Subarachnoid hemorrhage in Kashmir: Causes, risk factors, and outcome

Abdul Rashid Bhat, Mohammed AfzalWani, Altaf R. Kirmani
Department of Neurosurgery, Sher-i-Kashmir Institute of Medical sciences (SKIMS), Srinagar, Kashmir, India

ABSTRACT

Context: Kashmir, a snow bound and mountain locked valley, is populated by about 7 million ethnic and non-migratory Kashmiris who have specific dietary and social habits than rest of the world. The neurological disorders are common in Kashmiri population.

Aims: To study the prevalence and outcome of spontaneous intracranial subarachnoid hemorrhage (SAH) in Kashmir compared with other parts of the world.

Settings and Design: A retrospective and hospital based study from 1982 to 2010 in the single and only Neurosurgical Centre of the State of Jammu and Kashmir.

Materials and Methods: A hospital based study, in which, information concerning all Kashmiri patients was collected from the case sheets, patient files, discharge certificates, death certificates, and telephonic conversations with the help of Medical Records Department and Central Admission Register of Sher–i-Kashmir Institute of Medical Sciences, Kashmir India.

Statistical Analysis: Analysis of variance and students T-test were used at occasions.

Results: Incidence of SAH in Kashmiris is about 13/100,000 persons per year. SAH comprises 31.02% of total strokes and aneurysmal ruptures are cause of 54.35% SAHs. The female suffers 1.78 times more than the male. Total mortality of 36.60% was recorded against a good recovery of 14.99%. The familial SAHs and multiple aneurysms were also common. Intra-operative finding of larger aneurysmal size than recorded on pre-operative computed tomography (CT) angiogram of same patients was noteworthy. In 493 patients of SAH, the angiography revealed 705 aneurysms.

Conclusion: Spontaneous intracranial subarachnoid hemorrhage, due to aneurysmal rupture, is common in Kashmir, with worst outcome. Food habits like “salt-tea twice a day”, group-smoking of wet tobacco like “Jejeer”, winter season, female gender, hypertension, and inhalation of“Kangri” smoke are special risk factors of SAH, in Kashmiris. The plain CT brain and CT angiography are best diagnostic tools. The preventive measures for aneurysmal formation and rupture seems most promising management of future. The detachable endovascular aneurysmal occupying video assisted micro-camera capsules or plugs may be future treatment.

Key words: Aneurysms, Kashmir, outcome, risk factors, subarachnoid hemorrhage

Introduction

Spontaneous intracranial subarachnoid hemorrhage is the presence of blood in the subarachnoid space, bounded by the arachnoid and piamater, due to the rupture of either an aneurysm or arterio-venous malformation or due to hypertension or an unknown cause.[1] In 1924, London neurologist, Sir Dr. Charles P. Symonds (1890-1978), gave a complete account of all major symptoms of subarachnoid hemorrhage (SAH), and he coined the term “spontaneous subarachnoid hemorrhage.”[2] SAH is a form of stroke and comprises 1-7% of all strokes.[3] Spontaneous SAH is sudden in 90% cases, and is characterized by sudden onset, severe headache of bursting nature in 60% cases, usually in occipital region, irrespective of aneurysmal rupture. This can be diagnosed by standard tools like lumbar puncture, CT-scan, and four-vessel cerebral angiography.[4] The features of meningeal irritation, minimal neurologic findings of localizing value, and presence of blood in cerebrospinal fluid (CSF), along with headache, nausea, vomiting, and transient loss of consciousness are described by two-thirds of patients experiencing SAH.[5] Hippocrates in his “epidemics” described...
Asian Journal of Neurosurgery
Vol. 6, Issue 2, July-December 2011

Bhat, et al.: Subarachnoid hemorrhage in Kashmir

a patient with convulsions, paralysis of right arm, and loss of speech, in what Garrison believed was the first written description of aphasia. Hippocrates (331 BC) mentioned apoplexy several times in his aphorism as, “persons are mostly subjected to apoplexy between the ages of 40 and 60”.[6,8] The incidence of SAH in the United States of America (USA) on an average has remained constant at approximately 11 per 100,000 population, annually, while the deaths from SAH account for about 16 per 100,000 population.[5,9‑10] The assessed natural history of SAH in a patient states that there is just one chance of good recovery out of five and one chance of getting crippled by the disease, while asthere are three chances out of five to die sooner or later.[11] Even 10-15% die before reaching hospital, and those who survive, often have neurological or cognitive impairment.[12,13] The early concept of ‘aneurysms and SAH in India being uncommon, by B. Ramamurthi, has been proved wrong over time, but the real incidence of SAH in India, due to lack of exact epidemiological data, remains questionable.[14]

The Department of Neurosurgery, SKIMS, Kashmir, is a single tertiary neurosurgical care unit working under a uniform protocol in the State of Jammu and Kashmir, which has been equipped with all facilities to manage surgically, patients of subarachnoid hemorrhage, since its beginning in 1982. Yet, there is no study available on spontaneous intracranial subarachnoid hemorrhage in Kashmir. The present study, though hospital based, also serves as epidemiology of SAH in ethnic Kashmir in the absence of any other neurological center in the state of Jammu and Kashmir.

Materials and Methods

All (907) patients were Kashmiris who belonged to the north Indian province of Kashmir Valley, which is mountain‑locked from rest of the country (India), China, Pakistan, and Afghanistan, and goes under‑cover of a heavy snow blanket during winters with sub‑zero temperatures causing severe chill. The Department of Neurosurgery admitted all intracranial spontaneous subarachnoid hemorrhages (SAH) since Dec., 1982 either directly or as referred cases, from all the hospitals of the Kashmir Valley, for further management. This is a single Neurosurgical Unit in J and K, and has a uniform protocol to manage SAH patients which is backed by a methodical and advanced Medical Records Department to maintain the records. The patient case sheets, files, discharge certificates, and death certificates were retrospectively studied from January 1983 to June 2010. Based on the newer, better, and advanced investigations for the detection of SAH and its causes as well as for the ease of interpretation, the study was divided into three eras’ i.e. I, II, and III. The era I ‑ of carotid angiography ‑ spans from January 1983 to December 1992 (10 years). The era II ‑ of non‑contrast cranial computed tomography (NCCT) of brain, digital subtraction angiography (DSA), and prophylactic “Triple H” therapy ‑ stretches from January 1993 to December 2002 (10 years). The era III ‑ of computed tomography (CT)‑angiography and magnetic resonance imaging (MRI) ‑ extends from January 2003 to June 2010 (7‑1/2 years). The records showed that patients were triaged according to the history, and clinical status as assessed by the Glasgow Coma Scale (GCS), Hunt and Hess clinical grading, World Federation of Neurological Surgeons (WFNS) grading, and imaging status as assessed by the Fischer grading system (amount of blood on CT‑scan), DSA, two/four vessel carotid angiography, and CT‑angiography.15‑18 The protocol in managing all SAH patients was early surgery, if able to sustain protocol requirements, and (since 1993) prophylactic hypertensive hypervolemichemodilution “triple‑H” therapy.19 After resuscitation, either a lumbar puncture (LP) or plain CT scan brain followed by bilateral carotid angiography (holds true from Jan., 1983 to Dec., 2002) or immediate NCCT‑scan brain (if SAH was found, no lumbar punctures) followed by either DSA, bilateral carotid angiography, or CT‑angiography (from January 2003 to June 10). The SAH was recognized by the lumbar puncture and NCCT brain, while as the cause of SAH like an aneurysm or an arterio‑venous malformation, was identified on carotid angiography, CT‑angiography, and DSA. After angiography, revealed an aneurysm(s) or arterio venous malformations (AVMs), surgical intervention was performed, while cases with negative angiography were subjected to the repeat angiography. The patients with SAHs who had the past history suggestive of hypertension whether on regular or irregular treatment, irrespective of post‑SAH high or lower blood pressures, with negative carotid/CT angiography and again negative repeat angiography were labeled as hypertensive SAHs. All the SAHs with hypertension, or of unknown cause, were managed conservatively. The outcome was assessed by the most popular scale, Glasgow outcome Scoring (GOS) scale.[20] The data was compiled, results were analyzed, and the analysis of variance was applied as required.

Results

Incidence

Presuming the population of Kashmir, India, as 7 million (Census 2010‑11), observations revealed that the prevalence of SAH in Kashmir is about 13/100,000 persons per year. Analysis showed that a total of 2923 strokes were admitted over a period of more than 27 years, including 907 (31.02%) spontaneous SAHs (907 out of 2923) in the Department of Neurosurgery, Sher ‑ i ‑ Kashmir Institute of Medical Sciences (SKIMS), Kashmir, India [Table 1]. The incidence of aneurysmal SAH in the eras ‑ I (10 years), II (10 years), and III (7 ½ years) were 27.99% (138/493), 32.86% (162/493), and 39.14% (193/493), respectively, while the mortalities for all SAHs were 20.38% (68/332), 17.77% (59/332), and 9.33% (31/332) in theeras, I, II, and III, respectively. A total of 8.37% (76/907) patients with SAHs had the history or evidence of blood‑relations either
Table 1: Age, sex and causes of subarachnoid hemorrhages

| Age (years) | No. of strokes | No. of SAH | Sex | *Children | Causes |
|-------------|----------------|------------|-----|-----------|--------|
|             | Males | Females | *Children | Males | Females | *Children | Cases of aneurysms | Cases of AVMs | Hypertension | Idiopathic |
| 0-10        | 165   | 57      | 0        | 0 | 0 | 57 | 5 | 12 | 0 | 40 |
| 11-20       | 75    | 27      | 7        | 5 | 0 | 15 | 12 | 7  | 0 | 8  |
| 21-30       | 278   | 99      | 42       | 57 | 0 | 0  | 40 | 2  | 21 | 35 |
| 31-40       | 453   | 124     | 59       | 65 | 0 | 0  | 53 | 5  | 39 | 27 |
| 41-50       | 979   | 318     | 87       | 231 | 0 | 0 | 225 | 2  | 91 | 0 |
| 51-60       | 673   | 185     | 45       | 140 | 0 | 0 | 177 | 1  | 57 | 0 |
| 61-70       | 241   | 76      | 51       | 25  | 0 | 0  | 18  | 0  | 53 | 5  |
| 71-80       | 59    | 21      | 9        | 12  | 0 | 0  | 12  | 0  | 9  | 0  |
| Total       | 2923  | 907     | 300      | 535 | #72 | | 493 | 29 | 270 | 115 | 54.35% |

SAH - Subarachnoid hemorrhage; AVM - Arterio venous malformations; *Mean age-group=31 – 50 years. *Children were those patients who had an age of 18 years and less.

