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Management of Vasospasm after Aneurysmal Subarachnoid Hemorrhage

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ABSTRACT

Background: The loss of productive life-years after subarachnoid haemorrhage (SAH) is similar in magnitude to that of ischemic stroke. Cerebral vasospasm (VSP) is one of the complications of SAH which is, narrowing of the intracranial arteries several days after an aneurysmal SAH. **Objective:** This work aimed to assess the different management modalities of cerebral VSP which is a common complication of aneurysmal SAH.

Methods: This prospective study included 20 patients with aneurysmal SAH admitted to Neurosurgery Department, Tanta University Hospitals. For patients with vasospasm, Nimodipine was used, which is safe, cost-effective & decreases the risk of poor result & secondary ischemia. It is used prophylactically in all studied cases with SAH. Nimodipine is given either orally or continuous infusion. Endovascular techniques frequently play a role in the aggressive treatment of vasospasm as intra-arterial infusion of vasodilators (Nimodipine).

Results: There was a significant negative relationship among Modified fisher score & Glasgow coma scale (GCS) score on admission (r=-0.559, P=0.010), indicating that increasing Modified fisher score on admission is associated with lower GCs. The mean modified Rankin scale (MRS) improved over time from 0.80 ± 1.54 at 2 weeks to 0.55 ± 1.05 at 3 months and further to 0.20 ± 0.52 at 6 months and this difference was statistically significant (p=0.008). MRS score significantly improved with time (P=0.034). **Conclusions:** VSP following aneurysmal SAH can be devastating and thus prevention and treatment are crucial. Nimodipine is an effective therapy for managing VSP following aneurysmal SAH & endovascular nimodipine was effective for treatment and prevention of vasospasm.

Keywords: Management, Vasospasm, Aneurysmal subarachnoid hemorrhage.

INTRODUCTION

The loss of productive life years following a subarachnoid haemorrhage (SAH) is comparable to that following an ischaemic stroke at the community level. Prior estimates of the crude incidence of SAH ranged from 7.8/100.000 to 21.4/100.000 annually, with significant variation by sex, age, & geographic location [1]. SAH hall mark presenting symptom is thunder clapping headache, nausea, vomiting, fits and disturbed conscious level [2]. Cerebral vasospasm is one of the complications of SAH, which is narrowing of the intracranial arteries several days after an aneurysmal SAH [3]. The Fisher grade allows to predict morbidity & mortality in studied cases with SAH by classifying the volume of SAH in computed tomography (CT) scans [4]. The imaging modalities used for the detection of SAH include head CT, magnetic resonance imaging (MRI), CT angiography (CTA) & magnetic resonance angiography (MRA). The sensitivity of CT for detection of SAH is very high, reaching 100%. Lumbar puncture as urgent manoeuvre but must be done with specific precautions [5]. After non-contrast CT confirms SAH, digital subtraction angiography (DSA), which offers three-dimensional rotational images to detect lesions, is the gold standard for diagnosing intracranial aneurysms. It is particularly helpful if endovascular repair will be employed to treat a difficult aneurysm (large, calcified and thrombosed, or numerous aneurysms) [6].

While delayed cerebral ischaemia (DCI) is the recommended term to describe the post-SAH delayed clinical deterioration after other possible identifiable causes have been ruled out, angiographic cerebral vasospasm (VSP) is the recommended nomenclature for describing a demonstrated, transient narrowing of cerebral arteries on CT, MR, or DSA [7]. Treatment modalities of DCI include medical treatment: Euvolemia, calcium channel blockers (nimodipine), maintaining the normal haemoglobin level, and endovascular management; mechanically by angioplasty and chemically by intraarterial injection of calcium channel blockers (nimodipine) [8, 9].

The goal of this work was to evaluate the different management modalities of cerebral VSP which is a common complication of aneurysmal SAH.

PATIENTS & METHODS

This prospective study was carried out on 20 studied cases with aneurysmal SAH admitted to Neurosurgery Department, Tanta University Hospitals through the period from August 2023 to August 2024.

Exclusion criteria: Patients with traumatic SAH, with impaired renal & hepatic functions, with coagulation disorders and pregnancy.

Patient evaluation and management: We started the evaluation by taking a quick history, assessing the patient's airway, assessing their vital signs, & performing basic resuscitation. The Hunt & Hess scale was used to perform the initial clinical grading [10].

