"MULTIDETECTOR COMPUTED TOMOGRAPHIC ANGIOGRAPHY OF CEREBRAL VESSELS IN THE EVALUATION OF SUSPECTED NON-TRAUMATIC SUBARACHNOID HAEMORRHAGE-HOSPITAL BASED OBSERVATIONAL STUDY"

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ABBREVIATIONS

ACA	**:**	Anterior cerebral artery
ACOM	**:**	Anterior communicating artery
BA	**:**	Basilar artery
BBB	**:**	Blood Brain Barrier
CoW	**:**	Circle of Willis
CSF	**:**	Cerebrospinal fluid
CT	**:**	Computed tomography
CTA	**:**	Computed tomography angiography
DCI	**:**	Delayed cerebral ischemia
DSA	:	Digital Subtraction Angiography
FLAIR	**:**	Fluid attenuated inversion recovery
H & H	**:**	Hunt and Hess scale
HC	**:**	Hydrocephalus
HU	**:**	Hounsfield unit
IA	**:**	Intracranial aneurysms
ICA	**:**	Internal carotid artery
ICH	:	Intracerebral haemorrhage
ICP	**:**	Intracranial pressure
ISUIA	**:**	International study of unruptured intracranial aneurysms
IVH	**:**	Intraventricular haemorrhage
MCA	**:**	Middle cerebral artery
MDCT	**:**	Multidetector computed tomography

MIP	**:**	Maximum intensity projections
MPR	**:**	Multiplanar reconstructions
MRA	**:**	Magnetic resonance angiography
N/D ratio	**:**	Neck / dome ratio
PCA	**:**	Posterior cerebral artery
PCOM	**:**	Posterior communicating artery
SA	**:**	Subarachnoid space
SAH	**:**	Subarachnoid haemorrhage
VRT	**:**	Volume rendering technique

ABSTRACT

Subarachnoid hemorrhage is a serious neurological condition. About 85% of non-traumatic SAH are secondary to intracranial aneurysms rupture. It is associated with higher mortality of approximately 50 % overall and high survivor morbidity. Early detection and prompt treatment are necessary to reduce the disability.

CT brain is the preliminary investigation to confirm the presence of SAH in clinically suspected cases. Digital Subtraction Angiography (DSA) is considered the gold standard investigation for evaluating the exact source of bleeding and site of intracranial aneurysms. However, the invasiveness of the procedure and associated risk of permanent neurological complications is a necessity for alternate less invasive investigations.

Computed Tomographic Angiography (CTA) is a non-invasive and relatively less expensive procedure that can be performed in short time duration. Thus, it can be used as a primary imaging tool for evaluation of cerebral vascular diseases including aneurysms. The advantage also being, the study can be done as continuity of the initial brain CT for immediate diagnosis.

Objectives of the study:

1. To detect and characterize the intracranial aneurysms in patients with non-traumatic subarachnoid hemorrhage by computed tomographic angiography of cerebral vessels.
2. To determine the association between size, location and multiplicity of aneurysm and their effect on aneurysm rupture

MATERIALS AND METHODS

This is a prospective observational study carried out on 45 patients who underwent computed tomographic angiography of the cerebral vessels over a period of 1 year

duration at KLE's Dr. Prabhakar Kore Hospital & MRC, Belagavi. There were 20 males and 25 males, their ages ranged from 14 to 78 years with mean age of 54.8 years. CTA were interpreted to assess the presence of aneurysms and their morphological characteristics. The findings were analyzed to find the association between aneurysm size and location with their rupture risk.

RESULTS

Total of 38 aneurysms were detected in 33 of the 45 patients of which 5 patients had 2 aneurysms each and rest had solitary aneurysm. In 12 of the 45 patients no aneurysm or other identifiable causes of SAH were identified. MDCT excellently delineated the number, size, location and multiplicity of aneurysms. Of the 38 aneurysms, 31 were in the anterior circulation and 7 in the posterior circulation. Among the 31 aneurysms seen at the anterior circulation, there were 11 at anterior communicating artery, 8 at internal carotid arteries, 6 each at anterior cerebral and middle cerebral arteries. Among the 7 aneurysms seen at the posterior circulation there were one each at top of the basilar artery, basilar & posterior cerebral arteries and 2 each at the posterior inferior cerebellar & vertebral artery. Of the 38 ruptured aneurysms there were 2 (5%) giant aneurysms, 1(2%) large aneurysm, 19(50%) medium aneurysms, 10(26%) small aneurysms and 6(15%) very small aneurysms. The smallest aneurysm identified by MDCTA was 2.2 mm, and the largest was 27 mm (mean size of 6.31 mm). The N/D ratios of the aneurysms were calculated in 36 aneurysms and there were 6 aneurysms with narrow neck and 30 aneurysms with wide neck.

INTERPRETATION AND CONCLUSION

- This study showed MDCTA to be a highly useful investigation in the evaluation of patients with spontaneous non traumatic subarachnoid hemorrhage.
- Need for minimal patient co-operation, noninvasiveness of the procedure, quick scan duration and wide availability of the CT scanners make it the most suitable investigation in the acute setting of SAH.
- Majority of the ruptured aneurysms in our study were of small to medium size (<12 mm). The most common site for aneurysm rupture was anterior communicating artery.

  Hence, small aneurysms should not be considered safe and have to be considered for treatment specially when they are located at vulnerable sites like ACOM.
- The study also showed that size and location of aneurysm play an important role in their rupture risk and thus provide useful guide in the management of patients with aneurysmal SAH

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INTRODUCTION

Subarachnoid hemorrhage (SAH) is an emergency neurological condition that is characterized by presence of extravasated blood in the subarachnoid space which is an intermediate layer between the pial and arachnoid membranes of the meninges, which is normally filled with cerebro spinal fluid (CSF).

SAH most commonly results from head injury usually following road traffic accidents. Apart from trauma, the most frequent source of SAH is ruptured intracranial aneurysm that accounts for 80% of non-traumatic SAH [1]. SAH occurring secondary to aneurysmal rupture is a serious condition and is associated with higher mortality of approximately 50 % overall and high survivor morbidity despite various advanced treatment options. Higher mortality rates are associated with SAH and it contributes to about 5-10% of all cerebrovascular strokes [2].

About one third of survivors with subarachnoid hemorrhage are left with neurological deficits and require lifelong medical support. Low level of consciousness at admission, higher age of patient and thick subarachnoid clot at primary head computed tomography (CT) scan are poor prognostic factors [3,4].

There is widespread variation in the incidence of SAH from region to region. According to a systemic review the overall incidence was found to be about 9.1 per 100 000 person-years. The overall incidence ranged from 2 to 25 per 100 000 person-years [5].

Increased occurrence of aneurysmal subarachnoid hemorrhage has been observed in females. Female to male ratio is observed to be 2:1 [6].

Multiple intracranial aneurysms are noted in 15% to 20% of all cases. There is strong female predominance with multiple aneurysms. No definite identifiable cause has been found for intracranial aneurysm formation. A wide range of inherited and acquired factors predispose to their development and rupture. Occurrence of multiple aneurysms is related with vasculopathies such as Fibro muscular dysplasia besides various other connective tissue disorders. There is 10% incidence of associated aneurysms in Polycystic kidney disease.

The distinctive clinical presentation of SAH is abrupt onset, severe headache (thunder clap headache) that is usually accompanied by nausea, vomiting, neck stiffness or loss of consciousness. In the absence of the typical signs and symptoms SAH may be misdiagnosed as migraine or tension headaches in 50% of cases [7].

CT has been extensively used for initial diagnosis of SAH. If correctly performed, it is possible to identify SAH within 12h of symptom onset in almost 100% of cases and in 93% of cases within 24h [8,9].

Computed tomographic (CT) angiography of the intracranial vessels has also been a routine examination and is fully integrated in the imaging and treatment algorithm of SAH patients in many centers across the world. The greater advantage with CTA is that, it can be done immediately following the initial CT examination, thus reducing the examination period and providing quick diagnosis [10].

This study proposes to evaluate the role of Multi Detector CTA (MDCTA) in the detection of intracranial aneurysms causing non-traumatic SAH and also correlate their risk of rupture based on their size, location and multiplicity.

AIM AND OBJECTIVES OF THE STUDY:

AIM:

The aim of the study is to evaluate the site, size, shape and number of cerebral aneurysms detected by computed tomographic angiography of cerebral vessels.

OBJECTIVES:

1. To detect and characterize the intracranial aneurysms in patients with non-traumatic subarachnoid hemorrhage by computed tomographic angiography of cerebral vessels.

2. To determine the association between size, location and multiplicity of aneurysm and their effect on aneurysm rupture

REVIEW OF LITERATURE

ANATOMY

CEREBRAL VASCULAR ANATOMY:

The blood supply to brain is provided by a pair of internal carotid (ICA) and vertebral arteries (VA) which form anterior and the posterior circulation respectively. Intracranial ICA terminates by bifurcating into middle cerebral (MCA) and anterior cerebral arteries (ACA) to form anterior cerebral circulation. Posterior circulation is constituted by VA of either sides that fuse to form the basilar artery (BA) at the base of the pons which terminates into two posterior cerebral arteries (PCA).The vessels forming the anterior and posterior circulation of either sides are connected through the communicating arteries which together form a complete circle, circle of Willis (CoW).

Theoretically, this circle maintains a continuous blood supply into the affected cerebral parenchyma by forming pial collaterals in the event of trauma or diseases that affect one or more arteries supplying the territory.

CIRCLE OF WILLIS:

Thomas Willis is one of the greatest neuro anatomists of all time. He is greatly remembered for his description of the CoW as the arterial polygon at the base of the brain [11].

Though, Thomas Willis was not the first to describe the CoW, but he was the first to provide the functional significance and complete description of CoW and of this anastomosis as a safety measure against occlusive disease.

ANATOMY OF CIRCLE OF WILLIS:

A ring like interconnecting arterial polygon is formed by the arteries that are located at the base of brain to form the CoW. CoW encircles the pituitary stalk and is placed just anterior to the diencephalon adjacent to the intracranial portion of optic nerves, optic chiasm & optic tracts. Various variations are noted that include hypoplastic arteries or absent parts of the circle.

CoW plays major role in establishing connection between anterior and posterior circulation. ICA arises as terminal branch of common carotid artery from the bifurcation of carotid bulb at the level of C3 to C5 vertebrae, it then ascends up in the neck to enter the cranium through the carotid canal, a bony passage within the petrous temporal bone. It then bifurcates intracranially into anterior cerebral artery (ACA) and middle cerebral artery (MCA) bilaterally. Right and left ACA are connected by anterior communicating artery (ACOM) and the segment of ACA that is prior to the ACOM is labelled as A1 segment and subsequently followed by the A2, A3, A4 and A5 segments and the latter are also called as pericallosal segments. Both the ICAs, their two terminal branches and together with ACOM constitute the anterior cerebral circulation.

The posterior circulation comprises of bilateral vertebral arteries (VA), that arise from first segment of the subclavian arteries, traverse through the foramen transversarium in the cervical vertebrae and enter the cranium through the foramen magnum and at the base of pons both fuse to form basilar artery (BA). Basilar artery then ascends up in the pre pontine cistern and terminates at the upper border of pons by bifurcating into two posterior cerebral arteries (PCA).

The posterior communicating artery (PCOM) bilaterally connects the PCA to the internal carotid arteries, thus establishing the connection between posterior and anterior circulation.

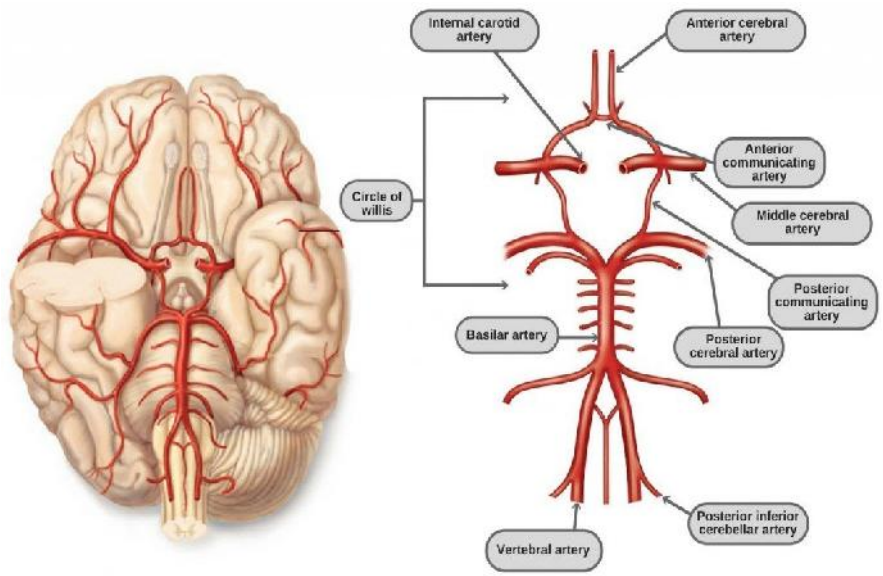


Figure 1: Schematic diagram of Circle of Willis

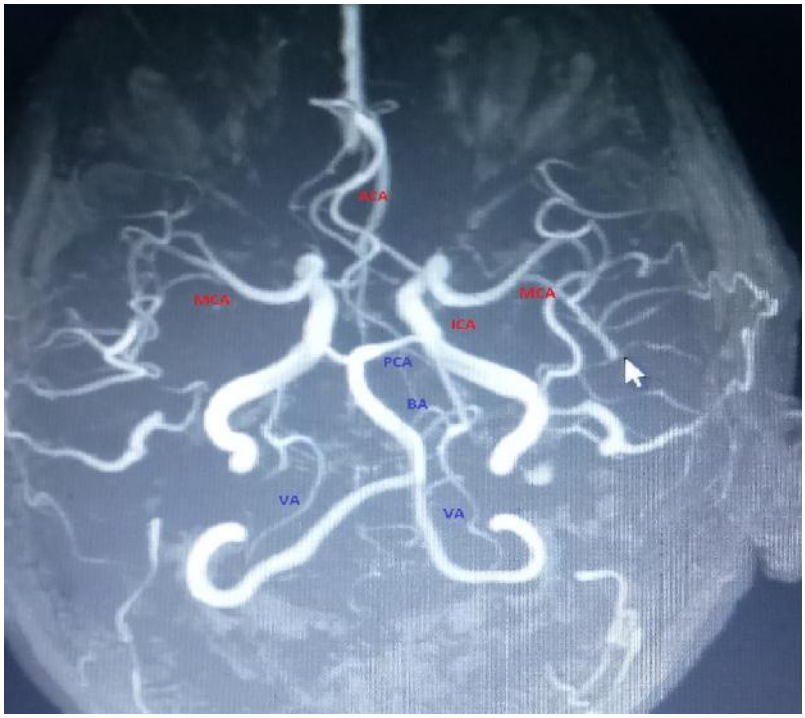


Figure 2: Angiographic anatomy of vessels forming the Circle of Willis

Intracranial arteries are divided into various anatomical segments and each arterial segment in the circle of Willis is named after their parent artery (ACA, MCA, PCA) and they are serially numbered in an increasing number after each division. A1, A2, A3, M1, M2 or P1, P2, P3 and so forth. A rich network of pial vessels interconnect the peripheral branches of these main arteries and thus establish the collateral supply across the territories.

