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Intracerebral Aneurysm Features, Natural History and Actual Treatment Approaches – Clips vs Coils

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Abstract

Introduction: Intracranial aneurysms (IA) represents a localized enlargement of a blood vessel caused by a cerebrovascular disorder in which the vessels' walls are weak, having a prevalence of 1-9% in the global population – the variability depends on hospital referral, neuroimaging findings and autopsy patterns. The pathology of IA is a dominant element in neurosurgical activity because of the multiple preoperative and management problems. IA represents a neurosurgical emergency, a life threatening pathology due risk of rupture (older age, hypertension and smoking being among rupture risk factors) and vasospasm development. Subarachnoid haemorrhage (SAH) associated with IA rupture has an incidence of 6-26 cases/100.000 people. Rupture mortality reaches up to 40% while only 30% of patients can fully recover. Main treatment options are represented by neurosurgical clips and endovascular coils, the former becoming increasingly popular. **Material & Methods:** Authors present **the personal experience of 701 IA consecutive cases – Prof AV Ciurea MD, PhD, operated on using open microsurgical approach** between 1 January 2000 and 1 January 2020 (20 years). **Results:** Most cases (319 cases – 45.5%) were reported between 41 and 50 years old, with male preponderance - 476 cases (67.9%). The symptoms were dominated by: headache 686 cases (97.86%), neck stiffness 658 cases (93.86%) focal neurologic deficits 498 cases (71.04%), seizures 364 (51.9%) etc., Hunt and Hess (H&H) grade II was dominated : 359 cases – 51.21%. There were 54 (7.7%) difficult cases with H&H IV and no H&H V cases operated on. The associated pathology was: systemic arterial hypertension (526 cases, 75%) and obesity/ hypercholesterolemia - (238 cases, 34%), ischemic cardiopathy - (119 cases, 17%), diabetes mellitus - (112 cases, 16%) etc. The common localization of intracranial aneurysms was the anterior communicating artery 170 cases (25%); the other locations were: medium cerebral artery - 105 cases (15%), posterior comunicating artery - 113 cases (16.11%), etc. All cases were total investigated by the same protocol: CT scan, DSA Angiography and operated (microsurgical approach clipping), as soon as possible after the subarachnoid hemorrhage (SAH).
The results of this cohort: Glasgow Outcome Scale (GOS - extension) in our data (at 6 months postoperative) shows: Upper Good Recovery (GR) 199 cases (28.44 %); Lower GR 198 cases (28.24 %); Upper MD 129 cases (18.40 %); Lower MD 107 cases (15.26 %); Upper SD 20 cases (2.85%); Lower SD 14 cases (1.99%); Vegetative state 7 cases (0.99%); Death 27 cases (3.85%). **Conclusions:** IA represents a neurosurgical emergency, a life threatening pathology due risk of rupture and vasospasm development. In this situation, the neurosurgical investigations must be made by the European protocol (CT and DSA Angiography) and as soon as possible the pacient must go in the operatory theatre. Main treatment options are represented by neurosurgical clips and endovascular coils, the former becoming increasingly popular.

Key words: intracranial aneurysm, rupture, rebleeding, vasospasm, microsurgical approach, surgical treatment, endovascular treatment, clips, coils

Abbreviations: IA – intracranial aneurysm, **SAH** – subarachnoid hemorrhage, **CVS** – cerebral vasospasm, **DSA** - Digital Substraction Angiography, **CT** - Computed Tomography, **MRI** - Magnetic Resonance Imaging, **WFNS** - World Federation of Neurosurgical Societies, **ACoA** - Anterior Communicating Artery, **PCoA** - Posterior Communicating Artery, **MCA** - Middle Cerebral Artery, **IH** - intracerebral hemorrhage, **IvH** - intraventricular hemorrhage, **SH** - subdural hemorrhage.

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Introduction and Demographic Data

Intracranial aneurysms (IA) represents a localized enlargement of a blood vessel caused by a cerebrovascular disorder in which the vessels' walls are weak.
IA can be classified by size, shape or vessel wall.