Early non contrast CT imaging was done and bleeding on the CT scan was quantified by using the Fisher scale [11]. In case the CT showed SAH, the patients did CTA immediately. Our patients were given initial medical treatment, and this was in the form of fluids starting from the first day, which was guided by cardiac function. Antiepileptic medications (levetiracetam). Antacid medications. Nimodipine, oral 2 tab (60 mg)/4 hrs or IV 1-2 mg/hr. Analgesics, Paracetamol 1000 mg/8

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hrs or Tramadol 100 mg amp/12 hrs. Patients were kept euvolemic to protect against vasospasm.

All patients after initial medical management and resuscitation were subjected to history taking and clinical examination (general & neurological). Also, routine laboratory investigations including coagulation profile, hepatic and renal functions. Serial evaluation of neurological function was done & preparation for angiography. In all our cases we did CTA immediately if the CT showed SAH. Conventional catheter angiography remained the gold standard for detection of intracranial aneurysms, in cases with negative CT and high suspicious for intracranial aneurysm, lumbar puncture and CSF xanthochrome level were considered in diagnosis.

Management of complications: Hydrocephalus: If the initial CT showed signs of hydrocephalus, a ventricular drainage was done. In patients without hydrocephalic changes in early CT, close follow up of conscious level was done and if hydrocephalic changes appeared, ventricular drainage was done.

Rebleeding: Every element linked to rebleeding (Valsalva & cough) was reduced. During ventriculostomy or lumbar puncture, rapid draining of a high volume of CSF was avoided. The amount of excessive stimulation was reduced. Short-acting medications were used to calm agitated individuals & reduce their headaches until they were drowsy but still receptive for neurologic status evaluation. Definitive prevention of re-bleeding was by definite treatment of the aneurysm either by endovascular treatment or surgically.

Vasospasm: Almost never before day 3 post SAH. Maximal frequency of onset during day's 6–8 post-SAH. Vasospasm was diagnosed radiologically including CT brain angiography and conventional catheter angiography. In our cases, Nimodipine was used prophylactically in all patients with SAH. Nimodipine was given either orally by a dose 60 mg/4 hrs or IV 1-2 mg/hr with continuous infusion.

Endovascular techniques: Endovascular techniques included transluminal angioplasty & intra-arterial infusion of vasodilators (Nimodipine) and balloon dilatation for focal vasospasm but in global vasospasm we used intra-arterial Nimodipine infusion. In our cases, in resistant cases intraluminal Nimodipine was given. All patients were performed in the neuro-angiography suite at Neurosurgery Department. All patients received either local or general anaesthesia. No pre-procedural systemic anticoagulation was administered. All studied cases underwent a single or double wall puncture using the usual transfemoral technique. Initial cerebral angiography was carried out using a 5 F diagnostic catheter & a high-resolution digital subtraction monoplane 3D rotational angiography machine for road mapping.

Exchange of 5-F diagnostic catheter (Bern) by 6-F guiding catheter & the guiding catheter placed in the appropriate position in the cervical segment of the internal carotid artery at the level of C2 cervical vertebra (Axis). The micro-catheters used were Excelsior SL-10; Boston Scientific or Echelon 10; ev3. The micro-catheter was shaped by steam and placed coaxially through guiding catheter and navigated into the aneurysm with the aid of 0.014-inch micro guide wire (Transend; Boston Scientific). Flush was prepared (500 ml normal saline, 5 ml nimodipine and 5000 IU heparin) and was connected to the guiding catheter with Y connector and another to the micro catheter to prevent spasm induced by micro-catheters. As for the direct bolus injection of nimodipine (3-5 ml + 15 cm normal saline) over 15-20 minutes guided by blood pressure.

Ethical Concerns: The research was done after approval from The Ethics Committee of the Faculty of Medicine, Tanta University Hospitals (approval code:36264MS285/8/23). The study adhered to the Helsinki Declaration throughout its execution. Informed written consents were obtained from the patients.

Statistical analysis

For each parameter, statistics were displayed & appropriate analysis was conducted based on the type of data collected. Data normality: The Shapiro-Wilk test was used to determine whether the data distribution was normal. Characteristic statistics: Numerical data were expressed as mean ± SD, median and range. The percentage & frequency of non-numerical data. The likelihood of outcomes: If a p value was ≤ 0.05 at the 95% confidence interval, it is deemed significant. Pearson correlation was done to estimate the degree of correlation between two quantitative variables.