BASIC HISTOLOGY:

The arteries in the brain exhibit similar basic histological features like other arteries throughout the body. The innermost layer is the tunica intima that is a single layer of flat epithelium, the middle layer tunica media is comprised of smooth muscle fibres and fibrous tissue and outer most layer is tunica adventitia that consists of loose connective tissue, vasa vasorum, nerves and fibrous tissue. There are also significant differences in cerebral vessels.

External elastic lamina is absent in intracranial arteries and they have relatively reduced percentage of elastic fibers within the middle and outer histological layers and hence reduced thickness of these layers [12].

This transition occurs at the point where arteries pierce the duramater. Hence, pathologies of the cerebral vasculature are different and one of the interesting example of this is aneurysms. Cerebral aneurysms are formed at points of high shear stress in the vessel wall.

ANATOMICAL VARIATIONS OF THE CoW:

A lot of anatomical variations are frequently observed in the CoW. A complete circle is visualized in only about approximately 45-48% of anatomical

specimens [13]. These variations consist of absence of vessels which may include hypoplasia or aplasia. Aplasia refers to lack of development of a segment of artery, whereas hypoplasia is the incomplete development of the vessel. The most common anomalies noticed are the hypoplasia of the vessels. The most usual sites of hypoplasia are the PCOM, followed by P1 segment, A1 segment and the ACOM with incidence of 10-53%, 6-18%, 2-12% and 2-9% respectively. Another common anomaly is the origin of PCA from the ICA termed as fetal origin, that is seen in 10-22% [13]. About 7% of the specimens show multiple anomalies and the most common is hypoplasia of two vessels. Either it is hypoplastic PCOM and ACA on the same side or hypoplastic PCOM of one side and the opposite side PCA [14].

There are some anomalies that are depicted in several arteries – these include duplication, fenestration and infundibulum.

Fenestration: It refers to the division of the lumen of a vessel into two separate and distinct channels that have their respective endothelial and muscularis layer. The adventitia may or may not be shared.

Duplication: It refers to two distinct arteries that originate separately and show no distal arterial convergence.

Infundibulum: It is a funnel shaped area of dilatation at the origin of an artery and has diameter of less than 2 mm. Clinical importance is that is has to be distinguished from aneurysm.

Anne Osborn in 1999 stated that the CoW is an arterial polygon, specifically a nonagon. The complete CoW consists of its “no part being either hypoplastic or absent” and it comprises of 10 component vessels. Anne Osborn also stated that

anatomic variations in the CoW are a rule, but not an exception. She also described that variants of CoW occur mostly in the posterior part, common site being PCOM.

SUBARACHNOID HEMORRHAGE:

Definition, Etiology and Epidemiology:

Subarachnoid hemorrhage (SAH) is a type of extra axial intracerebral hemorrhage that is characterized by presence of extravasated blood in the subarachnoid space which is intermediate layer between the arachnoid and pial membranes of the meninges, that is normally filled with cerebro spinal fluid (CSF).

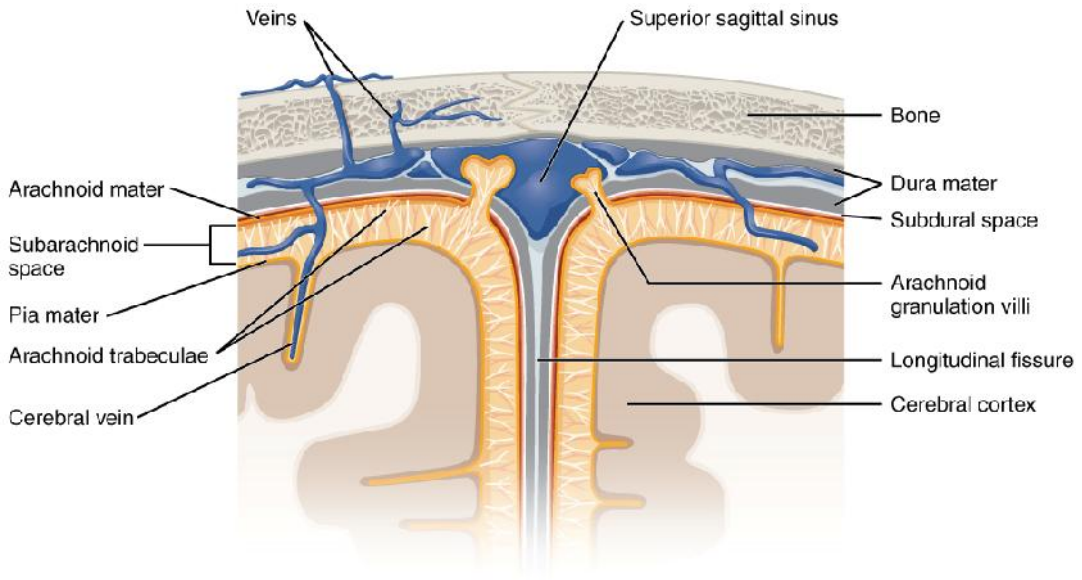


Figure 3 : Graphic representation of cross section through the skull showing different membranes of the brain and subarachnoid space

Etiology

SAH commonly results from trauma to the skull and is a common form of intracranial hemorrhage following head injury. However, SAH can also result from non-traumatic causes and the most common cause being rupture of cerebral

aneurysms in over 85% of cases, non-aneurysmal peri mesencephalic haemorrhage in 10%, and a variety of rare conditions arterio-venous malformations, peri mesencephalic hemorrhage, Dural arteriovenous fistulas, bleeding disorders, arterial dissections and alcohol abuse in 5% [1].

About half of the survivors with subarachnoid hemorrhage are dependent and left with neurological deficits and require lifelong medical assistance [15].

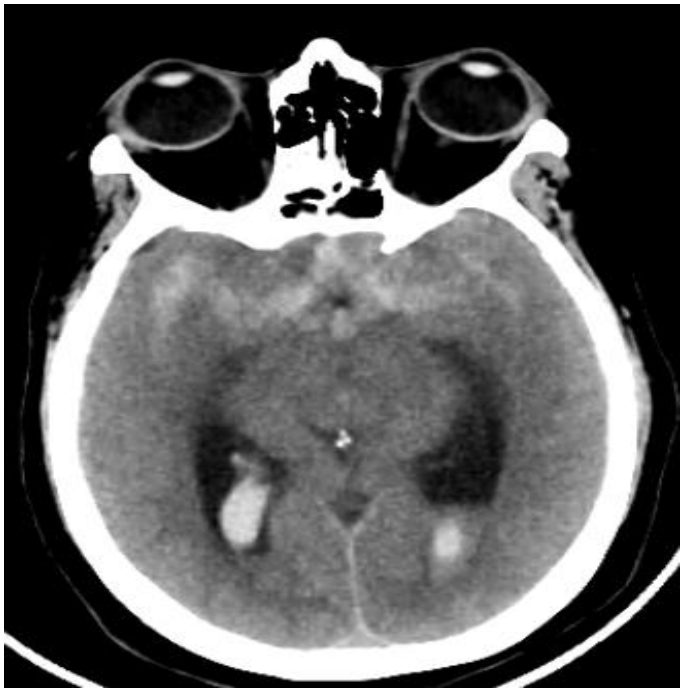


Figure 4 : Axial section of CT brain showing SAH secondary to rupture of ACOM aneurysm in the suprasellar cistern and bilateral sylvian fissures with intraventricular extension of the bleed

EPIDEMIOLOGY:

There exists wide variation in the incidence of SAH with respect to region, race and gender. According to a systemic review that included 51 studies from 21 countries the overall incidence was found to be about 9.1 per 100 000 person-years.

The overall incidence of SAH is about 2 to 25 per 100 000 person-years. There is variation in regional incidences of SAH ranging between 7 and 13 per 100 000 person-years as observed in majority of the studies. Highest incidence rates were observed in Japan and Finland and relatively lower rates were noted in South and Central America. Incidence in Japan is about 22.7 per 100 000 person-years, 19.7 in Finland, 4.2 in South and Central America, and rest of the regions had incidence of 9.1 per 100 000 person-years. Relatively higher incidence in Japan could be explained by genetic, environmental factors and greater older population and lower incidence in South and Central America can be due to relatively younger mean age of the population and other explanation can be due to racial differences in occurrence of SAH [5]. However, no specific explanation could explain the cause for variation in regional incidence of SAH.

Incidence of subarachnoid haemorrhage has remained relatively stable in high income countries over the past four decades. The incidence and proportional frequency of subarachnoid haemorrhage in low to middle income countries are significantly greater than the incidence and frequency in high-income countries.

There is difference in incidence of SAH between genders and is found to be relatively higher in women. This can be attributed to hormonal factors as higher incidence is observed after sixth decade. Female to male ratio is nearly 2:1 [6].

PATHOPHYSIOLOGY:

A series of events occur after the rupture of an aneurysm resulting in severe disturbance of brain function. The blood first spreads into the subarachnoid space, often associated with intraventricular extension. Initially, there is volume loading of

CSF space with blood that rapidly elevates the ICP and it typically reaches peak levels in 30 seconds [17]. This leads to development of blood clots in the cisternal and intraventricular compartments obstructing the cerebrospinal fluid (CSF) outflow and thus resulting in raised intracranial pressure and hydrocephalus.

Vasospasm is one of the most dreaded complication following aneurysmal SAH leading to cerebral ischemia and patient may develop neurological deficits, low level of consciousness and seizures. Following rupture of an aneurysm, there is physiologic response by the vessel to control bleeding. Vasospasm sets in few days after the SAH, typically from 4 to 14 days after the initial haemorrhage due to activation of various vasoactive mediators released from the dissolving blood clot in the subarachnoid space. Within four hours after SAH, there is disruption in blood-brain barrier (BBB) resulting in generalized brain edema [18].

ICH develops in about 20–30% of patients with aneurysmal SAH. Sometimes isolated ICH is noted without SAH [19]. Intraparenchymal hematoma is especially observed following rupture of ACOM aneurysms with hematoma in the frontal lobes.

Occurrence of non-traumatic subdural haemorrhage secondary to rupture of an aneurysm is uncommon, but some cases have been reported [20].

CLINICAL COURSE:

The most typical presentation of SAH is abrupt onset, severe and persistent headache as the first symptom. It is described by the patient as most terrible headache of life (Thunderclap headache) usually coupled with nausea, vomiting, neck stiffness, focal neurological deficits. Case fatality rate secondary to aneurysmal SAH varies between 8·3% to 66·7% and there is decrease in case fatality rate of 0·8% per year[21].

A patient´s neurological condition at admission is graded by The Hunt and Hess grading scale (H&H) which takes into account the symptoms, nuchal rigidity, any neurological deficits and state of consciousness of the patient [22].

The outcome in SAH patients depends on a variety of factors. Patient's clinical condition at admission to hospital, age of the patient, and the quantity of SA blood on the initial head CT are three most important factors [23]. Acute obstructive HC resulting from intraventricular extension of blood is associated with bad prognosis. The outcome is worse when there is associated ICH than in those with exclusive SA blood [24].

Apart from primary SA bleed and cerebral ischemia resulting from vasospasm, rebleeding is the major reason of death and disability [25]. Following the insult, about one- third of patients with SAH continue to be dependent [26].

DIAGNOSIS:

- **RADIOLOGICAL INVESTIGATIONS**

Computed tomography

CT imaging of the brain is the most widely performed radiological examinations. It is a quick and easily repeatable investigation and is widely available. It is possible to analyze different tissue components by measuring the density of CT voxel data and Hounsfield units (HU).

CT has a great accurateness to identify SAH and also provides extra information about parenchymal and other hemorrhage. On CT, fresh blood appears as hyperdense (white) area that approximately has HU of 60–75 units and thus provides

good contrast with adjacent tissues. CT also provides details regarding the various consequences of SAH such as HC, re-haemorrhage and ischemia. It is also a modality for follow up examination of SAH patients. Streak artifacts from surgical clips and coils may reduce the quality of image in follow-up imaging.

The ability of the CT to detect hemorrhage depends on the time interval from the symptom onset, the quantity of SAH, scanner resolution and also the radiologist's skill. False negative scans are observed with elapsing time. In up to 95% of patients, blood will be present in the SA space on the first day. Sidman *et al.* in their study found 100% sensitivity of third-generation CT scanners in detecting SAH when imaging was performed at or before 12 hours after symptom onset. After 12 hours of symptom onset, there was slight decrease in sensitivity to 82% [27].

Modern day CT scanners have greater sensitivity than earlier scanners. Lumbar puncture can be done to exclude SAH in the event of normal CT diagnosis [28]. Negative results of both CT brain and lumbar puncture rule out SAH [29].

GRADING SCALE FOR DISTRIBUTION OF SUBARACHNOID HEMORRHAGE

SAH is graded based on axial images of non-contrast CT. All the grading scales estimate the amount of SAH based on the thickness of SA blood.

The grading of SA blood helps to predict symptomatic vasospasm and ischemic events.

A grading scale was presented by Fisher et al. to estimate the amount of SAH to predict vasospasm. It is a well-established method and in use world-wide [30].

However, The Fisher grading scale did not differentiate ICH and IVH, thus faced criticism. A new revised grading scale was proposed by Claassen *et al.* (2001) based on the Fisher scale. It took into consideration the independent predictive value of SA blood and IVH for cerebral ischemia [31].

Table 1: FISCHER REVISED SCALE

Grade	Description
0	No SAH or IVH
1	Minimal/thin SAH, no IVH in either lateral ventricle
2	Minimal/thin SAH, with IVH in both lateral ventricles
3	Dense SAH, * no IVH in either lateral ventricle
4	Dense SAH, * with IVH in both lateral ventricles

*Completely filling ≥1 cistern or fissure

Magnetic resonance imaging

Owing to its high cost, less availability and also difficulties with restless patients, Magnetic resonance imaging (MRI) is thought to be of minimum value in the evaluation of acute SAH. However, later in the subacute phase, MRI is superior to CT and it also provides additional information about ICH, especially in those with normal CT and suspected subacute SAH. Fluid attenuated inversion recovery (FLAIR) sequence is found to be sensitive for diagnosing SAH which show sulcal hyperintensity [32]. Corresponding areas show signal dropout(blooming) on SWI sequence.

- **CEREBRAL ANEURYSMS:**

Aneurysms are pathological focal dilatations of the vessels of brain arterial system that are prone to rupture. A systematic review data of various autopsy studies from numerous countries has reported a prevalence of 0.4% and 3.6% in retrospective and prospective studies respectively [33].

These vascular abnormalities are explained by following presumed pathogenesis.

- Saccular or berry aneurysms comprise 90% of all cerebral aneurysms and are positioned at the bifurcation of the main arteries.
- Dolichoectatic, fusiform, or arteriosclerotic aneurysms constitute 7% of all cerebral aneurysms and these are abnormal elongated dilatations of proximal arteries.
- Mycotic aneurysms comprise 0.5% of all cerebral aneurysms, caused secondary to infections and are situated peripherally.
- Other types include neoplastic aneurysms and traumatic aneurysms

About 30% of cerebral aneurysm patients will have multiple aneurysms. Most of them are small and asymptomatic and present only after they suffer.

