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Cognitive Outcome After Endovascular Coiling in Patients with Subarachnoid Hemorrhage

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Abstract: *Cognitive performance after SAH is a very important factor affecting large group of patients surviving SAH and their quality of life.The study was designed to measure the effect of endovascular therapy on cognitive outcome of SAH patients.The patients were collected from Matareya Teaching Hospital, Cairo, Egypt, from January 2016 to April 2016 and reassessed at 3, 6 months after the acute stage of the disease.Thirty-six patients were evaluated and divided into 2 groups: Patient group: included 19 patients treated with endovascular coiling of intracranial aneurysm. Control group: included 17 patients managed conservatively. The Montreal cognitive assessment scale was used.*
It was concluded from the study that there is a significant number of the patients whom were studied suffered from cognitive dysfunction, but it couldn't be attributed to the method of treatment using endovascular coiling.
Keywords:*subarachnoid, cognitive, coiling, MoCA.*

I. Introduction

Subarachnoid hemorrhage accounts for 1 in 20 strokes and 85% of cases are caused by a ruptured intracranial aneurysm (Latimer et al, 2013).In recent years, survival rates improved among patients who experienced SAH (Morris *et al*., 2004).In patients presenting with spontaneous SAH, early diagnosis and detection of the underlying cause is considered critical in order to favor the prognosis (Kokkinis *et al.,* 2008).

Many patients, even those who were independent and had no focal deficits, had cognitive impairment after recovery from SAH (Hop *et al.,* 1998).Although cognitive impairments are most frequent within the first 3 months after SAH, recent studies demonstrated that residual cognitive impairments persist as long as 75 months after SAH and perhaps longer (Al-Khindi *et al*., 2010).

Many Researches Were Concerned With Recognition Of Factors Involved With Cognitive Deficit After SAH:

(Hadjivassiliou et al., 2001; Kreiter et al., 2002; Fontanella et al., 2003; Bellebaum et al., 2004; Benke et al, 2005; Manning et al, 2005; Ravnik et al., 2006; Samra et al., 2007; Martinaud et al, 2009; Sheldon et al., 2012) suggested that aneurysm location more with anterior circulation aneurysms especially ACA aneurysms & ACoA affect cognitive function more than posterior circulation aneurysms but (Haug et al., 2007; Al-Khindi et al., 2010; Sheldon et al., 2013) excluded any relation between aneurysmal Location and cognitive deficit.

(Kreiter et al., 2002; Ravnik et al., 2006; Sehba & Bederson, 2006; Proustet al., 2009; Al-Khindi et al., 2010; Sheldon et al., 2013) suggested that SAH resulted in diffuse, global damage to brain tissue, perhaps through a mechanism involving elevated intracranial pressure, reduction of cerebral blood flow and brain oxygenation, blood– brain barrier breakdown, and global cerebral edema that interfere with cognitive function.
The method of treatment by surgical clipping versus endovascular technique was researched by(Hadjivassiliou et al., 2001; Fontanella et al., 2003; Cheng et al., 2006; Frazer et al., 2007; Scott et al., 2010; Lanzino et al., 2013; Latimer et al., 2013) they suggested that in clipped patients, memory problems have been identified as being an enduring difficulty and executive functioning deficits and impaired concentration have also been sporadically reported. Versus lower rates with endovascular treatment as Endovascular coiling also (Mocco et al, 2006) suggested that aggressive early intervention, including emergency external ventricular drain placement and urgent open surgical management could contribute to cognitive deficit.

Delayed cerebral infarction was also suggested by (Chu Wong et al., 2013) that could be a possible risk factor.Cognitive deficits after SAH may be also associated with the presence and site of cerebral infarction. Left lateral and bilateral medial/basal frontal infarctions cause deficits in verbal memory. Impairments of expressive speech and language are associated with left lateral infarctions (Vilkki et al., 2004).