CASE PRESENTATION

Case 1: A 33-years-old female presented with sudden severe headache, nausea & vomiting, neck stiffness then fits occurred, which led to disturbance of consciousness level (DCL). The fits was resolved and the patient seeked medical attention after 3 days from symptoms. Physical examination revealed elevated blood pressure. The neurological examination showed that the patient Glasgow coma scale (GCS) was 13 with intact motor power with dysarthria (Hunt and Hess grade 3). CT brain revealed SAH (modified fisher grade 3). Cerebral diagnostic angiography showed anterior communicating artery aneurysm & vasospasm of left A1 part of anterior cerebral artery. Complications throughout the operation was during insertion of the coils blood clot formed and led to closing of the perforating vessels of the anterior cerebral as well as part of A2, integrillin (eptifibatide, which is antiplatelet drug) was injected locally at the site of blood clot. Post-operatively, patient was intact and conscious but with expressive aphasia CT and MRI was done for follow up of patient. MRI showed anterior cerebral artery territories infarction; patient was started in therapeutic dose of antiplatelet. Patient showed improvement of speech over a period of two weeks post-operative. Follow up was done by Modified Rankin scale: Within 2 weeks was grade 4 then follow up after 3 and 6 months was grade 0 (**Figure 1).**

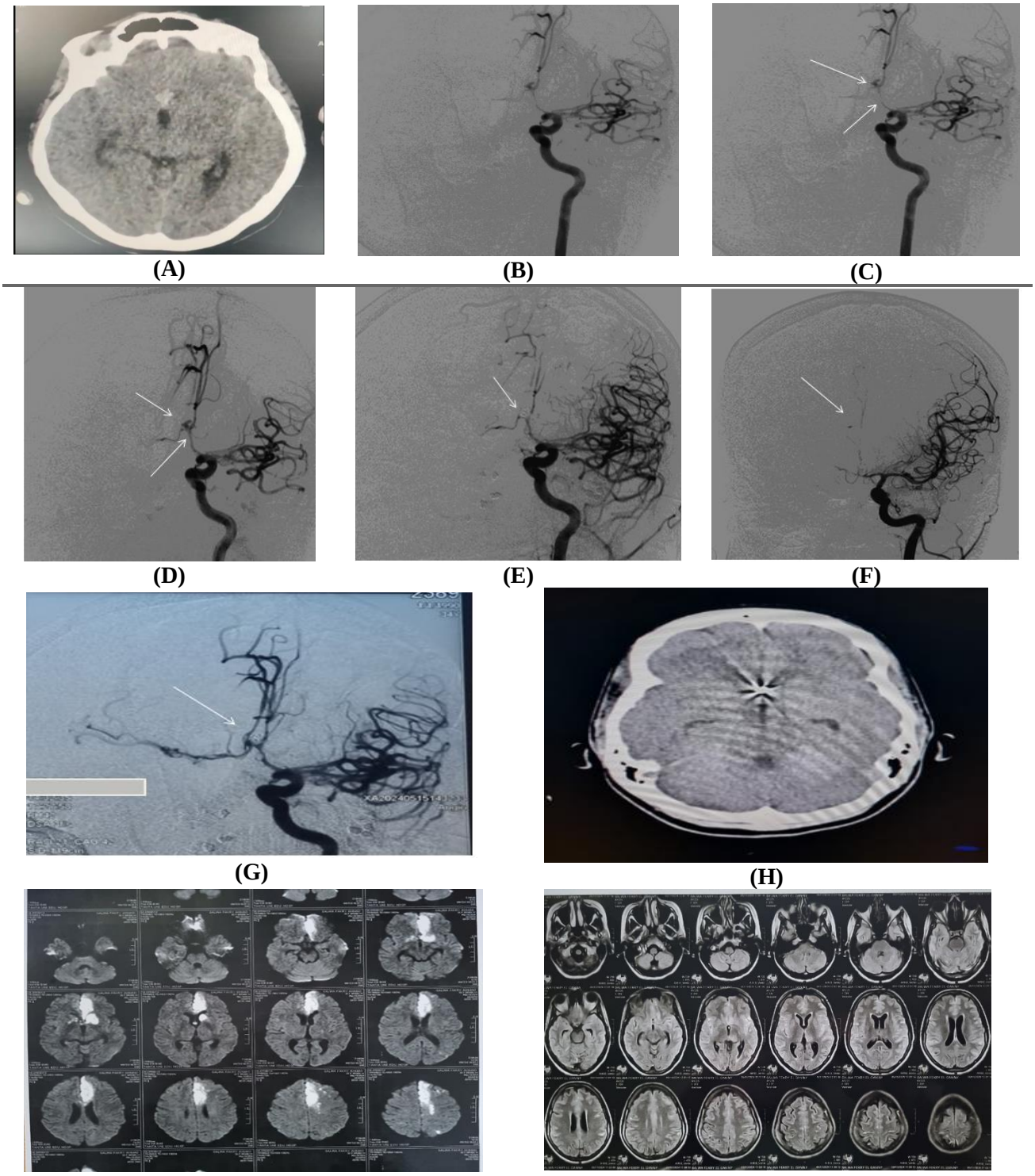

Figure 1: (A): CT brain showing blood in the subarachnoid space, **(B):** Cerebral diagnostic angiography showed anterior communicating artery aneurysm, **(C):** Before injection of intraarterial nimodipine, **(D):** After injection of intraarterial nimodipine, **(E):** Coiling of the aneurysm, **(F):** Blood clot formed leading to decrease flow in anterior cerebral, **(G):** After injection of integrillin and resolution of the blood clot, **(H):** Post-operative CT, **(I):** Post-operative MRI, **(J):** MRI follow up after 3 months showing resolution of ischemia.

Case 2: A 54-years-old male presented with sudden severe headache, nausea & vomiting, neck stiffness then followed by fits with DCL and left side weakness. Neurological examination showed that patient GCS of 14 with left side weakness and dysarthria (Hunt and Hess grade 3). CT brain showed SAH (modified fisher grade 1). Cerebral diagnostic angiography: Rt ICA angiogram AP view showed anterior communicating artery aneurysm and vasospasm of A2 part of anterior cerebral artery. Definitive management including endovascular coiling of the aneurysm and injection of intraarterial nimodipine (Rt ICA angiogram AP view showed resolution of vasospasm after nimodipine intra-arterial injection) (**Figure 2).**

