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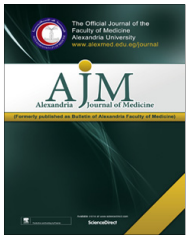

Evaluation of the role of ischemia modified albumin as a new biochemical marker for differentiation between ischemic and hemorrhagic stroke

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KEYWORDS

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Abstract *Objective:* To evaluate the role of the detection of ischemia modified albumin (IMA) level in the differentiation between ischemic and hemorrhagic cerebrovascular stroke.
Methods: Sixty elderly persons classified into three groups, 25 patients diagnosed with cerebral infarction, 15 patients diagnosed with cerebral hemorrhage, and 20 elderly healthy persons with matched age as control were enrolled in the study. IMA was measured using the available chemical method and computerized tomography (CT) was done for diagnosis of brain lesions.
Results: IMA was significantly higher in the patient group than in the control group. There was a positive significant correlation between age, albumin with IMA, ($P = 0.000$ and 0.037 respectively). However there was no statistical significant difference between sex and diagnosis cross tabulation (0.51). It was found that, IMA was statistically higher in the infarction group than the hemorrhage group ($P = 0.000$) and IMD index was statistically higher in the infarction group than the hemorrhage group ($P = 0.013$).
Conclusion: Our investigation in elderly patients suggests that IMA assay is a sensitive marker for early detection of ischemic and hemorrhagic stroke.
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1. Introduction

A stroke, previously known medically as a cerebrovascular accident (CVA) is the rabid loss of brain function(s) either focal or global due to disturbance in the blood supply to the brain, with symptoms lasting 24 h or longer or leading to death with no apparent cause other than of vascular origin.[1] The

Abbreviations: IMA, ischemia modified albumin; CT, computerized tomography; CVA, cerebrovascular accident; HGE, hemorrhage

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24 h limit differentiates stroke from transient ischemic attack, which is a related syndrome of stroke symptoms that resolve completely within 24 h.[2]

The incidence of stroke increases exponentially from 30 years of age, and etiology varies by age.[3] Advanced age is one of the most significant risk factors. 95% of strokes occur in people aged 45 years and older, and two-thirds of strokes occur in those over the age of 65 years. As the patient age increases the risk of dying increases.[4] Family members may have a genetic tendency for stroke or share a lifestyle that contributes to stroke.[5]

Stroke can be classified into two major categories: ischemic and hemorrhagic. About 87% of strokes are caused by ischemia and the remainder by hemorrhage. Some hemorrhages develop inside areas of ischemia (hemorrhagic transformation).[6] In ischemic stroke, blood supply to part of the brain is decreased, leading to dysfunction of the brain tissue in that area. There are four main reasons why this might happen: thrombosis (obstruction of a blood vessel by a blood clot formed locally), embolism (obstruction due to embolus formed elsewhere in the body),[7] systemic hypo perfusion (general decrease in the blood supply e.g. in shock) (global ischemia),[8] and venous thrombosis. Stroke without an obvious explanation is termed cryptogenic stroke and this constitutes 30–40% of all ischemic strokes.[9]

As regards hemorrhagic stroke, it is caused by accumulation of blood anywhere within the skull vault. A distinction is made between intra-axial hemorrhage (blood inside the brain either intra-parenchymal or intra-ventricular hemorrhages) and extra-axial hemorrhage (blood inside the skull but outside the brain e.g. epidural hematoma, subdural hematoma, and subarachnoid hemorrhage).[10]

A growing body of investigation supporting the potential of ischemia modified albumin (IMA) as a marker of ischemia is now available. Human serum albumin (HSA) is the most abundant protein in the blood with a mean concentration of 0.63 mmol/L. It is synthesized in the liver and has a half life of about 19 days. HSA has a unique structure and amino acid sequence which is specific to humans at its amino terminus (N-terminus).[11] Previous studies have shown the N-terminus of HSA to be the primary binding site for the transitional metals cobalt and copper. The HSA metal binding site is particularly susceptible to biochemical changes during ischemia compared to albumin from other species.[12]

The precise mechanisms for production of IMA during ischemia are not known, but have localized modification in the amino terminal of HSA during ischemia which leads to reduction in cobalt binding to this modified N-terminus.[13] Many reports indicate that the factors involved in ischemia that can induce these in vivo changes to albumin may include: acidosis, free radical damage, membrane energy dependent sodium and calcium pump disruption, reduced oxygen tension and free iron and copper ion exposure.[14] These conditions necessary for altering the metal binding site of HSA are known to occur within minutes of the onset of ischemia, and their effect on albumin could be detectable up to 6 h after the ischemic event.[15]

In the current study we tried to evaluate the detection of ischemia modified albumin level in the differentiation between ischemic and hemorrhagic cerebrovascular stroke.

2. Methods

From May 2011 to January 2013, this study was conducted in the Internal Medicine Department, Alexandria University Hospital, Egypt; after being approved from the local Research Ethics Committee, and informed consent was obtained from all participants. We studied 40 elderly persons recruited from the emergency department with a mean age of 49.0 ± 7.27 (range 40–75) years with cerebrovascular stroke (Group I included 25 patients with cerebral infarction and Group II included 15 patients with cerebral hemorrhage). In addition, 20 apparently healthy elderly persons (Group III) matched for age were included as controls. The exclusion criteria included cardiac diseases, cancer, infections, end stage renal disease, liver disease, uncontrolled diabetes mellitus and history of treatment of thyroid disease. All subjects were subjected to:

1. Full history taking.
2. Complete clinical examination especially for signs of cerebrovascular stroke (paresis, paralysis, loss of sensation, abnormal speech).
3. Radiological investigations: Computerized tomography (CT) for diagnosis of brain lesions.
4. Blood samples were collected by venipuncture tubes within two hours of arrival. These samples were sent to the laboratory and processed for: routine laboratory tests (liver function tests, renal function). Blood samples were collected before any heparin/thrombolytic treatment is started. Patients received routine institutional care according to their diagnosis blinded to the IMA results. IMA was measured using spectrophotometric albumin cobalt binding assay. The assay is based on the fact that ischemia causes changes in human serum albumin that are demonstrated by reduced exogenous cobalt binding. The concentration of IMA can be determined by addition of a known amount of exogenous cobalt ($CoCl_2$) to a serum specimen and measurement of unbound cobalt using a colorimetric assay after adding a coloring substance (dithiothreitol) which binds any excess (unbound) cobalt. An inverse relationship thus exits between the amount of albumin bound cobalt and the intensity of the color formation. All reactions were carried out at room temperature and in duplicate.

2.1. Test procedure

Addition of 200 μL of the patient serum, 50 μL of $CoCl_2$, followed by good mixing and incubation for 10 min were carried out. Then 50 μL of DTT working solution (1.5 g/L DTT solution) was added and mixed. After a period of 2 min incubation, 1.0 ml of 9.0 g/L solution of NaCl was added. The absorbance of this assay mixture was read at 470 nm using the 5010 Spectrophotometer (after reading the blank). Each sample was read in duplicate, with average absorbance taken and recorded in absorbance units (ABSU0) using PD-3035 Apel Japan. As regards result interpretation: cases with absorbance greater than 0.400 ABSU were considered positive for IMA, while cases with absorbance less than 0.400 ABSU were considered negative for IMA. IMA index was calculated by using

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