85% percent of saccular aneurysms develop in the CoW and they often rupture into the SA that account for 70 – 80% of non-traumatic SAH.

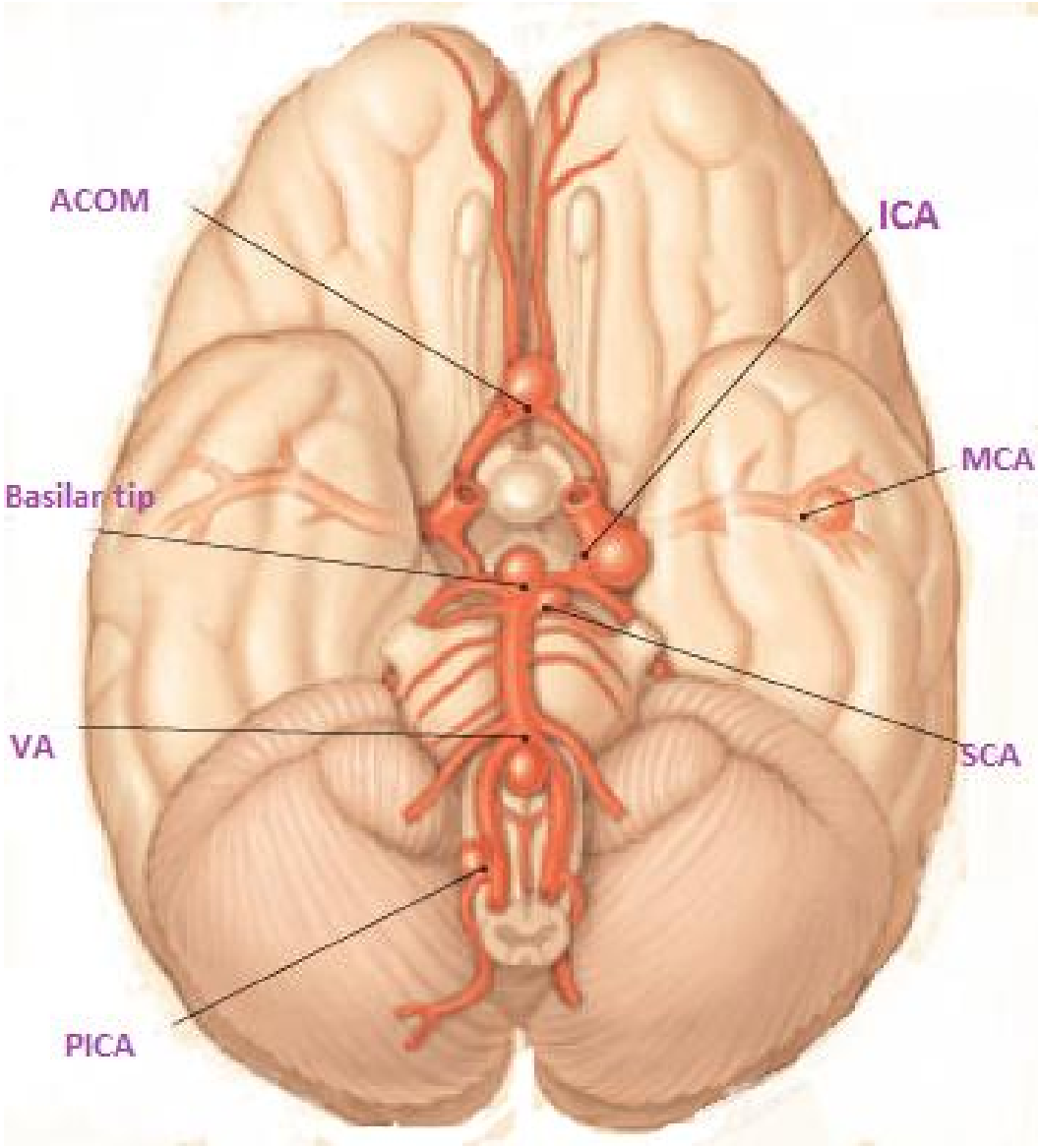


Figure 5: Schematic diagram showing most common sites of cerebral aneurysm

The most common site is the ACOM compromising of 35%, followed by ICA (30%-including the carotid artery, PCOM, and the ophthalmic artery), the MCA (22%), and lastly, the posterior circulation sites, most common being the top of the basilar artery [34].

RISK FACTORS:

Various factors predispose an individual to the development of aneurysms and their rupture.

These can be grouped as inherited and acquired factors or as Modifiable/ Non modifiable risk factors. Important non modifiable risk factors include genetic factors and various connective tissue disorders which account for about 5% cases [35.] Increasing age and family history of SAH predispose to aneurysm rupture. Increased frequency is observed in females across the various age spectrum and aneurysm rupture is about 1.6 times commoner in females than males. These predisposing conditions result in aneurysm formation and rupture by injury to vessel wall, preventing wall injury repair, and increased hemodynamic stress.

Non modifiable / Inherited risk factors include:

Various autopsy studies have shown increased risk of aneurysm formation in individuals with different inherited disorders. These include

- Autosomal dominant polycystic kidney disease
- Coarctation of the aorta
- Anomalous vessels
- Fibromuscular dysplasia
- Connective tissue disorders (Ex:Marfans and Ehlers- Danlos)
- High-flow states (Ex: vascular malformations)

Acquired risk factors include [36]:

- Older age
- Hypertension
- Cigarette Smoking
- Alcohol intake
- Oestrogen deficiency
- Hypercholesterolemia
- Carotid artery stenosis

A systemic review of various studies also showed that the same factors increased the risk of aneurysm growth over period of time and risk of rupture [37.]

Recent studies have also found influence of meteorological factors on aneurysm rupture. In a study by MeiHua li et al, the highest incidence of aneurysmal SAH was noted in winter between January and February and lowest in summer (July). However, these are not considered independent factors in evaluating risk of aneurysm rupture [38].

Morphological factors also have great influence on risk of aneurysm rupture. These include the size of the aneurysm, location, parent artery of origin and also the shape of aneurysm [39].

It is important to evaluate the association between aneurysm location and rupture risk because location of an aneurysm greatly predicts outcome of both surgical and endovascular management.

DIAGNOSIS AND IMAGING FINDINGS:

Different imaging modalities have been utilized for the diagnosis of aneurysmal rupture and to localize the aneurysm. Currently three imaging modalities are widely used and accepted, which include: Digital subtraction angiography (DSA), computed tomography angiography (CTA) and magnetic resonance angiography (MRA).

Conventional angiography or DSA is the gold standard modality for detecting aneurysms, but it is an invasive procedure and can be time consuming. With Modern catheter angiography there is overall 1% risk of transient and 0.5% risk of permanent neurological complications [40]. At any rate, overall re-rupture of the cerebral aneurysms occurs in 1-2% of cases during the angiography (DSA) procedure.

Other imaging modalities for detecting cerebral aneurysms are MR angiography (MRA) and CTA (CTA). MRA is safe, but less appropriate in the acute stage, because patients are usually restless or need extensive monitoring in the acute stage.

A review of studies that compared MRA and CTA in patients with recent SAH, showed a sensitivity of MRA to be 80% and specificity of 87% nearly similar to CTA. No significant difference was found between CTA and MRA for intracranial aneurysm detection [41]. Despite of its limitations, MRA is a feasible tool to detect aneurysms in relatives of SAH patients because of its non-invasive nature.

CT Angiography

CTA of the circle of Willis has proved to be an effective means of evaluating aneurysms and adds only little additional time to the routine unenhanced CT examination. It is a minimally invasive imaging technique that does not require arterial puncture or catheter insertion as in DSA, and the advent of MDCT have led to increased spatial resolution with shorter acquisition times. Furthermore, scan images from CTA can be viewed in unlimited projections, simplifying the aneurysm detection and characterization.

Various 2D and 3D post processing techniques aid in detecting the aneurysms. These include volume-rendering technique (VRT) as well as maximum intensity projections (MIP). Three- dimensional image post processing techniques like MIP demonstrate maximum arterial attenuation in the image i.e. contrast-filled arteries.

A meta-analysis of 21 studies that included 1251 patients showed a sensitivity of 93.3% and specificity of 87.8% for CTA when compared with DSA [42].

On the additional hand, MDCTA can detect aneurysms that were missed by conventional angiograph. Most of the centres are now successfully operating on ruptured aneurysm with CTA as the only imaging method [43].

In 1991, Shigeki Aoki et al studied intracerebral aneurysms and reported that all the aneurysms could be seen well on 3D-CTA. They reported that the reconstructed images from different directions were helpful in detailed evaluation of the direction of aneurysm, its proper shape, the size of neck and its connection with artery of origin. In cases of giant aneurysms, CTA was found superior to conventional angiography as dense opacification of the aneurysm superimposed on adjacent vessels

made identification of the neck of aneurysm difficult in conventional angiography. 3D CTA afforded several different angles to visualize the neck. Also, 3D CTA is useful in this situation to help determine a suitable angle on conventional angiography to demonstrate the neck and small vessels. The drawbacks of CTA quoted in the study-included difficulty in demonstrating small vessels. Patient movement also interfered with the quality of images. Hence, they recommended CTA as a compliment to DSA rather than as a replacement [44].

MANAGEMENT

In considering the management of aneurysms their risk of rupture must be weighed against the risks associated with the treatment options. Clear evidence-based strategies are lacking in the appropriate management of unruptured aneurysms. Three main available treatment options include: observation, endovascular coiling, and surgical clipping. Non-invasive serial monitoring can be done with MRA or CTA.

Endovascular treatment is safe relative to surgical treatment and which includes guiding a catheter into the cerebral vasculature through the femoral artery and via the ICA or vertebral artery. Under fluoroscopy guidance, as and when the catheter reaches the site of aneurysm, soft platinum coils will be deployed in the lumen of the lesion. These coils obliterate the lumen, initiate thrombus formation and thus occlude the aneurysm, and hence prevent future rupture [45]. But, wide neck and large size of the aneurysms provide difficulty for endovascular management.

Surgical clipping is used for treating intracranial aneurysms since long time. After craniectomy, a surgical clip is inserted at the junction of the normal artery and neck of the aneurysm. This method is found to be very effective [45]. However, it is an

invasive procedure and is associated with a greater risk of complications like any other surgery.

LITERATURE REVIEW

W Brinjikji et al [46] conducted a met analysis to examine the risk factors for growth of intracranial aneurysms and also examined the association between their growth and rupture.

They analyzed 21 studies that included 3954 patients with 4990 aneurysms. Various factors like age older than 50 years ($p < 0.01$), female gender ($p<0.01$), history of smoking ($p < 0.01$), non-saccular shape of aneurysm and aneurysm size were associated with higher growth rates. A rupture rate of 3.1% per year was noted with growth of aneurysm when compared to 0.1% rupture risk for stable aneurysms ($p < 0.01$). Hence, it is important to consider multiple anatomic and clinical risk factors in counselling the patient regarding the natural history of unruptured intracranial aneurysms.

Guang-Xian Wang et al [47] did a retrospective study that involved 379 patients that had 441 aneurysms and tried to analyze the potential risk factors to predict the aneurysmal rupture. Aneurysms located at ACOM, at the bifurcation site of arteries, wall irregularity, presence of daughter sac, aneurysm height, aspect ratio (AR), height to width ratio had positive correlation with rupture risk. Irregular aneurysm wall, bifurcation site of artery and high AR were analyzed with multivariate logistic regression model and they revealed increased rupture risk, whereas cerebral atherosclerosis, aneurysms at ICA and greater neck-width decreased the rupture risk

of aneurysm. The explanation can be that cerebral atherosclerosis or calcified walls result in slow flow rates entering the aneurysm and decrease the shear stress on walls.

Yasser Orz et al [48] conducted a study in order to determine the correlation between aneurysm size, location, multiplicity and their effect on rupture of aneurysm. They evaluated 81 patients that harbored 104 aneurysms. Retrospective analysis of various charts and radiological findings for aneurysm size, location, multiplicity and presentation was done. They concluded that aneurysm size and location have considerable role on their rupture risk. They also found that most common location of rupture of small aneurysms as ACOM and MCA was the commonest site for small unruptured aneurysms. As per their data majority of aneurysms ruptured at smaller size and no definite threshold size was found.

Ohashi Y et al [49] retrospectively evaluated the characteristics of small ruptured aneurysms in 280 patients. They examined the relationship between the size and location of ruptured intracranial aneurysms. In their series, the mean diameter of ruptured intracranial aneurysms was 7.6 mm. Aneurysms at ACOM and ACA ruptured at significantly smaller sizes than ICA or MCA. Patients with or poorly controlled hypertension had their aneurysms ruptured at significantly smaller sizes (mean 6.5 mm). Hence, they concluded that treatment decisions of unruptured aneurysms should not be only dependent on size of aneurysms. Even smaller aneurysms at vulnerable sites like ACOM and ACA tend to rupture. There is higher risk of rupture of small aneurysms in hypertensive patients.

Huibinkang et al [50] utilized self-controlled model to analyze the rupture risk of intracranial aneurysms by comparing the difference between two aneurysms in different locations within the same patient. 103 patients diagnosed with intracranial aneurysms between January 2011 and April 2015 were included in the study. Each patient had one ruptured and one unruptured aneurysm. Each aneurysm was evaluated for the presence of daughter sac, aneurysm neck, parent artery diameter, aneurysm height, width, location, the aspect ratio (maximum perpendicular height/average parent artery diameter) and width/height ratio.

By multivariate analysis, they identified that presence of daughter sac, maximum aneurysm height of >7mm, location of aneurysm at ACOM or PCOM were significantly associated with their rupture.

The usual view amongst neurosurgeons is that there is higher chance of aneurysm rupture with larger size of the aneurysm implying that very small aneurysms rupture rarely.

Parviz Dolati et al [51] in their study concluded that small aneurysms <5 mm are common causes of aneurysmal subarachnoid hemorrhage.

Hence it is highly recommended to look for other risk factors when they are found ruptured even before simply leaving them alone.

NON-ANEURYSMAL CAUSES OF NON TRAUMATIC SAH:

About 15 % of the spontaneous subarachnoid hemorrhages are secondary to various non aneurysmal causes.

These include

- Rupture of arterio venous malformation
- Perimesencephaic hemorrhage-This has been termed as angiography negative SAH as no identifiable cause can be found on CT angiography or either on DSA. It has good long-term outcome.
- Cerebral venous thrombosis
- Vasculitis
- Coagulation disorders and intake of various blood thinning agents

MATERIAL AND METHODS:

Source of data: Patients that are referred for CT Angiography of cerebral vessels to Department of Radio-Diagnosis at the KLE's Dr. Prabhakar Kore Hospital & MRC, Belagavi.

Method of collection of data:

(a) Study design: Hospital based prospective observational study.

(b) Sample size: Using the formula

$n = 4pq/d^2$

where p is prevalence of intracranial aneurysms (3.6%)

q is (1-p)

d is absolute precision of 6

The sample size is calculated to be 39.

The study will comprise of patients presenting with features of spontaneous subarachnoid hemorrhage who will be referred to the department during the period of study for CT angiography of cerebral vessels.

DURATION: January 1st 2018 to December 31st 2018.

(c) Inclusion criteria:

1. Patients of spontaneous subarachnoid hemorrhage suspected clinically or detected by CT Brain scan and referred to radiology department for cerebral angiography.