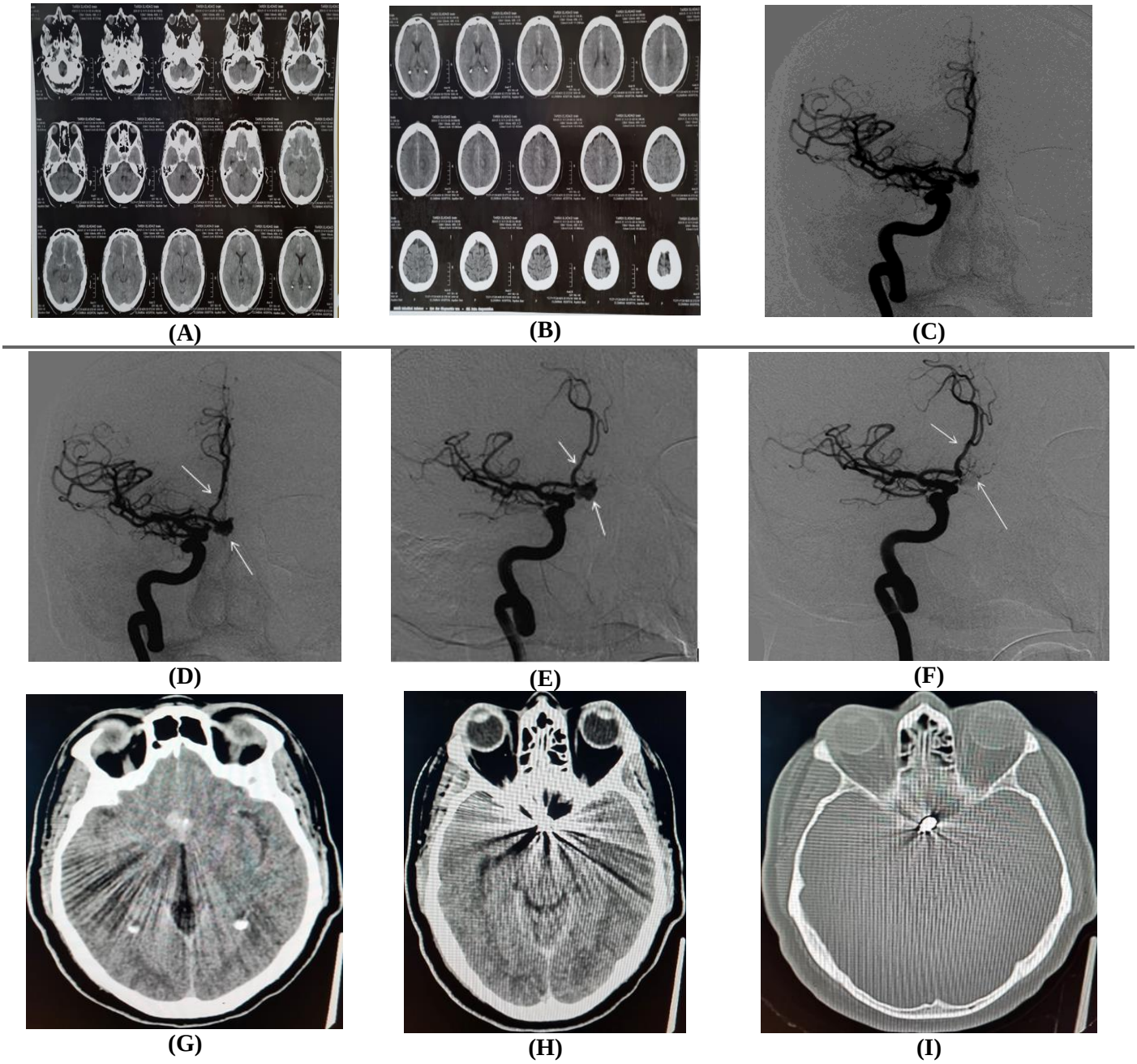

Figure 2: (A): CT brain scan was done showing subarachnoid hemorrhage (modified fisher grade 1), **(B):** CT brain scan was done showing subarachnoid hemorrhage (modified fisher grade 1), **(C):** Rt. ICA angiogram, AP view, showed anterior communicating artery aneurysm, **(D):** Before injection of intraarterial nimodipine, **(E):** After injection of intraarterial nimodipine, **(F):** Rt ICA angiogram AP view showed successful coiling of the aneurysm, **(G), (H), (I):** CT brain post-operative showing coils in the aneurysm.

RESULTS

Table (1) showed that demographic data, associated comorbidity and clinical presentation of the studied patients. The mean of GCS was 13.75. The mean of Hunt and Hess grade was 1.70. Among the studied patients, 7 (35%) studied cases had Hunt & Hess grade 1, 10 (50%) studied cases had Hunt & Hess grade 2 & 3 (15%) studied cases had Hunt & Hess grade 3. The Modified Fisher score distribution showed that the majority of subjects had a score of 3 (70.0%), while 15.0% of subjects had scores of 1 and 4 each.

Table (1): Demographic data, associated comorbidity, clinical presentation and Modified Fisher score distribution of the studied cases

			Total subjects (n=20)
Demographic data	**Age (years)**		55.30 ± 12.01
	Gender	**Female**	7 (35.0%)
		Male	13 (65.0%)
	Smoking		9 (45.0%)
Comorbidity	**HTN**		11 (55.0%)
	DM		3 (15.0%)
	RA		1 (5.0%)
Clinical presentation	**Speech**	**Aphasic**	1 (5.0%)
		Dysarthria	5 (25.0%)
		Intact	14 (70.0%)
	Disturbance of consciousness level		8 (40.0%)
	Vomiting		10 (50.0%)
	Headache		16 (80.0%)
	Weakness		5 (25.0%)
	GCS		13.75 ± 1.80
	Hunt and Hess Grade		1.70± 0.73
	Grade 1		7 (35%)
	Grade 2		10 (50%)
	Grade 3		3 (15%)
