

ROMANIAN
NEUROSURGERY

Vol. XXXV | No. 2 June 2021

Subarachnoid haemorrhage.
A critical neurosurgical emergency

Alexandra Bibiriță,
Daniel Teleanu,
Alexandru Vlad Ciurea



Subarachnoid haemorrhage. A critical neurosurgical emergency

Alexandra Bibiriță¹, Daniel Teleanu^{2,3},
Alexandru Vlad Ciurea^{4,5}

¹ Resident of Neurosurgery at Bucharest University Emergency Hospital, ROMANIA

² Chief of Neurosurgery Department at Bucharest University Emergency Hospital, ROMANIA

³ Assoc. Professor of Neurosurgery at "Carol Davila" University of Medicine and Pharmacy, Bucharest, ROMANIA

⁴ Chief of Neurosurgery Department and Scientific Director of Sanador Clinical Hospital, Bucharest, ROMANIA

⁵ Professor of Neurosurgery at "Carol Davila" University of Medicine and Pharmacy, Bucharest, ROMANIA

ABSTRACT

Subarachnoid haemorrhage (SAH) accounts for 3% of all strokes and is the cause of 5% of stroke mortality. SAH by rupture of cerebral aneurysm or arterial-venous malformation (AVM) remains the most devastating cerebrovascular disease. During admission for SAH, about 30-70% of patients suffer a rebleed, and from all rebleeds, about 90% lead to death no matter the treatment. Available current scales help predict the prognosis and guide the therapy. Considering that the lifestyle risk factors for SAH are of increasing prevalence, it is expected that it will affect even more people in the future. SAH should not be regarded as a disease but rather a set of events with devastating complications requiring adequate management from debut extending long after patient discharge.

EPIDEMIOLOGY

Subarachnoid haemorrhage stands for 3% of all cerebral vascular strokes and is responsible for the 5% mortality coming from CVAs. Incidence peak for SAH is 55-60 age group. During hospital admission for this pathology 30-70% of patients suffer a rebleed and approximately 90% of all rebleeds will end in death whatever the treatment.

PHYSIOPATHOLOGY AND AETIOLOGY

Subarachnoid haemorrhage occurs as a consequence of a blood vessel rupture in the subarachnoid space or near it. Following this rupture, blood will invade the space between the pia mater and the arachnoid,

Keywords

risk factors,
neurosurgical emergency,
CT,
angiography,
subarachnoid haemorrhage
scales,
cerebral vasospasm



Corresponding author:
Alexandra Bibirita

Bucharest University Emergency
Hospital, Romania

alexandra.bibirita@gmail.com

Copyright and usage. This is an Open Access article, distributed under the terms of the Creative Commons Attribution Non-Commercial No Derivatives License (<https://creativecommons.org/licenses/by-nc-nd/4.0/>) which permits non-commercial re-use, distribution, and reproduction in any medium, provided the original work is unaltered and is properly cited. The written permission of the Romanian Society of Neurosurgery must be obtained for commercial re-use or in order to create a derivative work.

ISSN online 2344-4959
© Romanian Society of
Neurosurgery



First published
June 2021 by
London Academic Publishing
www.lapub.co.uk

the subarachnoid space, which normally contains just cerebrospinal fluid.

Etiologically, which is considering the reason that led to the event, following types of SAH stand out – traumatic SAH or spontaneous.

The spontaneous form of SAH occurs in cases of high arterial blood pressure (hypertensive SAH) or is the direct consequence of an aneurysmal rupture, a cerebral or spinal arterial-venous malformation rupture or secondary to cervical-cerebral arteries dissection. [3,12] Actually about 85% of all SAH forms are due to the rupture of a cerebral aneurysm. Brain aneurysm typically occurs during spring and autumn and is considered directly linked to temperature changes, similar to CVAs. [2,12]

A rare form of spontaneous SAH is the idiopathic subarachnoid hemorrhage, in the presence of risk factors considered non-traditional and *modifiable* – alcoholism, smoking, cocaine or amphetamine abuse. Besides these forms, literature provides a series of traditional risk factors, which are not subject to medical or lifestyle intervention and thus called *non-modifiable*, frequently linked to SAH occurrence: family history of SAH, soft tissue disease (polycystic kidney disease, neurofibromatosis type I, Ehlers-Danlos syndrome mainly type IV and other collagen abnormalities), female sex (1.5 times the risk), African descent (2 times the risk), Japanese or Finnish descent, vasculitis, even more rare factors – parasitosis, Moya-Moya disease, eclampsia, blood disorders, coagulation disorders. [1,2,11,12] It is important to point out that as prevalence of the non-modifiable risk factors gets higher, it is expected that SAH incidence will increase in the following decades.