2. Patients who give consent to take part in the study.

(d) Exclusion criteria:

- As the study is primarily aimed at detecting intracranial aneurysms as a cause of subarachnoid hemorrhage, patients with subarachnoid bleed attributable to trauma, rupture of arterio-venous malformations, secondary to use of antiplatelet/ anticoagulant drugs will be excluded.

Ethical considerations: Approval for conducting the study was obtained from the institutional human ethics committee prior to the start of study. Only those participants willing to sign the informed consent were included in the study and Informed written consent was obtained from all the study participants. The risks and benefits associated with the study and the voluntary nature of participation were explained to the participants before obtaining consent. Confidentiality of the study participants was maintained.

Data collection tools: All the relevant parameters were documented in a structured study proforma.

METHODOLOGY:

The subjects were enrolled in the study after obtaining written informed consent for CT angiography of cerebral vessels.

A detailed history was taken in the form of a systematic proforma regarding patient age, sex, symptoms, past medical/surgical history.

After considering the inclusion and exclusion criteria, the patients were taken up for CT angiography of cerebral vessels.

CT angiography of cerebral vessels was done as per standard imaging protocol.

EQUIPMENT

All the patients were subjected to non-contrast CT followed by CT Angiography of cerebral vessels according to the institution protocol using a 64-slice CT scanner (Siemens) and 128- slice GE Revolution scanner with a collimation of 0.6 mm. The Scan range extended from the level of foramen magnum up to the cranial vault. A total of 90-100ml of non-ionic iodinated water soluble contrast, Iohexol (Omnipaque,350 mg I / ml) was injected through the antecubital vein with an 18-20 gauge intravenous cannula by using a mechanical power injector at the rate of 4-5 ml/s. By bolus tracking technique Arterial phase scanning was initiated when a threshold enhancement of 100HU was reached in the arch of aorta. The venous phase was acquired after 60-70 seconds delay following injection of contrast. All images were reconstructed with standard soft tissue algorithm. Source images were

reformatted using maximum intensity projection (MIP) and 3D volume rendered technique (VRT) for analysis.

Statistical Methods: All the qualitative variables are expressed in terms of frequency and proportion. Chi square test was used to analyse the association between the size and location of aneurysm with their rupture risk. Microsoft word and excel were used to generate graphs and tables.

Interpretation and Analysis:

The images were transferred to a work station that runs 3D software for further analysis.

Scans were analysed using the source images and various two-dimensional (2D) and three-dimensional (3D) postprocessing techniques, that included multiplanar reconstructions (MPR), maximum intensity projections (MIP) and volume rendering technique (VRT).

The evaluation of the circle of Willis and associated vascular pathologies was done.

The CTA findings were evaluated using following parameters:

SUBARACHNOID HEMORRHAGE:

Presence or absence of SAH was evaluated using Fisher's revised grading scale. The plain CT images were also assessed for distribution of subarachnoid haemorrhage-diffuse/ focal, presence of any midline shift, intraventricular extension of bleed, development of any hydrocephalus and intraparenchymal haemorrhage.

ANEURYSM:

Post processed images were carefully assessed for the Presence or absence of Aneurysm.

Each identified aneurysm was noted taking into consideration its artery of origin, exact site, aneurysm size, neck of the aneurysm and also its shape.

Anterior circulation aneurysms include those arising from: Anterior cerebral artery, anterior communicating artery, Pericallosal artery, Callosal marginal artery, Middle cerebral artery, Internal carotid artery and Posterior communicating artery.

Posterior circulation aneurysms include those arising from: Basilar artery, Top of basilar artery, Superior cerebellar artery, Posterior cerebral artery, Anterior inferior cerebellar artery and Posterior inferior cerebellar artery.

Aneurysms were classified into giant, large, medium, small and very small based on the following size criteria.

Size:

- Giant: ≥25mm.
- Large: ≥13-24mm.
- Medium: ≥5-12mm.
- Small: 3-5 mm.
- Very small: <3mm.

Aneurysms can be further classified into narrow and wide neck aneurysms based on Neck/Sac diameter.

- Narrow neck Aneurysm: Neck diameter / Sac diameter <1/3.
- Wide neck Aneurysm: Neck diameter / Sac diameter ≥ 1 /3.

RESULTS

Study population:

45 patients with non-traumatic subarachnoid haemorrhage that were referred for CT cerebral angiography were prospectively evaluated in this study.

Age and sex distribution of patients

Of the 45 study subjects, there were 20 males and 25 females. Males comprised 44 % and females 56% of the study group.

The age group ranged from 14 to 78 years with mean age of 54.8 years. Maximum patients (57%) were in the age group of 46-65 years and only 22% were less than 45 years.

Table 2: Descriptive analysis of gender group in the study (N =45)

Gender	Frequency	Percentage
Male	20	44%
Female	25	56%

Graph 1: Sex distribution among study group

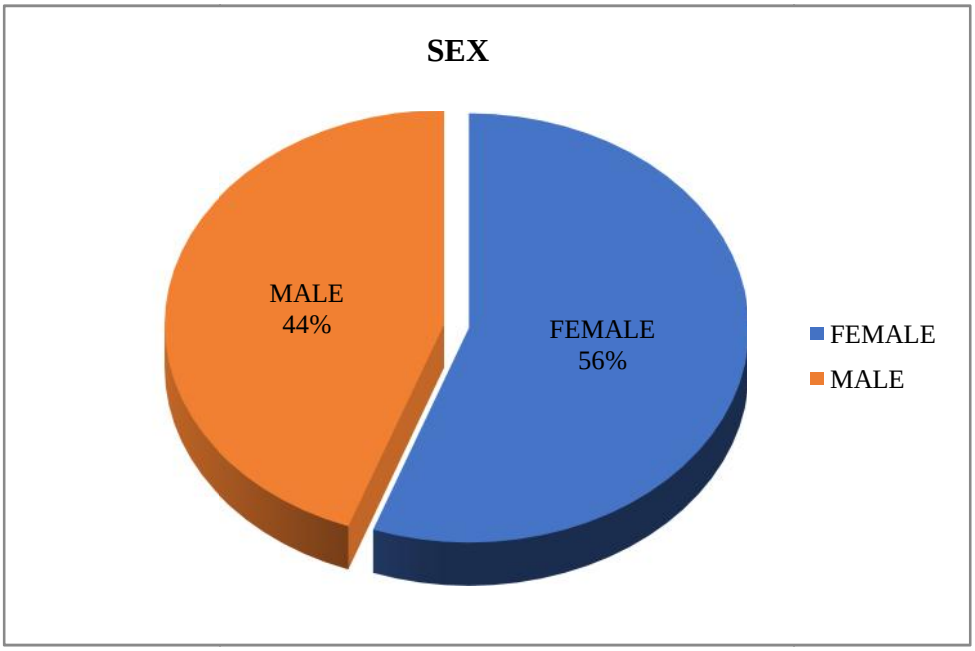


Table 3: Descriptive analysis of age distribution in the study(N=45)

Age group(yrs)	Frequency	Percentage
<25	1	2.2%
26-45	9	20%
46-65	26	57.7%
>65	9	20%

Graph 2: Age distribution among study group

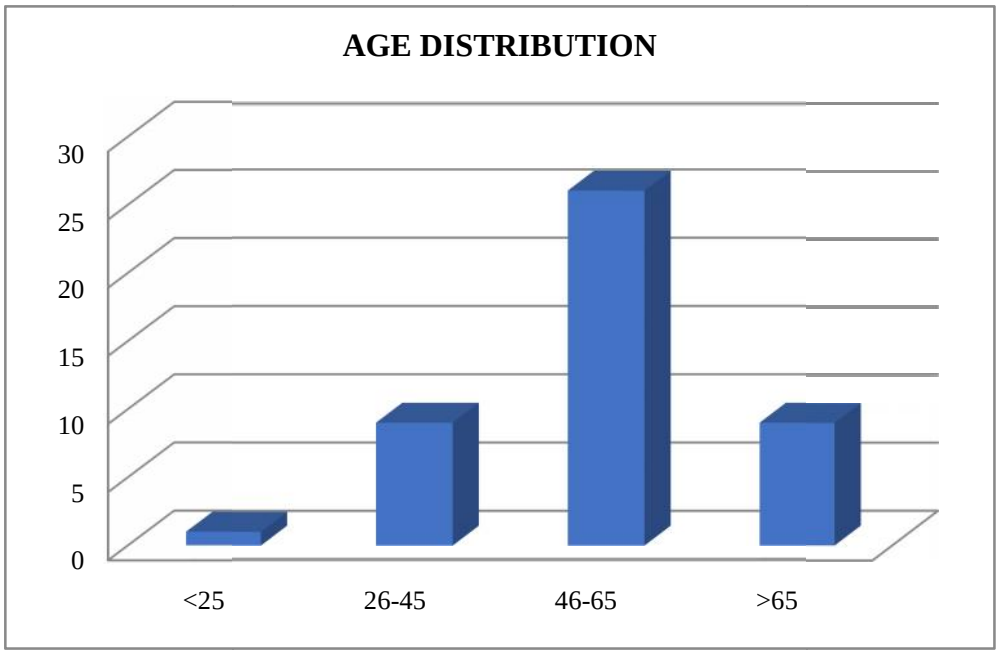
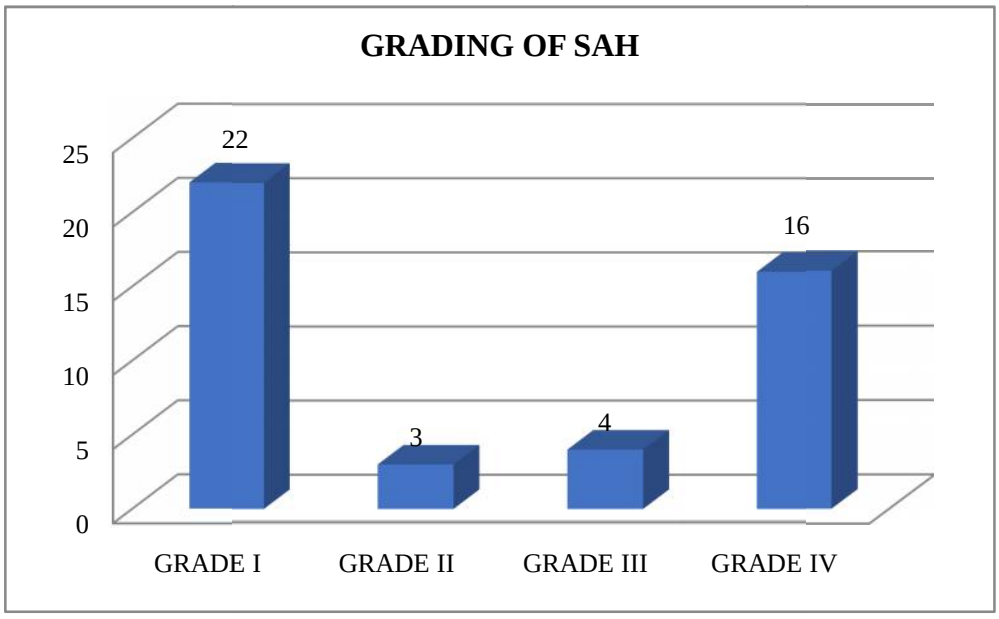


Table 4: Descriptive analysis of grades of SAH in the study group (N=45)

Grade of SAH	Frequency	Percentage
Grade I	22	48.8%
Grade II	3	6.6%
Grade III	4	8.8%
Grade IV	16	35.5%

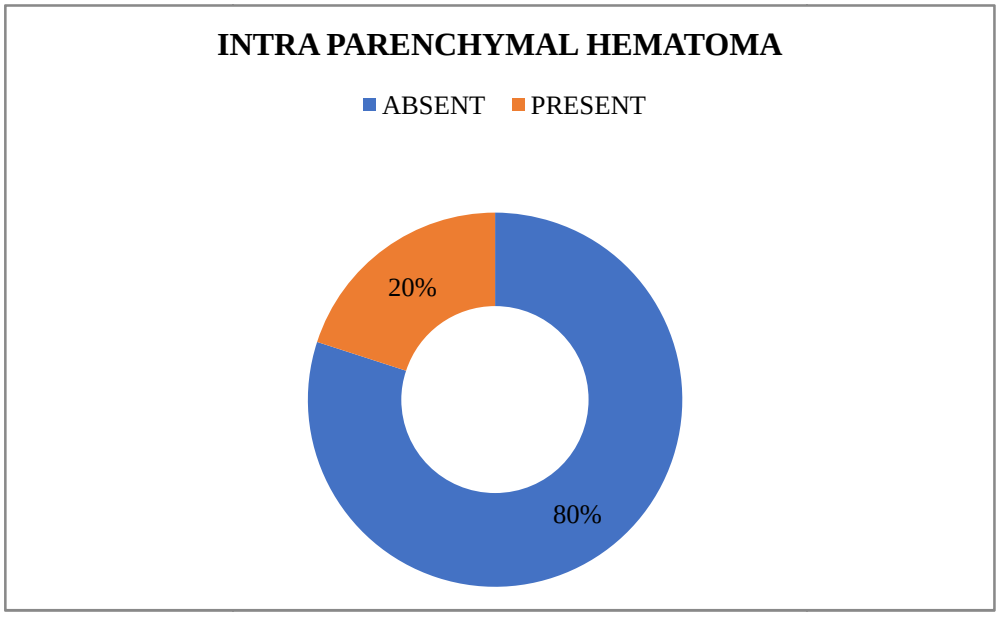
Among the 45 patients, there was almost equal distribution of SAH as diffuse (51%) and focal (49%).16(35%) of 45 patients were assessed to have grade 4 SAH, 4(8%) with grade 3 SAH, 3(6.6 %) with grade 2 SAH and rest of the 22(48%) patients with grade I SAH on non-contrast CT.

Graph 3: Grades of SAH in the study group



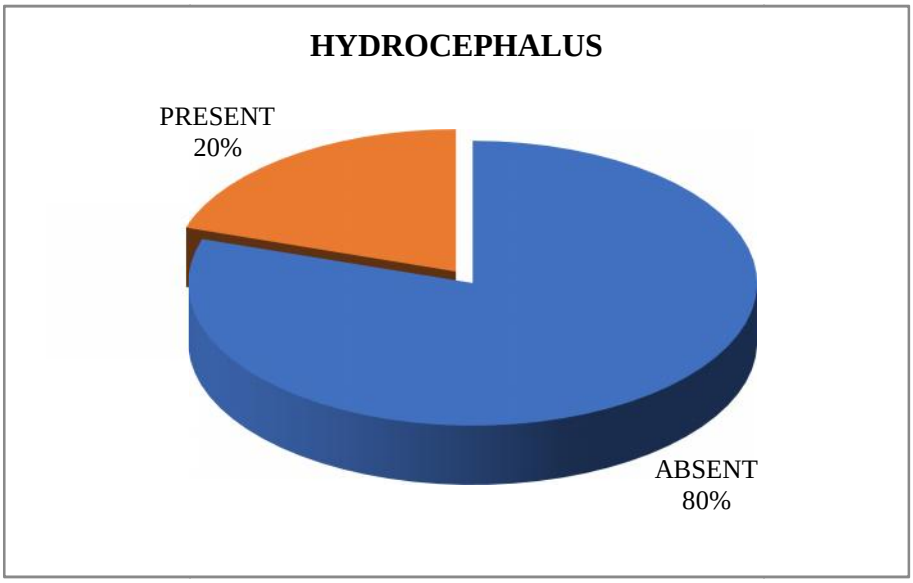
Intraparenchymal hematoma was noted in 9(20%) patients and in majority of the patients (80%) it was isolated SAH only.