Modified Fisher Score	**Modified Fisher Score**		2.80 ± 0.83
	Score 1		3 (15.0%)
	Score 3		14 (70.0%)
	Score 4		3 (15.0%)

Data are presented as mean ± SD or frequency (%). HTN: hypertension, DM: diabetes mellitus, RA: rheumatoid arthritis. GCS: Glasgow Coma Scale.

According to GCS, the mean GCS improved from 13.75 ± 1.80 at baseline to 14.55 ± 0.89 at 2 weeks & this difference was statistically significant (p = 0.011) (**Table 2).**

Table (2): Comparison of GCS change during follows up

	Baseline (n=20)	**2 weeks (n=20)**	**P value**
GCS	13.75 ± 1.80	14.55 ± 0.89	**0.011***

Data are presented as mean ± SD, GCS: Glasgow Coma Scale. * Significant as P-value ≤ 0.05.

All subjects had radiological vasospasm and 50% represented with clinical vasospasm. The most common aneurysm location in the studied patients was the anterior communicating artery (ACOM) aneurysm, seen in 50.0% of patients. Other types like middle cerebral artery (MCA) aneurysms were observed in 25.0% of cases, posterior communicating artery (PCOM) aneurysms were observed in 10% of cases, while less common aneurysms, including those in the basilar tip were present in small percentages of the study population. In terms of medical treatment, all subjects (100%) received nimodipine via endovascular injection. Among them, follow-up treatment included intravenous administration for 35% of subjects and oral administration for 65%. For the management approach of aneurysms, coiling was the dominant intervention (85.0%), with other methods like stent assisted coiling in 15% of subjects. Management-associated complications were noted in 55.0% of patients, with various medical treatments (external ventricular drain (EVD) and medical management) being applied as needed (Table 3)**.**