CLINIC OF SUBARACHNOID HAEMORRHAGE

Clinically the first symptom and nonetheless the most constant is severe headache with sudden onset – approximately 97% of cases, describes as a *thunderclap headache* and acknowledged by the patient as “the most intense headache I ever had”. [12]

A more particular situation is the *sentinel headache* – a sudden and severe pain that goes away, allowing for a symptom-free period with neglect of said episode, followed shortly by severe mental status alteration, paroxysmal phenomena known by neurosurgeons as “walk, talk and die patients.” [1,3]

Photophobia as an accompanying symptom is frequent, together with ocular symptoms such as subhyaloid hemorrhage, retinal or vitreous hemorrhage. In medical literature vitreous hemorrhage associated with SAH is known as Terson’s syndrome. [8]

Eye fundus examination may reveal papillary edema secondary to increased intracranial hypertension secondary to bleeding into the subarachnoid space. Vegetative phenomena such as emesis or syncope are common.

Sometimes, although not rarely the patient seeks medical attention after a loss of consciousness episode or for altered mental status, as neurological status on admission might vary from slight confusion to deep coma. Epileptic seizures occur frequently as about 20% of patients develop such symptoms in the first 24 hours since debut. They are considered to be a straight effect of increased intracranial pressure, associated hyponatremia or aneurysm site, especially when it involves sylvian arteries territory. [3,9]

Neurological examination reveals early meningeal signs: neck stiffness, headache, photophobia, ocular pain, emesis. Neck stiffness usually develops within 6 to 24 h from the debut of SAH. Within a few hours from debut signs such as Kernig, Brudzinki or bilateral Lassegue can appear or focal neurological signs. *Psychiatric acute onset* is more common and sometimes even specific among elderly patients. Some other accompanying signs or symptoms are acute urinary retention, diminished or abolished osteo-tendinous reflexes (usually after 4 to 6 h from the debut of the hemorrhage) and eventually focal neurological signs. Elderly patients, most commonly after 70 years old very often present with a far worse neurological status on admission. [9,12]

DIAGNOSIS

The elective diagnostic tool in SAH is the plain cerebral CT examination on arrival in the emergency room, which shows blood in the subarachnoid space. The Fisher grade is thus immediately established. This diagnostic imaging tool is nonetheless the actual gold standard in diagnosing the subarachnoid hemorrhage, its limitation being that when not enough time since the debut of the hemorrhage has passed, the first examination might turn out negative

and will become positive a few tens of minutes later. The Fisher grading system is based upon the results of the plain cerebral CT examination and it appreciates the risk of developing cerebral vasospasm. It is summarized in the table below. [14]

Fischer Grade	Presence of blood in the SA space	Other aspects	Vasospasm risk
Grade 1	No blood in the cisterns	No clots	21%
Grade 2	Blood <1 mm thickness	No clots	25%
Grade 3	Blood >1 mm thickness	+/- clots	37%
Grade 4	Rare blood, diffusely in the basal cisterns	Intraventricular hemorrhage or parenchymal	21-37%

Table 1. Fisher Scale (1982).

