length
 - **length of pectoralis minor:** (Kendall 2005)
R: slight shortness
L: slight shortness
 - **length test for teres major, latissimus dorsi, rhomboid major and minor muscles:** (Kendall 2005)
R: normal length
L: normal length
 - **length of medial and lateral shoulder rotators:** (Kendall 2005)
R: normal length
L: normal length
 - **length of carnial part of trapezius:**(Janda 2013)
R: grade 1 slight resistance on the shoulder
L: grade 1 slight resistance on the shoulder

3.6.8. Examination Of Muscles Tonicity (Palpation):

Name	Result
TRAPIZEUS	R: Normal tonic, no pain L: Normal tonic, no pain
DELTOID	R: Normal tonic, no pain L: Normal tonic, no pain
SUPRASPINATUS	R: slight hyper tonic, no pain L: Normal tonic, no pain
INFRASPINATUS	R: slight hyper tonic, no pain

	L: Normal tonic, no pain
BICEPS	R: slight hyper tonic, no pain L: Normal tonic, no pain
TRICEPS	L: slight hyper tonic, no pain R: Normal tonic, no pain
E CARPI RAD	L: slight hyper tonic, no pain R: Normal tonic, no pain
E CARPI ULNA	L: slight hyper tonic, no pain R: Normal tonic, no pain
F CARPI RAD	L: slight hyper tonic, no pain R: Normal tonic, no pain
F CARPI ULNA	R: slight hyper tonic, no pain L: Normal tonic, no pain
PECTORALIS MAJ	R: Normal tonic, no pain L: Normal tonic, no pain
PECTORALIS MIN	R: Normal tonic, no pain L: Normal tonic, no pain
RECTUS ABD	Normal tonic, no pain
QUADRATUS LUMB	Hypertonic, slightly painful
GLUTEAUS MAX	R: Normal tonic, no pain L: Normal tonic, no pain
PIRIFORMIS	R: Hyper tonic, painful L: Hyper tonic, painful
ADDUCTORS	R: Hyper tonic, Slightly painful L: Normal tonic, no pain
TIBIALIS ANTERIOR	R: slight hyper tonic, no pain L: Normal tonic, no pain

Table 13: Final Examination of muscles tonicity.

3.6.9. Final Joint Play Examinations:

Examination Of Upper Extremities Joints:

Joint name	Left	Right
Shoulder joint ventrodorsal-and caudal direction.	No Restriction	No Restriction
Shoulder blade against thoracic wall (scapula)	No Restriction	No Restriction
Acromioclavicular Joint ventrodorsal-craniocaudal direction	No Restriction	No Restriction
Sternoclavicular Joint ventrodorsal-craniocaudal direction	No Restriction	No Restriction
Elbow Joint medial-lateral direction	No Restriction	No Restriction
Radiocarpal Joint	No Restriction	No Restriction
Carpometacarpal Joint	No Restriction	No Restriction
Carpal Bones (distal/proximal rows of carpal bones)	Restricted in the direction of dorsal flexion	No Restriction
Carpometacarpal Joint of the thumb	No Restriction	No Restriction
Metacarpophalangeal Joints	No Restriction	No Restriction

Table 14: final examination of joint play upper extremities.

Examination Of Lower Extremities Joints:

Lower Extremity		
Sacroiliac joint	No Restriction	No Restriction
Patella (all directions)	No Restriction	No Restriction
Knee joint (medial \ lateral direction)	No Restriction	No Restriction

Tibiofibular joint (dorsal \ventral direction)	Restriction in ventral direction	No Restriction
Talocrural joint (dorsal \ventral direction)	No Restriction	No Restriction
Subtalar joint (dorsal \ventral direction)	No Restriction	No Restriction
Lisfranc's joint (dorsal \ ventral direction)	No Restriction	No Restriction
Chopart's joint (dorsal \ ventral direction)	No Restriction	No Restriction
Metatarsophalangeal Joints	No Restriction	No Restriction
Interphalangeal Joints	No Restriction	No Restriction

Table 15: final examination of joint play lower extremities.

3.6.10. Neurological Examination:

Deep tendon reflexes:

- **Knee jerk reflex:**
Physiological, in both sides.
- **Achilles tendon reflex:**
Right foot physiological
Left foot physiological
- **Biceps tendon reflex:**
In both biceps tendons, physiological.
- **Triceps tendon reflex:**
Right triceps tendon, physiological.
Left triceps tendon, physiological.
- **Wrist reflex:**
Physiological in both sides

Specific Neurological Test:

o Paretic test:

- ☐ **Mingazzini test upper extremities:**
patient could hold the upper extremities in a position correctly.
- ☐ **Mingazzini test lower extremities:**
patient was able to hold his legs in position correctly.

o Pyramidal Test: ☐

- **Trömner signs:** Negative in both hands. ☐
- **Hoffman test:** Negative in both hands. ☐
- **Oppenheim test:** Negative in both legs. ☐
- **Chaddock's Sign:** Negative in both legs. ☐
- **Babinski test:** Negative in both legs.

