ORIGINAL ARTICLE

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

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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 withother 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 factorsof 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

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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 anaverage 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			Causes			
			Males	Females	#Children	Cases of aneurysms	Cases of AVMs	Hypertension	Idiopathic
0-10	165	57	0	0	57	5	12	0	40
11-20	75	27	7	5	15	12	7	0	8
21-30	278	99	42	57	0	41	2	21	35
31-40	453	124	59	65	0	53	5	39	27
41-50	979	318	87	231	0	225	2	91	0
51-60	673	185	45	140	0	127	1	57	0
61-70	241	76	51	25	0	18	0	53	5
71-80	59	21	9	12	0	12	0	9	0
Total	2923	907	300	535	#72	493 54.35%	29	270	115

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. *Male: Female ratio of SAH=1.00: 1.78. *Aneurysmal distribution: Males=191; Females=297; Children=5. *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 populationper year. *SAH forms 31.02% (907/2923) of all strokes. *Age group of 41-60 years reveals most strokes (56.51%) and SAHs (55.45%)

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, 60.24% (297/493) female, and 1.01% (5/493) children, with the M/F ratio of 1.00: 1.55. Similarly, hypertensive SAHs, which consist of 29.76% (270/907) SAHs, mostly, i.e. 54.81% (148/270), come from the age group of 41-60 years. While 65.51% (19/29) of all AVM which form 3.19% (29/907) of all SAHs were found in the age group of 0-20 years. The idiopathic SAH, making 12.67% (115/907) of all SAHs, mostly, 53.91%, (62/115) involves the age group of 21-40 years. The mean age for all SAHs was 31-50 years [Table 1].

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 and3. 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 in the GCS score of 7-12 and WFNS grade 4 [Table 2]. Most of the hypertensive (56.66%=153/270), AVMs (75.86%=22/29), and idiopathic (60.86%=70/115) SAHs wereadmitted 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

(IV, V) of SAH compared withmales i.e.4.18% (18/907). 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].

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 warmthover 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 10.57% (83/785) scans, most of these, i.e. 55.42% (46/83), belonged to aneurysmal SAHs. There were 3.94% (31/785) patients with unilateral and unilobar or bilobar infarcts, of which, 48.38% (15/31) were aneurysmal SAHs [Table 4].

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	Grade					Deaths	Total
	I	II	III	IV	V		
Aneurysm cases	54	114	154	63	108	158	493
AVM cases	15	3	4	5	2	2	29
Hypertension	35	59	74	32	70	124	270
Idiopathic	8	42	22	27	16	48	115
Total	112	218	254	127	196	332	907
Males	54	111	97	20	188	101	300
Females	50	97	130	99	159	197	535
Children	8	10	27	8	19	34	72
Deaths	17	48	63	61	143	332	XXX
Total	112	218	254	127	196	332	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

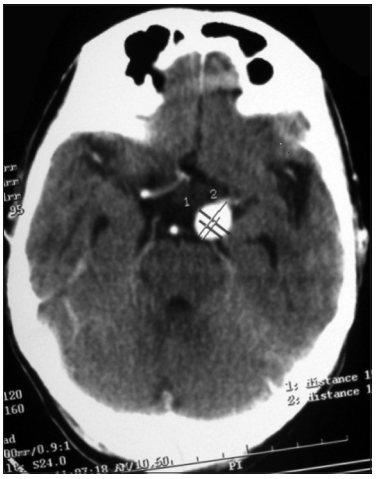

Figure 1: High attenuating lesion (aneurysm) of left internal carotid artery on plain CT-scan brain

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
					1	2	3	4	
Blood in subarachnoid space	353	7	68	54	0	258	224	-	482
Aneurysm as high attenuating lesion	164	0	0	0	-	-	-	-	164
Intra-ventricular hemorrhage	90	3	70	15	-	-	-	178	178
Intra-cerebral hemorrhage	45	6	61	13	-	-	-	125	125
Re-bleeding	188	2	6	3	-	-	-	-	199
Hydrocephalus	46	1	31	5	-	-	-	-	83
Infarct	15	1	10	5	-	-	-	-	31

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% (188/199) of all re-bleeds. *Re-bleeding caused 56.28% (112 deaths/199 re-bleeds) mortality, which comprises of 33.73% (112/332 deaths) of all deaths (36.60%=332/907)

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

$Parent artery	No. of aneurysms on parent artery only			Simultaneous aneurysms on associated arteries other than parent A.						Total cases (%)
				Same side of parent A.			Other side of parent A.			
	One	Two	Many	One	Two	Many	One	Two	Many	
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	2	38
Posterior Com. A.	5	0	0	3	0	0	2	0	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
						152(30.83%)				

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	21	38	5
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