(Mayer et al., 2002; Bellebaum et al., 2004; Mocco et al, 2006; Frazer et al., 2007), suggested that the hemorrhage itself and associated secondary brain damage, by a global toxic effect of widespread bleeding in the subarachnoid space. rather than the location of the aneurysm or the surgical intervention.

(Hadjivassiliou et al., 2001) suggested that outcome after SAH is primarily dictated by the hemorrhage possibly with some contribution from preexisting vascular disease.

(Bellebaum et al., 2004) evidenced that Patients with SAH of unknown origin show a cognitive pattern similar to patients who underwent clipping of a ruptured aneurysm. Also, patients with non-aneurysmal SAH develop cognitive deficits and impairments in daily life (Brand et al., 2014). Poor grade aneurysmal subarachnoid hemorrhage was also suggested by (Kreiter et al., 2002; Mocco et al, 2006).(Orbo et al., 2008) also suggested that the amount of subarachnoid blood is directly related to cognitive outcome.There was a strong link suggested by (Haug et al., 2007) between the degree of acute hydrocephalus (CSF drainage) and memory deficits, even 12 months after the ictus.

(Springer et al., 2009) found that any fever higher than 38.6 C during the first 2 weeks of hospitalization after acute SAH was an important risk factor for cognitive impairment.It is believed that changes in behavior, memory, etc. that represent long-term complications that cannot be explained by vasospasm alone. As a result of the global ischemic injury, secondary to raised ICP and decreased CBF, apoptosis has been shown to be widespread in the brain after SAH (Cahill et al., 2006).The time course of cognitive recovery after aneurysmal SAH is variable (Haug et al., 2007).

(Samra et al., 2007) suggested that Cognitive function, improved over time with significant improvement between 3 and 9 months and reached a plateau between 9 and 15 months after surgery. (Chu Wong et al., 2015) suggested that cognitive dysfunction might further improve even after 9 months. But, (Al-Khindi et al., 2010) demonstrated that residual cognitive impairments persist as long as 75 months after SAH and perhaps longer.The aim if this study is to assess effect of endovascular coiling on cognitive function in patients with acute subarachnoid hemorrhage.

II. Patients And Methods

This Study Was Conducted On Two Groups:

Patient group: A total of 32 patients were admitted to Matareya Teaching Hospital, Cairo, Egypt, with aneurysmal SAH between January and April 2016. All patients were managed with endovascular coiling of intracranial aneurysm. 13 patients were excluded as; Four patients (12.9 %) died within the acute stage of the disease, three excluded because of age older than 65 years, two was excluded because of dysphasia, one excluded due to sever visual impairment that interfered with the assessment testing and three were lost to follow-up. Thus, 19 subjects could be followed for the purpose of this study.

Control group*:* A group of 25 other patients with subarachnoid hemorrhage due to non-aneurysmal or unknown cause who undergo conservative treatment were admitted at the same time period. 8 patients were excluded from the study as; Two patient died during the acute stage of the disease, three excluded because of age older than 65 years one refused to participate in the study and two were lost to follow-up. Thus, 17 subjects could be followed for the purpose of this study.

Inclusion criteria:

- Patients confirmed to have acute SAH by non-enhanced CT brain or Lumbar Puncture.
- Both sexes were included in the study.
- Age between 20 years and 65 years old.

Exclusion criteria:

- Traumatic cause of subarachnoid hemorrhage.
- Past history of large or disabling lesion interfering withassessment scale**.**
- Patient with known past history of psychiatric disorder.
- Patient with decompensated metabolic disorder.

Methods: Patients were subjected to the following

- Patients diagnosed to have acute subarachnoid hemorrhage by CT brain or LP.
- Patients will be managed according to American Heart Association/American Stroke association guidelines (Connolly, et al, 2012).
- Aneurysmal subarachnoid hemorrhage Was identified by DSA or CT angiography.
- Spontaneous SAH was considered candidate for conservative management.
- Conservative management was chosen in non-aneurysmal SAH or aneurysms difficult to be treated due to technical factors.
- Demographic data of all patients was collected.
- Full medical history taking wasrecorded.
- Full neurological examination was done.
- Cognitive assessment using MoCA scale.