SAH forms 31.02% (907/2923) of all strokes. *Male: Female ratio of SAH=1.00: 1.78. *Aneurysmal distribution: Males=291; Females=297; Children=29. *AVM distribution: Males=4; Females=3; Children=19. *Presuming 7 million as population of Kashmir, India, the incidence/prevalence of SAH is 13/100,000 population per year. *SAH forms 31.02% (907/2923) of all strokes. *Age group of 41-50 years reveals most strokes (36.51%) and SAHs (55.45%) of all SAHs.

dead or being treated for SAH, and 55.26% (42/76) of these, were aneurysmal.

Age and sex

Age group of 41-50 years comprised of 35.06% (318/907) of all patients with 9.60% (87/907) male and 25.46% (231/907) female, though the age group most affected by the SAH was 21-60 years, comprising of 80.04% (726 patients) patients of a total of 907 SAHs, while 55.45% (503/907) patients of these were from the age group of 41-60 years. The age group least affected was, 11-20 years, with 2.97% (27/907) children/adult SAHs and elderly above 70 years with 2.31% (21/907) of all SAHs [Table 1]. The children in the age group of 0-10 years comprised 6.28% (57/907) of SAHs. The male formed 33.07% (300/907) and female 58.98% (535/907) of all SAHs, with a male/female ratio of 1.00: 1.78. However, the age group of 41-50 years comprised of maximum male i.e. 29% (87/300) and female i.e. 43.17% (231/535) of all SAHs, while, as most aneurysmal SAHs (225/493 = 45.63%) and hypertensive SAHs (91/270 = 33.70%) also belonged to the same age group. The aneurysmal SAHs comprised of 38.74% (191/493) male, and 60.24% (297/493) female, and 35.06% (318/907) of all SAHs. The age group of 41-50 years comprised of maximum male i.e. 29% (87/300) and female i.e. 43.17% (231/535) of all SAHs, while, as most aneurysmal SAHs (225/493 = 45.63%) and hypertensive SAHs (91/270 = 33.70%) also belonged to the same age group. The aneurysmal SAHs comprised of 38.74% (191/493) male, and 60.24% (297/493) female, and 35.06% (318/907) of all SAHs.

Signs and symptoms

The aneurysmal SAHs had 73.22% (361/493) headaches and hypertensive SAHs had 80% (216/270) headaches. The meningeal irritation was the most common sign, 68.46% (621/907), found in all SAHs, and 50.70% (250/493) in aneurysmal SAHs. The fundal abnormalities were 31.86% (289/907) common in all SAHs and 89.25% (241/270) common in hypertensive SAHs, while 16.43% (81/493) of aneurysmal SAHs showed fundal abnormalities. The 3rd nerve palsy was seen in 9.59% (87/907) of all SAHs and in 15.61% (77/493) of aneurysmal SAHs. The electrocardiographic (ECG) changes in the form of QT prolongation, Q waves, and cardiac dysrhythmias have been seen in 50.93% (462/907) of all SAHs and in 55.78% (275/493) of aneurysmal SAHs.

Neurological status on admission

All the SAHs were evaluated and graded on admission, by the clinical and radiological scores like GCS Score, WFNS grading, Hunt and Hess grading, and Fischer grading systems as follows.

Glasgow coma scale score and WFNS grading system

Nearly half of the 907 SAH patients, 49.28% (447/907), were admitted with a GCS Score of 13-14, and half of these, without a focal neurodeficit, thus fulfilling the WFNS grade criteria of 2 and 3. The aneurysmal SAHs were admitted mostly, i.e. 49.49% (244/493), with a GCS score of 13-14 and WFNS grade of 2 and 3. The best admission GCS score of 15, and WFNS grade of 1, was found only in 19.26% (95/493) aneurysmal SAHs. While the worst GCS score of 3-6, and WFNS grade 5, was applicable to 18.86% (93/493) SAHs caused by the aneurysms, comparatively small number (12.37% i.e. 93/493) of aneurysmal SAHs were admitted with a GCS score of 13-15 and WFNS grade of 1, 2, and 3.

Hunt and Hess grading system

About 36.38% (330/907) of all SAHs were admitted in Hunt and Hess good grades (I, II), resulting in a mortality of 19.69% (65/330). The SAHs with poor grades (IV, V), 35.61% (323/907), had worst mortality of 63.15% (204/323), and moderate grade (III) SAHs admitted, 28.00% (254/907), were having a death rate of 24.80% (63/254). By sex incidence, more female, i.e. 28.44% (258/907) were in poorer grades
Risk factors
Adults formed 92.06% (835/907) cases of all SAHs. Aneurysms were the leading cause, 54.35% (493/907), of all SAHs. The female outnumbered with 58.98% (535/907) SAHs, mostly postmenopausal, and depicted a male/female ratio of 1.00/1.78. Familial SAHs were found to occur in 8.37% (76/907), and most, 55.26% (42/76), of these were aneurysmal in origin. The largest number of SAHs, 77.39% (702/907), occurred, and got admitted in emergency, during chilly winter months of the year. Long (more than 10 years) history of tobacco smoking, in the form of cigarettes, huka (as JEJEER special to Kashmir), and pipes, was confirmed in 66.48% (603/907) patients. The use of sympathomimetic drugs like phenylpropanolamine, especially during chilly winters, was found in all cases. The history of intake of dietary salt in a high proportion, as Kashmiri “salt-tea, twice a day” is found in all 100% (907) SAHs. The use of “kangri” for continuous warmth over winters was found in 98% SAHs. The silent and continuous stream of coal smoke inhaled day and night under ‘pheran’ (robe) in winters may be a risk for SAH.

Investigations
Lumbar puncture
The lumbar puncture was performed in 69.45% (630/907) cases. Though, initially, every patient of SAH was diagnosed by lumbar puncture only, it was negative in 5.07% (32/630) cases. Thus, LP was diagnostic (sensitive) in 94.9% (598/630) SAHs.

Non-contrast CT-scan brain
The NCCT brain was performed in 86.54% (785/907) SAHs. The initial scan of 61.40% (482/785) SAH patients showed blood in subarachnoid space, and 73.23% (353/482) of these were aneurysmal SAHs. About 20.89% (164/785) SAHs, which underwent initial plain CT-scan brain, revealed aneurysms as high attenuating lesions [Figure 1]. The NCCT brain showed that of all intraventricular hemorrhages (IVHs), 50.56% (90/178) IVHs were from aneurysmal SAHs. The intracerebral hemorrhages (ICH), lobar, thalamic etc. were found on initial CT-scan in 15.92% (125/785) of SAHs, mostly in hypertensive and aneurysmal SAHs. The re-bleeding was confirmed in 25.35% (199/785) patients on CT-scan, and 94.47% (188/199) re-bleeds were aneurysmal SAHs. The hydrocephalus was found in 25.35% (199/785) scans, most of these, i.e. 55.42% (46/83), belonged to aneurysmal SAHs. There were 3.94% (31/785) (IV, V) of SAH compared with males i.e.4.18% (18/431). The aneurysmal SAHs (493 cases) were equally found in poor grade (34.68% = 171/493) and good grade (34.07% = 168/493) of Hunt and Hess grading system, with an overall mortality of 47.59% (158/332). Out of 29 SAHs due to AVMs, 24.13% (7/29) were in the poor grades, IV, V SAH, and 62.06% (18/29) had grade I and II SAH. But a total death rate of 0.60% (2/332) for AVMs was negligible as compared to aneurysmal, hypertensive, and idiopathic SAHs [Table 3].

Table 2: Admission GCS score and WFNS grading of SAH patients

| WFNS grade | GCS score | Aneurysms | AVM | Hypertension | Idiopathic | Total |
|------------|-----------|-----------|-----|-------------|------------|-------|
| 0          | No SAH    | 0         | 0   | 0           | 0          | 0     |
| 1          | 15        | 95        | 16  | 20          | 6          | 137   |
| 2          | 13-14, no deficit | 108       | 2   | 52          | 40         | 202   |
| 3          | 13-14+ deficit | 136      | 4   | 81          | 24         | 245   |
| 4          | 7-12      | 61        | 4   | 36          | 26         | 127   |
| 5          | 3-6       | 93        | 3   | 81          | 19         | 196   |
| Total      | 493       | 29        | 270 |            | 115        | 907   |

GCS - Glasgow coma scale; WFNS - World federation neurological surgeons; AVMs - Arterio venous malformation; SAH - Subarachnoid hemorrhage; Familial SAHs accounted for about 8.37% (76/907) of all subarachnoid hemorrhages and most of these, 55.26% (42/76), were aneurysmal SAHs which contributed 8.51% (42/493) to all aneurysmal SAHs.

Table 3: Clinical grading (Hunt-Hess scale) of SAH at admission related to cause and sex

| Variables | I | II | III | IV | V | Total |
|-----------|---|----|-----|----|---|-------|
| Aneurysms cases | 54 | 114 | 154 | 63 | 108 | 493   |
| AVM cases | 15 | 3   | 4   | 5  | 2  | 29    |
| Hypertension | 35 | 59  | 74  | 32 | 70  | 270   |
| Idiopathic | 8  | 42  | 22  | 17 | 16  | 115   |
| Total | 112 | 218 | 254 | 127 | 196 | 907   |
| Males | 54 | 111 | 97  | 20 | 188 | 1010  |
| Females | 50 | 97  | 130 | 99  | 159  | 535   |
| Children | 8  | 10  | 27  | 8  | 19  | 72    |
| Deaths | 17 | 48  | 63  | 61 | 143 | 332   |
| Total | 112 | 218 | 254 | 127 | 196 | 907   |

SAH - Subarachnoid hemorrhage; *Of all aneurysmal SAHs (493), 32.04% (158/493) died. *Poor clinical grade (IV, V) SAH has 63.15% (204/323) deaths.

