

THALAMIC ISCHEMIC STROKE

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Abstract: The objective of this research article is to provide an informed understanding on thalamic ischemic stroke and in particular the aetiology, pathophysiology and the symptomatic signs and characteristic features of a typical patient suffering from ischemic stroke. The thalamus provides an important role in executing functions such as ; memory, cognition, emotional behaviours, sleep-wake cycle, general cortical alert motor responses, processing fine and visualized sensory signals and relaying this information to the pre-motor cortex, motor cortex and the somatosensory cortex respectively. In addition to this, we will be particularly expanding on two aspects ; The diagnostic findings via computed tomography and magnetic resonance imaging and we will be looking at the typical signs and characteristics findings seen on these two types of imaging modalities. We will also be making an in depth analysis of the incidence of the cases of thalamic ischemic stroke and examine the influence if any, that the age of an individual has on the onset of ischemic stroke and to establish any trends between the age and onset of thalamic ischemic stroke. Furthermore, previous studies indicate that there is a substantial and ever developing link between the increases in age leading to the increase risk of an ischemic attack/stroke however; this link has yet to be proven due to the existence of environmental factors that can have a polymorphic influence on the development of thalamic ischemic stroke. The method taken in this article was intended to take an un-biased and randomized selection of a sample of patients who had previously suffered from an ischemic stroke of thalamic origin or were currently undergoing treatment for an ischemic stroke and to utilise different imaging procedures such as; computed tomography and magnetic resonance imaging to evaluate the severity and predict a cause of onset of the ischemic stroke. In addition to this, from a sample of 20 patients who had suffered from thalamic ischemic stroke, the results indicated that there was a direct correlation between the age and increased risk of developing thalamic ischemic stroke as the patients in our study were all of elderly origin ranging between the ages of 55-80. Furthermore, the characteristic signs upon evaluating the diagnostic images showed a pattern on C.T. as an ill-defined hypo-dense region of the thalamus or a general obscurity of the grey-white matter border between the adjacent internal capsule. On the other hand, in the diagnostic MRI images we could see a slightly impaired and hyper-intense grey-matter region and as the infarct develops with a higher degree of hyper-intensity can be seen. Overall, we can state that there is a generalized relationship with more elderly individuals at a higher risk of developing thalamic ischemic stroke/infarct, however, more research needs to be conducted in order to understand the pathophysiology and to have a greater depth of understanding of the diagnostic criteria found on C.T. / MRI that can be used to evaluate whether a patient is or has suffered from an thalamic ischemic stroke.

Keywords: ischemic stroke, thalamus, computed tomography, magnetic resonance imaging, diagnosis.

1. INTRODUCTION

To explain and understand the pathophysiology, signs and symptoms of thalamic ischemic stroke and to define whether there is a relationship between the age of a patient and the risk of developing a thalamic ischemic stroke. The target audience of this review article is for those individuals who have a background in neuroradiology or radiology as a whole and for medical students or individuals who have an expressed interest in the medical discipline in itself. The wide range of sources and journals utilised in this article are indeed appropriate and relevant for the evaluations we intend to make in this article. In addition, this review article will be a review of all the sources cited at the end of this review and it will consist of being an empirical view, based upon the clinical findings and observations of various renowned researchers as well as my own research. Generally, thalamic ischemic stroke is accompanied by a wide range of risk factors which can lead to an increased risk of developing an ischemic stroke specifically within the thalamic region of the brain and these include the following ; Age, sex, hypertension, diabetes mellitus, hypercholesterolemia, smoking and cardiac arrhythmias. Generally, the incidence of thalamic ischemic stroke amongst the population is 1 in every 6 deaths related to cardiovascular diseases and leading to stroke of the thalamic region. Every 40 seconds an individual suffers from a stroke in which every 4 minutes someone dies from a stroke with 85 % of these deaths due to a thalamic ischemic stroke in origin. An individual to suffer from a first stroke is at nearly 2 times higher in Afro-Caribbeans than in Caucasians. In addition to this, the diseases associated

with thalamic ischemic stroke include movement disorders such as athetosis, asterixis, vascular parkinsonism, tremor, chorea, dystonia, hemi ataxia and visual disturbances. All of the aggravating factors mentioned are important as they can increase the likelihood of an ischemic stroke to develop by 45-67%, which is a great increase in the risk of developing an ischemic stroke and can lead to the various developmental changes displayed within an individual such as vascular thalamic amnesia, changes in personality and attention.

