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PREDICTIVE FACTORS OF HEMORRHAGIC ISCHEMIC STROKE DETECTED BY CT AND MRI GRADIENT-ECHO T2 WEIGHTED IMAGE (GRE)

By

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ABSTRACT

Background: Risk of hemorrhage is significantly increased in large infarcts, with mass effect supporting the importance of edema for tissue damage and the deleterious effect of late reperfusion when edema resolves. In some instances, the rupture of the vascular wall secondary to ischemia-induced endothelial necrosis might cause an intra-infarct hematoma. Vascular rupture can explain very early hemorrhagic infarcts and early intra infarct hematoma (between 6 and 18 hours after stroke), whereas hemorrhagic transformation usually develops within 48 hours to 2 weeks. Intra cerebral hemorrhage (ICH) occurs as a result of bleeding from an arterial source directly into the brain parenchyma and accounts for 5–15% of all strokes.

Objective: The aim of this study was to determine the predictive factors of hemorrhagic transformation in patient with acute ischemic stroke detected by brain CT and MRI gradient echo T2 weighted image (GRE).

Patients and Methods: This was a cross sectional randomized prospective study carried out on 60 patients (32 males and 28 females), admitted at Neurology Department and Stroke Unit of Mostafa Kamel Military Hospital, within the first 24 hours of their symptoms. The study was carried out during the period from 1st of January 2018 till the end of December 2018.

Results: The results of this study showed that the size of infarction was small in 27 (45.0%) patients and large in 33 (55.0%) patients, the micro bleeds were found in 32 (53.3%) of the studied patients, and the hemorrhagic transformation was found in 11 (18.3%) of the patients. The results showed that there was a significant relation between micro bleeds and hypertension, diabetes mellitus, dyslipidemia, past history of stroke, cardiac disease and hemorrhagic transformation. The small size infarction showed a higher percent of micro bleeds. Regarding the hemorrhagic transformation, the risk factors were the size of infarction, anticoagulant use, hypertension, diabetic and cardiac diseases.

Conclusion: The most predictive factors of hemorrhagic transformation was the size of infarction (higher in large), micro bleed, anticoagulant use, hypertension, dyslipidemia and cardiac disease, while the most predictive factors of micro bleeds were the size of infarction (higher in small), age, hypertension, dyslipidemia, past history of stroke, diabetes mellitus, and cardiac disease.

INTRODUCTION

Stroke is defined as either symptoms lasting more than 24 hours or imaging of an acute clinically relevant brain lesion in patient with rapidly vanishing symptoms. Patients with symptoms lasting less than 24 hours but with infarction imaged by MRI have been reclassified as having stroke instead of transient ischemic attack *(Sacco et al., 2013).*

Advances in neuroimaging technology have resulted in a virtual explosion in the amount of pathologic information that can be obtained in the clinical stroke setting. This neuroimaging revolution has led to a much better understanding of cerebrovascular and tissue pathology, creating a wide array of opportunities for acute treatment and secondary prevention *(Malhotra and Liebeskind., 2017).*

Cerebral hemorrhage is one of the major complications of anti-coagulation and antiplatelet therapies instituted for prevention of ischemic stroke. The anticoagulant- related risk factors for cerebral bleeding were leukoaraiosis and age older than 65 years, in addition to the intensity of anticoagulation *(Saji et al., 2016).*

Risk of hemorrhage is significantly increased in large infarcts with mass effect supporting the importance of edema for tissue damage and the deleterious effect of late reperfusion when edema resolves. In some instances, the rupture of the vascular wall secondary to ischemia-induced endothelial necrosis might cause an intra-infarct hematoma. Vascular rupture can explain very early hemorrhagic infarcts and early intra infarct hematoma (between 6 and 18 hours after stroke), whereas hemorrhagic transformation usually develops within 48 hours to 2 weeks. Intra cerebral hemorrhage (ICH) occurs as a result of bleeding from an arterial source directly into the brain parenchyma and accounts for 5–15% of all strokes *(Campbell et al., 2019).*

It is of particular importance to extract such patients who are prone to bleeding complications after anticoagulant or antiplatelet therapy. The detection of micro-bleeds on T2 weighted MRI might be a direct indicator for such a risk *(Lane and Lip., 2010).* Asymptomatic micro-bleeds shown by gradient echo T2-weighted MRI are associated with small artery disease especially with intracerebral hemorrhage *(Arsava et al., 2013).*