Figure 1: High attenuating lesion (aneurysm) of left internal carotid artery on plain CT-scan brain

Table 4

| Variables | I | II | III | IV | V | Total |
|-----------|---|----|-----|----|---|-------|
| Aneurysm cases | 54 | 114 | 154 | 63 | 108 | 493   |
| AVM cases | 15 | 3   | 4   | 5  | 2  | 29    |
| Hypertension | 35 | 59  | 74  | 32 | 70  | 270   |
| Idiopathic | 8  | 42  | 22  | 17 | 16  | 115   |
| Total | 112 | 218 | 254 | 127 | 196 | 907   |
| Males | 54 | 111 | 97  | 20 | 188 | 1010  |
| Females | 50 | 97  | 130 | 99  | 159  | 535   |
| Children | 8  | 10  | 27  | 8  | 19  | 72    |
| Deaths | 17 | 48  | 63  | 61 | 143 | 332   |
| Total | 112 | 218 | 254 | 127 | 196 | 907   |

SAH - Subarachnoid hemorrhage; *Of all aneurysmal SAHs (493), 32.04% (158/493) died. *Poor clinical grade (IV, V) SAH has 63.15% (204/323) deaths.
Fischer grade classification on NCCT brain

The SAH of Fisher grade 2 (SAH less than 1 mm thick on CT scan) and 3 (more than 1 mm thick) appearance was found in 61.40% (482/785) scans. Out of all Fisher grade 2 and 3 SAHs, the aneurysms were the cause of 73.23% (353/482) patients. The Fisher grade 4 (any thickness with intraventricular hemorrhage or parenchymal extension) appearance was found in 38.59% (303/785) of those scanned for SAH, and of these, 44.55% (135/303) patients were aneurysmal SAHs. The NCCT brain for SAH was 100% sensitive in the study [Table 4].

Angiography

The commonest diagnostic tool to reveal the cause of SAH was angiography which detected more than 705 aneurysms in 493 patients with a patient/aneurysmal ratio of 1.00: 1.43 [Table 5], and 29 AVMs in 29 SAHs, thus angiography was 62.36% (522/837) accurate. This procedure also revealed diffuse and local vasospasm in 41.93% (351/837) SAHs and had a mortality of 0.59% (5/837). Out of all SAHs (907), about 92.28% (837/907) patients were subjected to different types of angiographies available in different periods of time from 1983 to 2010 [Table 6]. However, 7.71% (70/907) patients were not subjected to angiography due to their death before angiography or were allergic to the contrast material used, so that angiography was abandoned.

Carotid/vertebral angiography

After LP proved the presence of SAH, 48.18% (437/907) patients were subjected to the unilateral or bilateral carotid and vertebral angiography to detect any aneurysms or/and AVMs as a cause of SAH. The unilateral carotid angiography was performed in 19.90% (87/437), bilateral in 66.59% (291/437), and four-vessel (+ vertebral) angiography was performed in 13.50% (59/437) SAH cases. The carotid/vertebral angiography proved positive in 49.19% (215/437) SAHs and negative in 50.80% (222/437) cases, with a positive to negative ratio of 0.96: 1.00. However, vertebral angiography alone proved negative in 75.57% (44/59) cases, i.e. less accurate than carotid angiography. The carotid angiography detected single aneurysms in 137 SAH cases, and two or more than 2 aneurysms in 63 SAHs, which amounted to more than 263 aneurysms in 200 aneurysmal SAHs [Figure 2]. The carotid angiography detected 15 AVM cases. The local vasospasm was revealed in 34.09% (149/437) and diffuse vasospasm was found in 15.10% (66/437) SAH cases. The carotid/vertebral angiography led to 1.14% (5/437) deaths, mostly (three out of five) due to vertebral angiography [Table 6].

CT-angiography

About 24.49% (205/837) patients were subjected to the CT-angiography. The CT-angiography was negative in 16.58%

---

Table 4: CT-scan findings and SAH Fisher grades in 785 patients

| Findings                                | Aneurysm cases | AVM cases | Hypertensive | Idiopathic | Fisher grades | Total |
|-----------------------------------------|----------------|-----------|--------------|-------------|---------------|-------|
| Blood in subarachnoid space             | 353            | 7         | 68           | 54          | 0             | 258   |
| Aneurysm as high attenuating lesion     | 164            | 0         | 0            | 0           | -             | -     |
| Intra-ventricular hemorrhage            | 90             | 3         | 70           | 15          | -             | -     |
| Intra-cerebral hemorrhage               | 45             | 6         | 61           | 13          | -             | 125   |
| Re-bleeding                             | 188            | 2         | 6            | 3           | -             | -     |
| Hydrocephalus                           | 46             | 1         | 31           | 5           | -             | -     |
| Infarct                                  | 15             | 1         | 10           | 5           | -             | -     |

SAH: Subarachnoid hemorrhage; *: Initial SAH lead to 24.25% (220 deaths out of 907) deaths. *A total of 21.94% (199/907) patients re-bled. *Aneurysms re-bled 94.47% (388/411) of all re-bleeds. *Re-bleeding caused 22.28% (112 deaths/299 re-bleeds) mortality, which comprises of 39.73% (112/282 deaths) of all deaths (36.60%=332/907)

Table 5: Arterial site and number of aneurysms in 493 patients

| 1Parent artery                        | No. of aneurysms on parent artery only | Simultaneous aneurysms on associated arteries other than parent A. | Total (%) |
|---------------------------------------|----------------------------------------|---------------------------------------------------------------|-----------|
|                                       | One Two Many                            | Same side of parent A. Other side of parent A.                |           |
| Internal carotid A.                   | 65 8 5                                  | 8 4 2 5 4 2                                                   | 103       |
| Anterior Com. A.                      | 156 1 0                                | 4* 5 4 4* 2 2                                                | 178 (36.10) |
| Middle cerebral A.                    | 91 12 3                                | 5 6 4 4* 5 2                                                | 132 (26.77) |
| Anterior cerebral A.                  | 7 6 3                                  | 5 4 3 5 3                                                | 38        |
| Posterior Com. A.                     | 5 0 0                                  | 3 0 0 2 0                                                  | 10        |
| Basilar A.                            | 14 1 0                                 | 2* 3* 0 3* 2* 0                                            | 25        |
| Vertebral A.                          | 3 0 0                                  | 2 0 0 1 1 0                                                | 7         |
| Total                                 | 341 28 11                              | 29 22 13 24 17 8                                           | 493       |

493 SAH patients had more than 705 aneurysms, $: (Parent Artery); Artery with ruptured aneurysm A. Artery; Com. Communicating; #Side of proximal control; *Side opposite to proximal control; Any of the two sides and anterior com. artery, since basilar A. is in the midline
Table 6: Angiographic types and findings in 837 patients

| Type                        | No. of cases | Cases with aneurysms | Cases AVMs | Negative angiography | Vasospasm | Complications |
|-----------------------------|--------------|----------------------|------------|----------------------|-----------|---------------|
|                             |              | Single               | Two or more|                      | Local     | Diffuse       | Hemiparesis | Re-bleed | Deaths |
| Carotid angiography (+Vertebral angiography) | 437          | 137                  | 63         | 15                   | 222       | 149           | 66         | 21       | 35     | 5     |
| Unilateral                  | 87           | 36                   | 20         | 7                    | 24        | 4             | 8          | 0        |        |      |
| Bilateral                   | 291          | 93                   | 38         | 6                    | 154       | 12            | 18         | 2        |        |      |
| Four-vessel                 | 59           | 8                    | 5          | 2                    | 44        | 5             | 9          | 3        |        |      |
| CT-angiography              | 205          | 103                  | 59         | 9                    | 34        | 62            | 13         | 0        | 3      | 0     |
| D S A                       | 147          | 97                   | 29         | 3                    | 18        | 52            | 9          | 0        | 0      | 0     |
| MR-angiography              | 48           | 4                    | 1          | 2                    | 41        | 0             | 0          | 0        | 0      | 0     |
|                             | 341          | 152                  |            |                      | 263       | 88            |            |          |        |      |
| Total                       | 837          | 493                  | 29         | 315                  | 351       | 64            |            |          |        |      |

AVM - Arterio venous malformations; *Angiography detected more than 705 aneurysms in 493 patients, 29 AVMs in 29 patients with an accuracy of 62.36% (522/837) Aneurysmal distribution: Males=191; Females=297; M: F Ratio=1.00: 1.55; Children=5

MR angiography was the least used tool to discern the cause of SAH. Only 5.73% (48/837) SAHs were subjected to this imaging procedure with only 14.58% (7/48) accuracy and 85.41% (41/48) negativity [Table 6]. Among SAHs caused by aneurysms and AVMs, 8.31% (4/48) showed single and 2.08% (1/48) depicted double aneurysms, while 4.16% (2/48) patients had AVMs.

Causes

The analysis proved four major causes of SAH in the Kashmir valley. The aneurysmal rupture, as a cause of SAH, was found in about more than half the cases, i.e. 54.35% (493/907), of all SAHs. A total of 705 aneurysms were found in 493 patients with a patient/aneurysm ratio of 1.00:1.43. The aneurysmal SAHs comprised of 38.74% (191/493) male, 60.24% (297/493) female, and 1.01% (5/493) children, with the M/F ratio of 1.00: 1.55 [Table 1]. The hypertension caused 29.76% (270/907) of all SAHs. Neither history nor investigations revealed any cause for 12.67% (115/907) of the SAHs which were labeled as idiopathic. The AVMs lead to 3.19% (29/907) subarachnoid hemorrhages, and most, i.e. 65.51% (19/29), of these were

Digital subtraction angiography or catheter angiography

The DSA or catheter angiography was performed on 17.56% (147/837) with >87% accuracy in determination of the cause of SAH and was negative in 12.24% (18/147) patients. About 65.98% (97/147) patients had single aneurysm and 19.72% (29/147) SAHs revealed two or more than two (multiple) aneurysms that made it 155 aneurysms in 126 patients of SAH. The vasospasm was found in 36.58% (75/205) patients. The procedure had negligible complications [Table 6].