2. MATERIALS AND METHODS

In our study, computed tomography and magnetic resonance imaging modalities were utilised to effectively diagnose and precisely locate the ischemic stroke within the thalamic origin of the brain. Our study utilised 20 patients all ranging from the ages of 50 – 80 years of age who complained of symptomatic features associated with an thalamic ischemic stroke such as; loss of sensation, difficulties coordinating movements and balance, speech difficulties, visual disturbances, sleep disturbances, lack of interest and enthusiasm, memory loss who then underwent C.T. and MRI imaging to diagnose the cause and to exclude any other possibilities such as brain tumours, haemorrhagic stroke. Generally, each patient had a computed tomography image taken between 0 – 4.5 hours of onset of symptomatic features for differentiation between an ischaemic stroke and a haemorrhagic stroke. In addition to this, the imaging procedure is repeated approximately 7-8 days after the initial treatment plan to screen and identify whether the ischemic stroke is still present and in order to compare and contrast with the latter image taken at the initial investigation to ensure that the patient and stroke is responding well to the treatment. We then have consecutive images taken at specific time intervals at the 8 week mark from initial diagnosis and 6 months after the initial diagnosis to determine the sub-acute and the chronic stages of the thalamic ischaemic stroke.

3. RESULTS

From a sample of 20 patients who had suffered from thalamic ischemic stroke, the results indicated that there was a direct correlation between the age and increased risk of developing thalamic ischemic stroke as the patients in our study were all of elderly origin ranging between the ages of 55-80. Furthermore, the characteristic signs upon evaluating the diagnostic images showed a pattern on C.T. as an ill-defined hypodense region of the thalamus or a general obscurity of the grey-white matter border between the adjacent internal capsule. On the other hand, in the diagnostic MRI images we could see a slightly impaired and hyperintense grey-matter region and as the infarct develops with a higher degree of hyperintensity which can be seen. Overall, we can state that there is a generalized relationship with more elderly individuals at a higher risk of developing thalamic ischemic stroke/infarct, however, more research needs to be conducted in order to understand the pathophysiology and to have a greater depth of understanding of the diagnostic criteria found on C.T. / MRI that can be used to evaluate whether a patient is or has suffered from an thalamic ischemic stroke.

Figure A: CT of the brain. Bilateral lacunar thalamic ischemic stroke.

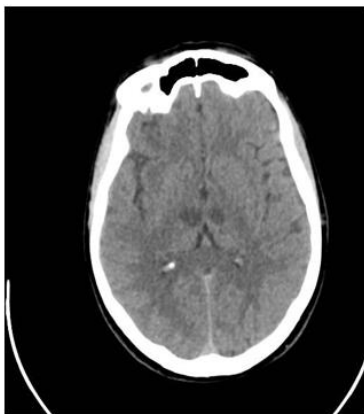


Figure B: Brain MRI. A patient with left thalamic infarction, following ischemic stroke in left MCA territory.

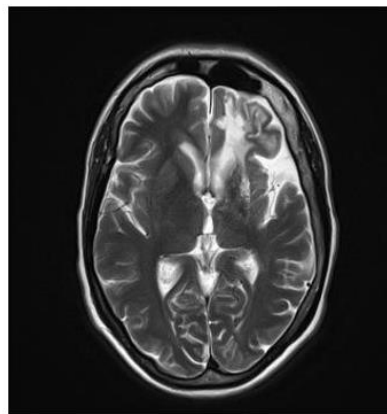
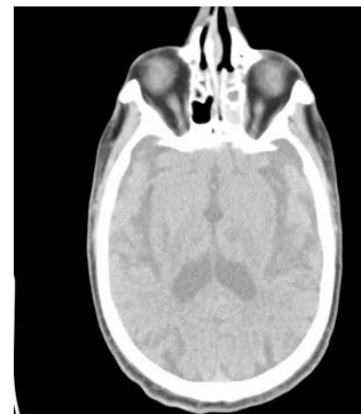


Figure C: CT of the brain - axial plane. Bilateral thalamic infarction in a subacute stage.