The micro bleeds are frequently detected in patients with cerebral infarction as well as in patients with intracerebral hemorrhage and even in small number of healthy individuals without stroke episodes *(Charidimou et al., 2012).* Gradient- echo T2-weighted MRI is uniquely sensitive to detect silent, old hemosiderin, but the clinical significance of such "micro bleeds" remains to be determined. Micro bleeds were defined as small, silent foci of signal loss on T2 weighted MRI other than the principle lesion(s) responsible for stroke episode *(Ovbiagele et al., 2013).*

Recognizing bleeding prone microangiopathy in stroke patients is of extreme clinical significance when treating hypertensive patients with or without episodes of intracerebral hemorrhage *(Shinohara et al., 2012).*

There is a pathological confirmation that the micro bleeds on Gradient- echo T2-weighted MRI represent hemosiderin deposits. The deposits may be a result of minor blood leakage through damaged blood vessels in addition to frank minor hemorrhage. What-ever the source, they may remain detectable for years. The micro bleeds are barely detectable with T2-weighted spin echo MRI and are not visualized with other conventional scan *(Haussen et al., 2012).*

The aim of this study was to determine the predictive factors of hemorrhagic transformation in patient with acute ischemic stroke detected by CT, MRI gradient echo T2 weighted image (GRE).

PATIENTS AND METHODS

This was a cross sectional randomized prospective study was carried out on 60 patients (32 males and 28 females), admitted at Neurology Department and Stroke Unit of Mostafa Kamel Military Hospital, with the first 24 hours of their symptoms. The study was carried out during the period from 1st. of January 2018 till the end of December 2018.

Inclusion criteria:

Patients within first time ischemic stroke, within the first 24 hours of their symptoms according WHO definition 2017 *(WHO 2017).*

Exclusion criteria:

1. Cerebral hemorrhage (subdural, extradural, intracerebral, subarachnoid hemorrhage) detect by initial CT brain.
2. Brain tumor and malignancy detect by initial CT brain.
3. Patients with contraindication for MRI, e.g. Pace maker.

All patients were subjected to:

A. Clinical assessment:

1. Stroke onset.
2. Full general and neurological history, and examination including demographic data: age, sex, occupation, family history of stroke, and history of stroke risk factors (as smoking, hypertension, diabetes mellitus, hyperchole-sterolemia, atrial fibrillation, and ischemic heart disease.
3. History of medications especially anticoagulants or anti platelets prior to the onset of stroke.
4. General and neurological examination.

B. Radiological assessment:

1. Computed tomography (CT) at admission for diagnosis and to exclude hemorrhage.
2. Magnetic resonance imaging (MRI) brain gradient echo T2 weighted image (GRE), within 24 hours from stroke onset.
3. Follow up computed tomography after seven days from onset or at any time of appearance of new symptoms, to detected hemorrhagic transformation.

MRI and CT recording were analyzed by radiologist to assess hemorrhagic transformation.

C. Laboratory assessment:

Laboratory investigations included complete blood picture, liver and renal function test, ESR, lipid profile, serum uric acid, PT, PTT and INR.

Ethical considerations:

Patients were consented by a written informed consent by patients or their close relatives before enrollment in this study.

This study was approved by ethical committee of Al-Azhar, Faculty of medicine (Cairo).

Statistical analysis:

The Data ware collected and entered into the personal computer. Statistical analysis was done using Statistical Package for Social Sciences (SPSS/version 21) software.

Arthematic mean, standard deviation, for categorized parameters, Chai square test was used, while for numerical data t-test was used to compare two groups. The level of significance was 0.05.

RESULTS

This study was carried out on 60 patients (32 males and 28 females) recruited from Neurology Department of Mostafa Kamel Military Hospital, within the first 24 hours of their symptoms, during the period from beginning of January 2018 till the end of December 2018.

The age of our patients ranged from 45-72 years, with a mean age 57.5±8.32 years. Males represented 53.3% of the patients, while females represented 46.7%. There were 25 (42.2%) patients had a positive family history of stroke, 14 (23.3%) patients had a past history of stroke. As regarding the comorbidity and risk factors, there were 25 (41.7%) smoker patients, hypertension was found in 30 (50.0%), patients, 20 (33.3%) were diabetic, dyslipidemia was found in 30 (50.0%), and cardiac disease in 15 (25.0%) patients.

The size of infarction was small in size in 27 (45.0%) patients, while the other 33 (55.0%) patients had a large infarction size.

Lacunar infarction was defined as a small deep lesion (usually 5 to15 mm in diameter) with high signal intensity on T2-weighted images, low signal intensity on T1-weighted, and FLAIR images with perilesional halo on FLAIR images, ruling out enlarged perivascular spaces and patchy leukoaraiosis *(Gyanwali et al., 2019).*

Of the 60 patients, 32 (53.3%) had micro bleeds. Regarding hemorrhagic transformation of the patients, it was found that 11 (18.3%) had a hemorrhagic transformation **(Table 1)**.

