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POSTSTROKE DEPRESSION IN RELATION TO DIFFERENT BRAIN LESIONS

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ABSTRACT

Objectives: **This study is aimed at the pathoanatomic correlates of depression in the postacute stage of patients with stroke.**

Methods: **Of a consecutive series of 47 stroke patients, with single demarcated unilateral lesions was selected. Clinical examination, neuroradiological, CT scan examination, and psychiatric assessment were performed within a 2-month period after the acute stroke. Depression was assessed with the Beck Inventory Scale (BIS) and DSM-IV-R criteria. The neuroradiological examination of all patients was performed on the same scanner, lesion location, lesion volume, and ventricle-to-brain ratio were analyzed.**

Results : **We found no significant differences in depression scores between patients with left and right hemisphere lesions and no correlation between the severity of depression and the anteriority and or the volume of lesion or brain atrophy. Major depressive disorders were only found in nine patients with left hemisphere lesions, all involving the basal ganglia, whereas none of the patients with right hemisphere stroke exhibited major depression.**

Conclusions: **Lesions in the vicinity of the left hemisphere basal ganglia tend to play a crucial role in the development of major depression after the acute stage of stroke. The pathophysiological implications of this finding are discussed.**

Key Words: stroke . brain lesion . depression . tomography .

INTRODUCTION

Depressive alterations after stroke have been a subject of widespread interest in the last decade. Although various hypotheses exist concerning the etiology of poststroke mood disorders, there is increasing evidence that poststroke depressive changes may have an organic basis due to biochemical derangement. Depressive stroke patients exhibit alterations of cortical receptor sensitivity[1] [2] and neurotransmitter metabolite concentrations in cerebrospinal fluid[3] as well as abnormalities in electrophysiological parameters (eg, shortening of rapid eye movement latency[4]). Grasso et al[5] were able to demonstrate a local decrease of cerebral blood flow in poststroke depressed patients. Interest in the relationship between lesion location and type and severity of depression has motivated a series of studies focusing on the pathoanatomic correlates of depressive disorders. Robinson and coworkers[6] [7] [8] demonstrated in a series of articles that left hemisphere (LH) lesions may be associated with a higher incidence of depression and that within the LH, severity of depression may be correlated with the distance between lesion and anterior pole of the hemisphere. Some groups were able to replicate these

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findings[9] [10] ; others found no significant correlation between lesion location and depressive alterations after stroke.[11] [12] [13] On the other hand, some data indicate a higher incidence of depression after right hemisphere (RH) lesions.[14] [15] Lesions localized in the left frontal lobe or basal ganglia seem to be more often associated with severe depressive disorders than lesions localized in other brain areas.[16] [17] [18] Other pathoanatomic parameters such as volume of lesion[19] [20] [21] [22] or cortical/subcortical atrophy[10] [23] showed no clear-cut association with type or severity of depressive disorders.

The study is aimed at the following 1: correlation of poststroke depression with well-defined pathoanatomic parameters of patients in the postacute stage of stroke…… 2: The differences between lesion location and type and severity of poststroke depressive disorders 3: The severity of depression correlate with the distance of the lesion to the anterior pole of the brain, the volume of the lesion,

SUBJECTS & METHODS

Subjects

Fourty seven (47) patients had been examined two months after stroke and selected according to the following inclusive criteria

(1) no history of psychiatric diagnosis or alcohol or drug abuse; (2) first single unilateral stroke event (no transient ischemic attack or prolonged reversible ischemic neurological deficit); and (3) no severe or consumptive concomitant disease. The main group was narrowed down to a subgroup of 47 patients who had been examined in the first 2 months after the stroke event and who fulfilled the following neuroradiological criteria: (1) all CT scans had been acquired with the same scanner and the same protocol; (2) all patients showed a single clearly demarcated lesion and no other signs of brain disease; and (3) no signs of space occupation due to midline shift or edema . All patients were initially admitted to the neurosurgical unit in AL-Hussain Hospital Most examinations were performed during inpatient treatment at the Neurological Clinic; The group consisted of 31 male and 16 female patients. Fifteen suffered from RH and 32 from LH stroke. The median age was 62 years (Table 1✦). None of the patients was treated with antidepressant medication or any drug with depression as a known side effect.

Methods

All patients were assessed with detailed neurological examinations and neuropsychological tests . Relevant demographic, psychiatric, and neuroradiological data are described in Table 1✦.