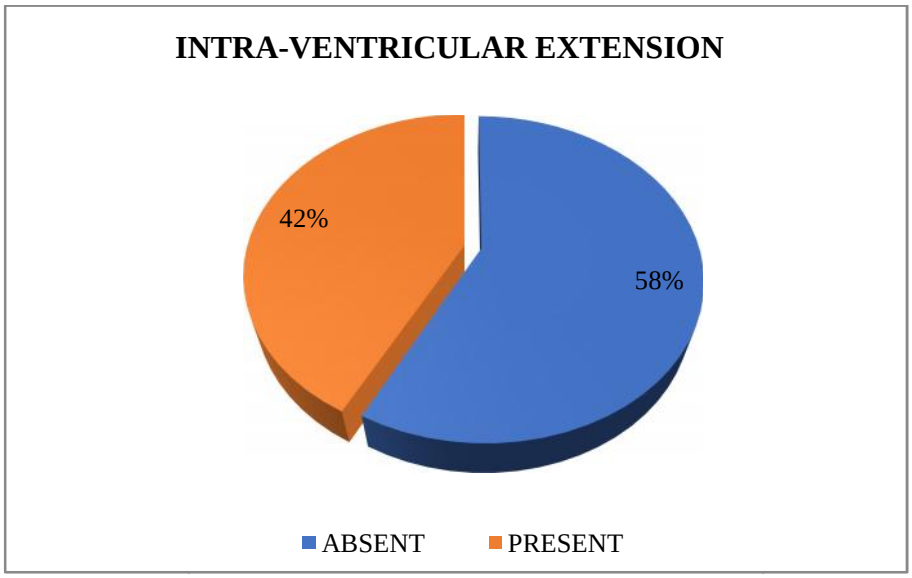
Graph 4: SAH associated with intraparenchymal hematoma



EVALUATION OF COMPLICATIONS DUE TO SAH

Graph 5: Graphical presentation of complications of SAH in the study group (N=45)





Among the 45 patients, mainly 2 complications were taken note of namely intraventricular extension and hydrocephalus.

Intraventricular extension was seen in 19 patients (42.2%) whereas presence of hydrocephalus was noted in only 9 patients (20%)

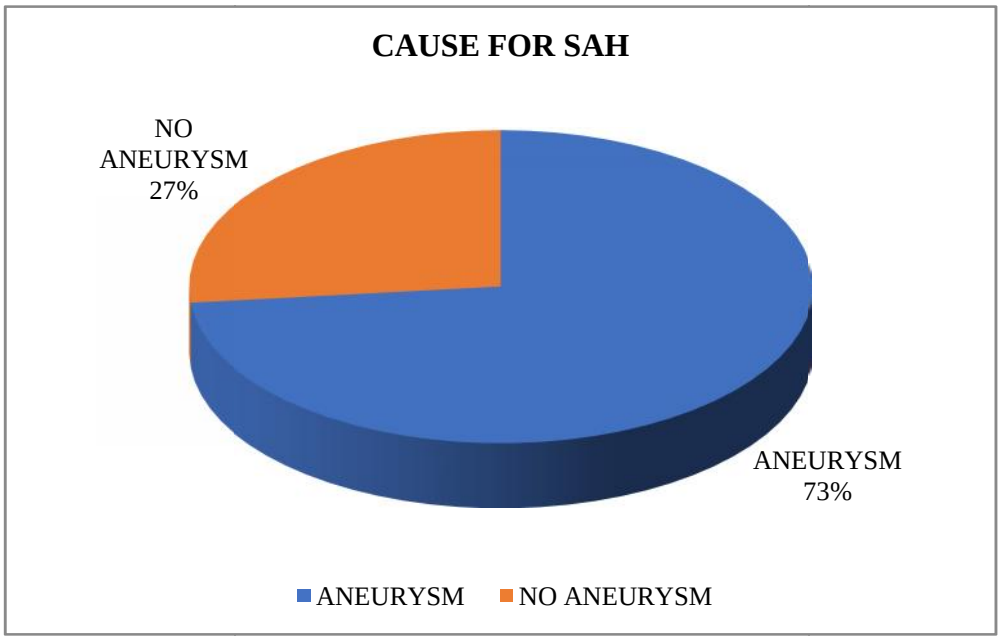
Table 5: Descriptive analysis of complications of SAH in the study group (N=45)

Complication	Frequency	Percentage
Intraventricular extension	19	42.2%
Hydrocephalus	9	20%

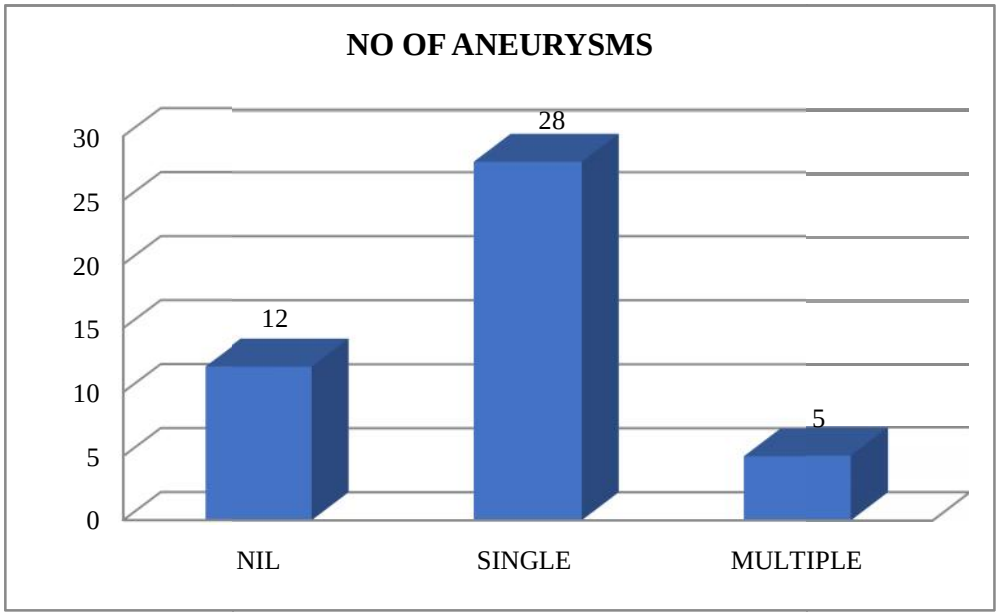
Evaluation of the cerebral aneurysms

Total of 38 aneurysms were detected on MDCTA in 33 of the 45 patients included in the study, of which 5 patients had 2 aneurysms each and rest had solitary aneurysm. In 12 of the 45 patients no aneurysm or other identifiable causes of SAH (AVMs or Dural fistula etc) were identified.

Graph 6: Graphical presentation of etiology of SAH in the study group (N=45)



Graph 7: Graphical presentation of multiplicity of aneurysms in the study group (N=45)

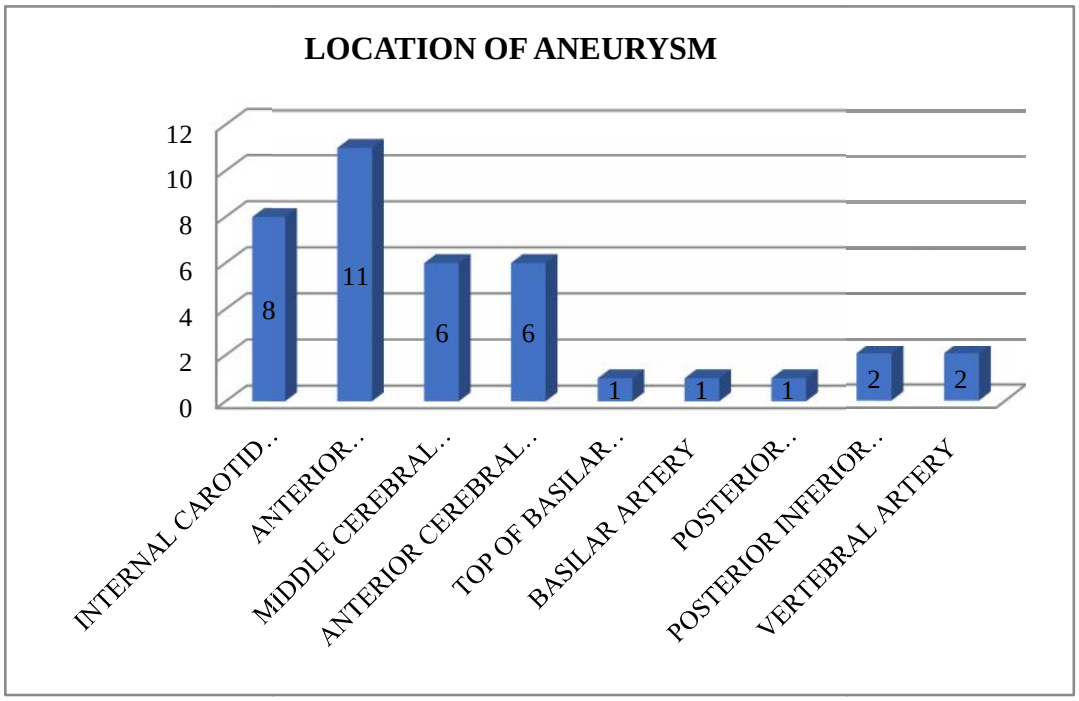


According to site of the aneurysm in our study, of the 38 aneurysms that we identified, 31 were in the anterior circulation and 7 in the posterior circulation. Among the 31 aneurysms seen at the anterior circulation, there were 11 at anterior communicating artery, 8 at internal carotid arteries, 6 at anterior cerebral arteries and 6 at middle cerebral arteries. Among the 7 aneurysms seen at the posterior circulation there were one each at top of the basilar artery, basilar & posterior cerebral arteries and 2 each at the posterior inferior cerebellar & vertebral artery.

Table 6: Descriptive analysis of Location of the cerebral aneurysms

Site	Incidence	Percentage (%)	Mean aneurysm size
Internal carotid artery	8	21.05263	6.03
Anterior communicating artery	11	28.94737	5.1
Middle cerebral artery	6	15.78947	3.9
Anterior cerebral artery	6	15.78947	4.95
Top of the basilar artery	1	2.631579	-
Basilar artery	1	2.631579	-
Posterior cerebral artery	1	2.631579	-
Posterior inferior cerebellar artery	2	5.263158	-
Vertebral artery	2	5.263158	-

Graph 8: Graph depicting location of aneurysms among study group



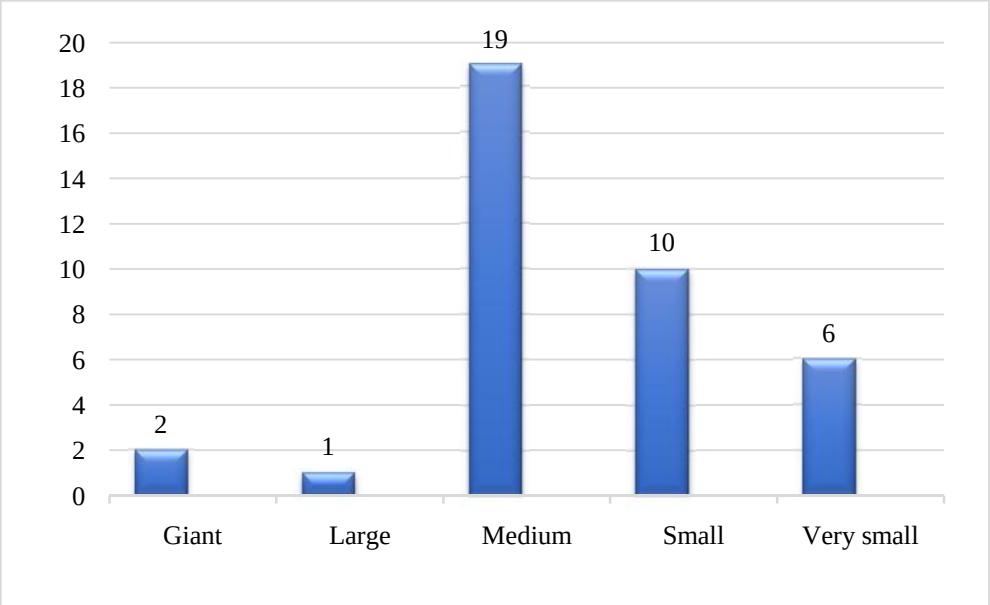
Of the 38 ruptured aneurysms in our study there were 2 (5%) giant aneurysms, 1(2%) large aneurysm, 19(50%) medium aneurysms, 10(26%) small aneurysms and 6(15%) very small aneurysms. The smallest aneurysm identified by MDCTA was 2.2 mm, and the largest was 27 mm (mean size of 6.31 mm).

The mean size of ruptured aneurysms was smaller in those at MCA. Of the ruptured aneurysms, majority (50%) of them were of medium size between 5 to 12 mm.

Table 7: Descriptive analysis of Size of aneurysms

Size of aneurysms	Incidence	Percentage (%)
Giant	2	5.263158
Large	1	2.631579
Medium	19	50.0
Small	10	26.315
Very small	6	15.78947

Graph 9: Graph depicting size category of aneurysms among the study group



Two out 38 aneurysms were fusiform aneurysms and it was not possible to measure dimensions of the neck in these aneurysms.

The N/D ratios of aneurysm were, therefore measurable in 36 aneurysms.

According to the neck of the aneurysm there were 6 aneurysms with narrow neck and 30 aneurysms with wide neck.