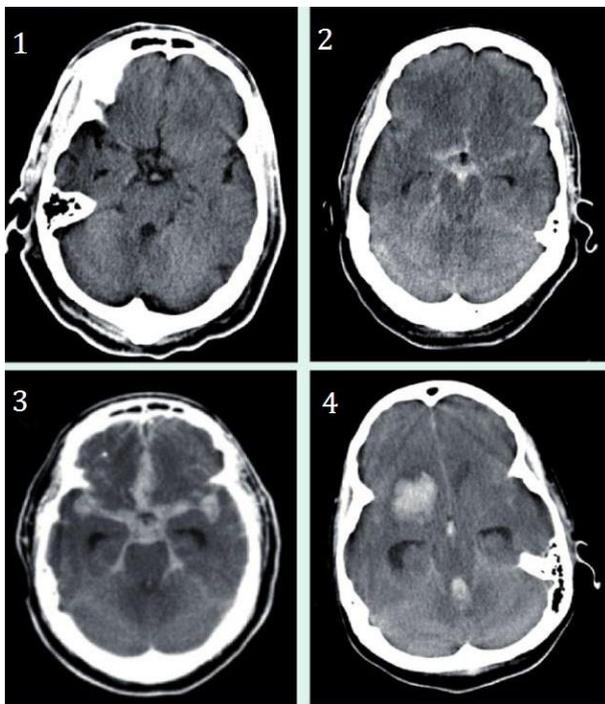


Figure 1. Cranio-cerebral CT axial sequences showing the 4 Fisher grades (1 – grade 1, 2 – grade 2, 3 – grade 3, 4 – grade 4). Source Future Neurol @ 2013 Future Medicine Ltd.

If the CT examination is negative but the clinical suspicion of SAH is still high, a lumbar puncture may be helpful for diagnostic purpose, but it is to be performed with every precaution and only after eye fundus examination excludes intracranial hypertension, in order to avoid cerebral herniation. This procedure can induce a rebleed if intracranial

pressure is too abruptly decreased, which is why a minimal amount of a CSF sample is to be drawn. [3,5] Lumbar puncture is according to available date the most sensitive diagnostic tool for SAH. A positive result consists of increased CSF pressure, xanthochromic CSF, more than 100 000 thousand erythrocytes per mm³, increased proteinorrhachia (>50 mg/dl) and normal or slightly decreased glycorrhachia (<50-70 mg/dl) [3,4,5].

Cerebral MRI imaging with FLAIR sequencing, CT angiography or MRI angiography do not add very much relevance to the SAH diagnosis in the first 24-48 hours following debut, but they prove to be excellent between day 3 and 7, with good premises for identifying a cerebral aneurysm and its anatomical features. [7]

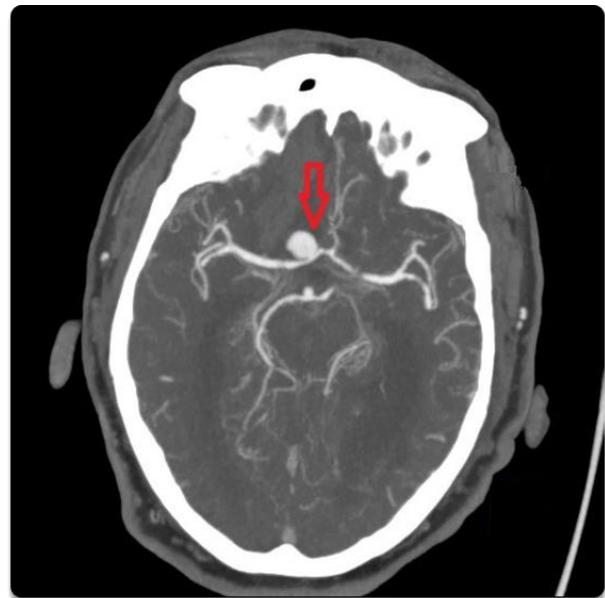


Figure 2. Axial enhancement cerebral CT sequence – red arrow points to an anterior communicating artery aneurysm – Collection of Neurosurgery I Clinic, Emergency University Hospital of Bucharest.

Digital subtraction angiography (DSA) - now a tool of capital importance in the management of subarachnoid hemorrhage – consists of selectively injecting contrast material using a catheter inserted into a large artery under radiologic screen. It is an extremely useful resource in the evaluation of cerebral aneurysms and it can also come in as a both diagnostic and therapeutic tool, providing the possibility of simultaneously embolizing the incriminated aneurysm or AVM.

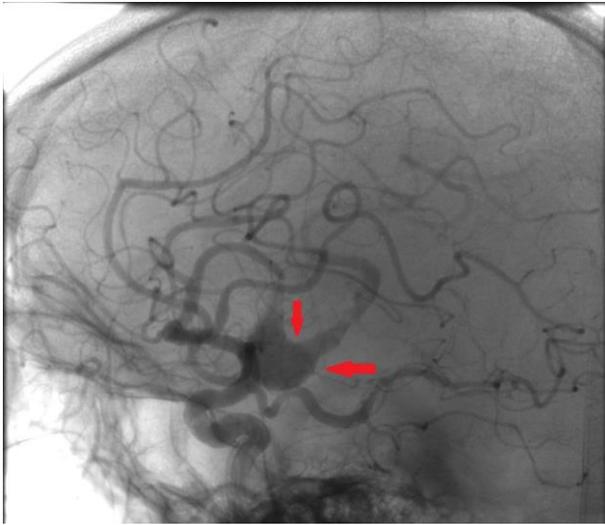


Figure 3. Right sylvian artery giant dissecting aneurysm near M1 and M2 segments junction. Right internal carotid angiography cliché. Collection of Neurosurgery I Clinic, Emergency University Hospital of Bucharest.