- Data analysis.

Cognitive assessment:

Cognitive assessment was done initially and at 3,6 months after acute subarachnoid hemorrhage and coiling using Montreal cognitive assessment scale.The Montreal Cognitive Assessment is a brief 30-point screening instrument that was developed and validated to identify patients with mild cognitive impairment.The MoCA-B Arabic version was used, which is the first cognitive screening tool designed specifically to detect MCI in low education or illiterate subjects (Julayanont *et al.,* 2015).

III. Statistical Analysis

IBM SPSS statistics (V. 20.0, IBM Corp., USA) was used for data analysis. Date were expressed as Mean±SD for quantitative parametric measures in addition to both number and percentage for categorized data.

The Following Tests Were Done:

1. Statistical presentation and analysis of the present study was conducted, using the mean X, standard deviation, Paired t-test and The Mann–Whitney test.
2. The Mann–Whitney test to study the association between each 2 variables or comparison between 2 independent groups as regards the categorized data, which was nonparametric.
3. Paired t-test to study 'before-after' test results un the same group.

Due to the small numbers in each treatment group, data was interpreted in relation to both statistical significance (p value < 0.05) and insignificant (p > 0.05).

IV. Results

Demographic Factors:

This study included 19 patients (11 males and 8 females) with acute aneurysmal SAH and treated with endovascular coiling of intracranial aneurysm. Of these, 11 were men and 8 were women, their age ranged between (27-62 years) with a mean (50.5 ± 10.3)

Control group included 17 patients with acute SAH due to non-aneurysmal or unknown cause who undergo conservative treatment, those patients matched with the age ranged between (29-63years) with a mean (49.7 ± 9.1) and sex, they were 9 males (53%) and 8 females (47%).

Years of Education:

There were 11 patients (58%) who had more than 6 years of education, and 8 patients (42%) with less than 6 years of education.

The control group included 8 patients (47%) with more than 6 years of education and 9 patients (53%)with less than 6 years of education.

Risk Factors:

There were 3 (16%) patients with hypertension, 3 (16 %) patients with diabetes mellitus, 2 (10 %) patients were hepatic, 1 (5%) patient with ischemic heart disease and patients 5 (26 %) patients were smokers.

Control group included 4 (23 %) patients with hypertension, 1 (6%) patients with diabetes mellitus, 1 (6 %) patients with ischemic heart disease,1 (0.6 %) patients was hepatic and 5 (29 %) patients were smokers.

Aneurysmal location:

In patients group, 10 (53%) patients were diagnosed to have ACoA, 4 (22 %) patients had PCoA, 2 (10%) patients had ICA, one (5%) patient had MCA, one (5%) patient had basilar artery aneurysm, and one (5%) patient had vertebral artery aneurysm.In control group 16 (94%) patients were diagnosed to have non aneurysmal SAH, one (6%) patient had multiple angiomatous malformations.

Baseline Cognitive Function:

Cognitive function was evaluated at the 1st few days of the disease using Montreal Cognitive Assessment Scale.The mean MoCA score in patients group 25.2 with SD 2.46 and the mean score in control group 25.5 with SD 1.2 as in **table (1).**

Table (1): patient and control groups as regarding baseline cognitive assessment.

	No.	Mean rank	Sum of ranks	U	W	Z	Sig.
P.	19	18.24	346.50	156.50	346.50	-.162	0.871
C.	17	18.79	319.50				

P.=patients group, c.= control group, no.= number, sig.=significance.