Figure 2: Right carotid angiogram revealing giant aneurysm of middle cerebral artery

Figure 3: CT-Angiography depicting multiple aneurysms of Acom.A, MCA and basilar artery
The familial subarachnoid hemorrhage was found in 8.37% (76/907) patients, and most of these, 55.26% (42/76), were of aneurysmal origin, making 8.51% (42/493) of all aneurysms. The familial SAHs were also found due to hypertensive (34.21%=26/76), idiopathic (9.21%=7/76), and AVM (1.31%=1/76) causes.

**Site and number of aneurysms (multiple aneurysms)**

The 493 aneurysmal SAH patients were harboring 705 aneurysms, detectable angiographically. The aneurysms were the most common cause of SAH in 54.35% (493/907) patients. The 69.16% (341/493) aneurysmal SAHs, each, harbored single aneurysm, and 30.83% (152/493) cases had two and more than two (multiple) aneurysms. The patients with middle cerebral artery aneurysmal rupture had most multiple aneurysms i.e. 31.02% (41/132) [Table 5]. The most common arterial site for aneurysms was anterior communicating artery (A Com A), accounting for 36.10% (178/493) of all aneurysmal SAHs. The A. Com. Artery contributed 87.64% (156/178) cases of single‑aneurysms and 0.56% (only one case i.e. 1/178) double‑aneurysm case to the study. The rest of the 11.79% (21/178) A. Com. A. aneurysm cases had a single parent artery aneurysm in association with aneurysms of the other arteries, on the same side of the proximal control (7.30%=13/178) and on the opposite side to that of the parent artery (4.49%=8/178) [Table 6]. The 26.77% (132/493) aneurysmal SAHs were caused by the middle cerebral artery (MCA) aneurysms. Among MCA aneurysmal SAHs, the patients with single MCA aneurysms accounted for 68.93% (91/132), and double aneurysms on the same parent artery occurred in 9.09% (12/132) patients. The patients with multiple (>2) aneurysms on the same parent (middle cerebral) artery accounted for 2.27% (3/132). The aneurysms on the other arteries in association with the parent artery (MCA) aneurysms, on the same side of the parent artery and on the opposite side to the parent artery, were found in 19.69% (26/132) patients. The aneurysms of internal carotid artery (ICA) were harbored by 20.89% (103/493) patients. The 63.10% (65/103) patients each, harbored a single aneurysm of ICA, while 7.76% (8/103) patients had double aneurysms on ICA, and multiple (>2) aneurysms were found on the parent artery (ICA) of 4.85% (5/103) patients. About 24.27% (25/103) patients with single ICA aneurysm each, had associated aneurysms on the other arteries of same and opposite sides to that of the parent artery. The anterior cerebral, posterior communicating, basilar and vertebral arteries had comparatively lower number of aneurysms [Table 5]. The commonest double aneurysm combination sites were anterior communicating and middle cerebral arteries, while commonest multiple (>2) aneurysm combination sites were anterior communicating, middle cerebral, and basilar arteries.

**Size of aneurysms**

The 493 aneurysmal SAHs with 705 aneurysms had variation in size.

(a) Small aneurysms (4 mm to 10 mm)=68.22% (481/705)
(b) Large aneurysms (11 mm to 25 mm)=27.80% (196/705)
(c) Giant aneurysms (>25 mm)=3.97% (28/705)

Half the giant aneurysms (50%=14/28) were found on the MCA, another one-fourth (25%=7/28) were detected in the internal carotid artery, and the rest (25%=7/28) occurred in the vertebra-basilar system [Figure 4]. Though small, large, or giant are absolute sizes, a parent artery‑related type of aneurysmal size grading was found much adjusting and relating, intra‑operatively. One finding is that an aneurysm of 5 mm is much smaller for internal carotid artery than for M2 or M3 segments of middle cerebral artery or for pericallosal artery.

**Familial SAHs and aneurysms**

The 8.37% (76/907) of all SAHs had evidence of blood relations to each other, and about 55.26% (42/76) of these were aneurysmal, contributing 8.51% (42/493) patients to aneurysmal cause of SAHs. The rest of the 44.73% (34/76) familial SAHs were of hypertensive (34.21%=26/76), idiopathic (9.21%=7/76), and AVM (1.31%=1/76) origin, amounting to 9.62% (26/270) hypertensive, 6.08% (7/115) idiopathic, and 3.44% (1/29) AVMs.

**Complications**

**Re-bleed**

The re-bleeding was confirmed by a CT scan in 25.35% (199/785) patients, leading to 56.28% (112/199) deaths. About 38.13% (188/493) aneurysmal SAHs re-bled and contributed 94.47% (188/199) patients to all re-bleeding [Table 4]. It was observed that after carotid angiography, increase in re-bleedings was 17.58% (35/199) [Table 6].

**Vasospasm**

The vasospasm was found in 40.20% (351/873) patients who had undergone angiography [Table 6]. The local vasospasm (related to parent artery of aneurysm and its branches) was depicted in 74.92% (263/351) and the diffuse vasospasm (related to all vessels) in 25.07% (88/351). The carotid angiography
showed vasospasm in 61.25% (215/351), CT angiography in 21.36% (75/351), and DSA detected vasospasm in 17.73% (61/351) patients. Of all cases (351), vasospasm due to the aneurysmal SAHs was found in 80.34% (282/351) patients.

Hydrocephalus
The hydrocephalus was found in about 10.57% (83/785) of those SAHs who underwent NCCT brain and more than half (55.42% = 46/83) of these were aneurysmal [Table 4].

Management
The 71.50% (649/907) SAHs underwent surgical and endovascular type of treatment, and 28.40% (258/907) of these hypertensive and idiopathic SAHs were treated conservatively. The surgical treatment included clipping, Ventriculo peritoneal (VP) shunts, external ventricular drainages (EVD), and carotid ligation (a few giant aneurysms). The 14.53% (130/907) SAH patients, with ventriculomegaly, were subjected to either external ventricular drainage (EVD) or/and VP shunts of mostly hypertensive and idiopathic origin. Endovascular treatment in the form of coiling/embolization and carotid ligation was required in 9.48% (86/907) aneurysms and AVMs [Table 7].

Clipping
The clipping was possible in 47.70% (433/907) of all SAHs, 0.88% (8/907) of these being AVMs, and the rest, 46.82% (425/907), aneurysms. Of all the aneurysmal SAHs (493), only 86.20% (425/493) aneurysms were clipped [Figure 5]. The single aneurysms were clipped in 96.23% (409/425) and the double aneurysms in 3.76% (16/425) patients. About 85.96% (153/178) Acom. A aneurysms, 98.48% (130/132) MCA and 85.44% (88/103) ICA aneurysms were clipped. The intraoperative aneurysmal size of same patient varied from the size on preoperative CT angiography by being 0.5-1.5 mm larger in case of distal anterior cerebral artery (DACA) and MCA aneurysms [Table 7].

Mortality
The initial episode of subarachnoid hemorrhage led to 24.26% (220/907) deaths and re-bleeding caused 12.35% (112/907) deaths. About 30.32% (275/907) died in the first two weeks and the remainder 6.29% (57/907) within the following four weeks. A total of 36.60% (332/907) patients of SAH died. Almost 1/3, 32.04% (158/493) of all aneurysms died, contributing 47.59% (158/332) to total mortality. The re-bleeding (112/332) contributed 33.73% to the overall mortality. Of all the deaths, there were 59.33% (197/332) female deaths, 30.42% (101/332) male deaths, and 10.24% (34/332) child deaths in SAH patients. The carotid angiography resulted in 1.14% (5/437) deaths, increasing the whole mortality by 1.50% (5/332) deaths [Tables 3, 6 and 7].

Outcome
Of all (907) SAH patients, 14.99% (136/907) SAHs showed good recovery, most of these were aneurysmal SAHs (69.11%; 94/136), although good recovery among aneurysmal SAHs was 19.06% (94/493). The severe disability and vegetative state occurred in 15.87% (144/907) and 12.56% (114/907) SAHs, respectively. However, more than 50% (58/114) patients in vegetative state were due to aneurysmal rupture [Table 7]. The hypertensive SAHs had a mortality of 45.92% (124/270), idiopathic SAHs 41.73% (48/115), aneurysmal SAHs 32.04% (158/493), and SAHs due to AVMs had 6.89% (2/29) mortality; a total SAH mortality of 36.60% (332/709).

Discussion
Incidence
SAH from a ruptured aneurysm accounts for approximately 5% of all strokes and incidence of SAH had remained stable

| Table 7: Management and Glasgow outcome scoring of SAH patients |
|----------------------|------------------|-----------------|-----------------|------------------|
| Variables            | Aneurysm | AVM | Hypertension | Idiopathic |
| Management           | 425      | 8   | 0             | 0             |
| a) Clipping          |          |     |               |               |
| b) Intervention      | 68       | 18  | 0             |               |
| c) EVD/VP            | 0        | 3   | 98            | 29            |
| d) Conservative      | 0        | 0   | 172           | 86            |
| Good recovery        | 94       | 8   | 31            | 3             |
| Moderate disability  | 119      | 8   | 21            | 33            |
| Severe disability    | 64       | 5   | 57            | 18            |
| Vegetative state     | 58       | 6   | 37            | 13            |
| Death                | 158      | 2   | 124           | 48            |
| Total                | 493      | 29  | 270           | 115           |

SAH - Subarachnoid hemorrhage; AVMs - Arterio venous malformation; *Embo: Embolization; #EVD: External Ventricular Drainage; SAH - Subarachnoid hemorrhage; VP: Ventriculo-Peritoneal; *Good recovery was seen in 14.99% (136 out of 907) of all SAH cases; 19.06% (94 out of 493) of aneurysms which formed 10.36% (94 out of 907) of all SAH cases