- **Size**
  - Small IA - diameter < 15mm;
    - Micro IA (Charcot Bouchard aneurysms) are usually located in blood vessels with a diameter smaller than 300 microns, usually concerning lenticulostriate vessels of the basal ganglia. They are associated with systemic arterial hypertension.
  - Large IA - diameter > 15mm
    - Large - diameter = 15 to 25 mm
    - Giant - diameter = 25 to 50 mm
    - Super giant - diameter > 50 mm
- **Shape**
  - Saccular IA
    - Because of the shape, they are often called "berry aneurysms" - round vessel enlargement. They are also the most common IA.
  - Fusiform IA
    - This type of IA represents both an elongation and distension of the entire vessel, often resulting from dissections when blood ruptures in the wall of an artery. They are referred to as dolichoectatic IA and have a smaller chance of rupture.
- **Vessel wall**
  - True aneurysms - in this category are included the IA that involves all three layers of an artery - intima, media and adventitia.
  - False aneurysms (pseudoaneurysms) - a collection of blood accumulated outside the vessel but still in contact with its outer layer due to the surrounding connective tissue which prevents blood from spreading out. Usually they evolve with either thrombosis or rupture in the surrounding spaces or cavities [1].

Aneurysm History

Giovanni Bastista Morgagni (1682-1771) is the author of the first autopsy of a patient who accused violent headache, an autopsy that highlighted a ruptured aneurysm. The first surgical ligation treatment of an aneurysm is attributed to Sir Victor Horsley (1855). An important step forward is then made by Harvey Cushing (1911), a pioneer of neurosurgery who pays extra attention to the clinical manifestations of aneurysms. The first clipping aneurysm was performed by Walter E Dandy in 1941. The number of operated aneurysms increased with the introduction of the operating microscope, improving the rigor of operations and, of course, the vital / functional prognosis. The contribution of Professor Mahmut Gazi Yasargil (1969), the founder of vascular microsurgery, in the treatment of intracranial aneurysms was major and revolutionary, both as a direct, rapid approach and through the introduction of the operating microscope [2]. In Romania, a major progress in the neurosurgical approach to aneurysms was made by. Prof. Leon Dănăilă (1982) who routinely introduced the operating microscope. Moreover, it has managed to find the most correct and effective approaches for all types of intracranial aneurysms, which has resulted in a significant decrease in mortality and morbidity in these diseases [3].
Another turning point in the diagnosis of aneurysms was the innovation of Egas Moniz, a Portuguese neurologist, who was one of the founders of cerebral angiography (1929). Subsequently, Sven Ivar Seldinger (Sweden 1953) made his contribution by introducing arterial catheterization and highlighting vascular malformations. Gradually, embolization methods also appeared by introducing obstruction materials at the level of the aneurysm. Embolization methods have evolved in a fascinating way, from the use of muscle fragments (Brooks 1930), removable latex balloons to modern materials such as coils - Guido Gugliemi and collaborators (he used for the first time titanium microfilaments which in addition to obturation of the aneurysm also performed electrothrombosis at the same time) [4], succeeding on the one hand the delimitation of the vessel by using stents, and on the other hand the introduction of substances that perfectly obstruct the aneurysm - the results of combining the two techniques led to exceptional performance - Jaque Moret and collaborators, in France [5]

Aneurysm Prevalence

IA has a **prevalence of 1-9%** in the global population, with higher rates among Finnish and Asian people and of 2% in patients without any associated risk factors. Unruptured IA are more prevalent with females (3:1) and elderly. When detected in children, boys are more affected than girls and they prevail posterior circulation (40-50%) [6]. Factors associated with higher prevalence are female gender, age higher than 30 years old, nationality (Chinese, Japanese, Finnish, Korean), arterial hypertension, wall stress, genetic factors (Polycystic kidney, Marfan's disease, Ehlers-Danlos syndrome etc.) and lifestyle (smoking, alcohol use, diabetes) [7] .

Unruptured Aneurysms

Unruptured IA have **1-2% rupture rate/year** (Japanese cohort studies reporting up to a 3.2% rupture rate/year). Subarachnoid haemorrhage (SAH) associated with IA rupture has an incidence of **6-26 cases/100.000 people**, with a female male ratio of 1.6:1 and a peak between 40 and 60 years old.
Factors associated with rupture of an unruptured IA are both patient and aneurysm related:

- Patient related: **female gender, arterial hypertension, age (>50-60 years), smoking, sentinel headaches, lifestyle and metabolic factors as well as genetic**
- Aneurysm related: size, location (basilar bifurcation, anterior and posterior communicating artery), multiplicity, aneurysm growth and multiplicity, clinical symptoms as well as inflamation.

Rupted Aneurysms

For a ruptured IA, the most important complications are rebleeding and vasospasm.