Table 8: Descriptive analysis of N/D ratio of aneurysms

N/D ratio of aneurysms	Incidence	Percentage (%)
Wide neck (≥1/3)	30	83.33333
Narrow neck (<1/3)	6	16.66667

Graph 10: Pie chart showing percentage of wide and narrow neck aneurysms in study group

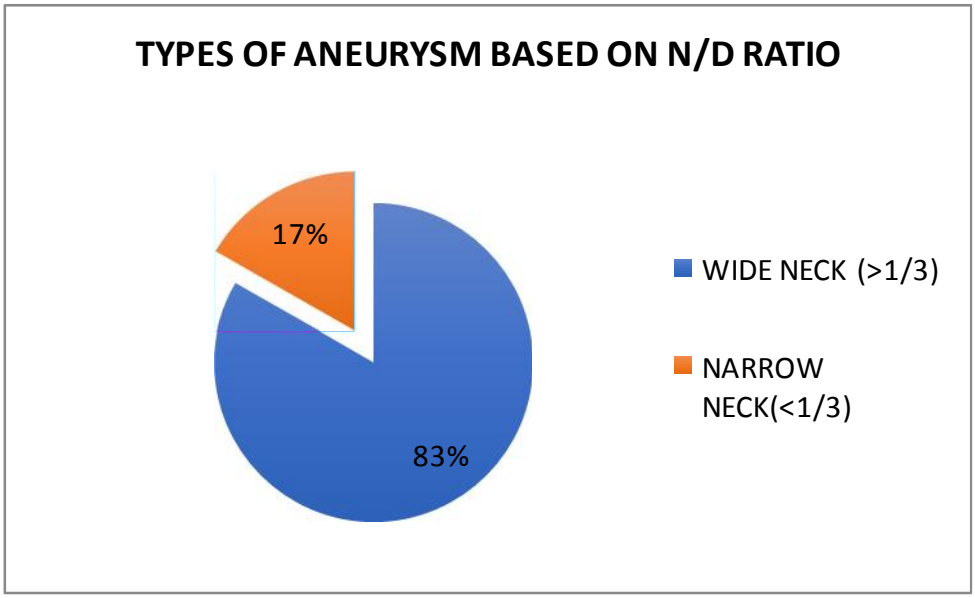


Table 9: Descriptive analysis of association between aneurysm location and size

SITE/SIZE	GIANT	LARGE	MEDIUM	SMALL	VERY SMALL	TOTAL
ACOM	0	0	7	2	2	**11**
ACA	0	0	3	2	1	**6**
ICA	0	1	4	1	2	**8**
MCA	0	0	1	4	1	**6**
PICA	0	0	1	1	0	**2**
VA	0	0	2	0	0	**2**
BA	1	0	0	0	0	**1**
TOP OF BA	0	0	1	0	0	**1**
TOTAL	**2**	**1**	**19**	**10**	**6**	**38**

CHI-SQUARE test was used to analyze the association between the aneurysm location and size. There is seen association between the size and location of the aneurysms with P value of 0.0017 (VERY SIGNIFICANT).

DISCUSSION

Non traumatic SAH is a devastating neurological condition and is associated with high survivor morbidity and mortality rates. Accurate management of the aneurysms demands the need to know the pattern of clinical presentation, risk factors leading to development and rupture of aneurysms and also the critical size predisposing to their rupture.

Patients in our study had typical presentation patterns with severe headache and vomiting. Few of the patients also had additional features of neurological deficits in the form of reduced motor power of the limbs. 45 patients that were clinically suspected to have SAH and its presence confirmed by Plain CT brain scan were included in the study. CT angiography was performed as per the study protocol. Angiography was performed immediately after confirmation of SAH on plain CT brain scans in majority of the patients with no time delay. Hence, CTA served as a quick and non-invasive tool in identifying the cause of non-traumatic SAH immediately after its confirmation with non-contrast brain CT scan.

CTA in evaluation of cerebral aneurysms:

The advanced technological developments of MDCT and specially the introduction of various post processing techniques have significantly improvised the spatial–temporal resolution and hence enable a minimally invasive and an accurate study in the assessment of intracranial aneurysms, even in the setting of acute SAH.

This was a prospective study, we included 45 patients who satisfied the inclusion and exclusion criteria and all patients underwent CTA and well tolerated the

procedure. All the 45 studies were assessed for presence of SAH and intracranial aneurysms, and there were no significant motion artefacts.

The 64 and 128 detector row CTA used in this study provided increased spatial resolution and Shorter acquisition times. The high-speed acquisition enabled an exclusively arterial phase acquisition. During DSA, most often the patient needs to be sedated who is already in a critical condition in order to obtain diagnostic images. In our study, Sedation was not essential for any of the CTA.

CTA is completed in very short duration of 10 minutes. In comparison, DSA takes a slightly higher scan duration and demands expert professional.

Of the total 45 participants that were included in the study, relatively higher incidence of SAH was identified in females (56%). Higher occurrence of SAH (57%) was noted between the age groups of 46-65 years with mean age of presentation being 54.8 years.

In our study, 38 aneurysms were found in 45 patients that presented with non-traumatic SAH, of that 36 were saccular aneurysms and 2 fusiform type. Rest of the 12 patients showed no definite identifiable cause of SAH on CTA.

Of the 33 individuals with aneurysmal SAH, 5 patients (15%) had more than one aneurysm and each of them had aneurysms at two locations. 28 of the study subjects presented only one aneurysm.

It was possible to identify aneurysms from even the ophthalmic and supraclinoid segments of the ICA (8 out of 38) attributable to various advanced post processing techniques such as VRT with multi planar reformations. Many other studies have shown decreased sensitivity in detecting these aneurysms due to their

close proximity to bony structures of the skull base, and may be overlooked or may have their location misinterpreted on MDCTA.

Tipper et al [52], in their study using 16-channel MDCTA studied 57 patients with suspected intracranial aneurysms and they detected 51 of 53 aneurysms, with sensitivity of 96% and specificity of 100%.

The size and location of the aneurysms are considered important deterministic factors in their risk of rupture. Various studies have reported critical sizes at which aneurysms have highest risk of rupture.

The mean size of ruptured aneurysms is 6.31 mm and the smallest aneurysm identified was 2.2 mm. The mean sizes of aneurysms at specific arteries were found to be 5.1 mm at ACOM, 3.9 mm at MCA, 4.95 mm at ACA and 6.03 mm at ICA.

A retrospective study of ISUIA, found small aneurysms to be safe with less risk of rupture. They found that aneurysms with size <10 mm had very less risk of rupture of about only 0.05%.

However, in our study, 42% of the ruptured aneurysms were of <5 mm in size and about 50% of them ruptured at size of 5-12 mm.

Similar findings were noted by Kassel and Torner et al [53] who found about 57% of the ruptured aneurysms between 5 to 10 mm in diameter approximately similar to 50% in our study. They also concluded that aneurysm size <10 mm needs to be considered seriously and those >5 mm to be treated at the earliest.

Of the 38 aneurysms, 11 (29%) aneurysms were at ACOM and 8(21%) at ICA.

Most common site of ruptured intracranial aneurysms was ACOM with mean size of aneurysm at the time of rupture to be 5.1 mm. Hence, ACOM is the most vulnerable site for aneurysm development and rupture mostly secondary to hemodynamic stress at this point. Thus, even small to medium sized aneurysms at these sites of sizes even <10 mm should be considered for surgical or endovascular management.

Joo et al [54] in their study also found ACOM to be the most common site of ruptured aneurysm and majority of them ruptured even before reaching sizes of 7 mm (in 71.8%) and 10 mm (in 87.9%).

In our study, second common site for aneurysm following ACOM was ICA, 8 aneurysms (21%) from various segments of ICA were noted followed by 6 aneurysms each in MCA and ACA. Less number of aneurysms were noted in the posterior circulation, 7 out of 38(18%) altogether.

Joeng et al [55] in their study found that ACOM aneurysms ruptured at smaller size than those at MCA. But, in our study MCA aneurysms ruptured at lesser mean size of 3.9 mm when compared to 5.1 mm in ACOM. And the mean size for aneurysm rupture was less than 7mm in any of the arteries.

Thus, it is important to note that any anterior circulation aneurysm is highly predisposed to rupture irrespective of its size and location.

Weibers et al [56] in their study have shown shrinkage in size of aneurysm following rupture, hence concluding that scans following rupture do not reflect the actual size of aneurysm.

But, Histopathological evaluation of the ruptured and unruptured aneurysms by Kotaoka et al [57] found no evidence that suggested aneurysm size reduction following its rupture. Hence it supports the evidence that even small aneurysms tend to rupture with no obvious change in size after their rupture.

In our study, 5(15%) patients had multiple aneurysms and 70% (7 out of 10) of them ruptured at sizes <5mm. Hence, it is not advisable to neglect smaller aneurysms when multiple aneurysms are found in a patient, especially when they are identified at most vulnerable sites like ACOM and ACA.

Of the 11 ruptured aneurysms at ACOM, 7 ruptured at sizes between 5-12 mm and 4 aneurysms at <5 mm size. At other sites also majority of aneurysms ruptured at sizes <12 mm with total 19 of 38 (50%) ranging between 5 to 12 mm and 16 (42.1 %) of size less than 5 mm.

Present study showed association between the aneurysm location and size with their rupture risk and was found statistically significant with P value of 0.0017.

Yasser Orz et al conducted a retrospective review and found that the most common location of ruptured small aneurysms was the anterior communicating artery, while the middle cerebral artery was the commonest site for small unruptured aneurysms.

The N/D ratios of aneurysm were measurable in 36 aneurysms of which 30 ruptured aneurysms had wide neck and 6 aneurysms were of narrow neck.

CONCLUSION

- This study showed MDCTA to be a highly useful investigation in the evaluation of patients with spontaneous non traumatic subarachnoid hemorrhage.

- Various post processing algorithms have improvised the technique and increased its accuracy in detecting even smaller aneurysms. CTA images provided useful information in deciding the treatment options of aneurysms and also offered pathway for surgeon.

- The results of this study support the use of CTA in routine clinical practice in the evaluation of nontraumatic SAH.

- Need for minimal patient co-operation, noninvasiveness of the procedure, quick scan duration and wide availability of the CT scanners make it the most suitable investigation in the acute setting of SAH.

- The study also showed that size and location of aneurysm play an important role in their rupture risk and thus provide useful guide in the management of patients with aneurysmal SAH. The most common site for aneurysm rupture was anterior communicating artery.

- Majority of the ruptured aneurysms in our study were at the ACOM and were of small to medium size (<12 mm). MCA aneurysms ruptured at lesser mean size followed by ACOM. Hence, small aneurysms should not be considered safe and have to be considered for treatment specially when they are located at common sites like ACOM.

SUMMARY

This was hospital based prospective observational study carried out in suspected cases of non-traumatic subarachnoid hemorrhage to evaluate the role of MDCTA in detecting intracranial aneurysms. MDCTA was found to be highly effective in detecting intracranial aneurysms as cause of SAH in 38 out of 45 patients. It was possible to identify aneurysms of size as small as 2.2 mm and also at difficult sites like supraclinoid segments of ICA. Thus, MDCTA serves as important tool in evaluation of patients with spontaneous SAH.

The size and location of the aneurysms were found to be important deterministic factors in their rupture risk with ACOM being the most common site of ruptured aneurysms and majority of the aneurysms had ruptured at sizes <12 mm. Thus, it is important to consider treatment of an aneurysm irrespective of its size with special consideration of vulnerable sites like ACOM.

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ANNEXURE I – CONSENT FORM

TITLE OF THE STUDY: "MULTIDETECTOR COMPUTED TOMOGRAPHIC ANGIOGRAPHY OF CEREBRAL VESSELS IN THE EVALUATION OF SUSPECTED NON-TRAUMATIC SUBARACHNOID HAEMORRHAGE-HOSPITAL BASED OBSERVATIONAL STUDY"

PRINCIPAL INVESTIGATOR: _____

INTRODUCTION AND PURPOSE:

Subarachnoid haemorrhage (SAH) is a major neurological emergency. Around 85% of non-traumatic SAH is caused by the rupture of intracranial aneurysms and are associated with high morbidity and mortality. Computed Tomographic Angiography (CTA) is a non-invasive, relatively less expensive procedure that can be used as a primary examination tool for imaging of cerebral vascular disease including aneurysms and other steno occlusive lesions.

PROCEDURE:

I request you to kindly participate in the study titled **"Multidetector computed tomographic angiography of cerebral vessels in the evaluation of suspected non traumatic subarachnoid haemorrhage-Hospital Based Observational Study"** at Dr. Prabhakar Kore charitable hospital and Medical Research Centre, Belgaum being conducted by _____, post graduate in Radiodiagnosis at J. N. Medical College Belgaum, Karnataka, under the guidance of _____, Professor, Dept. of Radiodiagnosis, J. N. Medical College, Belagavi.

We request you to participate in this study as you are eligible to be included. During the study you will be asked questions regarding your present and past medical history and you will be required to answer to the best of your knowledge. You will also be clinically examined as per the protocol drawn.

If you agree to participate in the study please furnish the details pertaining to the study.

BENEFITS:

- Noninvasive modality

COMPLICATIONS

No significant risk to the patient has been documented from CT angiography of cerebral vessels.

ALTERNATIVES:

If patient is not willing to take part in the study, his / her treatment or any other further investigations the patient wants to undergo, in future, in KLE will not be affected by his / her decision.

VOLUNTARY PARTICIPATION/WITHDRAWAL:

Taking part in this study is voluntary. I may choose not to take part in this study, or if I decide to take part I can later change my mind and withdraw from the study. My decision will not change the present or future health care or other services that I receive. The study doctor or the sponsor may stop my participation in this study. I will tell if any important new findings that may change my willingness to continue to take part. If I choose not to take part in the study, I will receive the standard treatment for patients with my condition.

COSTS:

NIL (The study is to be conducted on the participants who are advised Cerebral CT angiography by the referring consultant and the participants will bear the charges for it.)

Payment for Participation: No incentive will be paid to you for participating in this study.

COMPENSATION:

In the event that I become injured as a result of taking part in this study, treatment whatever available at KLE charitable hospital, Belagavi, will be offered to me. No reimbursement, compensation or free medical care is given.

CONFIDENTIALITY:

All information collected about me during the course of the study will be kept confidential to the extent permitted by the law. The code numbers will identify me in this research record. Information from this study may be published but my identity will be confidential in any publication.

QUESTION: If any enquiries in the future or in case of research related injury illness, you may contact following person.

Dr. Roopa Bellad
Professor of Pediatrics Chairperson, J.N. Medical College Institutional Ethical Committee for Human Subjects Research
Ph. No: 0831-2473777, Ext. 1529

CONSENT TO PARTICIPATE IN RESEARCH STUDY:

- I understand that I am participating in the study, which includes CT angiography of cerebral vessels.
- I confirm that I have read and understood the information in the patient information sheet. Procedure is explained to me in detail along with information about the advantages and disadvantages of taking part in the study. I have been given the opportunity to discuss all aspects of the trial, to ask questions and hereby consent to participation in the trial outlined above.
- I understand that the decision to take part in this study is completely voluntary and I am aware that I can choose to withdraw from the study at any point of time.
- I consent to the photographing or recording of the procedure to be performed including appropriate portions of my body, for medical, scientific or educational purposes provided my identity is not revealed in the pictures or by the descriptive texts accompanying them.
- I understand that there is no significant risk involved in the test that would be done in this study.
- No guarantee or assurance has given by anyone as to the results that may be obtained.
- My signature on this form signifies that I have willingly decided to participate after understanding the above information.