Figure 5: Intra-operative photograph of clipped left MCA aneurysm
at around 8 per 100,000 persons per year, over 35 years. In a small subset of studies, gender specific incidences were given, which indicated a higher incidence in women.[21] However, other study reports that SAH is a form of stroke and comprises 1-7% of all strokes.[3] The study at Sher-i-Kashmir Institute of Medical Sciences (SKIMS), Kashmir, observed that SAH formed 31.02% of all strokes and the overall incidence was about 13/100,000 population [Table 1]. The high proportion of SAHs compared to overall strokes in Kashmir may be due to racial or ethnic (population group of mountain locked Kashmir) factors. Though the incidence of aneurysmal SAH in the eras I (10 years), II (10 years), III (only 7½ years) were 27.99% (138/493), 32.86% (162/493), and 39.14% (193/493), the mortalities for all SAHs were 20.38% (68/332), 17.77% (59/332), and 9.33% (31/332) in eras, I, II, and III, respectively. The results differed in that initial (era I, 10 years) incidence was low, but mortality was high, and latter (era III, 7½ years) incidence was high, but mortality was low. This may be due to use of better and advanced diagnostic tools to detect aneurysmal SAH and improved care due to rise in learning curve of the treating neurosurgeons. Since community-based studies reported an incidence that ranged from 8.1 per 100,000 in Australia and New Zealand to 23 per 100,000 in Japan.[22,23] The lower incidence in some regions may be explained by racial differences, although in some studies the incidence of SAH in black populations was higher in comparison with white populations.[24,25] Incidences per 100,000 person-years were 22.7 (95% CI 21.9 to 23.5) in Japan, 19.7 (18.1 to 21.3) in Finland, 4.2 (3.1 to 5.7) in South and Central America, and 9.1 (8.8 to 9.5) in the other regions. There was wide variation in SAH incidence, ranging from 2 to 25 per 100,000 persons per year, with most regional incidences between 7 and 13 per 100,000 persons per year. The overall incidence of SAH is approximately 9 per 100,000 persons per year.[26] A large, multinational, World Health Organization study found that the age-adjusted annual incidence of SAH varied 10-fold between different countries, from 2.0 cases per 100,000 population in China to 22.5 per 100,000 in Finland.[27]

**Age and sex**

The Kashmir study revealed that male formed 33.07% and female 58.98%, of all SAHs, with a male/female ratio of 1.00: 1.78. However, the age group of 41-50 years comprised maximum male i.e. 29% (87/300) and female i.e. 43.17% (231/535) of all SAHs, while as most aneurysmal SAHs (225/493 = 45.63%) and hypertensive SAHs (91/270 = 33.70%) also belonged to the same age group. The aneurysmal SAHs comprised of 38.74% male, 60.24% female, and 1.01% children, with the M/F ratio of 1.00: 1.55 [Table 1]. SAH is reported to be 62% common in 5th and 6th decades of life. This is 54.3% common in female and 45.7% in male, while aneurysms are 59% common in female and 41% in male.[28] The incidence in women was 1.24 (1.09 to 1.42) times higher than in men; this gender difference started at age 55 years and increased thereafter. In the age group 25-45 years, incidence was significantly higher in men than in women, but in the age group 55-85 years, incidence was significantly higher in women than in men.[29] The studies have suggested that the gender difference is related to hormonal status, with incidence of SAH increasing with age, occurring most commonly between 40 and 60 years of age (mean age 50 years), but SAH can occur from childhood to old age, and is 1.6 times higher in women than in men.[30,31] The incidence of aneurysmal rupture is only 2-20/100,000 individuals/year.[32] Hemorrhage is more frequent in women than men (3:2 ratios) over the age of 40, but the reverse is true in those younger than 40 years.[33] Peak rupture rates occur between the ages of 50 and 60 years.[31] A series of 167 SAH patients is reported to have mean age ±SD 52.6 ± 14.1 and predominance (71%) of women.[32] The reasons for the overall higher incidence in women are not clear, but hormonal factors (including use of hormone replacement therapy) are a possible explanation.[33,34]

**Symptoms and signs**

The present study at SKIMS revealed that the most common symptoms were headache, 90.51%; loss of consciousness, 69.45%; weakness in a body part, 40.79%; visual disturbances, 29.98%; speech disturbances, 16%; and convulsions, 7.82%. The meningeval irritation was the most common sign, 68.46%, found in all SAHs, and 50.70% in aneurysmal SAHs. The fundal abnormalities were 31.86% common in all SAHs, and 89.25% common in hypertensive SAHs, while 16.43% aneurysmal SAHs showed fundal abnormalities. The 3rd nerve palsy was seen in 9.59% of all SAHs, and in 15.61% of aneurysm SAHs. The clinical presentation of aneurysmal SAH is one of the most distinctive in medicine. The sine qua non of SAH in an awake patient is the complaint of “the worst headache of my life,” described by 80% of patients who can give a history, but a warning sentinel headache is also described by 20% of patients.[35] Headache was reported in 10 to 48% of SAHs.[28,36] About 67.8% SAHs present with paralysis of a side, 75% loss of consciousness, while 78% patients had headache.[37] Fontanarosa retrospectively studied 109 patients with proven SAH and found headache in 74%, nausea or vomiting in 77%, loss of consciousness in 53%, and nuchal rigidity in 35%.[38] The speech disturbances ranged from 2 to 21.3%. Reportedly, visual disturbances occurred in 4 to 43% of patients.[37,38] A study reported meningeal irritation in 75%, 3rd nerve palsy in 14% and fundal abnormalities in 35% of SAH patients.[37] Seizures may occur in up to 20% of patients after SAH, most commonly in the first 24 hours and more commonly in SAH associated with intracerebral hemorrhage, hypertension, and middle cerebral and anterior communicating artery aneurysms.[39,40] More recent retrospective reviews report a low frequency of seizures ranging from 6 to 18%.[41] Other study showed 4% convulsions occurring in SAHs.[38] Delayed seizures occurred in 7% of patients in another series.[42]
Neurological status

The Kashmir study showed that 49.28% SAHs were admitted with a GCS score of 13-14 in WFNS grade 2 and 3. The worst GCS score of 6 and less was found in 21.60% cases i.e. WFNS grade 5. The aneurysmal SAHs were admitted mostly, i.e. 49.49%, with a GCS score of 13-14 and WFNS grade of 2 and 3. The best admission GCS score of 15 and WFNS grade of 1 was found only in 19.26% aneurysmal SAHs. The worst GCS score of 3-6 and WFNS grade 5 was applicable to the 18.86% SAHs caused by the aneurysms [Table 2]. The patient’s clinical status is assessed using the World Federation of Neurological Surgeons Scales and Hunt and Hess Scale.\(^{17,43}\) A study showed poor clinical condition on admission in 22% SAHs and amount of cisternal blood > median in 43% \(^{[32]}\). In the present study, 36.38% of all SAHs were admitted in Hunt and Hess good grade (I, II), resulting in a mortality of 19.69%. The SAHs with poor grade, (IV, V), 35.61%, had worst mortality of 63.15% and moderate grade (III) SAHs admitted, 28.00%, were having a death rate of 24.80%. The aneurysmal SAHs (493 cases) were equally found in poor grade (34.68%), and good grade (34.07%) of Hunt and Hess grading system with an overall mortality of 47.59%. The first scale of severity was described by Hunt and Hess in 1968, which showed 80% and 90% mortality in Hunt-Hess grade 4 and 5, respectively.\(^{[43]}\)

Risk factors

The present study observed that being an adult is a risk factor for SAH. Adults comprised of 92.06% of all SAHs. Aneurysms are the major cause of SAH with 54.35% of all SAHs. Familial SAHs occurred in 8.37% and mostly (55.26%) were aneurysmal in origin. However, it is reported that genetic factors explain only 10% of SAH, and most cases are attributed to smoking, hypertension, and excessive use of alcohol.\(^{[44]}\) Group smoking in the form of a special Kashmiri clay/copper water container with two wooden barrels, utilizing wet/dry tobacco called “JEJEER” is popular in Kashmir. This study revealed 66.48% patients were smokers. Also, the use of “Kangri” was noted in all SAHs. While warming in the cold season, the people of the Valley of Kashmir have a habit of using “Kangri” which uses slow and continuous burning coal in a baked clay pot over-woven by dried willow branches to hold it. Kangri emits silent and continuous stream of coal-smoke which is inhaled, day and night under “Pheran” (robe), by the most Kashmiris, and may be a risk for SAH. Racial differences have been reported in risk of SAH. Black Americans are at higher risk than white Americans.\(^{[45]}\) Multivariate models have found hypertension, smoking, and heavy alcohol use to be independent risk factors for SAH in the United States, Japan, the Netherlands, Finland, and Portugal.\(^{[46,50]}\) Sympathomimetic drugs, including cocaine and phenylpropanolamine are known risk factors for SAH.\(^{[51,52]}\) This study noted that phenylpropanolamine, as a decongestant and cold remedy, was much used during winters by all patients. The risk factors like smoking, female gender, hypertension, family history of cerebrovascular disease, and postmenopausal state are thought to be common for SAH and multiple aneurysms.\(^{[53]}\) The SKIMS study found females form more than half, 58.98%, of all SAHs, mostly postmenopausal, showing male:female ratio of 1.00: 1.78. There has also long been interest in the influence of meteorological and temporal factors on the incidence of SAH. Studies have provided variable results, but there appears to be a somewhat higher incidence of SAH in the winter months in the spring.\(^{[22,54]}\) Kashmir study revealed that most SAHs, 77.39%, got admitted during winters. Moreover, the use of high amount of dietary salt as “salt-tea, twice a day” by all patients has been a risk factor for hypertension and SAH.

Investigation

Lumbar puncture

Symonds described the use of lumbar puncture and xanthochromia in the diagnosis of SAH, and lumbar puncture showed evidence of hemorrhage in 3% of people in whom CT was found normal.\(^{[2,12]}\) The detection rate of SAH by lumbar puncture is reported to be 96.5%.\(^{[37]}\) The study at SKIMS confirmed the diagnostic sensitivity of lumbar puncture as 94.9%.