Aneurysm with sacs smaller than 5 mm are ideal for endovascular coiling, while for the rest of them the risk of recanalization or incomplete occlusion are considered too high, thus needing stent or balloon assisted coiling.

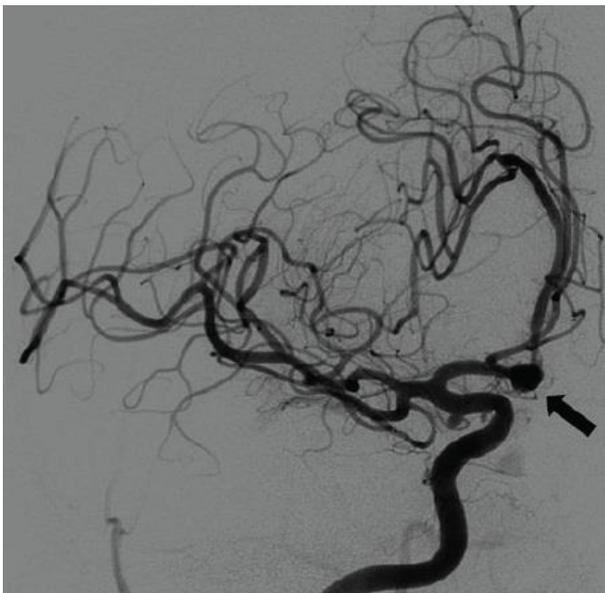


Figure 4. Digital subtraction angiography sequence – black arrows points to a anterior communication artery aneurysm – Open Source image.

MANAGEMENT

Once the diagnosis has been confirmed therapeutic measures are immediately started based on

patient's clinical and neurological status and in closed connections with SAH etiology. Diagnosis is further completed with other needed tests. The patient should be admitted into a neurosurgical service with dedicated intensive care unit in case of need of advanced vital function support.

Drug treatment consists of maintaining an optimal blood pressure of systolic BP < 160 mmHg, considering that high blood pressure values are associated with worsening SAH and rebleed risk. Vasospasm prevention is to be addressed by administering Nimodipine. Cerebral edema prevention, maintaining normal volemia and preventing hyponatremia are the next medical considerations, as even with maximal therapy they are extremely difficult to correct if they occur. Anti-seizure prophylaxis is mandatory because of the irritation of cerebral cortex by blood in the subarachnoid space. [6,9]

The neurological status of the patient suffering from SAH is to be appreciated with the help of Hunt & Hess scale and WNFS scale (World Neurosurgical Federation Society scale - 1988) and it is extremely important in the surgical indication. Classification into a Hunt & Hess (1968) grade is determined after clinical and neurological assessment as follows: [15]

Grade I: absent or mild headache, absent or minimal neck stiffness

Grade II: severe headache, franc neck stiffness, possible cranial nerves paresis

Grade III: confusion or lethargy, mild focal deficits

Grade IV: stuporous, hemiparesis forte, decerebrate

Grade V: comatose, decerebrate.

WFNS Scale is based upon Glasgow Comma Scale and the presence or absence of neurological deficits as such:

Grade I: GCS 15 points, no neurological deficits

Grade II: GCS 13-14 points, no neurological deficits

Grade III: GCS 13-14 points, present neurological deficits

Grade IV: GCS 7-12 points, whatever the neurological deficits

Grade V: GCS 3-6 points, whatever the neurological deficits.

Another scale of great clinical and therapeutical significance is the Fisher scale previously presented, with four degrees of severity, based upon computed-tomography aspects such as the presence of blood

in the ventricular system or parenchymal hematoma and thickness of subarachnoid blood.

The right moment to perform surgery in aneurysmal SAH is nowadays considered to be at 24-72 hours from debut in patients with Hunt & Hess grades 1 or 2 and consists of aneurysm securement through classical surgical clipping or endovascular coiling. Patients with a higher Hunt & Hess grade (altered mental status or neurological deficits) are to be admitted and monitored in the intensive care unit for vital functions support with the goal of obtaining a better neurological status in order for the aneurysm securement to be achievable with a more favorable risk/benefit balance.