Left untreated, a ruptured IA has 20-30% mortality in the first 2 weeks. The risk of rebleeding after a SAH reaches a peak in the first 24h **(4.2%)** - particularly in the first 6 to 8 hrs, having a cumulative risk of 19% in the first 2 weeks. After 6 months the risk reaches 50% and increases with 3% each year.

Vasospasm (CVS) represents a prolonged (although reversible) narrowing of cerebral arteries. It begins days after SAH, commonly between days 7 and 14. Pathogenesis of vasospasm consists of vascular smooth muscle contraction, endothelial injury and nitric oxide - endothelin1 imbalance (triggered by hemoglobin in subarachnoid space) as well as inflamation-mediated "remodeling" and narrowing of the arterial wall [8].

CVS represents the main cause of morbidity and death related to SAH. It leads to a neurological morbidity of 7% and an equal risk of death. CVS can be furtherly divided into 2 entities:

- Clinical CVS (also referred as delayed ischemic neurological deficit - DIND) - represents arterial stenosis causing cerebral ischemia and further clinical symptoms (which in turn may lead to irreversible neurological deficits)
- Angiographic CVS - arterial narrowing seen on vascular imaging. It can be detected several days after SAH and peaks between days 7 and 14.

Diagnosis of CVS relies both on clinical symptoms as well as imaging - Digital Substraction Angiography (DSA), CT and MRI with angiographic sequences, or even PET-CT as well as DWI and PWI MRI. Treatment follows two major paths: prevention (prophylactic balloon angioplasty, clot clearance, intrathecal vasodilators - nimodipine, magnesium, endothelin receptor antagonists, statins) or rescue treatment ("Triple H" therapy - hypervolemia, hypertension and hemodilution, intra-aortic balloon counterpulsation, endovascular reversal of vasospasm or intravenous milrinone) [8].

Aneurysms Etiology & Pathology

The exact etiology of IA development has not yet been accurately established. IA have a tendency to develop at arterial bifurcations, where there is a curve in the parent artery, in the angel described by it and a significant branch artery - in the direction that parent artery would have continued had the curve not been present[9].

Various processes, pathologies and genetic factors are to be incriminated:

- Congenital predisposition - a defect in the muscular layer of the arterial wall, often referred to as a medial gap
- Systemic hypertension
- Atherosclerosis
- Embolus - e.g. atrial mixoma
- Infectious - mycotic IA
- Traumatic
- Associated with other conditions - especially as part of genetic syndromes such as Autosomal Dominant Polycystic Kidney Disease, Fibromuscular Dysplasia, Marfan Syndrome, Ehlers-Danlos type IV Syndrome, Loeys -Dietz Syndrome, Moya Moya Disease or Sickle Cell Anemia.

IA Location

Location of IA is determined by several conjugate factors. Saccular IA represents 85% of all IA and are frequently located on major cerebral arteries - often in the branch apex where the hemodynamic stress on vessel wall peaks. Fusiform IA tend to develop more in the vertebrobasilar system. The location of saccular IA is as follows [9]:

- 85-95% in carotid system (anterior circulation), with 3 most common locations
  - Anterior Communicating Artery (ACoA) - 30%
  - Posterior Communicating Artery (PCoA) - 25%
  - Middle Cerebral Artery (MCA) - 20%
- 5-15% in posterior circulation (vertebrobasilar)
  - ~10% on basilar artery - basilar bifurcation (basilar tip), basilar-vertebral and basilar-superior cerebellar artery bifurcations as well as anterior inferior cerebellar artery.
  - ~5% on vertebral artery - most common on vertebral-posterior inferior cerebellar artery junction
- 20-30% patients present multiple IA

IA Pathological findings

In terms of pathological findings of IA, hemodynamic stress and arterial wall structure are often imbalanced. Contrary to what it may be expected, IA are associated with low shear stress - higher dome-neck ratios are in turn associated with lower shear stress.

In a normal arterial wall, hemodynamic stress and mechanical injury generates pads of myointimal hyperplasia, histologic finding similar with the one found in an IA wall. So, it can be stated that an aneurysm reacts to stress with a high cellular proliferation rate. Another aspect regarding IA is that smooth muscle cell become phenotypically oriented from contraction to proinflammatory and matrix oriented. Moreover, in comparison with normal artery tissue, there is a high expression and/or activity of matrix metalloproteinase (MMP-2 and MMP-9), capable of degrading both elastin and collagen - these findings are correlated with ruptured IA. In addition, although few arterial wall cells undergo apoptosis, in IA there are lots of apoptotic cell discovered. This also correlates with the high amounts of inflammatory cells discovered in the IA wall tissue - B and T-cell, macrophages, immunoglobulins and activated complement fractions.