Psychiatric Examinations

All patients were examined with the Beck Invenrtory Scale (BIC [24]), which was considered an adequate instrument for the assessment of the severity of depressive disorders in brain-damaged patients because of its lower weighting of cognitive and somatic items. all patients were classified according to the *Diagnostic and Statistical Manual of Mental Disorders,* edition 4, revised (DSM-IV-R) criteria[26] [27]. In the diagnosis of a dysthymic depression we had to ignore the 2-year criterion of DSM-IVR classification. Therefore, we use the operatively defined term "minor depression," which indicates that the respective patients otherwise fulfilled the DSM-IVR criteria of a dysthymic disorder with symptoms lasting less than 2 months.

Neuroradiological Examinations

All patients were studied with the same CT scanner under standardized conditions of data acquisition The demarcated infarctions were analyzed

with respect to the topography of lesion configuration We calculated the average distances of the anterior and posterior lesion borders to the frontal pole of the brain in each slice that contained a demarcated lesion. Lesions were classified as anterior, posterior, or nonclassifiable according to the definitions of Robinson et al.[8] Furthermore, the mean distance from the anterior lesion border to the frontal pole in percentage of overall anterior-posterior distance in each slice was calculated (ANTPER). For the assessment of cortical/subcortical atrophy, we performed planimetric measurements on the original CT data and calculated the lateral ventricle-to-brain ratio (VBR) contralateral to the side of the stroke lesion (according to the method of Starkstein et al[23] (28,29,30,31) ormed on the slice that showed the greatest width of the body of the lateral ventricles. Lesion volume was calculated in percentage of forebrain volume on standardized slices.

Statistical-Analysis

Data analysis was performed by nonparametric procedures with the use of rank correlation coefficients

RESULTS

Six patients (12.7%) showed a primary or secondary hemorrhagic stroke event; all others showed ischemic lesions. Sixty-three percent of all patients exhibited hemiparesis and 62% sensory disturbances. A visual field defect was diagnosed in 31% of the patients, and 62% exhibited facial weakness. There were no significant group differences between patients with RH and LH lesions with respect to motor or sensory deficits or visual field defects. Eighty-four percent of the patients with LH lesions showed aphasia, whereas none of the patients with RH stroke was aphasic. performed 1 month after aphasia onset, 15.6% of the LH group exhibited amnestic or Wernicke's aphasia, 25% exhibited Broca's aphasia, 18.8% exhibited global aphasia, and 9.4% demonstrated a nonclassifiable aphasic syndrome. There was no significant effect between groups in demographic variables such as age, education, or time since stroke.32

Psychiatric-Findings

A summary of the depression ratings is presented in Table 1✦. The median score on all rating scales was low, but a wide range indicated a bimodal distribution No significant difference of depression scores between LH and RH lesions. According to the DSM-IV-R criteria as modified above, 8 patients (17%) exhibited minor depression (5 [33%] RH lesions and 3 [9%] LH lesions), and 9 patients (19%) were diagnosed as major depressive; the latter all had LH lesions. To analyze the relationship between the clinical diagnosis and the severity rating of depression, which is presented in Table 2 A,B Patients with a clinical diagnosis of depression scored higher on rating scales whereas no significance could be established between the depression groups concerning the distribution of BIN scores.

Neuroradiological Findings

The median of slices that presented demarcated lesions was 4 (range, 1 to 8). There was no substantial difference in lesion topography between LH and RH lesions (Table 1✦) except for a greater temporal lobe involvement in patients with RH lesions (χ^2=7.5, *df*=1, *P*<.05). The thalamus was spared in all patients.

Associations Between Pathoanatomic Parameters and Depression

With respect to the lesion dichotomy as defined by Robinson et al,[8] we found that patients with lesions classified as anterior showed significantly higher depression scores than patients with posterior lesions in BIS rating scales (anterior lesions: BIS, median=8 [range, 0 to 16]; posterior lesions: BIS, median=11.5 [range, 8 to 15];