Examination Of Cranial Nerves:

Nerve No.	Nerve Name	Result
I	Ophthalmic	Physiological
II	Optic	Physiological
III	Oculomotors	Physiological
IV	Trochlearis	Physiological
V	Trigeminus	Physiological
VI	Abdducens	Physiological
VII	Facialis	Physiological
VIII	Vestibulocochlearis	Physiological
IX	Glossopharyngeal	Physiological

X	Vagus	Physiological
XI	Accesorius	Physiological
XII	Hypoglossal	Physiological

Table 16: Final Examination of cranial nerves

-Note: there was no impairment of the cranial nerves as the result of the cranial nerves examination showed.

.....

Sensation Examinations (Superficial sensation):

Dermatomes	Result
C5	Physiological
C6	Physiological
C7	Physiological
C8	Decreased
T1	Physiological
T2	Physiological
T3	Physiological

Table 17: superficial sensation examination

Note: we examined the deep sensation of the lower and upper extremities of the patient in both sides and we had Physiological results in all of them

3.6.11. ADL Evaluation

The patient could transfer himself alone without any external support. He still takes more than the usual time to wear his T-shirt and his slippers due to the difficulties to abduct and flex the whole arm also the disability of fine motor function but the improvement was noticeable. During eating he is using his right hand but he is using his effected hand to

hold for example the meat, not using the knife with it. In general, he focusses more on his affected hand and starting emphasis on using it and involve it on his daily life activities.

Barthel test for ADL evaluation:

Activity	Result
Stool incontinence	10\10
Urinary incontinence	10\10
Grooming	10\10
Toilet use	5\5
Feeding	5\10
Transfers	15\15
Walking	15\15
Dressing	5\10
Climbing stairs	10\10
Bathing	5\5
Sum of results: 90 /100	

Table 18: Final Barthel test.

ADL4 0-40 points: total dependence
 ADL3 45-60 points: marked dependence
 ADL2 65-95 points: slight dependence
 ADL1 95-100 points: independent

- The conclusion shows that the patient is still slight dependence on daily life activities as there is no big difference comparing with the initial test.

3.6.12. Final Examinations Conclusion

During the Final physical therapy examinations on the 10/2/2017 the result showed us the following findings:

The patient did not have any pain and he was generally happy about his progress but still complaining about the disability of moving the left wrist joint. The whole body was slightly shifted to the right side which influences the pelvis position to be elevated to the left side. The main results in gait examinations showed that the patient is more stable in walking, better gait pattern, more flexion on the hip and knee joint with swinging arms and the dorsiflexion of the ankle was noticeably increased as well.

In the direction of extension there was a marked limitation in the range of motion in left distal and proximal interphalangeal joints. Slight limitation in the extension direction was in left knee, hip, wrist and shoulder joints.

From the mental status point of view he was in a good condition. The patient was able to perform the Romberg I- III tests without any external support. Mingazzini tests for upper and lower extremities were negative. Pyramids track examinations show negativity results. The superficial sensation of the upper and lower limbs in the left side of the patient body was slightly decreased. He has physiological reflexes in the left side of his body during the deep tendon reflexes test. Finally, the patient is considered as a slight dependent patient according to the Barthel scale classification and he scored the same results with the results from the initial examinations.

3.7. Therapy Effect

From the results of the final examinations we found that the therapy was successful at some points considering the slow progression of the rehabilitation of this diagnose.

During my work with the patient, I avoid any overloading of work and the therapy sessions was to an acceptable level for the patient. The examinations and therapeutic procedures follow principles and methods provided by Kendall et al. (2005), Janda et al. (2013), Kolář et al. (2014) and Kabat with Bc. Tomáš Modlinger supervising and instructions. In details the negative and positive therapy effects are shown below:

Posture, Gait and stability examinations results shows many positive effects. First of all, increasing of the muscles strength on the affected side such as trapezius, deltoid, biceps brachii, triceps brachii, supinator, pronator and extensor digitorum muscles on the upper extremity and Gluteus maximus, Hamstrings muscles and Quadriceps femoris muscles on the lower extremity. Thanks to PNF techniques that I used and different exercises like SMT which also improved the patient's posture and his stability in gait

Second of all The movement of the trunk was more fluent (arms swinging to left and right sides) and the trunk is physiologically rotating while walking after it was in fixed position, the flexion of the hip and knee joint was increased the dorsiflexion of the ankle as well even the patient did lose his balance while performing Romberg test (feet apart\ feet together and with closed eyes).