As in **table (1),**There was no statistical difference between the patients and control regarding baseline cognitive evaluation using MoCA scale as (z = -0.162), which indicated that the two groups were homogenous before any application**.**

Neurological Complications:

During the acute stage; one (5%) patient was complicated by hydrocephalic changes, 2 (10%) patients were complicated by vasospasm. In control group one (6%) patient had hydrocephalic changes.

This Study Showed The Following Results:

There was no statistical difference between patients and control group as regarding cognitive outcome after 3 months after SAH. (P-value > 0. 05)

Table (2): Comparison between patients and control group as regard cognitive outcome 3 month after SAH.

	No.	**Mean rank**	**Sum of ranks**	**U**	**W**	**Z**	**Sig.**
p.	19	17.50	332.50	142.50	333.50	-0.609	Not sig.
c.	17	19.62	333.50				

P.=patients group, c.= control group, no.= number, sig.=significance.

In **table (2);** there is no statistical difference between patient and control groups using MoCA scale at 3 months follow up after SAH, as mean rank of patient group was (17.5) and for control group (19.62) and also (U, W, Z) were all non-statistically significant with (p value = 0.543).

There was no statistical difference between patients and control group as regarding cognitive outcome after 6 months after SAH (p value = 0.512).

Table (3): Comparison between patients and control group as regard cognitive outcome 6 month after SAH.

	No.	**Mean rank**	**Sum of ranks**	**U**	**W**	**Z**	**Sig.**
p.	19	17.37	330	140	330	-0.688	Not sig.
c.	17	19.76	336				

P.=patients group, c.= control group, no.= number, sig.=significance.

As in **table (3);** there is no statistical difference between patient and control groups using MoCA scale at 6 months follow up after SAH, as the mean rank of patient group was (17.37) and for control group (19.76) and also (U, W, Z) were all non-statistically significant with (p value = 0.512).

There was high statistical difference in patients group regarding cognitive outcome evaluated initially during the acute stage of SAH and after 3 months follow up using MoCA scale.

Table (4): Comparison between cognitive assessment initially and after 3 months follow up in patients group.

Gp.	No.	mean	SD	df	t	Sig.
In.	19	25.2	2.4	18	3.8	0.001
3 m.	19	23.9	2.97			

Gp. = group, No.-number, in.= initial assessment, 3m.= 3 months follow up

As in **table (4)** there was high statistical difference in patients group as regarding cognitive outcome evaluated initially during the acute stage of SAH and after 3 months follow up using MoCA scale in (0.001) mean MoCA score was higher in the initial evaluation than after 3 month follow up of the disease.

There was high statistical difference in patients group regarding cognitive outcome evaluated initially and after 6 months follow up using MoCA scale.

Table (5): Comparison between cognitive assessment initially and after 6 months follow up in patients group.

Gp.	**No.**	**mean**	**SD**	**df**	**t**	**Sig.**
In.	19	25.2	2.4	18	4.3	0.000
3 m.	19	23.1	3.5			

Gp. = group, No.-number, in.= initial assessment, 3m.= 3 months follow up

As in **table (5),** there was high statistical difference in patients group as regarding cognitive outcome evaluated initially during the acute stage of SAH and after 6 months follow up using MoCA scale in 0.000

Mean MoCA score was higher in the initial evaluation during the acute stage than after 6 month follow up of the disease.

V. Discussion

In recent years, survival rates have improved among patients who have experienced SAH (Morris et al., 2004).

Endovascular coiling was considered almost the most prominent method of management of intracranial aneurysm (Manning et al, 2005) and more accepted worldwide (Niimi et al., 2006).

Those surviving SAH often have cognitive problems such as memory deficits and language impairments (Wallmark et al., 2015).

There is a general consensus that cognitive deficits persist after SAH, outliving any improvements in residual physical deficits and ultimately leading to a permanent reduction in health-related quality of life (Brand et al., 2014).

The main purpose of this study was to explore whether the type of therapy (endovascular vs conservative) has an influence on the pattern and the severity of cognitive outcome in patients with SAH.