Table (3): Vasospasm related data, location of aneurysm and management of the studied patients

		Total subjects (n=20)	
Vasospasm	**Clinical Vasospasm**	10 (50.0%)	
	Radiological Vasospasm	20 (100.0%)	
Type of Aneurysm	**ACOM**	10 (50.0%)	
	ACOM, Basilar Tip	1 (5.0%)	
	Lt PICA	1 (5.0%)	
	MCA	5 (25.0%)	
	PCOM	2 (10%)	
	Basilar Tip	1 (5.0%)	
Complications	**Acute HCP**	3 (15%)	
	Delayed HCP	0 (0%)	
	Convulsions	3 (15%)	
	DIND & infarction	8 (40%)	
	Death	0 (0%)	
Management	**Nimodipine endovascular injection**	20 (100%)	
	Follow up treatment (Nimodipine)	**IV**	7 (35.0%)
		Oral	13 (65.0%)
	Type of management of aneurysm	**Coiling**	17 (85.0%)
		Stent assisted coiling	3 (15.0%)
	Management of associated complications	**EVD**	2 (10.0%)
		Medical management	9 (45.0%)

Data are presented as frequency (%). ACOM: anterior communicating artery, Lt PICA: left posterior inferior cerebellar artery, MCA: middle cerebral artery, PCOM: Posterior communicating artery, HCP: hydrocephalus, DIND: delayed ischemic neurological deficit, IV: intravenous, EVD: external ventricular drain, * Significant as P-value ≤ 0.05.

There was a significant negative relationship among Modified fisher score & GCS score on admission (r=-0.559, P=0.010), indicating that increasing Modified fisher score on admission is associated with lower GCS (**Table 4 & figure 3).**

Table (4): Correlation between modified fisher score and GCS score on admission

	GCS on admission	
	r	**P**
Modified fisher score on admission	-0.559	**0.010***

Data are presented as numbers. GCS: Glasgow Coma Scale. r: correlation coeffeicient, * for significant p value (<0.05).

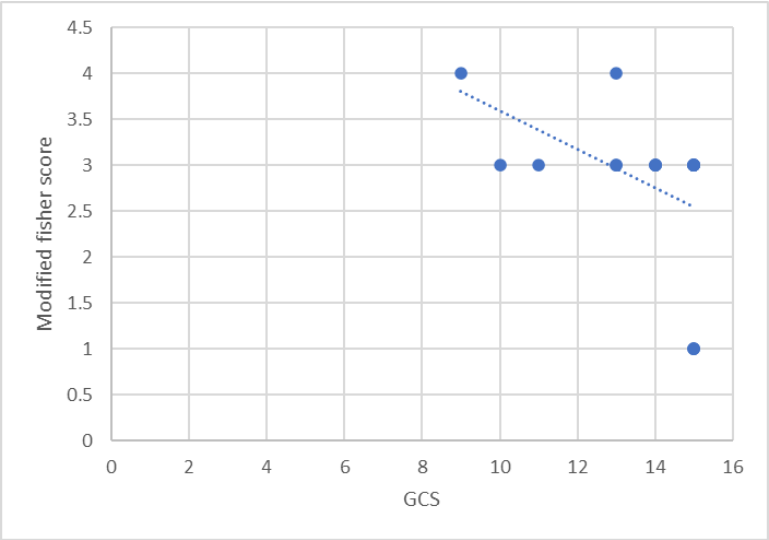

Figure (3): Correlation between Modified fisher score and GCS score on admission.

The mean Modified Rankin scale (MRS) improved over time from 0.80 ± 1.54 at 2 weeks to 0.55 ± 1.05 at 3 months and further to 0.20 ± 0.52 at 6 months & this difference was statistically significant (p=0.008). MRS score was significantly improved with time (P=0.034), as score 0 was significantly higher at 6 months (85%) compared to 65% and 70% at 2 weeks and 3 months respectively, whereas score 4 not observed either at 3 months and 6 months while 25% of the studied patients had score 4 at 2 weeks (**Table 5).**

Table (5): Changes in MRS in the follow-up periods after management

	2 weeks (n=20)	**3 months (n=20)**	**6 months (n=20)**	**P value**	**Pairwise Comparisons**
MRS	0.80 ± 1.54	0.55 ± 1.05	0.20 ± 0.52	**p=0.008***	p2 = 0.059, **p3 = 0.042*** **p4 = 0.038***
Score 0	13(65.0%)	14 (70%)	17 (85%)	**P=0.034***	----
Score 1	1 (5.0%)	1 (5.0%)	2 (10%)		
Score 2	0 (0%)	3 (15%)	1 (5.0%)		
Score 3	1 (5.0%)	2 (10%)	0 (0%)		
Score 4	5 (25%)	0 (0%)	0 (0%)		

Data are presented as frequency (%). MRS: Modified Rankin scale * for significant p value (<0.05).