Atherosclerotic plaques, often detected in relation with IA, are also highly known to promote inflammation and cell proliferation, hence the relation with apoptosis and often implication in aneurysm rupture.

Familial aggregation of IA highly suggests a genetic role in the development of this disease - this also being the strongest risk factor for SAH. Genetic and functional studies have clearly defined one initiating mechanism - high risk genetic variants may lead to the loss or alteration of THSD1 protein function which in turn may lead to an altered endothelial cell adhesion to the extracellular matrix.

From a histologic point of view, IA wall tissue displays several characteristics:

- Loss/rupture of the internal elastic lamina
- Myointimal hyperplasia - which leads to intimal thickening
- Muscle fibers disarray

- Depletion of cellular components
- Irregularities in the intimal surface
- Atherosclerotic plaques can be sometimes detected.

Clinical Features

IA can be asymptomatic, in this case being discovered by chance, or can present as neurological deficits or SAH - when it ruptures. SAH is the most frequent manifestation of IA, usually being accompanied by intracerebral haemorrhage (IH), intraventricular haemorrhage (IvH) or even, in 2-5% of the cases, subdural haemorrhage (SH). SAH is often accompanied by headache, vomiting, neck stiffness loss of consciousness which can lead to comatose state, seizures and sometimes 3rd cranial nerve palsy and fever. Clinical symptoms are graded using Hunt & Hess grading scale or WFNS grading scale and Fischer Score on CT [9].

Other than major rupture, there are other "warning signs" which may lead to IA suspicion:

- Mass effect - brain stem compression, cranial neuropathy or pituitary gland/stalk compression (which in turn leading to endocrine disturbances)
- Sentinel haemorrhage (a minor haemorrhage) - the latency between sentinel haemorrhage and actual SAH is the shortest - ~ 10 days
- Homonymous Hemianopia or amaurosis fugax - due to small infarcts or transient ischemia
- Seizures
- Headache
  - Around 25% of the patients describe sentinel headache which precedes IA rupture. It can be triggered by aneurysm expansion, partial thrombosis or intramural bleeding.
  - IA rupture is accompanied by "thunderclap headache", often described as "the worst headache of my life", vomiting, neck stiffness and loss of consciousness.
- Cranial Nerve Palsy
  - Oculomotor palsy - extraocular muscle palsy, ptosis and fixed midriatic pupils
  - Visual loss - due to compressive optic neuropathy (ophtalmic artery aneurysms) which leads to nasal quadrantanopsia, chiasmal compression and facial pain syndromes.

In some particular locations, clinical symptoms may indicate the existence of an IA. In middle cerebral artery (MCA) aneurysm the patient may display hemiparesis, visual field impairment as well as epileptic seizures. Moreover, in posterior cerebral artery (PCA) or vertebral artery (VA) clinical examination may detect oculomotor palsy and brain stem compression while in aneurysms of cavernous part of internal carotid artery (ICA) patient may develop cavernous sinus syndrome (ophthalmoplegia, proptosis, ocular and conjunctival congestion, trigeminal sensory loss and Horner's syndrome).

Diagnosis

The "golden standard" in terms of IA diagnosis is **Digital Substraction Angiography (DSA)**, although angiographic sequences of CT and MRI are rapidly gaining ground. However, despite still being the primary diagnostic tool, DSA has a risk of transient ischemic stroke (TIS) (1%), inguinal hematoma (1-2%) and stroke (0.01%)[10].

Angiographic CT has a sensitivity and specificity of 96-98%, varying for IA < 3mm to 90-94% reaching up to 100% for IA > 4 mm. The main advantage of angiographic CT is its low duration as well as bony structure visualisation. On the other hand, the drawbacks are lack of sensitivity for ICA in the skull base and cavernous sinus which fills with contrast substance.

Angiographic MRI has 70-99% sensitivity and 100% specificity. Most frequently, physicians use the three-dimensional time of flight sequence (3D TOF). Its cons are represented by increased duration of image acquisition and high sensitivity to movement.

Lumbar puncture is indicated only when the event happened at least 3 days prior to hospital admission or in case of angiography CT showing no evidence of IA despite clinical manifestations, but is rarely used these days due to the risk of cerebral herniation. Lumbar puncture analysis may detect red blood cells in the cerebral spinal fluid (CSF) and xanthochromia [11].