Participant's Name/ legally authorized _____
representative

Signature _____

Name and signature of witness _____

Name and signature of interviewer _____

Date:

Place:

ANNEXURE-II

PROFORMA FOR DATA COLLECTION

S. No:

NAME: _____

AGE :_____

OP/IP NO : _____

MOBILE NO :_____

ADDRESS: _____

_____

CT NUMBER: _____

CHIEF COMPLAINTS:

HISTORY OF PRESENTING ILLNESS:

OTHER RELEVANT HISTORY IF ANY:

CT BRAIN PLAIN FINDINGS:

SUBARACHNOID HEMORRHAGE:- PRESENT/ ABSENT

IF PRESENT-

DISTRIBUTION: DIFFUSE/ FOCAL

MIDLINE SHIFT: PRESENT/ ABSENT

INTRAVENTRICULAR EXTENSION: PRESENT/ ABSENT

HYDROCEPHALUS: PRESENT/ ABSENT

INTRAPARENCHYMAL HAEMORRHAGE: PRESENT/ ABSENT

IF PRESENT-

LOCATION:

SIZE:

GRADES OF SUBARACHNOID HEMORRHAGE:

FISCHER REVISED SCALE

Grade	**Description**
0	No SAH or IVH
1	Minimal/thin SAH, no IVH in either lateral ventricle
2	Minimal/thin SAH, with IVH in both lateral ventricles
3	Dense SAH,* no IVH in either lateral ventricle
4	Dense SAH,* with IVH in both lateral ventricles

*Completely filling ≥1 cistern or fissure

ANY OTHER FINDINGS

CTA OF CEREBRAL VESSELS:

- ANEURYSMS: PRESENT/ ABSENT

IF PRESENT-

1. NUMBER:

2. LOCATION:

3. SIZE:

Neck:-

Dome:-

4. MORPHOLOGY (SHAPE):

ANNEXURE-III- ETHICAL CLEARANCE LETTER

K.L.E.UNIVERSITY'S
JAWAHARLAL NEHRU MEDICAL COLLEGE,
NEHRU NAGAR, BELAGAVI-590010 (KARNATAKA-INDIA)
(Accredited 'A' Grade by NAAC)

Website: http//www.jnmc.edu
E-Mail : dome@jnmc.edu

Phone: (+ 91-(0)831 Office : 2471350
Principal: 2471701
Fax No. +91 (0)831 – 2470759

Ref: MDC/DOME/ 06 **Date: 22/11/2017**

REG NO.BS0117002

Sub: Institutional Ethical Clearance for the study.

With reference to the above, we wish to inform you that your proposed research project titled **"MULTIDETECTOR COMPUTED TOMOGRAPHIC ANGIOGRAPHY OF CEREBRAL VESSELS IN THE EVALUATION OF SUSPECTED NON TRAUMATIC SUBARACHNOID HAEMORRHAGE – HOSPITAL BASED OBSERVATIONAL STUDY",** is ethical and justifiable. The proposed research project has been cleared by the JNMC Institutional Ethics Committee on Human Subjects Research.

(Dr. Arathi Darshan)
Member Secretary
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

(Dr. Roopa M Bellad)
Chairman,
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

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ANNEXURE IV – FIGURES

REPRESENTATIVE CASES

CASE 1

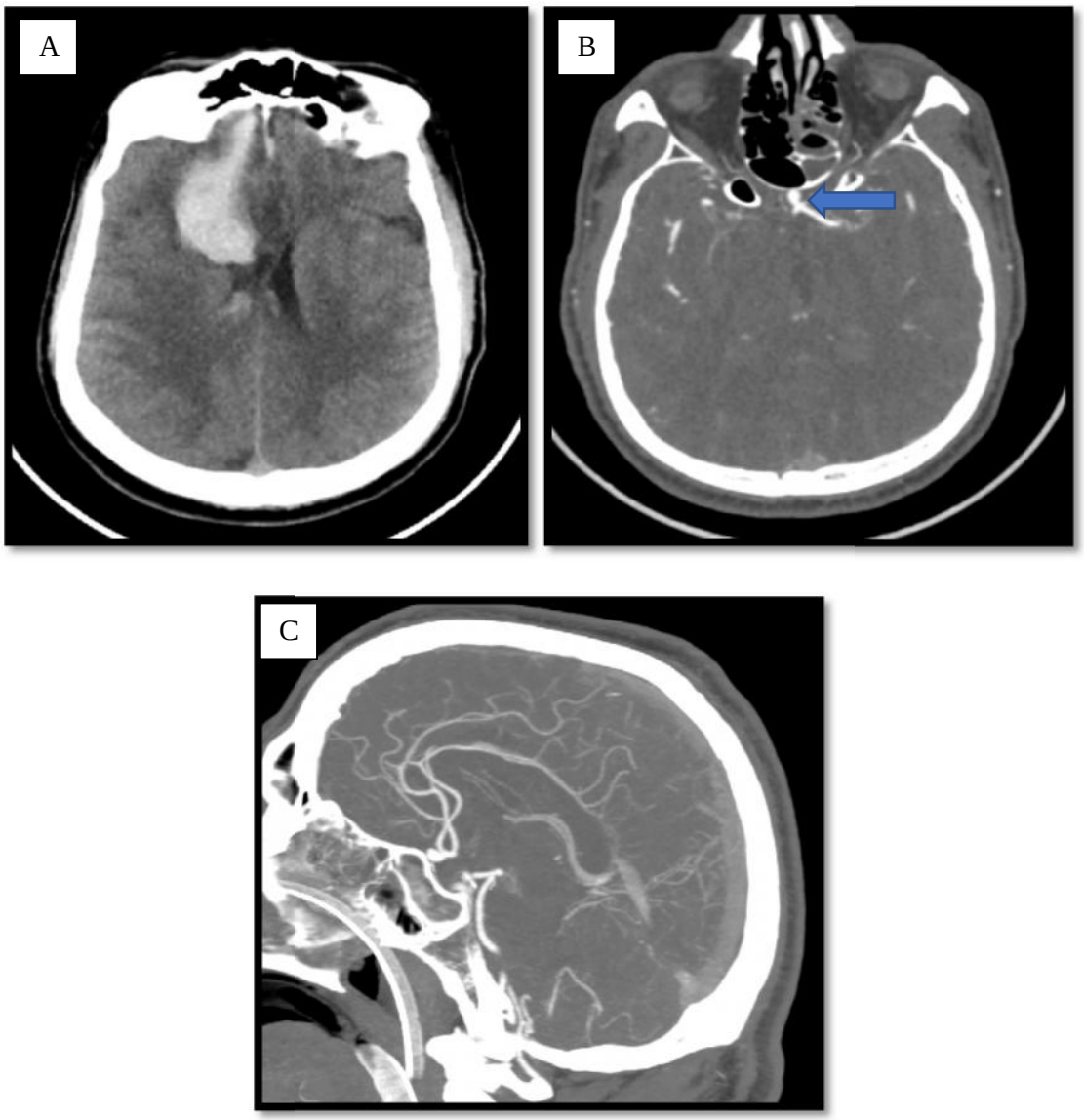


Figure 6: 56 year old male patient with a ruptured ACOM aneurysm. A. Plain CT brain showing SAH and intraparenchymal hematoma in the right frontal region, B. Arterial phase C. Sagittal MIP of CTA showing ruptured ACOM aneurysm

CASE 2

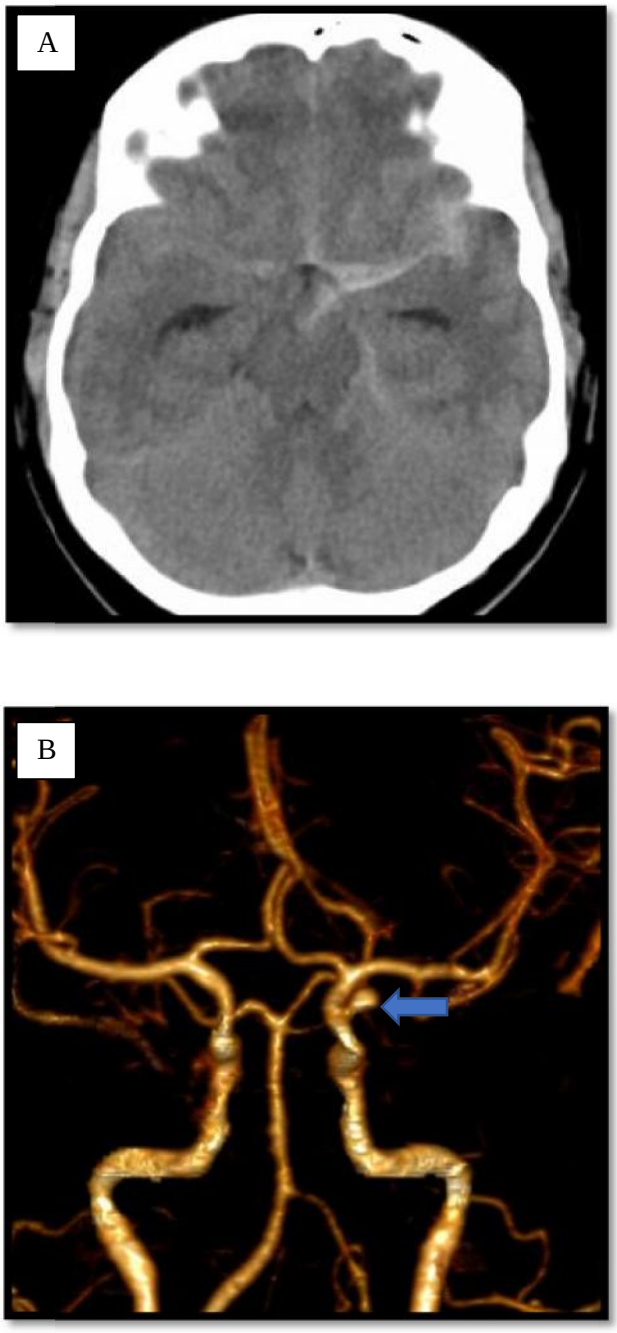


Figure 7: 55year female patient with sudden onset severe headache. (A). unenhanced CT showing diffuse subarachnoid haemorrhage, Fisher's revised grade 3. (B) Anterior VRT showing a saccular aneurysm at the supraclinoid segment of the left ICA.

CASE 3

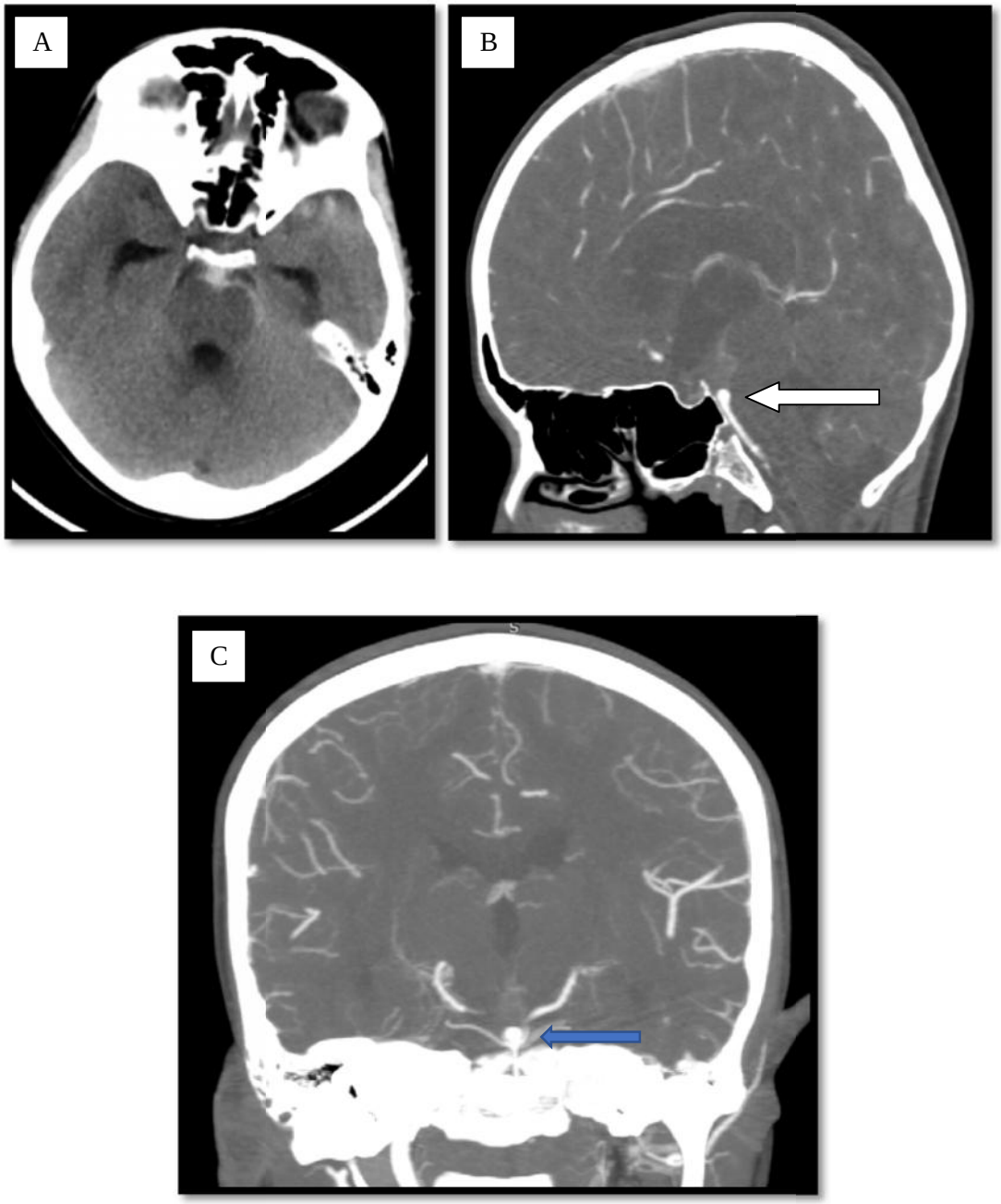


Figure 8: 41-year-old male with basilar tip aneurysm (A) Axial precontract CT showing subarachnoid haemorrhage (B) sagittal arterial phase (C) MIP showing saccular aneurysm at the top of the basilar artery