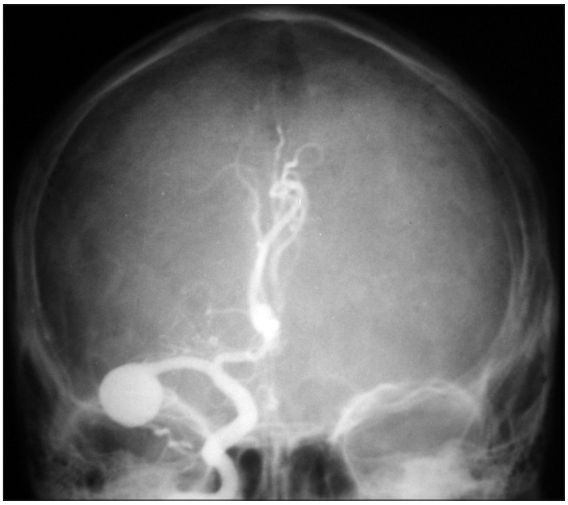

Figure 2: Right carotid angiogram revealing giant aneurysm of middle cerebral artery

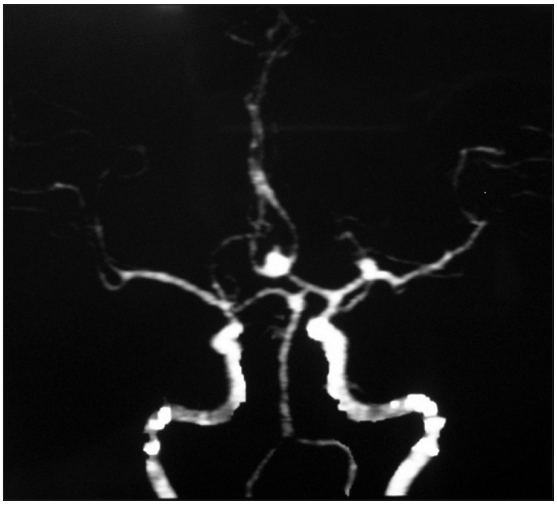

Figure 3: CT-Angiography depicting multiple aneurysms of Acom.A, MCA and basilar artery

(34/205) and positive in 83.42% (171/205) patients, with an accuracy of >83%. More than half of the investigated patients, 50.24% (103/205) had single and 28.78% (59/205) had two or more than two (multiple) aneurysms, thereby detecting more than 221 aneurysms in 162 patients [Figure 3]. CT-angiography also depicted 4.39% (9/205) AVMs. The vasospasm was found in 36.58% (75/205) patients. The procedure had negligible complications [Table 6].

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 aneurysms that made it 155 aneurysms in 126 patients of SAH. The 2.04% (3/147) patients who underwent DSA proved to be the AVMs. The DSA detected vasospasm in about 41.49% (61/147) patients and the procedure had no complications [Table 6].

MR-angiography

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

children. 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 (4.49%=8/178) of the proximal control [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 to 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

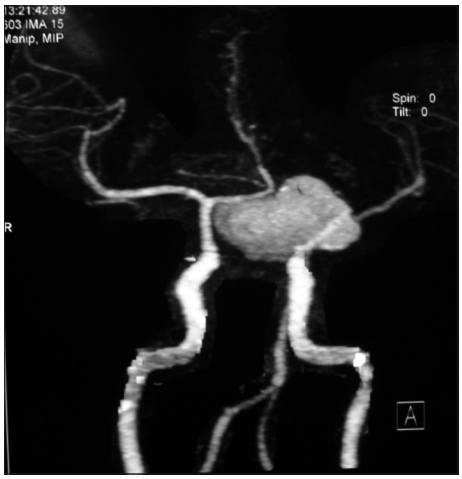

Figure 4: CT-angiogram showing giant aneurysm of left ICA bifurcation and vasospasm

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 27.58% (8/29) AVMs were trapped by clipping. The intraoperative aneurysmal size of same patient varied from the size onpreopertive CT angiography by being 0.5-1.5 mm larger in case of distal anterior cerebral artery (DACA) and MCA aneurysms [Table 7].

Table 7: Management and Glasgow outcome scoring of SAH patients

Variables	Aneurysm	AVM	Hypertension	Idiopathic	Total
Management	425	8	0	0	433
a) Clipping					
b) Intervention coiling/ embo./ carotid ligation	68	18	0	0	86
c) EVD/VP shunt as definitive treatment	0	3	98	29	130
d) Conservative	0	0	172	86	258
Good recovery	94	8	31	3	136
Moderate disability	119	8	21	33	181
Severe disability	64	5	57	18	144
Vegetative state	58	6	37	13	114
Death	158	2	124	48	332
Total	493	29	270	115	907

SAH - Subarachnoid hemorrhage; AVMs - Arterio venous malformation; *Embo: Embolization; #EVD: External Ventricular Drainage; SAH - Subarachnoid hemorrhage; VP: Ventriculo- Peritonial; *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