Separate analysis of LH and RH lesions revealed that only patients with LH anterior lesions scored significantly higher in the respective depression. The correlation analyses included all patients and the entire range of depression scores. One could argue that, particularly in the postacute stage of stroke, physical or neuropsychological symptoms related to the stroke event and symptoms produced by depression are highly confounded, and that low depression scores do not reflect any degree of depression at all. We investigated this problem and the psychometric properties of depression rating scales used in studies with stroke patients elsewhere.[33] In the present study we reanalyzed our data excluding all patients scoring on fewer than three BIS scales and presenting fewer than six positive scores. Twenty-eight patients (18 with LH and 10 with RH lesions) remained in the statistical analysis. The correlation coefficients obtained did not differ from the data reported above: We found no significant correlation between the anteriority of lesion , and BIS sum scores. As described above, we found major depressive disorders only in patients with LH lesions. In an additional step, we evaluated the lesion topography of those patients. This patient group consisted of 6 men and 3 women with a median age of 58 years (range, 43 to 79 years) and a median BIS score of 11.5 (range, 8 to 15). the lesion configurations. Seven lesions were classified as anterior, and two lesions were nonclassifiable. Lesion volume ranged from 0.22% to 18.66% (median, 2.02%).

Superimposition revealed an area of maximal overlap in the left lenticular nucleus that was included in the lesions of 6 patients with LH lesions and major depressive disorders. Patients with RH lesions and minor depression also presented an overlap in subcortical (mainly in opercular) areas but no clear-cut maximum. Accordingly, patients with lesions of the LH basal ganglia or lesions in the LH lenticulostriate or anterior choroidal artery area of vascular supply showed a significantly higher frequency of major depressive disorders and scored significantly higher on depression rating scales BIS compared with patients with lesions in all other territories of vascular supply.

DISCUSSION

This study analyzed the relationship between depressive disorders in the postacute stage of stroke with pathoanatomic data and measurements based on CT scan examinations. We found no significant differences between depression scores in LH and RH lesions and no remarkable correlation between the severity of depression and the anteriority or the volume of lesion or cortical/subcortical brain atrophy. However, there was an association between lesion location and DSM-IV-R diagnosis of major depressive disorders. Nine of 13 patients with LH lesions that involved the lentiform nucleus exhibited major depression.

One interesting result of our present study was that we found no differences in depression severity between RH and LH lesions, whereas major depressive disorders were only found in patients with LH stroke. Furthermore, we found no essential correlation between pathoanatomic measurements and depression severity ratings. Åström et al,[10] however, found no significant association between depression and brain atrophy in the postacute stage of stroke, whereas in a follow-up study 3 years later brain atrophy was demonstrated to contribute significantly to major depressive alterations. The most critical point of the study of Åström et al, however, is that brain atrophy was based on diagnoses by evidence. The results of all correlations between degree of depression and

pathoanatomic measurements in the present study indicate that the occurrence of depressive disorders after stroke reflects neither a pure "left frontal pathology" nor a simple volume effect of the brain tissue damaged. A measurement such as anteriority of lesion location does not reflect neuroanatomic data and seems somewhat superficial. However, specific lesion location may prominently determine the pathogenesis of poststroke depressive alterations. The most striking result of our present study is the finding that lesions of the LH basal ganglia seem to play a crucial role in the production of major depressive disorders in the postacute stage after stroke. This result replicates the findings of a previous study of our group[22] that reported a significant overlap of lesions in LH basal ganglia structures in acute stroke patients with aphasia and major depression. Although the configuration of the core lesion in that study differed from the lesion configuration reported in the present study, the left basal ganglia were involved in all acute aphasics with depressive disorders. Some other studies have assigned an important role to lesions of the basal ganglia in poststroke depression. Alexander and Lo Verme[34] investigated patients with subcortical aphasia and found that only 2 of 9 patients with thalamic lesions but 4 of 6 patients with putaminal lesions showed medium to severe depressive disorders. Starkstein et al[17] demonstrated that patients with a stroke in the area of the LH basal ganglia exhibit significantly higher depression scores compared with RH basal ganglia or LH and RH thalamic lesions. Moreover, patients with a pure depressive disorder after stroke more often demonstrate an involvement of the LH basal ganglia compared with patients with poststroke anxiety disorders.[18]