DISCUSSION

In our study, the demographic data of the studied subjects showed that the average age of participants was 55.30 years. The majority of the subjects were males (65.0%). In the study, a slightly higher percentage of participants were non-smokers (55.0%) compared to smokers (45.0%). **Nassar *et al.*** [12] showed in aneurysmal SAH patients in which the age of patients 50.6 ± 9.8 years, however the study disagreed to ours as they found that patients included 23 (60.5%) females, 15 (39.5%) males. Furtherly, in a study by **Pavelka *et al.*** [13] revealed that 386 patients (49.7%) developed vasospasm within the first 3-21 days following aneurysmal sub arachnoid hemorrhage (ASAH). **Slettebø *et al.*** [14] found that there were about 58.2% current smokers.

In our study total of 75% of patient had associated comorbidities, more than half of the participants had hypertension (55.0%), while a smaller proportion had diabetes mellitus (15.0%) and rheumatoid arthritis (5.0%). **Nassar *et al.*** [12] who noted that in 38 aneurysmal SAH patients, 18 (47.4%) were hypertensive and 7 (18.4%) were diabetics (total of 65.8%)**.**

The clinical presentation of the studied subjects revealed that most participants had intact speech (70.0%). DCL was present in 40.0% of patients. Vomiting occurred in only 50.0% of subjects, while headache was the main symptom, affecting 80.0% of participants. The mean GCS was 13.75 with a median of 14.50. The mean Hunt and Hess grade was 1.70. Among the studied patients, 7 (35%) of studied cases had Hunt & Hess grade 1, 10 (50%) of studied cases had Hunt & Hess grade 2 & 3 (15%) of studied cases had Hunt & Hess grade 3. Moreover, **Rouanet and Silva** [15] claimed that the most frequent presenting symptom is a headache, which is typically characterised as the worst headache one has ever experienced. This headache is sudden & peaks in intensity in no more than an hour. Ten to forty percent of studied cases get a warning leak or sentinel episode, which is a similar headache that occurs 2 to 8 weeks before the bleeding. The chance of death or impairment increases fourfold when a sentinel episode occurs, however none of the studied cases in our research showed these warning indicators. seventy-seven percent of instances result in nausea & vomiting, fifty-three percent in loss of consciousness, thirty-five percent in meningismus & ten percent in localised impairments.

In our study group the Modified Fisher Score distribution showed that the majority of subjects had a score of 3 (70.0%), while 15.0% of subjects had score of 1 and also 15.0% of subjects had score of 4. All subjects underwent a range of imaging investigations, including CT brain, cerebral CT angiography, and diagnostic cerebral angiography. **Zhao *et al.*** [16] in their multi-center observational study showed that 305 (34%) of patients had modified Fisher grade of 3 & 4.

In our study all patients had radiological vasospasm and 50% represented with clinical vasospasm. **Donaldson *et al.*** [17] found that the incidence of cerebral arterial vasospasm following aneurysmal SAH varies widely from nine to ninety three percent.

In our research the most common aneurysm site in this group was ACOM aneurysm, seen in 50.0% of patients. Other types like MCA aneurysms were observed in 25.0% of cases, while less common aneurysms, including those in the basilar tip and PCOM were present in small percentages of the study population. **Sattari *et al.*** [18] study showed that ACOM was the most common site for both ruptured & unruptured aneurysms, & ACOM aneurysms account for 37% of all intracranial aneurysms which corelates with our results. On the other hand, **Nassar *et al.*** [12] had different results where angiography showed that the ruptured aneurysmal sites were the anterior communicating (23.7%), MCA (36.8%), and posterior communicating (21.1%).

Concerning the complications, 15.0% of subjects had early convulsions and there was no late convulsions as all patients received a prophylactic dose of antiepileptics and 15.0% had acute HCP, which was managed with EVD. Zero% of the patients had chronic hydrocephalus due to the low grade of the subarachnoid hemorrhage, rebleeding and death was zero% in our patient group due to early intervention where the cases that were referred to our center were in good condition with low Hunt and Hess grades and small sample size.