Exception to this rule occurs when SAH of any etiology is accompanied by large parenchymal hematoma (Fisher grade IV) that come with vital risk to the patient and it requires emergency intervention whatever the Hunt & Hess grade. A similar situation occurs when patient's life is endangered on the short-term due to intracranial hypertension (secondary to acute obstructive hydrocephalus following SAH) - a external ventricular drainage is placed together with an *intracranial pressure monitoring device* - in order to obtain clinical and neurological amelioration until definitive treatment of the cause that led to SAH is possible. [9, 10]

Following surgery, after securing the aneurysm whose rupture produced the subarachnoid hemorrhage, together with general medical measures and complications prevention, a transcranial Doppler ultrasound is needed in order to appreciate the blood flow through the main cerebral arteries and possible vasospasm, as well as control cerebral CT examination for the verifying of occlusion devices used and for the visualizing the aspect of the ongoing SAH. In cases of drug resistant vasospasm *intraarterial endovascular vasodilator therapy* may be used - vasodilator agents are selectively injected directly into the cerebral arteries under angiographical control.

COMPLICATIONS

In the postoperative period medical treatment measures must be continued. Considering the risk of rebleed, which stand at 30-70% despite adequate treatment and knowing that 90% of rebleed lead to death, postsurgical patients must be monitored in the intensive care unit.

Despite these therapeutic resources, the major risk of devastating complications particularly difficult to treat persists - cerebral vasospasm and late cerebral ischemia, vegetative phenomena of central origin, parenchymal hematoma, rebleed, electrolytes imbalance, normal pressure hydrocephalus. [10]

The risk of developing cerebral vasospasm stands present since the debut of SAH but is considered to be at its highest between days 3-7 and extending up to 3 weeks. It is very accurately appreciated with the help of the Fisher grading system, risk correlation being proportionate with a higher grade.

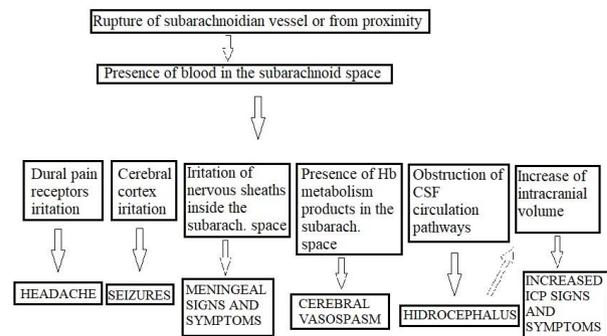


Figure 5. Résumé of SAH complications and their mechanism. Personal illustration.

NEUROLOGICAL REHABILITATION

The treatment of subarachnoid hemorrhage is a multidisciplinary one, step-by-step and one which address simultaneously the etiological factor, the worsening factor and at the same time the prevention of complications. [2,12] For a favorable outcome and prognosis, and also for survivors' rehabilitation a quick and adequate diagnosis is required. It should happen in a primary neurosurgery center with radiology and intensive care optimal facilities available at all times. The prognosis of these patients and their neurological rehabilitation require the right cooperation between the medical team consisting of emergency room physician - radiologist - interventionist - neurosurgeon - intensivist - neurologist and rehabilitation physician.

CONCLUSIONS

A curative *treatment* of subarachnoid hemorrhage is out of the question at the moment, with it being not actually a disease but rather a course of pathological events that develop in a chain like manner and that

imply a series of serious consequences over cerebral structures, both short term and long term.

Methods of treatment of SAH complications are currently under development, and even though they are based on hypotheses decades old, some of them are highly debatable according to some authors, such as subarachnoid space washout-out with drugs designed to prevent vasospasm. [13]

Considering that about 15-20% of patients that suffer from SAH decease before getting to medical attention of any kind according to WHO data, and that 40% of survivors from SAH of any kind will have permanent neurological deficits, two things stand out: early identification of high suspicion SAH cases is critical and referral to an *emergency neurosurgical service* is of paramount need.

ABBREVIATIONS

AVM – arterial-venous malformation;
BP – blood pressure;
CSF – cerebrospinal fluid;
CVA – cerebrovascular accident;
ICP – increased intracranial pressure;
WHO – World Health Organization.