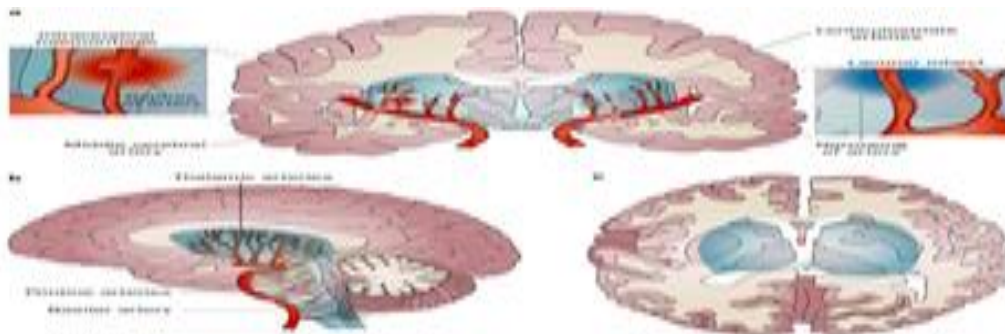


4. DISCUSSION

A thalamic ischaemic stroke is a type of lacunar stroke, which refers to a stroke in the deep part of your brain mainly involving the thalamus. A lacunar stroke is the most common type of ischaemic stroke resulting from an occlusion of small penetrating arteries that provide blood to the brain's deeper structures. There are various risk factors which can lead to an increased risk of developing a lacunar stroke and these include; hypertension which can lead to an ischemic stroke as the persistently high blood pressure can cause a narrowing and diseases of the blood vessels leading to a predisposition of ischemic strokes due to damaging of the inner lining of the blood vessels causing an obstruction of blood flow and oxygen to the brain hence resulting in an ischemic stroke. In conjunction to this, diabetes mellitus is another characteristic syndrome that can lead to an ischemic stroke as the raised blood glucose levels can lead to stiffening of the blood vessels and can cause a build-up of fatty deposits. These changes can lead to a characteristic blood clot eventually leading to a stroke. In relation to hypercholesterolemia, it is a situation whereby there is an increased circulation of LDL and VLDL in the systemic circulation and the increased levels of fatty acids in the bloodstream can lead to the predisposing of a thromboembolism or an atherosclerotic plaque thereby depriving the brain of adequate supply of blood and oxygen leading to an ischemic stroke.

Below is an image representing a part of the brain's circulation with the idea of representing visually the effect and the various ways in which the lacunar stroke begins. As we can see, there are several different vessels which are of a higher risk to developing an occlusion or becoming progressively narrower due to external environmental stimuli and these include the : inferior, median, superior ramus of the paramedian arteries which is a branch of the basilar artery, the lateral communicating branch of the posterior choroidal artery which is a branch of the posterior cerebral artery, tubulothalamic branch of the posterior communicating artery a branch from the posterior cerebral artery and the medial and infero-lateral communicating branches of the posterior choroidal arteries a branch of the posterior cerebral artery. The image below indicates when we have a narrowing in the artery due to aforementioned risk factors, the supply of blood to a specific part of the brain parenchyma is significantly reduced and can lead to cortical and subcortical necrosis, resulting in compression and deviation of the surrounding brain tissues leading to the development of the various CNS signs and symptoms seen in patients.

Figure 1: A transverse section through the brain illustrating how a lacunar infarct develops in the brain.



In terms of the development of a lacunar stroke in the thalamus it occurs in a theory proposed by Koffler, who stated that lacunes are derived from an occlusion of a single deep penetrating artery that arises directly from constituents of the circle of willis, cerebellar arteries and basilar arteries but with a higher incidence of cases presented within the basilar artery origin. The two proposed mechanisms are microatheroma and lipohyalinosis. The microatheroma is the more common pathology leading to a lacunar infarct, it is where an atheroma in the artery blocks the orifice of the penetrating artery known as a luminal atheroma or it involves an atheroma existing at the origin of the penetrating artery called a junctional atheroma. Thereby this pathology leads to hypo-perfusion whereby we have inadequate amount of blood flowing through the vaso-occluded arteries and thus a lacunar infarct formation. On the other hand, lipohyalinosis is a cerebral small vessel disease affecting the small arteries, arterioles and capillaries of the brain. It is characterized by vessel wall thickening and a resultant reduction in luminal diameter. This occlusion is assumed to be due to hypertension induced in the acute stage by fibroid necrosis, which would lead to a lacunar infarct. We also have an alternative proposed mechanism that can be a cause of the formation of a lacunar infarct. This is endothelial dysfunction, capillary permeability or an enhanced blood-brain-barrier permeability which enables leakage of blood contents, promoting gliosis and formation of white-residual like matter opacities seen on magnetic resonance imaging. Below is a figure displaying the possible micro-circulatory routes in which the infarct

formation can take place within the brain's circulatory system and we have a C.T. image of a middle-aged male suffering from a lacunar infarct in the left thalamus, which can be seen as a hyperattenuated region within the sub-cortical region of the thalamus.