Plain CT-brain

The sensitivity of CT in the first 12 hours after SAH is 98% to 100%, declining to 93%, at 24 hours, and to 57 to 85%, six days after SAH.\(^{[15,56]}\) Computed tomography (CT scan) of the brain has a high sensitivity and will correctly identify over 95% of cases.\(^{[13]}\) The ruptured aneurysms in 21.1% SAH patients were detected as high attenuating lesions on plain CT-scan.\(^{[37]}\) The SKIMS study revealed 100% sensitivity of plain CT scan brain and 20.89% SAHs, which had high attenuating lesions on CT-scan, were confirmed aneurysms by angiography.

Carotid angiography

In study at Kashmir, 48.18% patients were subjected to carotid angiography. The unilateral carotid angiography was performed in 19.90%, and bilateral in 66.59% patients, and detected single aneurysms in 137 SAH cases and multiple aneurysms in 63 SAHs, which amounted to more than 263 aneurysms in 200 aneurysmal SAHs. A study reports that adequate carotid angiography detects berry aneurysms in 80% of those SAHs which are CT-negative.\(^{[4]}\) The carotid angiography has high positive results with a positive to negative ratio of 3.50: 1.00.\(^{[1]}\) In a series of SAH patients, carotid angiography was performed unilaterally in 38.2%, and bilaterally in 17% patients with 7.8% complications.\(^{[37]}\) The SKIMS study shows carotid angiography positive in 49.19% SAHs with a positive/negative ratio of 0.96/1.00, with a mortality rate of 1.14%.

CT angiography

Kashmir study proves that CT-angiography (CTA) was positive in 83.42% patients with an accuracy of > 83% and negative in 16.58%. About 50.24% had single and 28.78% had multiple
aneurysms, thereby detecting more than 221 aneurysms in 162 patients. Authors of a study have reported sensitivity of CTA for aneurysms between 77 and 100% and specificity between 79 and 100%. For aneurysms of 5 mm in size, CTA has sensitivity between 95 and 100% compared between 64 and 83%, when aneurysms are <5 mm. Among aneurysms detected on CTA and then undergoing surgery, 100% correlation was observed between CTA and catheter angiography. Velthuis and colleagues found that CTA is equal to catheter angiography in 80 to 83% of cases. In 74% of patients, catheter angiography performed after CTA did not reveal any additional information. CTA has been shown to be effective in determining the presence of severe vasospasm, but is less accurate in detecting mild and moderate vasospasm. The SKIMS study showed that CTA detected vasospasm in 36.58% patients. DSA or selective catheter cerebral angiography is currently the standard for diagnosing cerebral aneurysms as the cause of SAH. Approximately, 20 to 25% of cerebral angiograms performed for SAH will not indicate a source of bleeding. Repeat angiography after one week will show a previously unrecognized aneurysm in an additional 1% to 2% of cases. At SKIMS, DS-angiography detected single aneurysms in 65.98% patients and multiple aneurysms in 19.72% SAHs, which disclosed 155 aneurysms in 126 patients. DSA proved 87.76% sensitive and detected vasospasm in about 41.49% patients.

MR angiography

The sensitivity of three-dimensional time-of-flight magnetic resonance angiography (MRA) for cerebral aneurysms is between 55 and 93%. With aneurysms 5 mm, the sensitivity is 85% to 100%, whereas the sensitivity of MRA for detecting aneurysms <5 mm drops to 56%. The present study disclosed 14.58% accuracy for detecting aneurysms in SAH patients. Among SAHs caused by aneurysms and AVMs, 8.31% showed single and 2.08% depicted double aneurysms. This low accuracy could probably be due to the fact that firstly MRA was not available in the first 20 years of study at SKIMS. Secondly, only selected SAHs with negative angiography were subjected to the MRA-study due to cost factor. Thirdly, CT-angiography has become surgeon and patient friendly due to its accuracy and ease of doing as compared to MRA.

Causes

The SKIMS study disclosed the causes of SAH as 54.35% aneurysms, 29.76% hypertensive bleeds, 12.67% unknown-cause, and 3.19% AVMs. Locksley reports the causes of spontaneous SAH in a study as 51% aneurysms, 6% AVMs, 15% hypertension, and 22% idiopathic. Another author reported 36.7% SAHs due to aneurysms, 7.1% AVMs, 9.4% hypertension, and 46.8% as unknown causes. In one study, 85% cases of spontaneous SAH are caused by rupture of a cerebral aneurysm. Rinkel et al. reports that 15-20% spontaneous SAH has no detectable cause on the first angiogram.

Site of aneurysms

The study at Kashmir observed that angiography detected 705 aneurysms in 493 aneurysmal SAH patients. The anterior communicating artery (Acom.A) accounted for 36.10% aneurysms, middle cerebral artery (MCA) 26.77%, internal carotid artery (ICA) 20.89%, anterior cerebral artery (ACA) 7.70%, and vertebra-basilar (VB) arteries accounted for 6.49% aneurysms. A study by Pakarinen, 1967, revealed 40% aneurysms on the Acom.A, 36% on the MCA, 34% on the ICA and Pcom.A, and 6% aneurysms on the ACA. However, Locksley 1966 in a study reported 38.1% aneurysms on ICA and Pcom.A, 30.3% on Acom.A, 20.9% MCA, 5.8% ACA, and 5.5% aneurysms on the VB arteries. Tandon, India, 1988, detected 45.9% aneurysms on the ICA and Pcom.A, 24.8% Acom.A, 17.3% MCA, 10.2% ACA, and 1.08% aneurysms on the VB arteries. Another series showed 42.9% aneurysms on the ICA-Pcom.A complex, 25.7% aneurysms on the Acom.A, 18.7% aneurysms on the MCA, 8.5% on ACA, and 4% aneurysms on the VB-system. The SKIMS study also found the present classification of aneurysmal-size like small, large, and giant as absolute. The study found a new parent-artery related aneurysmal size grading accurate and intra-operatively adjusting. For example, two aneurysms of 5 mm size (small) each, located separately on the larger internal carotid artery and smaller M2 segment of MCA or DACA, is found intra-operatively small for the internal carotid artery and larger for the DACA or M2 of MCA.

Multiple aneurysms

A series of studies reported multiple aneurysms in patients of spontaneous SAH, at the rate of 9%, 18.5%, and to 20%, . The study in Kashmir found that 30.83% aneurysmal SAHs had multiple aneurysms accounting for >364 aneurysms and 69.16% aneurysmal SAHs harbored single aneurysms, i.e. 341 aneurysms. Thus, 493 aneurysmal SAHs carried >705 aneurysms in all.

Familial aneurysms

True familial intracranial aneurysm syndrome occurs when two first- through third-degree relatives have intracranial aneurysms. This is associated with SAH at a younger age, a high incidence of multiple aneurysms, and hemorrhages among siblings and mother-daughter pairings. In family members with the familial intracranial aneurysm syndrome, the risk of harboring an unruptured aneurysm was 8%. A study of 23 families with familial SAH found that having three affected relatives tripled the risk of SAH. When MRA was used to screen 8680 asymptomatic individuals for intracranial aneurysms, the overall incidence of aneurysms was 7.0%, but rose to 10.5% in those with a family history of SAH. However, another MRI study reported that 4% of relatives of sporadic SAH patients had aneurysms. In a large case control study, family history was found to be an independent risk factor for SAH. The study at SKIMS found that 8.37% of all SAHs...
had evidence of first through third degree blood relations to each other, and about 55.26% of these were aneurysmal, contributing 8.51% patients to aneurysmal SAHs. The rest of the 44.73% familial SAHs were of hypertensive (34.21%), idiopathic (9.21%), and AVM (1.31%) in origin.

**Re-bleeds**

Pakarinen found re-bleeding in 51.7% of SAH patients with 77.5% mortality. Several prospective follow-up cohorts have demonstrated that the risk of re-bleeding with conservative therapy is between 20 and 30% for the first month, after hemorrhage.[73] The risk of re-bleeding is highest immediately, following hemorrhage (4 to 6% over the first 24 hours), and declines over the next few days.[74] Re-bleeding was reported in 14% in a study of 167 patients.[72] At SKIMS, re-bleeding occurred in 25.35% SAH patients, leading to 56.28% deaths of re-bleed cases.

**Vasospasm**

The Kashmir study observed vasospasm in 40.20% of SAHs. The local vasospasm was more (74.92%) prevalent than local entity. The vasospasm was found in 30% patients of SAH in a study from India.[75] After aneurysmal SAH, angiographic vasospasm is seen in 30 to 70% of patients, with a typical onset three to five days after the hemorrhage, maximal narrowing at 5 to 14 days, and a gradual resolution over to four weeks.[76] The secondary cerebral ischemia occurred in 27% of a series.[72] The 1980s saw the introduction of triple H therapy as a treatment for delayed ischemia due to vasospasm, and trials with nimodipine in an attempt to prevent this complication.[76,77]

**Hydrocephalus**

The study at Kashmir found 10.57% SAH cases with hydrocephalus. Acute hydrocephalus (ventricular enlargement within 72 hours) is reported to occur in 20 to 30% of patients, and is more frequent in patients with poor clinical grade and higher Fischer scalescores.[78] Hydrocephalus was reported in 14.1% of SAH patients in a study.[75] Some SAH patients with acute hydrocephalus may benefit from early placement of a ventricular drain at the initial hospital.[79]

**Treatment**

Before 1970, carotid ligation was commonly used to treat recently ruptured intracranial aneurysms. Now, it is used for aneurysms that cannot be treated by direct surgical clipping or coil embolization.[80] A recent review by Taylor et al. from several series concluded that the risk of re-bleeding was lower than expected after carotid ligation for untreated ruptured aneurysms.[81] The Italian neurosurgeon, Dr. Guido Guiglielmi, introduced his endovascular coil treatment in 1991.[82] The International Subarachnoid Aneurysm Trial (ISAT) showed that in the aneurysms of anterior circulation (Acom.A/ACA), the likelihood of death or being dependent on others for activities of daily living was reduced (7.4% absoluteriskreduction, 23.5% relativerrisk reduction) if endovascular coiling was used as opposed to surgery.[83] Aneurysms of the VBcomplex and posterior cerebral artery are hard to reach surgically and are more accessible for endovascular management.[84-85] Aneurysms in the cavernous segment of the internal carotid artery are also difficult to treat with surgery, but may be treated relatively easily with coil embolization.[86] The SKIMS series required endovascular treatment in the form of coil embolization and carotid ligation in 9.48% aneurysms and AVMs (Table 7). Because of their morphology, MCA aneurysms can be difficult to treat by coil embolization, and surgical results for these aneurysms are often reported as more favorable.[85,87] The study at Kashmir revealed that out of all aneurysmal (493) SAHs, only 86.20% patients were clipped. The single aneurysms were clipped in 96.23% and double aneurysms in 3.76% patients (Table 7). About 85.96% Acom.A aneurysms, 98.48% MCA, and 85.44% ICA aneurysms were clipped. This study noted that a specific size of an aneurysm on the CT-angiography, grew larger intra-operatively by 0.5-1.5 mm, especially in distal anterior cerebral artery and MCA aneurysms. This may be due to dividing of arachnoid membrane that forms cicatrized and tight adhesions around the aneurysm to reduce its size and shrink it after its rupture. The size variation can also be the cause of difference in the measurement of intraluminal aneurysmal diameter on angiography and external diameter of aneurysm intraoperatively. This was more a finding noted in delayed surgery. The Kashmir study found 14.53% SAH patients having IVH, ventriculomegaly, and hydrocephalus were subjected to EVD either or/and VP shunts.