CONFLICTS OF INTEREST

The authors have no conflicts of interest to declare.

REFERENCES

1. Kameda-Smith M, Aref M, Jung Y, Ghayur H, Farrokhyar F, Determining the diagnostic utility of lumbar punctures in CT negative suspected subarachnoid hemorrhage: A Systematic Review and Meta-analysis, *World Neurosurg.* 2020; 148: e27-e34.
2. Kellermann I, Kleindienst A, Hore N, Buchfelder M, Brandner S., Early CSF and Serum S100B, Concentrations for Outcome Prediction in Traumatic Brain Injury and Subarachnoid Hemorrhage, *Clin Neurol Neurosurg.* 2016; 145:79-83.
3. Nadkarni NA, Maas MB, Batra A, Kim M, Manno EM, Sorond FA, Prabhakaran S, Naidech AM, Liotta EM, Elevated Cerebrospinal Fluid Protein Is Associated with Unfavorable Functional Outcome in Spontaneous Subarachnoid Hemorrhage, *J Stroke Cerebrovasc Dis.* 2020 29(4):10460.
4. Lenhart M, Bretschneider T, Gmeinwieser J, Ullrich OW, Schlaier J, Feuerbach S, Cerebral CT angiography in the diagnosis of acute subarachnoid hemorrhage, *Acta Radiol.*, 2013, 38(5):791-6.
5. Raevis J, Elmalem VI, Pseudotumor cerebri syndrome causing a terson like syndrome, *Am J Ophthalmol Case Rep.* 2020; 20: 100993.
6. Vivancos J, Gilo F, Clinical management guidelines for subarachnoid haemorrhage. Diagnosis and treatment, *Neurologia*, 2014;29(6):353-70.
7. Wu L, Chen G, Signaling Pathway in Cerebral Vasospasm After Subarachnoid Hemorrhage: News Update, *Acta Neurochir Suppl.* 2016; 121:161-5.
8. Veldeman M, Albanna W, Weiss M, Conzen C, Schmidt TP, Schulze - Steinen H, Wiesmann M, Clusmann H, Schubert GA, Invasive neuromonitoring with an extended definition of delayed cerebral ischemia is associated with improved outcome after poor-grade subarachnoid hemorrhage, *J Neurosurg.* 2020; 15:1-8.
9. Veldeman M, Albanna W, Weiss M, Conzen C, Schmidt TP, Clusmann H, Schulze-Steinen H, Nikoubashman O, Temel Y, Schubert GA, Treatment of Delayed Cerebral Ischemia in Good-Grade Subarachnoid Hemorrhage: Any Role for Invasive Neuromonitoring?, *Neurocrit Care.* 2020.
10. Probst MA, Hoffman JR, MD, Computed Tomography Angiography of the Head is a reasonable next test following a Negative Non-contrast Head Computed Tomography in the Emergency Department work up of Subarachnoid Hemorrhage, *Ann Emerg Med.* 2016 June; 67(6): 773–774.
11. Hughes JD, Bond KM, Mekary RA, Dewan MC, Rattani A, Baticulon R, Kato Y, Azevedo-Filho H, Morcos JJ, Park KB, Estimating the Global Incidence of Aneurysmal Subarachnoid Hemorrhage: A Systematic Review for Central Nervous System Vascular Lesions and Meta-Analysis of Ruptured Aneurysms, *World Neurosurg.* 2018;115:430-447.
12. Flemming KD, Lanzino G, Management of Unruptured Intracranial Aneurysms and Cerebrovascular Malformations, *Continuum (Minneap Minn).* 2017 Feb;23(1, Cerebrovascular Disease):181-210.
13. Kodama, Namio & Sasaki, Tatsuya & Kawakami, Masahisa & Sato, Masahiro & Asari, Jun. (2000). Cisternal irrigation therapy with urokinase and ascorbic acid for prevention of vasospasm after aneurysmal subarachnoid hemorrhage. Outcome in 217 patients. *Surgical neurology.* 53. 110-7; 53(2):110-7.
14. Lindvall P, Runnerstam M, Birgander R, Koskinen LO. The Fisher grading correlated to outcome in patients with subarachnoid haemorrhage. *Br J Neurosurg.* 2009 Apr;23(2):188-92.
15. Rosen DS, Macdonald RL. Subarachnoid hemorrhage grading scales: a systematic review. *Neurocrit Care.* 2005;2(2):110-8.