INTRODUCTION

Cerebrovascular stroke is the second leading cause for both mortality and disability; as proved by Disability-Adjusted Life Years (DALYs) quantify both premature mortality (YLLs) and disability (YLDs) in Egypt, to be 10.5 % of all mortality causes ***(Institute for Health Metrics and Evaluation, 2010)***.

The phrase "time is brain" emphasizes that human nervous tissue is rapidly lost as stroke progresses and emergent evaluation and therapy are required. The typical patient loses 1.9 million neurons each minute in which stroke is untreated ***(Saver, 2006)***.

It has been estimated that 15 minutes delay in stroke management means delay 1 month of disability free life on the long term prognosis of the patient ***(Sandy et al., 2010).***

The typical journey of the stroke patient starting from his point to reach Emergency Medical Services (EMS), to have the Triage and referral to either to Primary Stroke Centers (PSC) or Comprehensive Stroke Centers (CSC), as like as taking the decision of potential eligibility of either intravenous rTPA administration or interventional thrombolysis, including the physical general assessment, neurological assessment and imaging process should be less than 3 hours. Door to drug is to be less than 60 minutes and door to Stroke Unit admission is to be less than 3 hours ***(Sandy et al., 2015).***

Phasic delay in providing an optimum service for the stroke patient includes pre-hospital delay and in-hospital delay.

Although, pre-hospital delay comprised the majority of time loss from symptom onset to potential treatment, the in-hospital times from emergency department arrival to being seen by an emergency department physician, initiation and interpretation of a Computed Tomography (CT) scan, and being seen by a neurologist were consistently longer than recommended ***(AHA/ASA Guideline, 2013)***.

As like as a substantial number of in-hospital stroke patients experience a long delay between symptom recognition and a neurological evaluation. While medical personnel are usually notified very soon after an in-hospital stroke is recognized, such patients often do not receive a rapid neurological evaluation ***(Evenson et al., 2001).***

Thorough neurological assessment of patients experiencing acute stroke is critical for accurate diagnosis, treatment and care throughout patient track towards proper care ***(Alberts et al., 1993).***

Consistent use of a standardized assessment tool designed for stroke patients assists in the achievement of lesser time of assessment and management ***(Alberts et al., 1993).***

The National Institutes of Health Stroke Scale (NIHSS) is a systematic assessment tool that provides a quantitative

measure of stroke-related neurologic deficit. The NIHSS was originally designed as a research tool to measure baseline data on patients in acute stroke clinical trials. Now, the scale is also widely used as a clinical assessment tool to evaluate acuity of stroke patients, determine appropriate treatment, and predict patient outcome ***(Richardson et al., 2006).***

A criticism of the NIHSS relates to its validity in certain non-dominant-hemisphere stroke syndromes. It is well recognized that an individual can score 0 on the NIHSS, despite having evidence of ischemic stroke, particularly in the posterior circulation territory ***(NIHSS, 1999).*** Examination of the component subscales of the NIHSS reveals a focus on limb and speech impairments and relatively little attention to, for example, cranial nerve lesions ***(Martin-Schild et al., 2011).*** There are radiological correlates, when quantifying extent of cerebral damage for a specified NIHSS score; the median volume of right-hemisphere strokes is larger than the volume of left-hemisphere strokes, suggesting non-dominant strokes are required to be more severe to reach the same grading on the NIHSS ***(Sato et al., 2008; Woo et al., 1999).***

An Emerging trend to complement the role of the NIH stroke scale score with another score primarily assessing the non-dominant hemispheric functions in the initial assessment of the stroke patient has been started in the world's stroke institutes searching for what they called "the holy complementary stroke scale" ***(Harrison et al., 2013).***

Also, this expansion of the NIHSS may be especially valuable in settings where imaging is not available or not possible, to better estimate lesion volume.

Among other scales assessing non-dominant hemispheric functions, Cookie Theft Picture Description Test (originally as a part of Boston Diagnostic Aphasia Examination), showing provisionally valuable benefit for the initial assessment of an acute ischemic stroke ***(Turc et al., 2016).***

As a pilot study for assessing role of quantitative analysis for such scale done by Turc and Others showing statistical significance. However, such application especially in different population still needs validation and subsequent modification and culture adaptation ***(Turc et al., 2016).***

Similar quantitative analysis of the picture description tasks for patients having right hemispheric functions deficits is integrated within the Comprehensive Aphasia Test (CAT) ***(Daniel et al., 2016)*** under the Spoken Picture Description Subscale which has been modified and culturally adapted for the Egyptian Population and been validated very recently ***(Bruce, 2010).***

The more accurate and rapid prediction of the site, size and side of the lesion using validated tools, the more efficient and suitable scale to be clinically implemented ***(Abou El-Ella et al., 2013).***

Finding standardized values for either whole brain or hemispheric volume and change correlations with age and different brain injuries is still a point of progress for further scientific research. Although, primary data were recently released as the Virginia Institute of Neuropsychiatry Patent Application correlates the whole brain volume and its changes through age with which we can correlates the manually determined ischemic lesion volume from MRI series in order to roughly estimate the relational lesion volume to the whole expected brain volume influencing further clinical decisions ***(Ross et al., 2016).***

Based on above mentioned correlates, we hypothesize that combining the traditional NIH stroke scale with the Spoken picture description subscale of the modified Comprehensive Aphasia Test could predict the volume, site and side of the acute ischemic stroke without being imaging aided and with no time added to initial assessment ***(Sims et al., 2009).***

Aim of the Work

To assess the additive predictive value of the picture description tasks as in Spoken picture description subscale of the modified Comprehensive Aphasia Test to that of the NIHSS as regards the main characteristics of the acute ischemic stroke; site, size and side.

Chapter 1

CEREBROVASCULAR STROKE

History of def.

"Apoplexy" was the first term used to describe "very acute nontraumatic brain injuries", firstly used by Hippocrates circa 400 BC and been used for more than 2000 years when William Cole in 1689 first introduce the word "stroke" to refer to the temporary vascular-related episodes of brain dysfunction with no then specific differnation between the infarction and the transient ischemic attack ***(Cole, 1869; Hippocrates, 1939).***

In 1970, the World Health Organization has released the definition of stroke to be "rapidly developing clinical signs of focal (or global) disturbance of cerebral function, lasting more than 24 hours or leading to death, with no apparent cause other than that of vascular origin." ***(Aho et al., 1980).***

History of TIA

An Ad Hoc Committee on Cerebrovascular Disease in 1975, defined Transient Ischemic Attacks to be episodes of temporary and focal dysfunction of vascular origin, which are variable in duration, commonly lasting from 2 to 15 minutes, but occasionally lasting as long as a day (24 hours) which don't leave no persistent neurological deficit." ***(Ad Hoc Committee, 1975).***

By 2002, this definition has been changed by an expert committee who proposed a new definition: "A TIA is a brief episode of neurologic dysfunction caused by focal brain or retinal ischemia, with clinical symptoms typically lasting less than one hour, and without evidence of acute infarction." ***(Albers et al., 2002).***

In 2009, another expert committee of the AHA/ASA published a scientific statement defining TIA and recommending evaluation has agreed to define the "transient ischemic attack (TIA) as a transient episode of neurological dysfunction caused by focal brain, spinal cord, or retinal ischemia without acute infarction." This definition set new link between TIA and stroke ***(Easton et al., 2009).***

Because of the variability of duration, there is now general agreement that a fixed time designation should not be the primary distinguishing factor between stroke and TIA. Time should be a secondary consideration when adequate imaging is unavailable. Time range frequencies could be a part of commentaries on these definitions ***(Sacco et al., 2013).***

The link between TIA and the Stroke

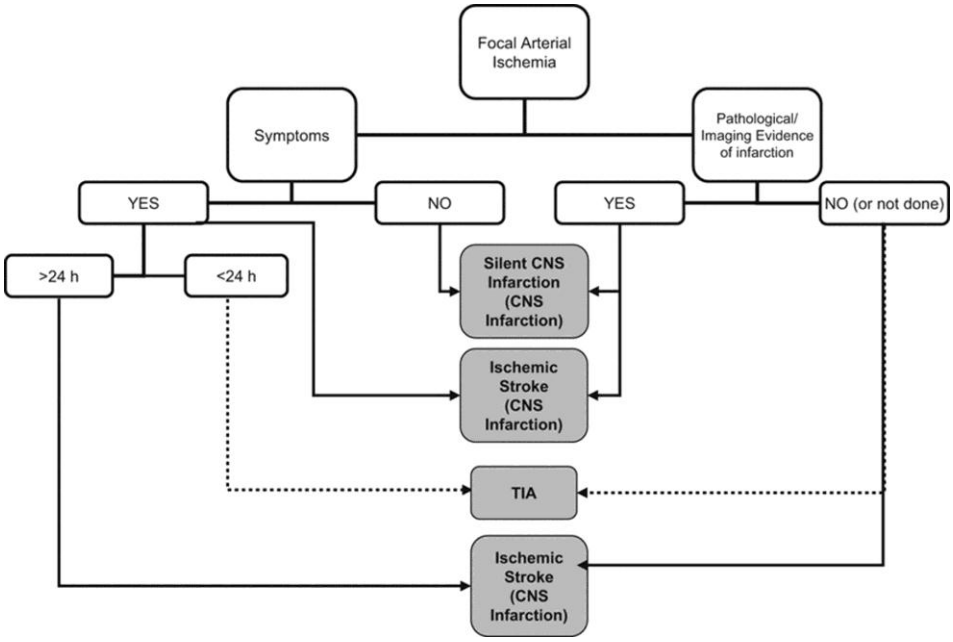
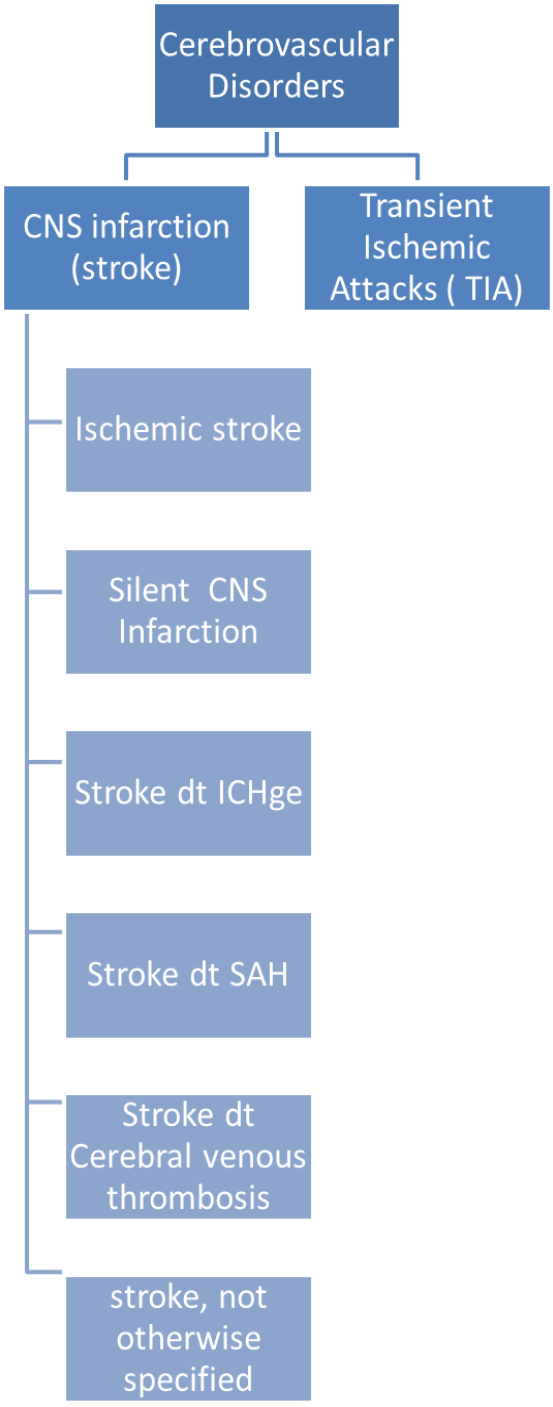


Figure (1): Hierarchy for diagnosis of acute ischemic events ***(Sacco et al., 2013).***

In the *International Classification of Diseases* (*ICD*) system, the cerebrovascular disorders were chiefly classified as TIA, cerebral ischemic stroke, ICH, or SAH to emphasize the intimate relation between the cerebrovascular stroke and the transient ischemic attacks as being different stereotypes of cerebrovascular ischemic accidents ***(Centers for Disease Control and Prevention Web site, 2001).***

The great role the duration of neurological symptoms and signs in the early definitions of stroke and TIA became controversial in the recent studies, using clinical observation and modern brain imaging, that shown that the duration and

reversibility of brain ischemia are variable. Brain tissue that is deprived of needed nutrients can, in some patients, survive without permanent injury for a considerable period of time—several hours or even, rarely, days—while in most other individuals, irreversible damage (infarction) occurs quickly. Because of this variability of duration, there has been a general agreement that a fixed time designation should not be the primary distinguishing factor between stroke and TIA. Time should be a secondary consideration in case of unavailability of adequate imaging. However, time range frequencies could be a part of commentaries on these definitions.


Cerebrovascular Disorders
CNS infarction (stroke)
Transient Ischemic Attacks (TIA)
Ischemic stroke
Silent CNS Infarction
Stroke dt ICHge
Stroke dt SAH
Stroke dt Cerebral venous thrombosis
stroke, not otherwise specified

Final def. of CNS infarction

In 2013, an expert committee of the AHA/ASA published a scientific statement defining proposed the most updated available definition of CNS infarction to be:" Brain, Spinal Cord, or Retinal Cell Death Attributable to Ischemia, Based on Pathological, Imaging, Other Objective Evidence, and/or Clinical Evidence ***(Sacco et al., 2013).***

As a result the definition of ischemic stroke is to be proposed as an episode of neurological dysfunction caused by focal cerebral, spinal, or retinal infarction with evidence of CNS infarction as defined previously ***(Sacco et al., 2013).***

The term "stroke" should be broadly used to include all of the following

Definition of CNS infarction

CNS infarction is brain, spinal cord, or retinal cell death attributable to ischemia, based on

1) Pathological, imaging, or other objective evidence of cerebral, spinal cord, or retinal focal ischemic injury in a defined vascular distribution.

2) Clinical evidence of cerebral, spinal cord, or retinal focal ischemic injury based on symptoms persisting ≥24 hours or until death, and other etiologies excluded. (Note: CNS

infarction includes hemorrhagic infarctions, types I and II; see "Hemorrhagic Infarction.")

Definition of ischemic stroke

An episode of neurological dysfunction caused by focal cerebral, spinal, or retinal infarction. (Note: Evidence of CNS infarction is defined above).

Definition of silent CNS infarction

Imaging or neuropathological evidence of CNS infarction, without a history of acute neurological dysfunction attributable to the lesion.

Definition of intracerebral hemorrhage

A focal collection of blood within the brain parenchyma or ventricular system that is not caused by trauma. (Note: Intracerebral hemorrhage includes parenchymal hemorrhages after CNS infarction, types I and II—see "Hemorrhagic Infarction").

Definition of stroke caused by intracerebral hemorrhage

Rapidly developing clinical signs of neurological dysfunction attributable to a focal collection of blood within the brain parenchyma or ventricular system that is not caused by trauma.

Definition of silent cerebral hemorrhage

A focal collection of chronic blood products within the brain parenchyma, subarachnoid space, or ventricular system on neuroimaging or neuropathological examination that is not caused by trauma and without a history of acute neurological dysfunction attributable to the lesion.

Definition of subarachnoid hemorrhage

Bleeding into the subarachnoid space (the space between the arachnoid membrane and the pia mater of the brain or spinal cord).

Definition of stroke caused by subarachnoid hemorrhage

Rapidly developing signs of neurological dysfunction and/or headache because of bleeding into the subarachnoid space (the space between the arachnoid membrane and the pia mater of the brain or spinal cord), which is not caused by trauma.

Definition of stroke caused by cerebral venous thrombosis

Infarction or hemorrhage in the brain, spinal cord, or retina because of thrombosis of a cerebral venous structure. Symptoms or signs caused by reversible edema without infarction or hemorrhage do not qualify as stroke.

Definition of stroke, not otherwise specified

An episode of acute neurological dysfunction presumed to be caused by ischemia or hemorrhage, persisting ≥24 hours or until death, but without sufficient evidence to be classified as one of the above.

Epidemiology and global burden

In the most recent estimate for the global burden of the cerebrovascular stroke in 2013 just released February 2017, it was addressed that stroke is the second most common cause of deaths (11.8% of all deaths) worldwide, after ischemic heart disease (14.8% of all deaths), and the third most common cause of disability (4.5% of DALYs from all cause) after ischemic heart disease (6.1%), with nearly estimate of 6.5 million deaths per year. This is accompanied by incidence of new stroke cases worldwide of 10.3 million new cases every year ***(Abd-Allah and Moustafa, 2014).***

Comparing the state of stroke between 1990 and 2013, GBD showed an important discriminative issue between the changed trend of mortality and disability rates and the corresponding absolute number change as measured by new strokes. As the stroke mortality and DALYs rates have declined (measured per 100000 person) from 142 and 2431, respectively, in 1990 to 110 and 1807, in 2013 and at the same time the absolute number of people who died from stroke,