**Mortality**

SKIMS study recorded 36.60% mortality of SAH patients. About one-third of all aneurysmal patients i.e. 32.04% died, thus aneurysmal deaths contributed 47.59% to total mortality. About 33.73% of all deaths were those SAHs who died of re-bleeding. A study noted that initial bleeding episode of spontaneous SAH leads to 30% deaths in first 24 hours, 40% in first week, 60% at 6 months, and 70% at five years.[73] The mortality rate for SAH, in the 1966 Cooperative Study on Intracranial Aneurysms (CSIA), was 50% at 29 days and 33% SAH patients admitted through emergency department.[84] In a population-based study by Broderick et al., the 30-day mortality rate among all patients who suffered SAH was 45%, with the majority of deaths occurring in the first days after SAH.[85] Recurrent hemorrhage remains a serious consequence of aneurysmal SAH with a case fatality rate of 70% for persons who re-bleed.[81] Death rate of 17% in a total of 167 patients is observed.[32] Up to half of all cases of SAH are fatal, and 10-15% die before reaching a hospital.[12] The mortality for SAH is between 40 and 50%.[84] It is reported that a mortality of 77.5% occurred in patients of SAH who had re-bleed.[12] The initial hemorrhage can be devastating, and up to a quarter of patients die before reaching medical attention.[80] At SKIMS, the initial episode of subarachnoid hemorrhage led to 24.26%
deaths, and re-bleeding caused 12.35% deaths. About 30.32% SAHs died in the first two weeks, and the remaining 6.29% died within the following four weeks. This high mortality in SKIMS study could be due to late presentation of the patient directly to the Neurosurgical Care Unit, delayed referral from a peripheral hospital, and mal-transportation of a semi-comatose patient, without intubation and without securing airway, due to lack of trained drivers and attendants.

**Outcome**

The outcome for patients with SAH remains poor, with population-based mortality rates as high as 45% and significant morbidity among survivors. Perhaps the most meaningful and simplest measure of the effect of these deficits is whether the patient is able to return to his or her previous occupation. The study at Kashmir found good recovery in 14.99% SAH patients, most of these were aneurysmal SAHs 10.36%. Cross et al. found that two factors associated with better outcomes in the high-volume hospitals were greater use of endovascular services and a higher percentage of patients transferred from other hospitals. The outcome of 167 SAHs in a series is reported as 17% deaths; vegetative or severe disability, 15%; moderate disability, 25%; and good recovery, 43%. At SKIMS, about 19.95% SAHs of all causes ended up in moderate disability. The severe disability occurred in 15.87% and vegetative state was found in 12.56% SAHs. A total of 36.60% of all SAHs died.

**Conclusion**

The prevalence of aneurysmal SAH in ethnic Kashmiris is high owing to ‘typical diet and habit’ related risk factors and possible genetic factors. SAH, with an incidence of 13/100,000 per year, represents 31.02% of all strokes, a mortality of 36.60%, and good recovery of only 14.99% in Kashmir. The aneurysmal SAH at 54.35% is the commonest cause of mortality and morbidity. The female gender and thosein the age group of 40 to 60 years are at highest risk. The familial SAHs and multiple aneurysms in single patients have been recognized. The intra-operative size of middle cerebral and distal anterior cerebral artery, aneurysms have not been found to match the pre-operative assessed size on any angiogram. Moreover, the present classification of aneurysmal size has been found absolute and un-adjustable and not parent-artery related. The future of SAH and aneurysmal management is hidden in research for prevention rather than treatment, which is a compulsion. The future treatment may include permanentendovascular aneurysmal occupying detachable video assisted micro-camera capsules, plugs, or seals.

**References**

1. Pakarinen S. Incidence, aetiology and prognosis of primary subarachnoid hemorrhage: A study based on 589 cases diagnosed in a defined Urban Population during a defined period. ActaNeuroScand 1967;29:1-128.
2. Symonds CP. “Spontaneous subarachnoid hemorrhage”. Q J Med 1924;18:93-122.
3. Feigin VL, Rinkel GJ, Lawes CM, Algra A, Bennett DA, Van Gijn J, et al. Risk factors for subarachnoid hemorrhage: An update systemic review of epidemiological studies. J Stroke 2005;36:2773-80.
4. Gilroy J, Meyer JS. Cerebrovascular disease – subarachnoid hemorrhage. In: Gilroy J, Stirling Meyer J, (Editors) in Medical Neurology, 3rd ed. New York: McMillon publishing co., Inc; 1979; pp. 574-86.
5. Sahs A, Perret GE, Locksley HB, Nishioka H. Intracranial aneurysms and subarachnoid hemorrhage. Philadelphia: J.B. Lippincott; 1969.
6. Clarke E. Appoplexy in Hippocratic writings. Bull Hist Med 1963;37:301.
7. McHenry LC. Garrison’s History of Neurology. E Clarke on ‘Hippocratic Aphorisms on Apoplexy’ Springfield Illinois, Charles C Thomas 1969, pp 19-20.
8. Hippocrates Aphorisms. Translated by Sprendel C, London; 1755.
9. Smith RR, Up-Church JJ. Monitoring anti-fibrinolytic therapy in subarachnoid hemorrhage. J Neurosurgery 1973;38:339-44.
10. Ingall TJ, Wisnant JP, Wiebers DO, O’Fallon WM. Has there been a decline in subarachnoid hemorrhage mortality? Original contributions. Stroke 1989;20:718-24.
11. Ask-Upmarks E, Ingavar D. A follow-up examination of 138 cases of subarachnoid hemorrhage. Acta Med Scand 1950;138:15-31.
12. vanGijn J, Kerr RS, Rinkel GJ. Subarachnoid haemorrhage. Lancet 2007;369:306-18.
13. Suarez JI, Tarr RW, Selman WR. Aneurysmal Subarachnoid hemorrhage. New England J Med 2006;354:387-96.
14. Ramamurthi B. Are subarachnoid hemorrhages uncommon in India. Neurology India 1965;13:42-3.
15. Jennett B, Bond M. Assessment of outcome after severe brain damage. Lancet 1975;1:480-4.
16. Hunt W, Hess R. Surgical risk as related to time of intervention in the repair of intracranial aneurysms. J Neurosurg 1968;28:14-20.
17. Drake CG. Report of World Federation on Neurological Surgeons committee on a universal subarachnoid hemorrhage grading scale. J Neurosurg 1988;68:985-6.
18. Fisher CM, Kistler JP, Davis JM. Relation of cerebral vasospasm to subarachnoid hemorrhage visualized by CT-scanning. Neurosurgery 1980;6:1-9.
19. Origitano TC, Wascher TM, Reichman OH, Anderson DE. Sustained increased blood flow with prophylactic hypertensive, hypervolemic, hemodilution (“triple-H” therapy) after subarachnoid hemorrhage. Neurosurgery 1990;27:729-40.
20. Teasdale G, Jennett B. Assessment of coma and impaired consciousness, a practical scale. Lancet 1974;2:81-4.
21. Feigin VL, Lawes CM, Bennett DA, Anderson CA. Stroke epidemiology: A review of population-based studies of incidence, prevalence, and case-fatality in the late 20th century. Lancet Neurol 2003;2:43-53.
22. The Australasian Co-operative Research on Subarachnoid Haemorrhage Study Group, (Writing Committee: Anderson C, Hankey G, Jamrozik K, Dunhabin D). Epidemiology of aneurysmal subarachnoid haemorrhage in Australia and New Zealand: Incidence and case-fatality from the Australasian Co-operative Research on Subarachnoid Haemorrhage Study (ACROSS). Stroke 2000;31:1843-50.
23. Inagawa T, Takechi A, Yahara K, Saito J, Moritake K, Kobayashi S, et al. Primary intracerebral and aneurysmal subarachnoid hemorrhage in Izumo City, Japan, part I: Incidence and seasonal and diurnal variations. J Neurosurg 2000;93:958-66.
24. Lavados PM, Sacks C, Prina L, Escobar A, Tossi C, Araya F, et al. Incidence, 30-day case-fatality rate, and prognosis of stroke in Iquique, Chile: A 2-year community-based prospective study (PISCIS project). Lancet 2005;365:2206-15.
25. Kissela B, Schneider A, Kleinidorf D, Khoury J, Miller R, Alwell K. Stroke in a biracial population: The excess burden of stroke among blacks. Stroke 2004;35:426-31.
26. De Rooij NK, Linn FH, Van der Plas JA, Algra A, Rinkel GJ. Incidence of subarachnoid haemorrhage: A systematic review with emphasis on region, age, gender and time trends. J NeurolNeurosurg Psychiatry 2007;78:1365-72.
27. Ingall TJ, Asplund K, Mahonen M, Bonita R. A multinational comparison of subarachnoid hemorrhage epidemiology in the WHO MONICA stroke study. Stroke 2000;31:1054-61.