Treatment

In order to opt for the most suitable treatment approach, a better understanding of aneurysm natural history is mandatory.

Unptured IA

For unruptured IA, the prevalence is 1% to 9% in the global population. The risk of rupture is considered by most of the studies to be around 1%. The International Study of Unruptured IA (ISUIA) was conducted upon 1937 unruptured IA, being a reference study in the field. The patients were then divided into 2 groups: Group 1 - 727 patients, Group 2 - 722 patients. With a mean follow-up duration of 8.3 years the results showed that in Group 1 the cumulative rate of rupture was 0.05%/year - IA < 10mm and 1% - IA > 10mm (IA > 25 mm had a rupture rate of 6%). In the 2nd group, the cumulative rupture rate was 0.5% - IA < 10mm and 1% - IA > 10 mm. For the 1st group, in relation with rupture proved to be size and location (basilar tip, vertebral-basilar, posterior cerebral, and posterior communicating artery aneurysms having a higher risk of rupture). In the 2nd group location and increasing age proved to be predictors related with rupture [11, 12].

Lee and colleagues conducted a 7404 patients study followed on a period of 3 years. Rupture rate was reported at 0.9%/year, highest in the first year after IA diagnosis. The only risk factor for rupture proved to be old age [13].

Unruptured Cerebral Aneurysm Study (UCAS) evaluated 5720 patients. The risk of annual rupture was 0.95% which increased with IA size: 1.13% with 5-6 mm IA, 3.35% with 7 to 9 mm, 9.09% with 10 to 24 mm and 76.26% with sizes > 25 mm [13].

Greving and colleagues conducted an analysis on 8382 patients in order to determine predictors of IA rupture. The risk of rupture at 1 year was 1.4% and 3.5% at 5 years [14] .

While data for unruptured IA are not so well defined, when it comes to ruptured IA the information is more conclusive as well as based on randomized controlled trials. For this kind of IA the results are split into 2 groups - short term (from admission to 6 month from SAH) and long term (after 6 months from SAH).

For the short term results, rupture related factors can be further divided into patient and aneurysm related factors.

- Patients related factors - **clinical grade on admission** (the higher H&H grade the higher mortality risk; for H&H grade V there is 95% mortality risk), **gender, age, hypertension**, **time from hemorrhage** (patients seen immediately there is a 40% chance of 1-month survival; seen after 24 hours the rate increases to 60%, reaching a survival 1-month rate peak of 80% if the patient does not receive medical attention in the first 7 days), molecular an genetic profile as well as environmental factors (smoking was positively associated with survival after SAH).
- Aneurysm related factors - **aneurysm location** (VA location - 60.9% mortality, multiple IA - 47% mortality while ACA - 33.7% mortality rate) and **rebleeding (**poor clinical grade, intracerebral, subdural and intraventricular hematoma and systolic arterial pressure are associated with superacute rebleeding - < 8 hours; clinical grade and aneurysm characteristics - location, maximal diameter, configuration and intra-aneurysm blood pattern, are associated with acute rebleeding - days after initial SAH).

The long term results (after 6 months) shows that the average rebleeding rate is 3%. The overall mortality associated with delayed haemorrhage is estimated to be 60%. Epileptic seizures, complication associated with conservative treatment, varied according to the aneurysm location: 5% for ACA, 10% for PCA and 25% for MCA [3].
Treatment approaches for IA have radically shifted since the increasingly adoption of endovascular therapies. Hence, the debate whether open microsurgical approach or endovascular techniques is better suited became a popular topic in the field of vascular neurosurgery. The decision has to be individualised, taking into account not only aneurysm characteristics, rupture risk but also patient status and preferences [10].

Endovascular techniques display a wide range of options that physician can choose from:

- Thrombosing coils
  - Gugliemi Detachable Coils – titanium coils or electrical coils
  - Matrix Detachable Coils
- Stent assisted treatment - vascular stents and coils (Neuroform and Enterprise stent); it provides a desynchronization between arterial pulse of originating artery and IA, it consolidates the originating artery and promotes intimal cell proliferation and growth along the IA neck.
- Single vascular stent (in some cases even double stents are used).
- Blood flow diverter stent (PED - pipeline embolization device) - it reduces the amount of blood flow which reaches the IA, promoting aneurysm thrombosis
  - This type of endovascular device showed promising results in two major studies. Pipeline embolization device for IA treatment (PITA) showed 93.3% complete aneurysm occlusion at angiographic follow-up [12], while Pipeline for uncoilable or failed IA (PUFS) showed a 99.1% successful device placement rate on internal carotid artery, petrous segment (which is often inaccessible for open microsurgical approach) [15,16] .