Delayed ischemic neurological deficit and infraction was observed in 35.0% of the participants, while acute cerebral infarction with resultant permanent focal neurological deficient didn't occur in our cases as the severity of the bleeding was high (low grade fisher). In one of our cases a blood clot formed during deployment of the coils in the aneurysm with was dislodged in the contralateral A2 and resulted in front lobe infarction (hemiparesis & aphasia), the patient received intraoperative rapid acting antiplatelet, which helped in the restoration of the circulation in the anterior cerebral artery territory and follow up of the case revealed complete recovery after 3 months. In addition, **Vasconcellos de Oliveira Souza *et al.*** [19] demonstrated that 92% of patients with aneurysmal SAH experienced at least one complication, including hydrocephalus (34.4%), intracranial hypertension (33%), infection of unknown origin (20.8%), hypernatraemia (20.8%), hyponatraemia (19.8%), delayed cerebral ischemia-related infarction (18.7%), pneumonia (18.4%), acute kidney injury (16.5%) and seizures (11.8%). **Germanwala AV *et al.*** [20] studied 473 cases with aneurysmal SAH. Just thirteen percent of their studied cases had symptomatic hydrocephalus compared to nineteen percent who had radiographic hydrocephalus, of which only about two-thirds were symptomatic. Additionally, in the early phases, about half of their studied cases with symptomatic hydrocephalus

recovered on their own. Additionally, they noted that a small percentage of studied cases with aneurysmal SAH benefit from either temporary or permanent CSF diversion. Of thirty-two studied cases treated with external ventricular drainage or a shunt, seventy-eight percent showed improvement.

In terms of medical treatment, all subjects (100%) received nimodipine via endovascular injection. Among them, follow-up treatment included intravenous administration for 35% of subjects and oral administration for 65% the patient received nimodipine for 21 days after the initial bleeding, which led to improvement of the vasospasm in 95% of patients and didn't leave a permanent neurological deficit. As for the management approach of aneurysms, we adopted the technique of endovascular intervention where coiling was the dominant intervention (85.0%), and stent assisted coiling in 15% of subjects. Management-associated complications were noted in 55.0% of patients, with various medical treatments (EVD and medical management) being applied as needed. Furthermore, **Hao *et al.*** [21] assured our observation as they stated that nimodipine was used to control cerebral VSP, which is one of the major reasons for severe disability & mortality in studied cases with aneurysmal subarachnoid hemorrhage.

In our study, the mean GCS improved from 13.75 ± 1.80 at baseline to 14.55 ± 0.89 at 2 weeks & this difference was statistically significant (p = 0.011). **Chalard *et al.*** [22] conducted a retrospective study where they supported our results as they revealed improvement of GCS of patients with aneurysmal SAH gradually after management.

The mean MRS improved over time from 0.80 ± 1.54 at 2 weeks to 0.55 ± 1.05 at 3 months and further to 0.20 ± 0.52 at 6 months and this difference was statistically significant (p=0.008). MRS score was significantly improved with time (P=0.034), as score 0 was significantly higher at 6 months (85%) compared to 65% and 70% at 2 weeks and 3 months respectively, whereas score 4 not observed either at 3 months or 6 months while 25% of the studied patients had score 4 at 2 weeks. **Shen *et al.*** [23] observed improvement of MRS over time in studied cases with aneurysmal SAH.

LIMITATIONS: The research was limited by a single-center study, a short follow-up time & a very small sample size, all of which ultimately reduced the statistical power of the analysis & made the findings less generalisable.

CONCLUSIONS

Vasospasm following aneurysmal SAH can be devastating and thus prevention and treatment are crucial. Our study casted the light on the associated comorbidities like DM and high prevalence of hypertension between studied cases with aneurysmal subarachnoid hemorrhage. Nimodipine is an effective therapy for managing vasospasm following aneurysmal subarachnoid hemorrhage. Therefore, further investigations with larger and variable sample size for more accurate results, longer duration of follow-up to detect the long-term possible complications and multi-center study are recommended.

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Conflict of Interest: Nil.

ACOM	Anterior communicating artery,
aSAH	Aneurysmal sub arachnoid hemorrhage
CSF	Cerebrpspinal fluid
CT	Computed tomography
CTA	CT angiography
DCI	Delayed cerebral ischemia
DCL	Disturbance of consciousness level
DIND	Delayed ischemic neurological deficit
DM	Diabetes mellitus
DSA	Digital subtraction angiography
EVD	External ventricular drain
GCS	Glasgow coma scale
HCP	Hydrocephalus
HTN	Hypertension,
IV	Intravenous
Lt PICA	Left posterior inferior cerebellar artery,
MCA	Middle cerebral artery,
MRI	Magnetic resonance imaging
MRS	Modified Rankin scale
PCOM	Posterior communicating artery,
RA	Rheumatoid arthritis
SAH	Subarachnoid hemorrhage
VSP	Vasospasm

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