28. Locksley HB. Natural history of subarachnoid hemorrhage, intracranial aneurysms and arteriovenous malformations: Based on 6368 cases in the cooperative study. J Neurosurg 1966;25:219-39.

29. vanGijn J, Rinkel GJ. Subarachnoid haemorrhage: Causes and management. Brain 2001;124:249-78.

30. Rinkel GJ, Djibuti M, Algra A, van Gijn J. Prevalence and risk of rupture of intracranial aneurysms: A systematic review. Stroke 1998;29:251-6.

31. Okhuma H, Fujita S, Suzuki S. Incidence of aneurysmal subarachnoid hemorrhage in Shimokita, Japan, from 1899 to 1998. Stroke 2002;33:195-9.

32. Ynte MR, Slooter AJ, Bardoel A, Frijns CJ, Rinkel GJ, Wijmenga C. Genes and outcome after aneurysmal subarachnoid hemorrhage. J Neurol 2005;252:417-22.

33. Longstreth WT, Nelson LM, Koepsell TD, Van Belle G. Subarachnoid hemorrhage and hormonal factors in women. A population-based case-control study. Ann Intern Med 1994;121:168-73.

34. Mhurchu CN, Anderson CS, Jamrozik KD. Hormonal factors and risk of aneurysmal subarachnoid hemorrhage. An international population-based, case-control study. Stroke 2001;32:606-12.

35. Bassi P, Banderà R, Loiéro M, Togioni G, Mangoni A. Warning signs in subarachnoid hemorrhage: A cooperative study. ActaNeurolScand 1991;84:277-81.

36. Longmore M, Wilkinson I, Turmezei T, Cheung CK. Oxford Handbook of Clinical Medicine, 7th ed. Oxford: Oxford University Press;2007. p. 841.

37. Tandon PN. Subarachnoid hemorrhage in India: An Epidemiological study. ICMR Bull 1988;18:33-8.

38. Fontanarosa PB. Recognition of subarachnoid hemorrhage. Ann Emerg Med 1989;18:199-205.

39. Sundaram MB, Chow F. Seizures associated with spontaneous subarachnoid hemorrhage. Can J Neurol Sci 1986;13:229-31.

40. Ohm J. Hypertension as a risk factor for epilepsy after aneurysmal subarachnoid hemorrhage and surgery. Neurosurgery 1990;27:578-81.

41. Lin CL, Dumont AS, Lieu AS, Yen CP, Hwang SL, Kwan AL, et al. Characterization of perioperative seizures and epilepsy following aneurysmal subarachnoid hemorrhage. J Neurosurg 2003;99:978-85.

42. Claassen J, Peery S, Kreiter KT, Hirsch LJ, Du EY, Connolly ES, et al. Predictors and clinical impact of epilepsy after subarachnoid hemorrhage and surgery. Neurosurgery 2009;65:235-43.

43. Hunt WE, Hess RM. Surgical risk as related to time of intervention in the repair of intracranial aneurysms. J Neurosurg 1968;28:14-20.

44. Ruijgrok YM, Buskens E, Rinkel GJ. Attributable risk of common and rare determinants of subarachnoid hemorrhage. Stroke 2001;32:1173-5.

45. King JT Jr, Berlin JA, Flamm ES. Morbidity and mortality from subarachnoid and intracerebral hemorrhages in blacks as compared with whites. N Engl J Med 1992;326:733-6.

46. Alberts MJ, Quinones A, Graffagnino C, Friedman A, Roses AD. Risk of aneurysmal subarachnoid hemorrhage: A international cooperative study. J Neurosurg 1966;25:219-39.

47. Van Gijn J, van Dongen KJ. The time course of aneurysmal subarachnoid hemorrhage on early CT: Is lumbar puncture still needed after a negative scan? J NeurolNeurosurg Psychiatry 1995;58:357-9.

48. Van Gijn J, van Dongen KJ. The time course of aneurysmal subarachnoid hemorrhage on early CT: Is lumbar puncture still needed after a negative scan? J NeurolNeurosurg Psychiatry 1995;58:357-9.

49. Klein HG, Kato A, Daellenbach K, Sander O, Kaste M. Intracerebral hemorrhage and hormonal factors in women. A population-based, case-control study. Stroke 2001;32:606‑12.

50. Velthuis BK, Rinkel GJ, Rams M, Witkamp TD, Berkelbach van der, Spreen JK, et al. Subarachnoid hemorrhage: Aneurysm detection and preoperative evaluation with CT angiography. Radiology 1998;208:423-8.

51. Alperico RA, Patel M, Casey S, Jacobs B, Maguire W, Decker R. Evaluation of the circle of Willis with three-dimensional CT angiography in patients with suspected intracranial aneurysms. AJNR Am J Neuroradiol 1991;12:1517-8.

52. Anderson GB, Ashforth R, Steinke FE, Findlay JM. CT angiography for the detection of cerebral vasospasm in patients with acute subarachnoid hemorrhage. AJNR Am J Neuroradiol 2000;21:1011‑5.

53. Cioffi F, Pasqualin A, Cavazzoni P, Da Pian R. Subarachnoid hemorrhage of unknown origin: Clinical and tomographical aspects. ActaNeurolChir (Wien) 1989;97:31-9.

54. Forster DM, Steiner L, Hakanson S, Bergvall U. The value of repeat pan-angiography in cases of unexplained subarachnoid hemorrhage. J Neurosurg 1978;48:712-6.

55. Atlas SW. Magnetic resonance imaging of intracranial aneurysms. Neuroimaging Clin N Am 1997;7:709-20.

56. Alberts MJ, Quinones A, Graffagnino C, Friedman A, Roses AD. Risk of intracranial aneurysms in families with subarachnoid hemorrhage. J Neurosurg 1978;48:712-6.

57. Datta A. Subarachnoid hemorrhage: A preventable disease with a heritable component. Stroke 2002;33:1321-6.

58. Henderson WG, Tomer JC, Nibbelink DW. Intracranial aneurysms and subarachnoid hemorrhage: Report on a randomized treatment study, IV-B. Regulated bed rest: Statistical evaluation. Stroke 1977;8:579-89.

59. Winn HR, Almaani WS, Berga SL, Jane JA, Richardson AE. The Treatment of ischemic deficits from vasospasm with intravascular volume expansion and induced arterial hypertension. Neurosurgery (September) 1982;11:337-43.
77. Pickard JD, Murray GD, Illingworth R, Shaw MD, Teasdale GM, Foy PM, et al. Effect of oral nimodipine on cerebral infarction and outcome after subarachnoid haemorrhage: British aneurysm nimodipine trial. BMJ 1989;298:636‑42.

78. Suarez‑Rivera O. Acute hydrocephalus after subarachnoid hemorrhage. SurgNeurol 1998;49:563‑5.

79. Schievink WI, Wijdicks EF, Piepgras DG, Chu CP, O’Fallon WM, Whisnant JP. The poor prognosis of ruptured intracranial aneurysms of the posterior circulation. J Neurosurg 1995;82:791‑5.

80. Larson JJ, Tew JM Jr, Tomsick TA, van Loveren HR. Treatment of aneurysms of the internal carotid artery by intravascular balloon occlusion: Long‑term follow‑up of 58 patients. Neurosurgery 1995;36:26‑30.

81. Taylor W, Miller JD, Todd NV. Long‑term outcome following anterior cerebral artery ligation for ruptured anterior communicating artery aneurysms. J Neurosurg 1991;74:51‑4.

82. Guglielmi G, Viñuela F, Dion J, Duckwiler G. Electrothrombosis of saccular aneurysms via endovascular approach. Part 2: Preliminary clinical experience. J Neurosurg 1991;75:8‑14.

83. Molyneux AJ, Kerr RS, Yu LM, Clarke M, Sneade M, Yarnold JA, et al. International subarachnoid aneurysm trial (ISAT) of neurosurgical clipping versus endovascular coiling in 2143 patients with ruptured intracranial aneurysms: A randomised comparison of effects on survival, dependency, seizures, rebleeding, subgroups, and aneurysm occlusion. Lancet 2005;366:809‑17.

84. Raaymakers TW, Rinkel GJ, Ramos LM. Initial and follow‑up screening for aneurysms in families with familial subarachnoid hemorrhage. Neurology 1998;51:8‑14.

85. Johnston SC, Wilson CB, Higashida RT, Dowd CF, McDermott MW, et al. Endovascular and surgical treatment of unruptured cerebral aneurysms: Comparison of risks. Ann Neurol 2000;48:11‑9.

86. Halbach VV, Higashida RT, Dowd CF, Urwin RW, Balousek PA, Lempert TE, et al. Cavernous internal carotid artery aneurysms treated with electrolytically detachable coils. J Neuroophthalmol 1997;17:231‑9.

87. Regli L, Uske A, De Tribolet N. Endovascular coil placement compared with surgical clipping for the treatment of unruptured middle cerebral artery aneurysms: A consecutive series. J Neurosurg 1999;90:1025‑30.

88. Cross DT 3rd, Tirschwell DL, Clark MA, Tuden D, Derdeyn CP, Moran CJ, et al. Mortality rates after subarachnoid hemorrhage: Variations according to hospital case volume in 18 states. J Neurosurg 2003;99:810‑7.

89. Broderick JP, Brott TG, Duldner JE, Tomsick T, Leach A. Initial and recurrent bleeding are the major causes of death following subarachnoid hemorrhage. Stroke 1994;25:1342‑7.

90. Heros RC, Kistler JP. Intracranial arterial aneurysm‑an update. Stroke 1983;14:628‑31.

91. Sundt TM Jr, Kobayashi S, Fode NC, Whisnant JP. Results and complications of surgical management of 809 intracranial aneurysms in 722 cases: Related and unrelated to grade of patient, type of aneurysm, and timing of surgery. J Neurosurg 1982;56:753‑65.

92. Rosen DS, Macdonald RL. Subarachnoid hemorrhage grading scales: A systematic review. Neurocrit Care 2005;2:110‑8.

How to cite this article: Bhat AR, AfzalWani M, Kirmani AR. Subarachnoid hemorrhage in Kashmir: Causes, risk factors, and outcome. Asian J Neurosurg 2011;6:57‑71.

Source of Support: Nil, Conflict of Interest: None declared.