Also the joint play examinations showed some good result since some joints restrictions were released such as: Shoulder blade against Thoracic wall (released in the direction of depression), Lisfranc's joint (released in both directions dorsal\ ventral) and Carpal bones in the direction of palmar flexion as well.

Finally, positive sign of the therapeutic procedure was the improvement of ROM in all directions of most left body joints. This improvement is due to the active and passive ROM exercises. The following Table shows the passive ROM examination comparison:

Initial ROM examination	Final ROM examination
Shoulder: S: 35-0-150 F: 140-0-0 T: 120-0-10 R: 60-0-60	Shoulder: S: 35-0-160 F: 140-0-20 T: 120-0-15 R: 70-0-70
Elbow: S: 0-0-135	Elbow: S: 0-0-145

Wrist: S: 40-0-70 F: 20-0-35	Wrist: S: 55-0-55 F: 15-0-35
Forearm: R: 35-0-55	Forearm: R: 70-0-70
Hip: S: 15-0-120 F: 30-0-10 R: 25-0-30	Hip: S: 20-0-130 F: 35-0-15 R: 35-0-35
Knee: S: 0-0-135	Knee: S: 0-0-140
Ankle: S: 15-0-30 R: 10-0-30	Ankle: S: 20-0-40 R: 15-0-35

Table 19: ROM comparison

The ROM was increased almost in all the joints of the left side of the body due to the improving of the muscles strength and our instruction to be aware and emphasis on involving his left hand in all daily activities

3.8. Prognosis

The effect of the therapy as it discussed above shows generally good prognosis in comparison with the duration of my practice with the patient and for the slow rehabilitation progress of this diagnose in general. However, me and my patient experience the effect of the therapy and we both were satisfied with the results.

As I experienced from the practical course the prognosis was not that huge but noticeable, and I believe that such kind of diagnosis will need more time to improve. However, I expect my patient to have a great prognosis with the time due his strong motivation, wellness and hardworking also by following home-exercises, changing his lifestyle, continuing the long rehabilitation program and regular self-therapy as he is performing now.

4. Conclusion:

The result we had with the patient was not as I expected but it was satisfying for both of us due to the improvement of his overall condition and increasing of the function, strength and ability of his hand which keeps him motivated and happy with the result. Also the cooperation and energy that I received from the patient throughout my work at the hospital made me more confidence about the effect of my therapy.

At the end I can say that the benefits that I had from this period of time which I was spending with patients at Kladno hospital were amazing since I really knew how a professional physiotherapist would treat the patient and how communicate with them in a real way and to experience how the full day of my career will be like in the future.

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Abbreviations

ACI: Anterior circulation infarction

ADC: Apparent diffusion coefficient

ADL: Activities of daily living

AHA: American Heart Association

ASA: American Stroke Association

AIS: Acute ischemic stroke

AICA: Anterior inferior cerebellar artery

ATP: Adenosine triphosphate

CADASIL: Cerebral arteriopathy, autosomal dominant, with subcortical infarcts and leukoencephalopathy

CVA: Cerebrovascular accident

FLAIR: Fluid-attenuated inversion recovery

HCPs: Healthcare providers (HCPs)

CT: Non-contrast computed tomography

MCA: Middle cerebral artery

MRA: Magnetic resonance angiogram

MRI: Magnetic resonance image

MIP: Maximum intensity projection

NIHSS: National Institutes of Health Stroke Scale

Occupational therapy: OT

ICA: Internal carotid artery

PCA: Posterior cerebral artery

PET: Positron emission tomography

PICA: Posterior inferior cerebellar artery

SPECT: Single-photon emission computed tomography

rt-PA: Recombinant tissue-type plasminogen activator

TIA: Transient ischemic attacks

TOAST: Trial of ORG 10172 in Acute Stroke Treatment

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UNIVERZITA KARLOVA
FAKULTA TĚLESNÉ VÝCHOVY A SPORTU
José Martího 31, 162 52 Praha 6-Vešelavín

Application for Approval by UK FTVS Ethics Committee

of a research project, thesis, dissertation or seminar work involving human subjects

The title of a project: Case study of a patient after ischemic stroke

Project form: bachelor

Period of realization of the project: February 2017

Applicant: Albarrak Ahmad

Main researcher: Albarrak Ahmad

Supervisor (in case of student's work): doc. PaedDr. Dagmar Pavlů, CSc.