The role of lesions of the LH basal ganglia and/or their surrounding white matter in the pathophysiology of poststroke depression explained by The modern view of functional neuroanatomy of emotional behavior favors complex and multiple interactions of cortical and subcortical brain structures.[35] [36] Within these networks of neuronal activity not only may specific lesions of the cortex or subcortical ganglia evoke disorders of emotional behavior but also the disruption of ascending or descending neuronal pathways. Noradrenergic activation,[8] neurochemical changes of serotonergic receptors,[1] and the interruption of dopaminergic pathways ascending from the ventral tegmental area[22] have been implicated in the pathogenesis of poststroke depressive disorders At the present time there is no conclusive evidence that one neurotransmission system plays a dominant role in the development of poststroke depression. However, most of the implied neuronal pathways have to transit the basal ganglia and surrounding white matter. Therefore, lesions of the basal ganglia and their vicinity affect different neurotransmission systems and may cause serious cortical remote effects.[5] Damage of the basal ganglia and surrounding white matter may produce a significantly higher frequency of depressive disorders simply because these structures are the most important subcortical/cortical gateway. This hypothesis, however, does not explain the finding of higher frequencies of depressive alterations after LH basal ganglia lesions. A positron emission tomographic study based on 5-hydroxytryptamine receptor binding demonstrated a significantly lower (compensatory) upregulation of cortical 5-hydroxytryptamine receptors in patients with LH compared with RH lesions.[1] It s hypothesize that RH lesions lead to a greater depletion of biogenic amines that results in an ipsilateral compensatory upregulation, whereas no or only moderate upregulation occurs after LH lesions. However, presently there is little

corroborative evidence for a lateralized effect of biochemical changes after cerebral lesions, and the lateralization of depressive disorders after basal ganglia lesions still remains unclear.

In the present study we were able to demonstrate that patients with LH basal ganglia lesions are more likely to exhibit major depression than patients with RH lesions. This result shows that at least in the postacute stage after stroke, depressive alterations can be mediated by organic factors. Moreover, our data show that simple dichotomies such as anterior or posterior lesions do not have a significant value in terms of pathoanatomic considerations of poststroke mood disorders. However, other variables also influence the development of depression after stroke. Illness perception, coping styles, or psychosocial changes all can lead to psychoreactive induced depression. We have discussed these variables in a multitime and multifactor model of depression after stroke elsewhere.[43]

Limitations Of Study

Several limitations of the present study should be noted. Because we examined a highly selected population of patients, our results cannot be generalized to the population of stroke patients. We investigated only patients within a 2-month period after the acute stroke event. Because we insisted on including only patients who were examined with the same neuroradiological procedure and who showed a first unilateral single demarcated lesion, our series became small. Additionally, male patients were overrepresented in our study, and the patient group was relatively young compared with epidemiological data. Although these limitations impair the generalizability of our results, the questions we addressed in our study required a homogeneous subgroup that was carefully selected according to the described inclusion criteria.

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Table 1. Demographic, Psychiatric, and Pathoanatomic Features of the Study Population

	All N	RH Lesions N	LH Lesions N
No. of patients	47	15	32
Median age, y (range)	62 (24-79)	63 (43-75)	60 (24-79)
Sex ratio (M\F)	31:16	9:6	22:10
Median time after onset, month	1.0 month	0.75 month	1.0 month
Aphasia, No. (%)	27 (57)	0	27 (84)
Depression scores, median (range)			
BIN (Beck Inventory Scale)	15 (0-28)	18 (1-14)	14 (0-17)
DSM-IV-R diagnoses, No. (%)			
No depression	30 (64)	10 (67)*	20 (63)
"Minor depression"	8 (17)	5 (33)	3 (9)
Major depressive disorder	9 (19)	0 (0)	9 (28)
Lesion location, No. (%)			
Frontal	20 (43)	6 (40)	14 (44)
Parietal	19 (40)	5 (33)	14 (44)
Temporal	18 (38)	10 (67)	8 (25)
Occipital	5 (11)	2 (13)	3 (9)
Basal ganglia	19 (40)	6 (40)	13 (41)
Thalamus	0 (0)	0 (0)	0 (0)

RH indicates right hemisphere; LH, left hemisphere; BIC (Beck Inventory Scale) , DSM, *Diagnostic and Statistical Manual of Mental Disorders*, edition 4, revised;
*** p < 0.05**

Table 2 – A demonstration of frequency of depression in patients with post stroke

	Observe No	%
Depression	17	36
No depression	30	64
Total	47	100

P> 0.001

Table 2 – B severity of depression in poststroke

Validity	Non depressed	Frequency 30	% 64	V % 64
Depressed	Minor = (mild+moderate)	8	17	17
Patient	Sever	9	19	19