Table (1): Microbleeds and hemorrhagic transformation of the studied patients group

Parameters \ Counts	Number	Percent
Radiological assessment (microbleeds)		
Yes	32	53.3
No	28	46.7
Hemorrhagic transformation		
Yes	11	18.3
No	49	81.7

It was found that there was a significant relation between hemorrhagic transformation and size of infarction, the larger the size of infarction the higher percentage of hemorrhagic transformation ($p<0.05$). Also, there was a significant relation between hemorrhagic transformation and anticoagulant used.

There was a significant relation between hypertension and hemorrhagic transformation. The majority of hypertension patients had a hemorrhagic transformation ($p<0.05$). There was a significant relation between hemorrhagic transformation and dyslipidemia. The patients with dyslipidemia had significantly a higher percentage of hemorrhagic transformation ($p <0.05$). It was found that there was a significant association between cardiac patients and positive hemorrhagic transformation ($p <0.05$). Also, there was no significant relation between hemorrhagic transformation and DM, ($p>0.05$), **(Table 2)**.

Table (2): Effect of different risk factors on hemorrhagic transformation

Variables \ Hemorrhagic transformation	Negative "n=49"		Positive "n=11"		X^2 p
	No.	%	No.	%	
Size of infarction					
Small	26	53.1	1	9.1	7.017
Large	23	46.9	10	90.9	0.008*
Anticoagulant					
No	48	98.0	6	54.5	18.81
Yes	1	2.0	5	45.5	0.0001*
Hypertension					
No	28	57.1	2	18.2	5.45
Yes	21	42.9	9	81.8	0.019*
Diabetes mellitus					
No	33	67.3	7	63.6	0.056
Yes	16	32.7	4	36.4	0.813
Dyslipidemia					
No	27	55.1	3	27.3	2.78
Yes	22	44.9	8	72.7	0.095
Cardiac					
No	44	89.8	1	9.1	32.45
Yes	5	10.2	10	90.9	0.0001*

It was found that there was a significant relation between micro bleeds and hemorrhagic transformation. The majority of negative micro bleeds were negative hemorrhagic transformation ($p <0.05$). Also, it was found that there was a significant relation between micro bleeds and size of infarction. The smaller size of infarction the higher the presence of micro bleeds related with the positive micro bleeds ($p <0.05$).

There was a significant relation between micro bleeds and dyslipidemia. The patients with dyslipidemia significantly had micro bleeds micro bleeds ($p <0.05$). There was a significant association between cardiac patients and positive micro bleeds ($p <0.05$). Also, there was a significant relation between past history of stroke and incidence of micro bleeds, hypertension and micro bleeds, the majority of positive micro bleeds had a hypertension ($p <0.05$).

No significant relation between anticoagulant and micro bleeds, between smoking and micro bleeds, and between micro bleeds and DM **(Table 3)**.

Table (3): The association between different risk factors and micro bleeds

Variables \ Microbleeds	Positive "n=32"		Negative "n=28"		X^2 p
	No.	%	No.	%	
Hemorrhagic transformation					
Negative	21	65.6	28	100	9.20
Positive	11	34.4	0	0.0	0.0024*
Size of infarction					
Small	20	62.5	7	25.0	8.48
Large	12	37.5	21	75.0	0.004*
Anticoagulant					
No	27	84.4	27	96.4	2.41
Yes	5	15.6	1	3.6	0.120
Smoking					
No	17	53.1	18	64.3	0.768
Yes	15	46.9	10	35.7	0.381
Past history of stroke					
No	18	56.3	28	100.0	13.32
Yes	14	43.7	0	0.0	0.0002*
Hypertension					
No	6	18.8	24	85.7	26.79
Yes	26	81.2	4	14.3	0.0001*
Diabetes mellitus					
No	20	62.5	20	71.4	0.535
Yes	12	37.5	8	28.6	0.464
Dyslipidemia					
No	7	21.9	23	82.1	21.69
Yes	25	78.1	5	17.9	0.001*
Cardiac					
No	19	59.4	26	92.9	8.93
Yes	13	40.6	2	7.1	0.0028*

DISCUSSION

The results of our study showed that the size of infarction was small in 45.0% patients, and large in 55.0% of patients. The radiological assessment of the studied patients group was found in 53.3% patients had micro bleeds.

In agreement with our results, *Lee et al (2018)* found that the size of infarction ranged from 2-10 ml. The small and large infarction was divided in the studied cases without significant difference.

Also, in our study, the hemorrhagic transformation of the patients was found that 18.3% patients had a hemorrhagic transformation. In agreement with our study, *Li et al (2017)* found that the incidence of hemorrhagic transformation among cerebral micro bleeds was 13.8%

In our study, the results showed that there was a significant relation between micro bleeds and hemorrhagic transformation. The majority of negative micro bleeds was negative hemorrhagic transformation. Also, there was a positive relation between micro bleeds and size of infarction, the small size related with the positive micro bleeds. No significant relation between past history of sroke and micro bleeds.

These results were in agreement with *Lovelock et al (2010)* and *Charidimou et al (2012* who found that micro bleeds have been associated with stroke history in patients taking anticoagulant medications in cross-sectional case-control, case-case comparisons and small case series studies, suggesting that there may be a stronger predictor of anticoagulant-associated ICH than leukoaraiosis.

In this study, there was a significant relation between hypertension and micro bleeds. The majority of hypertensive patients had micro bleeds.

Lioutas et al. (2017) showed that hypertension was frequent in intracerebral hemorrhage and lacunar infarction. Therefore, the presence of multiple micro bleeds suggests that the microangiopathy has reached an advanced stage, in which the blood vessels are prone to bleeding. Another explanation may be cerebral amyloid angiopathy, which is also a small artery disease in the non-hypertensive elderly, presenting lobar hemorrhage with the coexistence of lacunar infarcts and white matter lesions.

There was a significant relation between micro bleeds and DM. From 20 diabetic patients, 14 patients were positive micro bleeds.

Several studies have reported that CMBs are significantly associated with DM, atherosclerosis, lacunar infarct, and stroke. In addition, it may contribute to cognitive impairment. Therefore, CMBs serve as an independent risk factor for the progression of MCI to dementia *(Hong et al., 2017)*. The clinical symptoms of cerebral hemorrhage are primarily dependent on the location and number of CMBs. The progression of vascular cognitive impairment may be accelerated with an increase in the number of CMBs *(Nardone et al., 2011)*. With the emergence of cerebral hemorrhage, the severity of cognitive impairment also increases, which suggests an accumulation effect *(Ihn et al., 2013)*.

In our study, it was found that there was a significant relation between micro bleeds and dyslipidemia, the patients with dyslipidemia was significantly had positive micro bleeds.

Cholesterol, diabetes mellitus, and smoking have also been reported as risk factors for micro bleeds, but the results differ according to the study. Some studies have shown hypercholesterolemia lowers the risk of micro bleeds, and statin therapy increases the risk of cerebral hemorrhage *(Ducrocq et al., 2013)* in meta-analysis results showed the opposite. Cystatin C, a renal function indicator, has been associated with deep and infratentorial CMB, as well as chronic kidney disease, implicating it as a risk factor for CMB *(Hackam et al., 2011* and *Oh et al., 2014)*.

Yates et al. (2014) found that the development of new micro bleeds associated with the progression of ischemic vascular brain lesions. Concurrent progressions of hemorrhagic and ischemic vascular brain lesions support the hypothesis of a common pathophysiologic pathway for these lesions.

It was found that there was a significant association between cardiac patients and positive micro bleeds ($p < 0.05$).

Evidence that micro bleeds also associate with ischemic or occlusive brain disease was found in meta-analysis of clinical studies *(Charidimou et al., 2013)*.

Naganuma et al. (2015) found that CMBs statistically significant predictors of ICH, and they have confirmed previous observations of a predominance of lobar CMBs in patients with ICH. *Haussen et al. (2012)* suggested that the presence of micro bleeds reflects a more diffuse pathologic process in the brain by showing that white matter changes surround the actual micro bleeds.

In this study, there was a significant relation between use of anticoagulant and hemorrhagic transformation. This was in agreement to Lovelock et al. (2010). Antithrombotic drug use and anticoagulant drugs increase risk of micro bleed and hemorrhagic transformation *Lovelock et al. (2010)*. They showed that the use of oral anticoagulant drug associates with an increased risk of developing new micro bleeds. More importantly, they found proof that a higher maximum INR, and fluctuations of INR during the initiation period of anticoagulant use were both associated with higher prevalence of micro bleeds. *Charidimou et al. (2011)* showed that CMBs are markers of more advanced cerebrovascular disease and severe underlying microangiopathy, and presence of leukoaraiosis is a risk factor for warfarin-related ICH, CMBs might contribute to increase the risk of warfarin-related ICH. *Ge et al. (2011)* found that, in patients with ischemic cerebrovascular disease only (i.e. IS or TIA), CMBs were more frequent in aspirin users compared with non-users especially after long exposure to aspirin (greater than 5 years) although a confounding effect of cerebrovascular disease could not be completely eliminated.