Project description: The case study of a patient after ischemic stroke affected the right part of the brain conducted under the expert supervision of an experienced physiotherapist. The patient is having his rehabilitation course in neurological department at Oblastní Nemocnice Kladno. The methods that are used with the patient by the researcher are based on the knowledge earned from the bachelor program in physiotherapy in UK FTVS.

Ensuring safety within the research: For that particular research the researcher does not use invasive methods. All the precaution and risk prevention are followed according to the hospital rules, policies and procedures signed documentation. The rehabilitation regimes are designed, prescribed and approved by the responsible physician, all the methods are used with the patient were under the responsible supervision of Mgr. Tomas Modilger.

Ethical aspects of the research: All the members and participants in the research project are adults and non-vulnerable. All the personal data are anonymized and will be stored in an anonymous form.

Informed Consent: attached

It is a duty of all participants of the research team to protect life, health, dignity, integrity, the right to self-determination, privacy and protection of the personal data of all research subjects, and to undertake all possible precautions. Responsibility for the protection of all research subjects lies on the researcher(s) and not on the research subjects themselves, even if they gave their consent to participation in the research. All participants of the research team must take into consideration ethical, legal and regulative norms and standards of research involving human subjects applicable not only in the Czech Republic but also internationally.

I confirm that this project description corresponds to the plan of the project and in case of any change, especially of the methods used in the project, I will inform the UK FTVS Ethics Committee, which may require a re-submission of the application form.

In Prague, 13, February, 2017

Applicant's signature:

Approval of UK FTVS Ethics Committee

The Committee: Chair: doc. PhDr. Irena Parry Martínková, Ph.D.

Members: prof. PhDr. Pavel Slepíčka, DrSc.

doc. MUDr. Jan Heller, CSc.

Mgr. Pavel Hráský, Ph.D.

Mgr. Eva Prokešová, Ph.D.

MUDr. Simona Majorová

The research project was approved by UK FTVS Ethics Committee under the registration number: 064/2017

Date of approval: 14.2.2017

UK FTVS Ethics Committee reviewed the submitted research project and **found no contradictions** with valid principles, regulations and international guidelines for carrying out research involving human subjects.

The applicant has met the necessary requirements for receiving approval of UK FTVS Ethics Committee.

UNIVERZITA KARLOVA
Fakulta tělesné výchovy a sportu
José Martího 31, 162 52, Praha 6
Stamp of UK FTVS
- 20 -

Signature of the Chair of
UK FTVS Ethics Committee

INFORMOVANÝ SOUHLAS

Vážená paní, vážený pane,

v souladu se Všeobecnou deklarací lidských práv, zákonem č. 101/2000 Sb., o ochraně osobních údajů a o změně některých zákonů, ve znění pozdějších předpisů, Helsinskou deklarací, přijatou 18. Světovým zdravotnickým shromážděním v roce 1964 ve znění pozdějších změn (Fortaleza, Brazílie, 2013) a dalšími obecně závaznými právními předpisy Vás žádám o souhlas s prezentováním a uveřejněním výsledků vyšetření a průběhu terapie prováděné v rámci praxe na¹, kde Vás příslušně kvalifikovaná osoba seznámila s Vaším vyšetřením a následnou terapií. Výsledky Vašeho vyšetření a průběh Vaší terapie bude publikován v rámci bakalářské práce na UK FTVS, s názvem²

Získané údaje, fotodokumentace, průběh a výsledky terapie budou uveřejněny v bakalářské práci v anonymizované podobě. Osobní data nebudou uvedena a budou uchována v anonymní podobě. V maximální možné míře zabezpečím, aby získaná data nebyla zneužita.

Jméno a příjmení řešitele Podpis:.....

Jméno a příjmení osoby, která provedla poučení³ Podpis:.....

Prohlašuji a svým níže uvedeným vlastnoručním podpisem potvrzuji, že dobrovolně souhlasím s prezentováním a uveřejněním výsledků vyšetření a průběhu terapie ve výše uvedené bakalářské práci, a že mi osoba, která provedla poučení, osobně vše podrobně vysvětlila, a že jsem měl(a) možnost si řádně a v dostatečném čase zvážit všechny relevantní informace, zeptat se na vše podstatné a že jsem dostal(a) jasné a srozumitelné odpovědi na své dotazy. Byl(a) jsem poučen(a) o právu odmítnout prezentování a uveřejnění výsledků vyšetření a průběhu terapie v bakalářské práci nebo svůj souhlas kdykoli odvolat bez represí, a to písemně zasláním Etické komisi UK FTVS, která bude následně informovat řešitele.

Místo, datum

Jméno a příjmení pacienta Podpis pacienta:

Jméno a příjmení zákonného zástupce⁴

Vztah zákonného zástupce k pacientovi Podpis:

