شبكة المعلومات الجامعية

التوثيق الإلكتروني والميكروفيلم

بسم الله الرحمن الرحيم

HANAA ALY

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جامعة عين شمس

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قسم

نقسم بالله العظيم أن المادة التي تم توثيقها وتسجيلها
علي هذه الأقراص المدمجة قد أعدت دون أية تغيرات

يجب أن

تحفظ هذه الأقراص المدمجة بعيدا عن الغبار

HANAA ALY

INTRODUCTION

Stroke is the third most leading cause of death worldwide after coronary heart disease and cancer especially ischemic infarcts which comprise one of the most common devastating disorders ***(Lozano et al., 2010).***

Ischemic stroke is characterized by sudden loss of blood circulation to an area of the brain, resulting in a corresponding loss of neurologic function. Acute ischemic stroke is caused by thrombotic or embolic occlusion of a cerebral artery and is more common than hemorrhagic stroke ***(Edward et al., 2018).***

The association of hyperglycemia and brain injury had already been described by **Claude Bernard in 1849**, for a longtime, hyperglycemia was understood only as an epiphenomenon due to the stress of an acute injury. Its impact on the neurological recovery was ignored for a long time and the increase in blood glucose level was understood as an adaptive response to provide glucose for an exclusive glucose consuming tissue ***(Hamilton et al., 1995).***

Hyperglycemia is common during acute ischemic stroke. Several studies have shown admission blood glucose is elevated in >40% of patients with acute ischemic stroke, most commonly among patients with a history of diabetes mellitus ***(Gentile et al., 2006).***

Scales that measure neurological deficits or specific body functions can be used especially well for triage and to guide acute treatment decisions. The NIHSS, for example, is a valuable tool for initial assessments of patients with stroke in emergency departments, hospitals, or in the pre-hospital setting and is predictive of subsequent resource use and long-term outcome ***(Schlegel et al., 2003).***

The role of clinicians in stroke management is not confined to treatment and the confidence of the patient and family can be greatly enhanced by the ability to offer an accurate prognosis. Reliable prognosis allows better planning for supportive care, more accurate information to be given to relatives and resources to be allocated in a more efficient way ***(Liu et al., 2007).***

The question is whether it is possible to determine a patient's prognosis based on the data available during an initial hospitalization for stroke. Because of the complex nature of stroke, many variables may play a role in determining prognosis. Among these variables, hyperglycemia is of special importance ***(Flint and Smith, 2004).***

Aim of the Work

The aim of the work was to study the glycemic status after acute ischemic stroke and to assess the influence of glycemic status in the stroke outcome.

Chapter 1

ACUTE STROKE

Definition

The WHO has defined stroke as a clinical syndrome characterized by rapidly developing symptoms and signs of focal, and at times global (for patients in coma), loss of cerebral function, with symptoms lasting more than 24 hours or leading to death, with no apparent cause other than that of vascular origin. The definition excludes transient ischemic attack (TIA), which is defined as rapidly developed clinical signs of focal or global disturbance of cerebral function lasting less than 24 hours, with no apparent non-vascular cause ***(Hatano et al., 2009)***

With more widespread use of magnetic resonance with diffusion weighted imaging (MRI with DWI) for the brain, up to one third of patients with symptoms lasting ≤ 24 hours have been found to have an infarction. This has led to a new tissue-based definition of TIA: a transient episode of neurological dysfunction caused by focal brain, spinal cord, or retinal ischemia, without acute infarction ***(Easton et al., 2009).***

The proposed new definition for TIA has the problem that brain imaging does not correlate particularly well with pathological infarction: Computed Tomography of the brain (CT-Brain) may be normal in clinically definite stroke, silent

infarction may occur and imaging sensitivity is highly dependent on both imaging method and area of the brain being examined ***(Pendlebury et al., 2009).***

Risk factors

Important but unmodifiable risk factors for stroke include age, ethnicity, sex, family history, and genetics:

1. Older age, particularly age >80 years ***(Grysiewicz et al., 2008)***
2. Race and ethnicity, with risk higher for blacks than for whites ***(Howard et al., 2013).***
3. Sex, with risk higher at most ages for men compared with women, except for ages 35 to 44 years and >85 years, where women have a similar or higher risk than men ***(Bushnell et al., 2014).***
4. Family history and genetic disorders, with a higher risk for monozygotic twins and those with genetic disorders such as sickle cell disease or cerebral autosomal dominant arteriopathy with subcortical infarctions and leukoencephalopathy ***(Meschia et al., 2011).***

The major modifiable risk factors for stroke are the following ***(Harmsen et al., 2006):***

1. Hypertension
2. Diabetes mellitus

3. Smoking
4. Hyperlipidemia
5. Physical inactivity

Two important mechanisms of ischemic stroke are amenable to effective secondary prevention: ***(O'Donnell et al., 2016).***

1. Atrial fibrillation
2. Carotid artery stenosis

The risk of stroke is particularly increased in patients with two or more risk factors. Control of atherosclerotic risk factors is important for the primary and secondary prevention of stroke. Control of risk factors also reduces the risk of coronary events, a common comorbidity in patients with cerebrovascular disease ***(Bushnell et al., 2014).***

Types:

Acute ischemic stroke subtypes are often classified, based upon the underlying cause, into the following categories: ***(Chung et al., 2014).***

1. Large artery atherosclerosis
2. Cardioembolism
3. Small vessel occlusion
4. Stroke of other, unusual, determined etiology
5. Stroke of undetermined etiology

Determination of the type of stroke can influence treatment to be used. Most strokes in the United States, approximately 87%, are ischemic. The majority, approximately 60%, of all new ischemic strokes are either large-artery atherosclerosis, cardioembolic, or small vessel diseases ***(Grysiewicz et al., 2008).***

Pathophysiology of Acute Ischemic Stroke:

Ischemic strokes are due to a reduction or complete blockage of blood flow. This reduction can be due to decreased systemic perfusion (e.g., low blood pressure, heart failure, or loss of blood), severe stenosis, or occlusion of a blood vessel ***(Caplan, 2009).***

Decreased systemic perfusion due to systemic hypotension may produce generalized ischemia to the brain. This is most critical in the border zone (or watershed) areas, which are territories that occupy the boundary region of two adjacent arterial supply zones. The ischemia caused by hypotension may be asymmetric due to preexisting vascular lesions. Areas of the brain commonly affected include the hippocampal pyramidal cells, cerebellar Purkinje cells, and cortical laminar cells ***(Barnett et al., 1998).***

Thrombosis refers to obstruction of a blood vessel due to a localized occlusive process within a blood vessel. The obstruction may occur acutely or gradually. In many cases,

underlying pathology such as atherosclerosis may cause narrowing of the diseased vessel. This may lead to restriction of blood flow gradually, or in some cases, platelets may adhere to the atherosclerotic plaque forming a clot leading to acute occlusion of the vessel ***(Garcia et al., 1998).***

Atherosclerosis usually affects larger extracranial and intracranial vessels. In some cases, acute occlusion of a vessel unaffected by atherosclerosis may occur because of a hypercoagulable state ***(Caplan, 2009).***

Embolism refers to clot or other material formed elsewhere within the vascular system that travels from the site of formation and lodges in distal vessels causing blockage of those vessels and ischemia. The heart is a common source of this material, although other arteries may also be sources of this embolic material (artery to artery embolism). In the heart, clots may form on valves or chambers. Tumors, venous clots, septic emboli, air, and fat can also embolize and cause stroke. Embolic strokes tend to be cortical and more likely to undergo hemorrhagic transformation, probably due to vessel damage caused by the embolus ***(Caplan et al., 2006).***

Lacunar infarction occurs as a result of small vessel disease. Smaller penetrating vessels are more commonly affected by chronic hypertension leading to hyperplasia of the tunica media of these vessels and deposition of fibrinoid material leading to lumen narrowing and occlusion. Lacunar

strokes can occur anywhere in the brain but are typically seen in subcortical areas. Atheroma can also encroach on the orifices of smaller vessels leading to occlusion and stroke ***(Ringelstein and Zunker, 1998)***.

Non-atherosclerotic abnormalities of the cerebral vasculature, whether inherited or acquired, predispose to ischemic stroke at all ages, but particularly in younger adults and children. These can be divided into non-inflammatory and inflammatory etiologies. The major nonatherosclerotic vasculopathies associated with ischemic stroke are: ***(Furie et al., 2007)***

1. Arterial dissection.
2. Fibromuscular dysplasia.
3. Vasculitis.
4. Moyamoya disease.
5. Sickle cell disease arteriopathy.
6. Focal cerebral arteriopathy of childhood.

National Institutes of Health Stroke Scale (NIHSS) (Table 1)

The NIHSS is a 15-item impairment scale, which provides a quantitative measure of key components of a standard neurological examination ***(Appelros and Terent, 2004).***

The scale assesses level of consciousness, extraocular movements, visual fields, facial muscle function, extremity strength, sensory function, coordination (ataxia), language (aphasia), speech (dysarthria), and hemiinattention (neglect) ***(Lyden et al., 2001).***

An additional item that measures distal motor function has been used in a few drug trials, but is not widely used in ongoing research or in clinical practice ***(Kasner, 2006).***

The NIHSS was designed to assess differences in interventions in clinical trials, although its use is increasing in patient care as an initial assessment tool and in planning post-acute care disposition ***(Schlegel et al., 2004).***

Table (1): National Institute of Health Stroke Scale (Maximum = 42), (Minimum = Zero) ***(Adams et al., 2003)***

Response	(Score)	Response	(Score)
Level of consciousness		Motor arm (left and right) no drift	(0)
Alert	(0)	Drift before 10 seconds	(1)
Drowsy	(1)	Falls before 10 seconds	(2)
Stupors	(2)	No effort against gravity	(3)
Coma	(3)	No movement	
Response to level of consciousness questions:		**Motor leg (left and right)**	
		No drift	(0)
		Drift before 10 seconds	(1)
Answers both correctly	(0)	Falls before 10 seconds	(2)
Answers one correctly	(1)	No effort against gravity	(3)
Answers neither correctly	(2)	No movement	(4)
Response to level of consciousness commands		**Ataxia**	
		Absent	(0)
Obeys both correctly	(0)	One limb	(1)
Obeys one correctly	(1)	Two limbs	(2)
Obeys neither correctly	(2)		
Pupillary response		**Sensory**	
Both reactive	(0)	Normal	(0)
One reactive	(1)	Mild	(1)
Neither reactive	(2)	Severe loss	(2)
Gaze		**Language**	
Normal	(0)	Normal	(0)
Partial gaze palsy	(1)	Mild aphasia	(1)
Total gaze palsy	(2)	Severe aphasia	(2)
		Mute or global aphasia	(3)
Visual fields		**Facial palsy**	
No visual loss	(0)	Normal	(0)
Partial hemianopsia	(1)	Minor paralysis	(1)
Complete hemianopsia	(2)	Partial paralysis complete paralysis	(2)
Bilateral hemianopsia	(3)		(3)
Dysarthria		**Extinction/inattention**	
Normal	(0)	Normal	(0)
Mild	(1)	Mild	(1)
Severe	(2)	Severe	(2)

Role in clinical practice and research

Neurologists spend several years in training to learn the fine points of the neurological examination. However, the first clinician to assess a patient with stroke, typically in an emergency department, is seldom a neurologist. Non-neurologist physicians, medical students, nurses, and other health practitioners often have difficulty mastering and performing the neurological examination, and as a result, their attempts to perform a neurological examination on a patient with acute stroke can be prolonged and unfocused. The NIHSS offers a more expeditious approach since it can be effectively implemented by all types of health-care providers, with excellent reliability and validity, after only a few hours of training ***(Goldstein and Samsa, 1997).***

Clinicians can then use the scale for initial evaluation, providing quick and accurate assessments of stroke-related deficits, which are easy to communicate with other clinicians, ultimately saving valuable time in triage and treatment of the patient. Video training and certification of NIHSS administration with digital video are available on DVD (digital video disc) or free online and seem highly effective ***(Lyden et al., 2005).***

Widespread training offers an advantage in multicenter clinical trial logistics, where standardized use by all