Figure 2: An image representing the arterial circulation of the brain

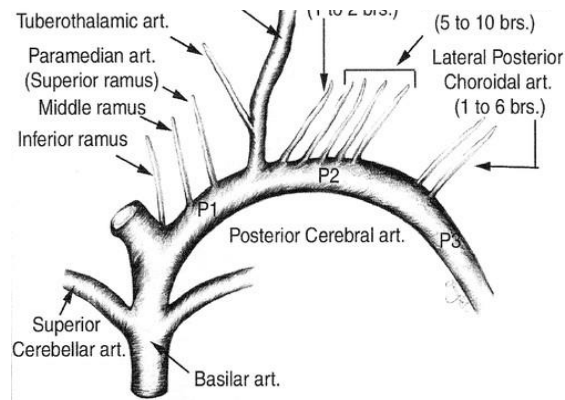
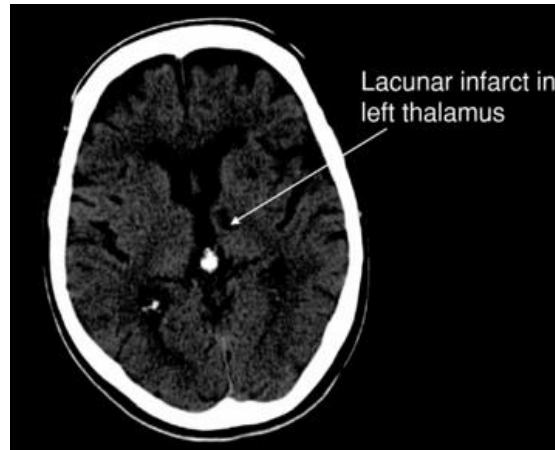


Figure 3: A C.T. image presenting a left lacunar infarct in the thalamic region of the brain.



Furthermore, the signs and symptoms that can be seen in a patient suffering from a lacunar infarct can be categorized into four groups/stages. These symptoms and syndromes can occur in a sudden, progressive or a fluctuating manner. The first group is a pure motor stroke or hemiparesis, which has a typical location within the posterior limb of the internal capsule, basilar portion of the pons and the corona radiata. It is presented by hemiparesis or hemiplegia that affects the face, arm or leg of the side of the body opposite to the location of the infarct and we can have a presentation of dysarthria, dysphagia. The second group is ataxic hemiparesis, which can be located in the posterior limb of the internal capsule, basilar part of the pons, red nucleus, corona radiata. It displays a combination of cerebellar and motor symptoms including weakness and clumsiness on the ipsilateral side of the body, it affects the leg more than the upper limbs and it is more commonly known as homolateral ataxia and crural paresis. The third group is a pure sensory stroke that can be located in the contralateral thalamus, internal capsule and corona radiata. It is marked by numbness on one side of the body, which can in the long-term lead to a development of tingling, pain, burning and unpleasant sensation on the contralateral upper portion of the body. The final group is a mixed somatosensory type whereby this infarct can be located in the thalamus, adjacent posterior limb of the internal capsule, lateral pons. It includes a syndrome called the lacunar syndrome, which is typically presented with hemiparesis or hemiplegia with sensory impairment in the contralateral side of the body.

There are a possible different number of combinations that can be employed in the treatment of the lacunar-based infarct. One of these is the administration of tissue plasminogen activator such as alteplase within 3 to 4.5 hours of stroke onset if the patient does not have any contraindications such as co-morbidities, which can complicate the effectiveness of the tissue plasminogen activator; this whole process is called thrombolysis. A high dosage of aspirin can be given via intravenous administration within 48 hours of identifying the lacunar infarct as it acts as a blood thinner and anti-coagulator. Consequently, anti-coagulants such as warfarin and heparin can also be administered for both short and long-term treatment of the lacunar infarct. For the prevention of long-term reoccurrences, medical regimens and plans are used to target and correct the underlying risk factors of lacunar infarcts such as hypertension, diabetes mellitus and cigarette smoking. The occupational and physical therapy interventions are used in the rehabilitation of a lacunar stroke, so for those patients who have suffered from an lacunar infarct or stroke and are treated with the aim of recovery or the best physical position to lead an independent life. The physiotherapy program includes a range of motions of the joints of the paretic limb using passive ranges of motions using splints and braces to treat complications such as contractures and spasticity.

5. CONCLUSION

Overall, thalamic stroke is classified as a general debilitating and progressively worsening condition whereby there are large majorities of the population being affected and it stems either from pre-existing comorbidities or due to lifestyle factors. Currently, the prevention and treatment plans for stroke and in particular thalamic ischaemic stroke

are limited to the administration of medications and rehabilitative medicine, however, emerging trials such as deep brain stimulation are being tested to prove the efficacy and effectiveness of this type of treatment.

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