Poststroke Depression In Relation To Different Brain Lesions

	Total	**47**	**100**	**100**

According to DSM IV

: Major Depressive Episode

١) كيف كان مزاجك خلال تلك الفترة؟ وهل كان يتغير خلال اليوم الواحد؟ وكيف كان يلاحظ الاخرون؟ وكم استمر ذلك؟

٢) هل كنت تجد متعة في الفعاليات التي تمارسها يوميا؟ وهل كان لديك اهتمام بها؟

٣) هل حصل ان زاد او نقص وزنك ؟ وكم هو مقدار الزيادة او النقصان؟ وما هي الفترة التي استغرقها؟

٤) هل حصل ان زادت او قلت شهيتك للطعام؟ وهل لاحظ الآخرون ذلك؟ وكم استغرق ذلك؟

٥) هل كان هناك تغير في نومك؟ وكيف كان التغير؟ وهل كان يحصل يوميا؟ وكم استمر؟

٦) هل كان يحصل لك تميج او بطء نفسي وحركي؟ وهل لاحظ الاخرون ذلك؟ وكم استمر هذا؟

٧) هل كان لديك شعور بعدم جدوى الحياة؟ وهل كنت تشعر بالذنب؟ وما هو؟

٨) هل حصل وان قلت قدرتك على التفكير او اتخاذ القرارات؟ وهل حصل ذلك يوما؟ وكم استمر ذلك؟

٩) هل كانت تراودك افكار الموت او افكار انتحارية؟ وهل حاولت الانتحار او كانت لديك خطة للانتحار؟ وما هي؟

١٠) هل حصل وان فقدت شخص او شيء عزيز قبل حدوث هذه التغيرات؟

النموذج المختصر لقائمة بيك للاكتئاب

الاسم :

العمر :

المستوى التعليمي :

التاريخ :

بيانات اخرى :

تعليمات : في مايلي مجموعات من العبارات ، الرجاء ان تقرأ كل مجموعة على حده ، ثم قم بوضع دائرة حول رقم العبارة (0 او 1 او 2 او 3) والتي تصف حالتك خلال الاسبوع الحالي في ذلك اليوم.

تأكد من قراءة عبارات كل مجموعة قبل ان تختار واحدة منها، وتاكد من انك قد اجبت على كل المجموعات.

(1)
0 انا لا اشعر بالحزن والكآبة.
1 انا اشعر بالحزن والكآبة.
2 انا مكتئب او حزين طول الوقت ولا استطيع ان انزع نفسي من هذه الحالة.
3 انا حزين او غير سعيد لدرجة انني لا استطيع تحمل ذلك.

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(2)
0 انا لست متشائما على وجه الخصوص، او لست مثبط الهمة فيما يتعلق بالمستقبل.
1 انا اشعر بأن المستقبل غير مشجع.
2 انا اشعر بأن ليس لدي شيء اتطلع اليه في المستقبل.
3 انا اشعر بان المستقبل لا امل فيه وان الاشياء لا يمكن ان تتحسن.

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(3)
0 انا لا اشعر بأنني شخص فاشل.
1 انا اشعر بانني فشلت اكثر من الشخص المتوسط.
2 كلما اعود بذاكرتي إلى الوراء، كل ما استطيع اراه في حياتي... الكثير من الفشل.
3 انا اشعر بأنني شخص فاشل تماما (كوالد، والدة، زوج، زوجة).

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(4)
0 انا لست غير راضي.
1 انا اشعر بالملل اغلب الوقت.
2 انا لا احصل على الاشباع او الرضا من أي شيء بعد الان.
3 انا غير راضي عن كل شيء.

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(5)
0 انا لا اشعر بأنني آثم او مذنب.
1 انا اشعر بأنني رديء او لا قيمة لي اغلب الوقت.
2 انا اشعر بالذنب او الاثم تماما.

3 انا اشعر كما لو انني رديء جدا او عديم القيمة.

(6)
0 انا لا اشعر بخيبة الامل في نفسي.
1 . انا فاقد الامل في نفسي.
2 انا مشمئز من نفسي.
3 انا اكره نفسي.

(7)
0 انا ليست لدي أي افكار للاضرار بنفسي.
1 انا لدي افكار للاضرار بنفسي ولكن لا انفذها.
2 انا اشعر انه من الافضل ان اموت.
3 لو استطعت.. لقتلت نفسي.