This study compared 2 groups of patients who suffered from acute SAH regarding the effect of the method of treatment applied to those patients on cognitive outcome.

All patients were collected from Matareya Teaching Hospital and diagnosed to have acute SAH by non-contrast CT brain.

All patients were admitted to Neurology ICU at least for the first week of manifestations.

As soon as possible, the baseline cognitive function was assessed using MoCA.

All patients were managed medically with measures that could prevent vasospasm as hydration, hypertension and oral Nimodipine treatment for 21 days and according to the recent Guidelines for the Management of Aneurysmal Subarachnoid Hemorrhage (Connolly et al., 2012).

All patients were subjected to Diagnostic Cerebral Angiography to assess the cause of subarachnoid bleeding.

Some patients were found to have aneurysmal SAH and were managed with endovascular coiling, others were found to have non- aneurysmal SAH and managed with conservative treatment and one patient was found to have multiple angiomatus malformations that required no surgical or endovascular interventions and was managed conservatively.

A group of patients who had endovascular coiling was compared against a group managed conservatively.

Patients subjected to Conservative management were chosen as a control (or comparison group) over patients managed with surgical clipping, as used by some investigators; Hadjivassiliou et al. (2001), Fontanella et al.(2003), Cheng et al., (2006), Frazer et al. (2007), Scott et al. (2010), Lanzino et al. (2013) and Latimer et al. (2013), to avoid the craniotomy factor affecting cognitive function in the method of clipping, and also because clipping was rarely used now due to the advancement of endovascular techniques over open craniotomies.

Almost all of the patients managed with conservative treatment were- non intentially- found to have non-aneurysmal SAH.

Many patients with SAH are not capable of completing cognitive assessment test as a result of poor clinical condition, or severe headache from mental exercise.

Patients with severe headache were postponed till relative improvement of their condition.

Consequently, the findings in this research only pertain to SAH survivors in relatively good condition.

After management during the acute stage of the disease, the patients were discharged and followed during the following 6 months.

Cognitive function was screened at 3 and 6 months after onset of the disease.

A MoCA score less than 26 was used to indicate the presence of at least mild cognitive impairment (Nazem *et al.*, 2009).

Some investigators have raised concern about the methods used to determine cognitive impairment.

MoCA, as a screening tool, has the advantages of brevity and ease of administration. A recent review concluded that MoCA compared favorably with the MMSE as a screening test that is sensitive to the milder forms of cognitive impairment that often accompany cerebrovascular disease.

Koski (2013), provides some evidence that the MoCA covers the range of content that is required for the assessment of cognitive impairment in cerebrovascular disease, with the exception of mental processing speed.

We found that this cognitive test, have been used in many investigations, and suited for the majority of patients with SAH. The Arabic version of the test have been used in our study for use of Egyptian patients.

In the present study, we found residual cognitive impairments in both groups at the time of the follow-up examinations done at 3 and 6 months, with a significant proportion of patients continuing to have at least mild to moderate cognitive deficits.

Cognitive changes were observed in both patient groups. Patients treated with the endovascular technique as well as patients treated with the conservative management were impaired. These deficits are likely to be associated with the SAH condition.

9 of 19 patients managed with endovascular coiling were found to have cognitive deficit, and 7 of 18 patients managed with conservative treatment were found to have cognitive dysfunction.

Comparing both groups indicated that there is no difference regarding the method of treatment of SAH.

There is an unsolved debate in the SAH outcome concerning the degree to which the acute characteristics of SAH contribute to cognitive outcome.

Kreiter *et al.*, (2002) obtained a significant linkage between cognitive dysfunction (in particular in executive functioning) and Hunt and Hess Grade 3 or higher, as well as the presence of a thick layer of subarachnoidal blood on preoperative CT scan.