CONCLUSION

The risk factor of hemorrhagic transformation was large size of infection, hypertension, anticoagulant use and cardiac disease, while the most predictive factors of micro bleeds were the age of the patient, size of infarction, past history of stroke, hypertension, dyslipidemia, diabetes mellitus, anticoagulant use and cardiac disease.

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عوامل التنبوء بالتحول النزيفى في الاحتشاء الدماغي الحاد بواسطة الفحص بالأشعة المقطعية والرنين المغناطيسي منحدرالممال

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خلفيــة البحــث: تُعــرف الســكتة الدماغيــة علــى أنهــا إمــا أعــراض تســتمر لأكثــر مــن 24 ســاعة أو تصــوير لآفــة دماغيــة حــادة ذات صــلة ســريريًا فــي المــريض الــذي يعــاني مــن أعــراض التلاشــي الســريع. كمــا تتضــمن المرضــى الــذين يعــانون مــن أعــراض تــدوم أقــل مــن 24 ســاعة ولكــن مــع رصــد احتشــاء فــى تصــوير المــخ بــالرنين المغناطيســي بــدلاً مــن تســميتها نوبــة قصــور بالــدورة الدمويــة المخيــة. ويعــد النــزف الــدماغي أحــد المضــــاعفات الرئيســــية للعلاجــــات المضــــادة للــــتجلط والمضــــادة للصــــفيحات التــــي وضــعت للوقايــة مــن الســكتة الدماغيــة. مــن عوامــل الخطــر التــي تســاعد علــى حــدوث التحــول النزفــي المخــي النــزف المخــي الــدقيق الكامــل وارتفــاع ضــغط الــدم ومضــادات التجلط وزيادة نسبة الدهون بالدم.

الهــدف مــن البحــث: هــو تحديــد العوامــل التنبؤيــة للتحــول النزفــي فــي المرضــى الــذين يعــانون مــن الســكتة الدماغيــة الحــادة التــي تــم الكشــف عنهــا بواسطةالتصــوير المقطعــي، التصوير بالرنين المغناطيسي منحدر الممال.

المرضــى وطــرق البحــث: أجريــت الدراســه علــى 60 مريضــا (32 مــن الــذكور و28 مــن الإنـــاث)، تـــم قبـــولهم بقســـم الأعصـــاب ووحـــدة الســـكتة الدماغيـــة فـــي مستشـــفى مصـــطفى كامـــل العســـكري مـــع أول 24 ســـاعة مـــن أعراضـــهم. أجريـــت الدراســـة خـــلال الفترة من بداية يناير 2018 حتى نهاية ديسمبر 2018.

النتـــائج: أظهـــرت نتـــائج هـــذه الدراســـة أن حجـــم الاحتشـــاء كـــان صـــغيرا لـــدى 27 (45.0٪) مريضـــا و33 مريضـــا (55.0٪). تـــم العثـــور علـــى نزيـــف دقيـــق فـــي 32 (53.3٪) مـــن المرضـــى الخاضـــعين للدراســـة،ووجد التحـــول النزفـــي فـــي 11 (18.3٪) مـــن المرضـــى ، أوضـــحت النتـــائج وجـــود علاقـــة ذات دلالـــة إحصـــائية بـــين وجـــود نـــزف المـــخ الـــدقيق الكامـــل والتحـــول النزفـــي كمـــا أوضـــحت النتـــائج ارتفـــاع نســـبة وجـــود نـــزف المـــخ الـــدقيق الكامـــل والتحـــول النزفـــي لـــدى مرضـــى ارتفـــاع ضـــغط الـــدم ومـــرض البـــوال الســـكري والمرضـــي الـــذين يســـتخدمون مضـــادات الـــتجلط. كمـــا أظهـــرت الدراســـة ارتفـــاع نســـبة حـــدوث نـــزف المـــخ الـــدقيق الكامـــل والتحـــول النزفـــي للمرضى الذين كان لهم تاريخ سابق لحدوث جلطات المخ.

الاســتنتاج: يعــد تقــدم العمــر وارتفــاع ضــغط الــدم ومــرض البــوال الســكري وارتفــاع نســبة الــدهون فــي الــدم هــي اهــم عوامــل لحــدوث نــزف المــخ الــدقيق الكــامن بينمــا كبــر حجــم الجلطــة ونــزف المــخ الــدقيق الكامــل وارتفــاع ضــغط الــدم واســتخدام مضــادات التجلط هي اهم العوامل المسببة للتحول النزفي المخي.