(8)
0 انا لم افقد اهتمامي بالناس.
1 . انا اقل اهتماما بالناس مما تعودت ان اكون من قبل.
2 انا فقدت اغلب اهتماماتي بالناس، ولدي مشاعر قليلة تجاههم.
3 انا فقدت كل اهتماماتي بالناس، ولا اهتم بهم على الاطلاق.

(9)
0 انا اتخذ القرارات بنفس الجودة كما تعودت ان اتخذها من قبل.
1 انا احاول تأجيل اتخاذ القرارات.
2 انا لدي صعوبة شديدة في اتخاذ القرارات.
3 انا لا استطيع اتخاذ أي قرار بعد الآن.

(10)
0 انا لا اشعر بأنني اظهر اسوأ مما اعتدت ان اظهر به من قبل.
1 انا مشغول وقلق على انني اظهر كبير السن او غير جذاب.
2 انا اشعر بان هناك تغيرات ثابتة في مظهري تجعلني اظهر بطريقة غير جذابة.
3 انا اشعر بانني قبيح او كريه المنظر.

(11)
0 انا استطيع العمل بنفس الجودة كما تعودت من قبل.
1 ابذل بعض الجهد لكي ابدأ العمل في بعض الاشياء.
2 يجب علي ان ادفع نفسي بقوة لاقوم بأي شيء.
3 انا لا استطيع ان اقوم بأي عمل على الاطلاق.

(12)
0 انا لا اجهد اكثر مما تعودت من قبل.
1 انا اشعر بالاجهاد بسهولة اكثر مما تعودت من قبل.
2 انا اشعر بالاجهاد من أداء أي شيء.
3 انا في منتهى الاجهاد لدرجة اني لا أستطيع عمل أي شيء .

(13)
0 شهيتي للأكل ليست أسوأ من المعتاد.
1 شهيتي للأكل ليست جيدة كما كانت.

2 شهيتي اصبحت أسوأ الآن.

3 انا ليست لي شهية للأكل نهائيا.

تقدير درجة الاكتئاب باستخدام معيار بيك	
الدرجة	الاكتئاب
0 – 4	لا يوجد- الحد الادنى
5 – 7	معتدل- غير حاد
8 – 15	متوسط
16 +	شديد

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Poststroke Depression In Relation To Different Brain Lesions

اكتئاب ما بعد السكته (الصدمة الدماغية) وعلاقتها بأضرار الدماغ المختلفة

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الملخص :

الأهداف: تهدف الدراسة الى تحديد العلاقة بين الاضرار التشريحية المرضية والاكتئاب لمرضى السكتة (الصدمة) الدماغية

الطرق: تم أنتخاب (٤٧) مريض بصورة متوالية مصابين بضرر محدد أحادي الجانب . وأجري لهم الفحص السريري ، الفحص الشعاعي بمفراس الدماغ ، والتقويم النفسي ،خلال شهرين مابعد السكتة الدماغية . تم تقويم الاكتئاب بمقياس بيك ، ومعايير الاكتئاب للمصنف الامريكي الرابع. علما أن الفحص الشعاعي العصبي (مفراس الدماغ) لجميع المرضى تم بنفس التقنية موقع الضرر ، حجم الضرر ونسبة البطين – الى – الدماغ تم تحليلها جميعاً

النتائج: لم يتم أستخراج أختلاف واضح في معدل درجات الاكتئاب بين المرضى المصابين في الفص الايسر والمرضى المصابين في الفص الايمن ، ولاتوجد علاقة بين شدة الاكتئاب وامامية (الفص الامامي) او حجم الضرر او ضمور الدماغ .اضطرابات الاكتئاب العظمى موجودة لدى تسعة مرضى فقط كان لديهم ضرر الفص الايسر للدماغ . معظم المرضى كان لديهم ضرر في العقدة القاعدية ، بينما لا يوجد أي مريض مصاب بضرر الفص الايمن للدماغ يعاني من نوبة اكتئاب عظمى .

الأستنتاج: الاضرار المجاورة الى العقدة القاعدية للفص الايسر للدماغ تلعب دورا حاسما في نشوء نوبات الاكتئاب العظمى بعد السكتة (الصدمة) الدماغية .

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