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

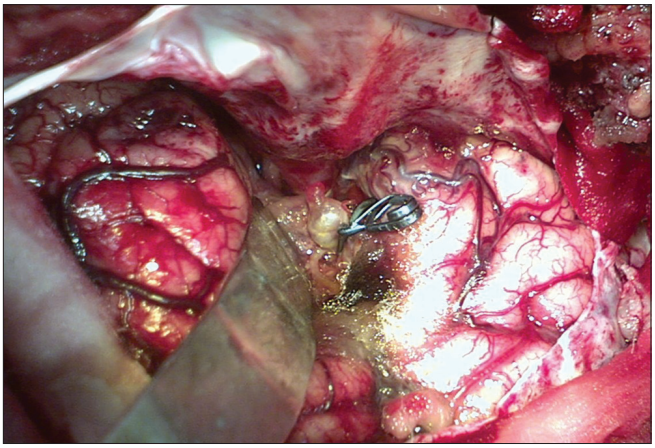

Figure 5: Intra-operative photograph of clipped left MCA aneurysm

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 yeas) 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 studiesreported an incidence that ranged from 8.1 per 100,000 in Australiaand 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 foundthat the age-adjusted annual incidence of SAH varied 10-foldbetween 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.[26] The studieshave suggested that the gender difference is related to hormonalstatus, with incidence of SAH increasing withage, occurring most commonly between 40 and 60 years of age (mean age 50 years), but SAH can occur from childhood to oldage, and is 1.6 times higher in women than in men.[29,30] The incidence of aneurysmal rupture is only 2-20/100,000 individuals/year.[27] 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.[22] 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 meningeal 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 aneurysmal SAHs. The clinical presentation of aneurysmal SAH is one of the mostdistinctive in medicine. The sine qua non of SAH in an awakepatient is the complaint of "the worst headache of my life,"described by 80% of patients who can give a history, but a warningor 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.[28,37] 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 hoursand more commonly in SAH associated with intracerebral hemorrhage, hypertension, and middle cerebral and anterior communicatingartery aneurysms.[39,40] Morerecent retrospective reviews report a low frequency of seizuresranging from 6 to 18%.[41] Other study showed 4% convulsions occurring in SAHs.[28] Delayed seizures occurredin 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 coalin 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 riskthan white Americans.[45] Multivariate models have found hypertension, smoking, and heavyalcohol 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 postmenopausalstate 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 monthsand 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.[55,56] Computed tomography (CT scan) of the brain has a high sensitivity and will correctly identify over 95% of cases.[12] The ruptured aneurysms in 21.1% SAH patients were detected as high attenuating lesions on plain CT-scan.[57] 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.[57] 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 aneurysmsare <5 mm.[58] Among aneurysms detected on CTA and then undergoing surgery, 100% correlation was observedbetween CTA and catheter angiography. Velthuis and colleagues found that CTA is equal to catheter angiography in 80 to 83% of cases.[59] In 74% of patients, catheter angiography performed after CTA did not reveal any additional information.[60] CTA has been shown to be effective in determining the presence of severe vasospasm, butis less accurate in detecting mild and moderate vasospasm.[61] 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.[62] Repeat angiography after one week will show a previously unrecognized aneurysm in an additional 1% to 2% of cases.[63] 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%.[64] 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.[28] Another author reported 36.7% SAHs due to aneurysms, 7.1% AVMs, 9.4% hypertension, and 46.8% as unknown causes.[37] In one study, 85% cases of spontaneous SAH are caused by rupture of a cerebral aneurysm.[12] Rinkel *et al*. reports that 15-20% spontaneous SAH has no detectable cause on the first angiogram.[65]

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.[1] 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.[28] 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.[37] 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.[57] 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%.[28,57,66] 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.[67] This is associated with SAH at a younger age, a high incidence of multiple aneurysms, and hemorrhages among siblings and mother-daughter pairings.[68] In family members with the familial intracranial aneurysm syndrome, the risk of harboring an unruptured aneurysm was 8%.[69] A study of 23 families with familial SAH found that having three affected relatives tripled the riskof SAH. When MRA was used toscreen 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.[70] However, another MRI study reported that 4% of relatives of sporadic SAH patients had aneurysms.[71] In a large case control study, family history was found to be an independent riskfactor for SAH.[72] 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.[1] 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.[32] At SKIMS, re-bleeding occurred in 25.35% SAH patients, leading to 56.28% deaths of re-bled 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.[37] 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 tofour weeks.[75] The secondary cerebral ischemia occurred in 27% of a series.[32] 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.[57] Some SAH patients with acute hydrocephalus may benefitfrom 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 waslower 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% relativerisk 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.[83-85] Aneurysms in the cavernous segment of the internal carotid artery are also difficult to treat withsurgery, 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 aneurysmscan be difficult to treat by coil embolization, and surgical results for these aneurysms are often reportedas 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] Themortality rate for SAH, in the 1966 Cooperative Study on Intracranial Aneurysms (CSIA), was 50% at 29 days and 33%SAH patientsadmitted through emergency department.[88] 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.[89] Recurrent hemorrhage remains a serious consequence of aneurysmal SAH with a case fatality rate of 70% for persons who re-bled.[28] 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%.[48] It is reported that a mortality of 77.5% occurred in patients of SAH who had re-bled.[1] The initial hemorrhage can be devastating, and up to a quarter of patients die before reaching medical attention.[90] 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-comatosed 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.[29,91] Perhaps the most meaningful and simplest measure of the effect of these deficits is whether the patient is able to return tohis or her previous occupation.[92] 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 percentageof patients transferred from other hospitals.[88] 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%.[32] 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.

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