SAHs can cause global lesions: The first minutes after SAH are characterized by an exponential increase in intracranial pressure (ICP), which serves to stop the bleeding. As a consequence of the increased ICP, blood circulation in all basal arteries is dramatically reduced and global ischemic lesions may occur. The duration of the increased ICP and the reduced circulation can vary inter-individually, but normally two to four minutes of increased pressure are required to stop the bleeding. The variability in the mechanism leading to diffuse brain damage may help to understand the variability in the severity of cognitive deficits as a consequence of SAH (Bellebaum et al., 2004).

Sheldon *et al.*, (2013) Stated that There was no effect of aneurysm variables (location or surgical intervention), on cognitive impairment after SAH.

Delayed cerebral infarction was also suggested by Chu Wong *et al.*, (2013) that could be a possible risk factor.

Orbo *et al.*, (2008) also suggested that the amount of subarachnoid blood is directly related to cognitive outcome.

There was a strong link suggested by Haug *et al.*, (2007) between the degree of acute hydrocephalus (CSF drainage) and memory deficits.

Mayer *et al.*, (2002) suggested that the hemorrhage itself and associated secondary brain damage, by a global toxic effect of widespread bleeding in the subarachnoid space. rather than the location of the aneurysm or the surgical intervention.

Samra *et al. (*2007) and Martinaud *et al*. (2009) suggested that aneurysm location the most acceptable factor affecting cognitive dysfunction.

Frazer *et al.*, (2007) disagreed with this results as they found that there are minimal differences in the long term cognitive outcomes between endovascular coiling and surgical Clipping but they suggested that both methods of intervention could be attributed to cognitive dysfunction.

Kremer *et al.*, (2002) denoted that incomplete endovascular occlusion or regrowth of an aneurysm can lead to rebleeding with a significant impact on functional outcome.

Our results provide preliminary evidence that SAH itself may result in cognitive deficits.

The result from this research could be attributed to:

The large variability in the prevalence of cognitive deficits can be partially attributed to the heterogeneous nature of SAH. Patients differ with respect to aneurysm location, infarction location, severity of hemorrhage, and incidence of delayed hydrocephalus and angiographic vasospasm.

Possible toxic substances and global brain damage that could be affected in patients with SAH regardless etiological finding or method of treatment.

The clinical condition at the time of discharge from the hospital has been found to be valuable with respect to later cognitive impairment Presently, we could not reproduce this finding, possibly because few of our patients had neurological deficits at the time of discharge.

Neurological complications also should be considered to be a cause of cognitive dysfunction as hydrocephalus and vasospasm but this could not be certain due to few number of patients in the sample that suffered from those complications and also due to affection of cognitive function in other patients not complicated by hydrocephalus or vasospasm.

The amount of blood collected in subarachnoid space and modified Fisher score at presentation could be related to cognitive outcome after the disease.

Possible medical comorbidities could be considered also as a risk factors as long standing diabetes mellitus.

Study limitations:

First, cognitive function was assessed and compared by MoCA, not a comprehensive neuropsychological battery.

Second, the number of patients did not allow subgroup analysis such as with location of cerebral aneurysm.

There was a limitation found during the study that was the low mean years of education among the patients so the MoCA basic test was used.

Possible pitfalls during assessing the baseline cognitive function using MoCA could be found as the patients' performance could be affected by the headache that had its maximum intensity during the acute stage of the disease.

The researcher also found more variables that could affect the results as location of aneurysm or non-aneurysmal cause of SAH that was recommended considering in the future studies.

The timing of endovascular intervention and the timing of initial assessment using MoCA in relation to the first days of the disease, could not be regarded in this study due to variable timing of presentation of the patients after the onset of the disease, to the hospital. But initial assessment and endovascular therapy was applied as soon as possible.

VI. Conclusion

It was concluded from the study that there is a significant number of the patients who suffered from cognitive dysfunction, but it couldn't be attributed to the method of treatment using endovascular coiling.

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