CASE 4

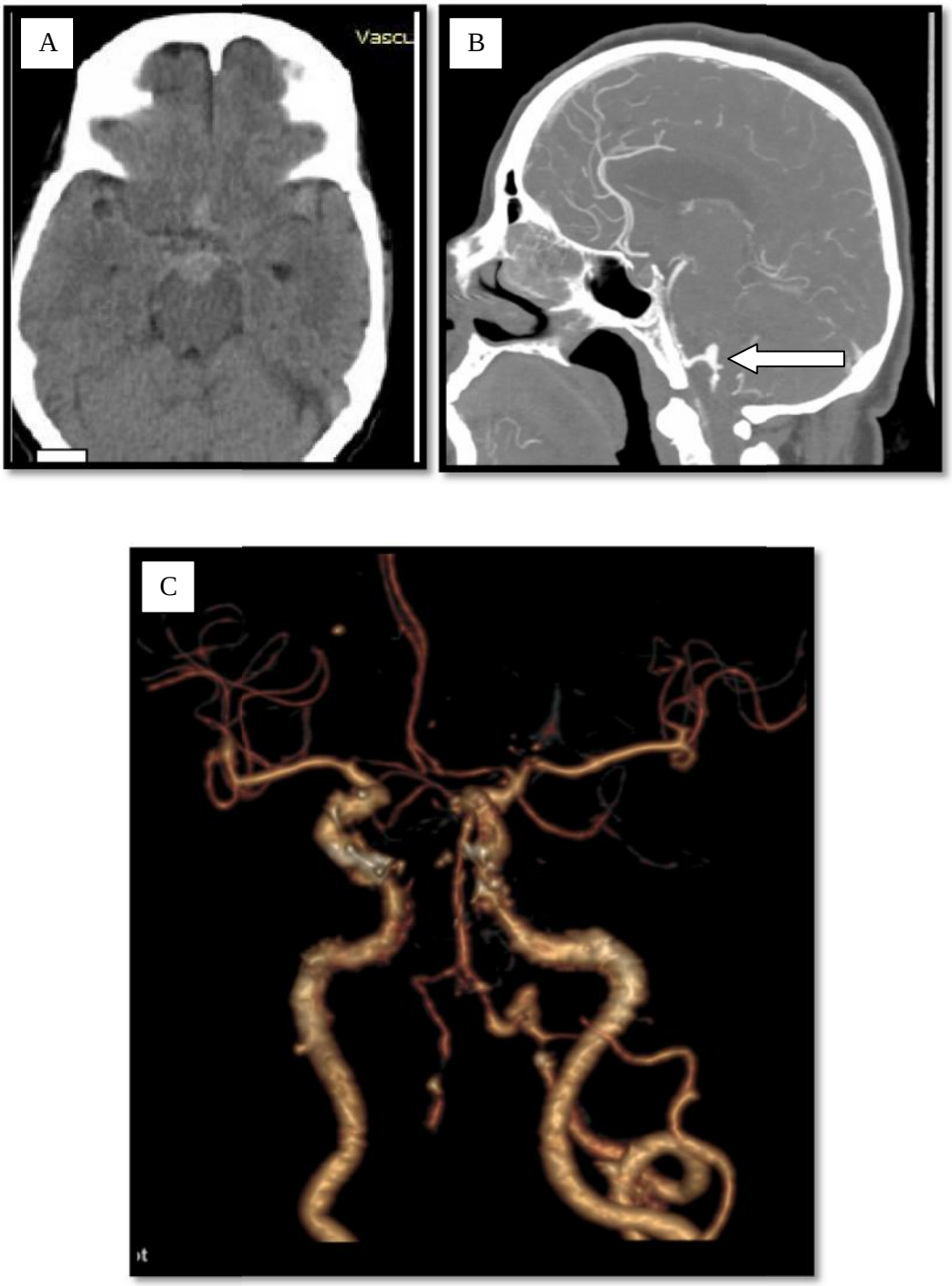


Figure 9: 54-year-old male with aneurysm of V4 segment of Left vertebral artery (A) Axial precontract CT showing subarachnoid haemorrhage (B) sagittal MIP arterial phase (C) VRT showing saccular aneurysm of V4 segment of Left vertebral artery

CASE 5

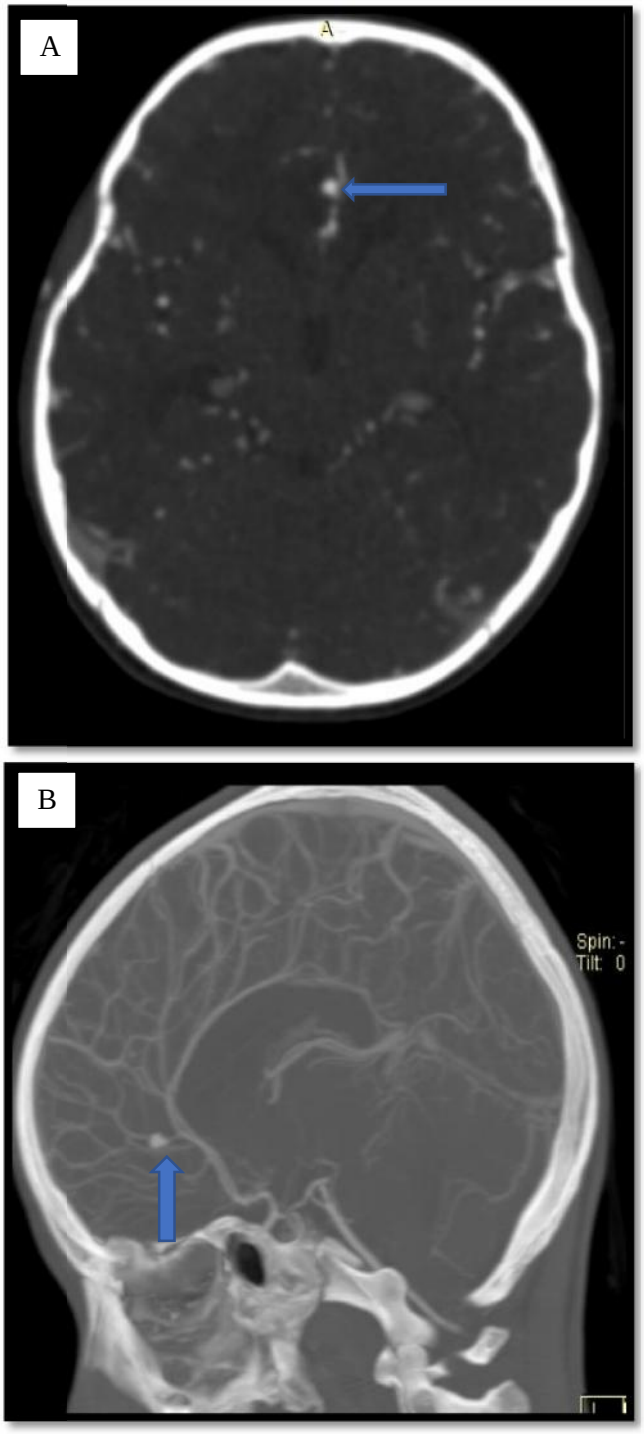


Figure 10: (A) arterial phase axial CTA. (B). Sagittal MIP showing a small saccular aneurysm arising from the pericallosal segment of left ACA.

ANNEXURES V - MASTER CHART

SL.NO	Age	Sex	SAH Focal /Diffuse	Midline shift	Intraventricular extension	Grade of SAH	Hydrocephalus	Intraparenchymal hemorrhage	No. of aneurysms	Aneurysm site	Thrombus	Size	Neck	Dome	N/D Ratio
1	56	M	Focal Cisternal	Absent	Absent	1	Absent	Absent	1	ACOM	Absent	4 x 3 mm	1.8 mm	4 mm	0.45
2	70	F	Diffuse	Absent	Absent	3	Absent	present	1	ACOM	Absent	6 x 5 mm	2.0 mm	6 mm	0.3
3	65	F	Focal Cisternal	Absent	Absent	1	Absent	Absent	1	ACOM	Absent	5 x 4 mm	3.0 mm	5 mm	0.6
4	52	M	Diffuse	Absent	Present	4	Present	Present	1	A3 segment of Rt ACA	Absent	8.0 x 7.0 mm	1.8 mm	8 mm	0.22
5	47	F	Diffuse	Absent	Absent	1	Absent	present	1	At bifurcation of Rt MCA	Absent	3.0 x 2.1 mm	1.4 mm	3 mm	0.4
6	64	F	Diffuse	Absent	Absent	1	Absent	Absent	0						
7	58	M	Diffuse	Absent	Present	2	Absent	Absent	1	Lt anterior choroidal artery	Absent	6.5 x 3.8 mm	3.5 mm	6.7 mm	0.52
8	14	M	Diffuse	Absent	Present	4	Absent	Absent	1	Lt VA V4 segment	Absent	7 x 4 mm	2.0 mm	7 mm	0.28
9	45	F	Focal Cisternal	Absent	Absent	1	Absent	Absent	0						
10	60	F	Focal Cisternal	Absent	Absent	1	Absent	Absent	1	Lt paraophthalmic ICA	Absent	13 x 10 mm	5.8 mm	13 mm	0.44
11	48	F	Focal Cisternal	Absent	Absent	1	Absent	Absent	1	ACOM	Absent	2.2 x 1.6 mm	2.2 mm	2.4 mm	0.9
12	60	F	Focal Cisternal	Absent	Absent	1	Absent	Absent	2	Distal A2 segment of azygous ACA, M2 segment Lt MCA	Absent	6 x 3 mm , 3x3mm	4.0 mm	6 mm	0.6,0.3
13	50	M	Focal Cisternal	Absent	Absent	1	Absent	Absent	1	ACOM	Absent	6.2 x 4.2 mm	3.2 mm	6.2 mm	0.5
14	45	F	Diffuse	Absent	Present	4	Present	Absent	2	Distal A2 segment of azygous ACA	Absent	3.2 x 2.2 mm	1.5 mm	3.2 mm	0.46
										Rt ICA Paraophthalmic region	Absent	2.6 x 2.4 mm	1.5 mm	2.6 mm	0.57
15	55	F	Focal Cisternal	Absent	Absent	1	Absent	Absent	0						
16	35	F	Focal Cisternal	Absent	Absent	1	Absent	Absent	1	Azygous ACA with Distal ACA	Absent	2.4 x 2.2 mm	1.5 mm	2.4 mm	0.6
17	54	F	Focal Cisternal	Absent	Absent	1	Absent	Absent	0						
18	75	M	diffuse	Absent	Present	4	Absent	present	1	M2 segment of Lt MCA,Inferior division	Absent	7.5 x 6.0 mm	4.2 mm	7.5 mm	0.5
19	48	F	Focal Cisternal	Absent	Absent	1	Absent	Absent	0						
20	40	F	Focal Cisternal	Absent	absent	1	Absent	Absent	1	Ophthalmic seg of Lt ICA	Absent	5.0 x 3.0 mm	2.2 mm	5.2 mm	0.42
21	54	M	Focal Cisternal	Absent	Absent	1	Absent	Absent	1	V4 Left VA	Absent	7.1 x 6.9	3.8 mm	7.2 mm	0.52
22	75	F	Diffuse	Absent	Present	4	Absent	Absent	2	ACOM	Absent	4.0 x 2.3 mm	2.1 mm	4.0 mm	0.5
										Distal M1 Rt MCA	Absent	2.6 x 2.0 mm	1.3 mm	2.7 mm	0.48

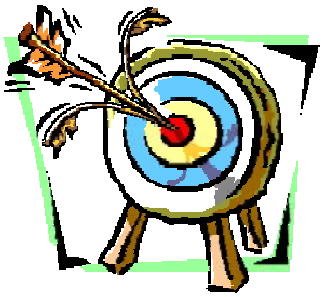
SL.NO	Age	Sex	SAH Focal /Diffuse	Midline shift	Intraventricular extension	Grade of SAH	Hydrocephalus	Intraparenchymal hemorrhage	No. of aneurysms	Aneurysm site	Thrombus	Size	Neck	Dome	N/D Ratio
23	70	M	Focal cisternal	Absent	Absent	1	Absent	Absent	0						
24	78	M	Focal cisternal	Absent	Absent	1	Absent	Absent	0						
25	56	M	Diffuse	Absent	Present	4	Present	Absent	0						
26	52	M	Focal cisternal	Absent	Present	2	Absent	Present	1	A5 Left ACA	Absent	4.0 x 3.1 mm	2.1 mm	4.0 mm	0.5
27	60	F	Focal Cisternal	Absent	Absent	1	Absent	Absent	1	Rt Supraclinoid ICA	Absent	1.4 x 1.3 mm	1.2 mm	1.5 mm	0.8
28	65	M	Focal Cisternal	Absent	Absent	1	Absent	Absent	1	ACOM	Absent	8.0 x 7.0 mm	4.8 mm	9.0 mm	0.53
29	50	F	Diffuse	Absent	Present	4	Present	Absent	1	ACOM	Absent	7.0 x 4.0 mm	3.7 mm	7.2 mm	0.51
30	44	F	Diffuse	Absent	Present	4	Absent	Absent	1	Rt PICA	Absent	5.7 x 4.5 mm	2.0 mm	5.9 mm	0.33
31	52	M	Focal	Absent	Absent	1	Absent	Absent	0						
32	55	F	Diffuse	Absent	Present	4	Present	Absent	1	Lt Supraclinoid ICA	Absent	6.2 x 5.4 mm	3.0 mm	6.5 mm	0.46
33	68	F	Diffusel	Absent	Absent	3	Absent	present	1	At bifurcation of Rt MCA	Absent	4.2 x 4.0 mm	3.2 mm	4.2 mm	0.76
34	60	F	Diffuse	Absent	Present	4	Present	Present	1	ACOM	Partially Thrombosed	2.2 x 1.7 mm	1.4 mm	2.2 mm	0.63
35	48	F	Diffuse	Absent	Present	4	Present	Absent	2	ACOM	Absent	5.0 x 4.0 mm	2.1 mm	5.2 mm	0.4
										Left Paraophthalmic	Absent	9.0 x 4.0 mm	1.8 mm	9.0 mm	0.2
36	70	M	Diffuse	Absent	Present	4	Present	Absent	2	Distal Rt ACA	Absent	6.1 x 5.1 mm	2.6 mm	6.2 mm	0.4
										Supraclinoid Rt ICA	Absent	4.6 x 4.0 mm	2.8 mm	4.6 mm	0.6
37	37	M	Diffuse	Absent	Present	4	Present	Absent	0						
38	41	M	Diffuse	Absent	Absent	3	Absent	Absent	1	Basilar tip	Absent	5.2 x 4.0 mm	2.2 mm	5.2 mm	0.4
39	62	M	Diffuse cisternal	Absent	Absent	3	Absent	Absent	0						
40	68	F	Focal Cisternal	Absent	Absent	1	Absent	Absent	1	Fusiform aneurysm of Rt PCA	Absent	26mm			
41	59	M	Diffuse cisternal	Absent	Present	4	Absent	Absent	1	Lt PICA	Absent	4.0 x 3.2 mm	2.1 mm	4.0 mm	0.5
42	71	F	Focal cisternal	Absent	Present	2	Absent	Absent	0						
43	45	M	Diffuse cisternal	absent	present	4	absent	present	1	ACOM	Absent	6.5 x 4.1 mm	3.1 mm	6.5 mm	0.47
44	40	F	Diffuse cisternal	present	Present	4	Absent	Present	1	M1 seg of Right Middle cerebral artery	absent	3.6 x 3.5 mm	2.7 mm	3.6 mm	0.75
45	49	M	Focal	Absent	Absent	1	Absent	Absent	1	Fusiform aneurysm of Basilar artery		27mm			

ANNEXURE-VI

KEY TO MASTER CHART

M	**:**	Male
F	**:**	Female
ACA	**:**	Anterior cerebral artery
ACOM	**:**	Anterior communicating artery
BA	**:**	Basilar artery
MCA	**:**	Middle cerebral artery
ICA	**:**	Internal cerebral artery
PCA	**:**	Posterior cerebral artery
PICA	**:**	Posterior inferior cerebellar artery
VA	**:**	Vertebral artery

Introduction



Objectives

Review of Literature

Methodology

Results

Discussion

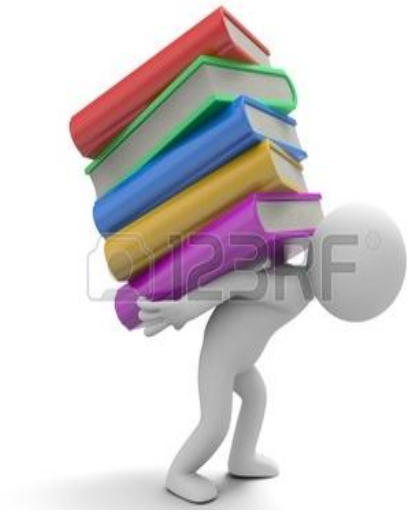
Conclusion

Summary

Bibliography

Annexure-I

Annexure-II



Annexure-III

Annexure-IV

Annexure-V

Annexure-VI