At the opposite end, the **open microsurgical approach** is based primarily on vascular clips that fully obliterates the aneurysm from circulation - there are various clips models (McFadden VariAngle, Yasargil, Harnesniemi, Sugita).

Ruptured IA

One of the most important studies to evaluate safety and efficiency of the two approaches is International subarachnoid aneurysm trial (ISAT), a randomized trial conducted on 2143 patients - 1070 in the clipping group and 1073 in the coiling group. It aimed to determine poor outcome (modified Rankin Score (mRS) = 3-6) at 1 year follow-up. 97.3% of total IA were located in the anterior circulation (ACoA - 50.5%, ICA - 32.5%, MCA - 14.1). The results at 1 year follow-up showed that 23.7% of coiled treated patients and 30.6% of clipped patients met the aforementioned outcome (mRS = 3-6). Additionally, rebleeding rate at 1 year was 0.15% for endovascular approach and 0.07% for surgical approach [19].
The Barrow Ruptured Aneurysm Trial (BRAT), also a randomized study, had mainly the same aim as the previous one, mainly to determine safety and efficiency of these 2 methods. There were 239 patients surgically treated and 233 treated using coils. At 1 year follow-up, poor outcome (mRS = 3-6) was observed in 33.7% of the patients surgically treated compared with coiling techniques which resulted in just 23.2% poor outcome. Probability of reintervention was 4.49% with clipping while for coiling it reached up to 10.6% (more than twice as much). At 3 year follow-up, IA located on the posterior circulation proved to have a better outcome if treated by endovascular techniques. Reintervention rate at this point was 5% for clips and 13% for coils. During the same 3 year interval, complete thrombosis of aneurysm went initially from 85% to 87% for clips while it dropped from 58% to 52% for coils. At 6 years follow-up the occlusion rate was 96% for clips compared to 48% for coils. Overall, the reintervention rate was calculated at 4.6% for clips and 16.4% for coils. It is worth mentioning that surgical approach showed more versatility than endovascular one as approximately 30% of IA were inoperable by endovascular approach so the neurosurgeons opted for open microsurgery [20] .
A Chinese study conducted by Li and colleagues, presented results on 192 randomized patients. Cerebral infarction rate was measured reaching 21.7% for clips and 12.8% for coils, while aneurysm occlusion was 83.7% for clips and 64.9% for coils, both parameters measured at 1 year follow-up [21] .

Two meta-analysis conducted by Lanzino and his colleagues and Li et al. have also offered conclusive results. The first one showed that poor outcome at 1 year follow-up was higher in the surgical group rather than embolization one, with no mortality difference detected among the two. In the first month after treatment, rebleeding rates were lower in the coiling group [22]. Li and colleagues demonstrated the same results as the previous meta-analysis regarding poor outcome, a rebleeding rate of 1% for clips and 2-3% for coils and occlusion rates of 84% for clips and 66.5% for coils. They also found that vasospasm proved to be more common after clipping (43.1%) than coiling (43.1%) [23].
Another study further researched treatment associated vasospasm and observed the patient who undergo clipping developed localised vasospasm (around the rupture site) while those treated by endovascular approach demonstrated progressive distal vasospasm [24] .

Surgical timing

Is another aspect worth focussing on. It is divided into 4 categories:

- Immediate surgery (< 24h)
- Early surgery (24 - 72h)
- Delayed (3-10 days post SAH)
- Late surgery (10-14 days post SAH)

Immediate IA obliteration can improve outcome by eliminating rebleeding, especially for endovascular treatment, representing the actual approach. In a single center study, it was concluded that patients treated in the first 24h had lower incidence of poor outcome compared with those operated on after 24h. Moreover, there was also an absolute risk reduction in poor outcome and death if coiling can be performed in the first 24h [25,26]. However, immediate surgery is not recommended for multiple aneurysms in a single stage procedure, giant aneurisms, poor medical condition, posterior circulation aneurysms and low vascular surgery experience.
Delayed surgery is preferred for more complex lesions such as giant IA or in cases for which prolonged periods of temporary occlusion are expected to achieve aneurysm occlusion.
In conclusion, earlier IA occlusion has reduced the impact of rebleeding on patient's outcome, for both surgical and endovascular techniques. Additionally, it also allows more aggressive and earlier management of cerebral vasospasm [27].

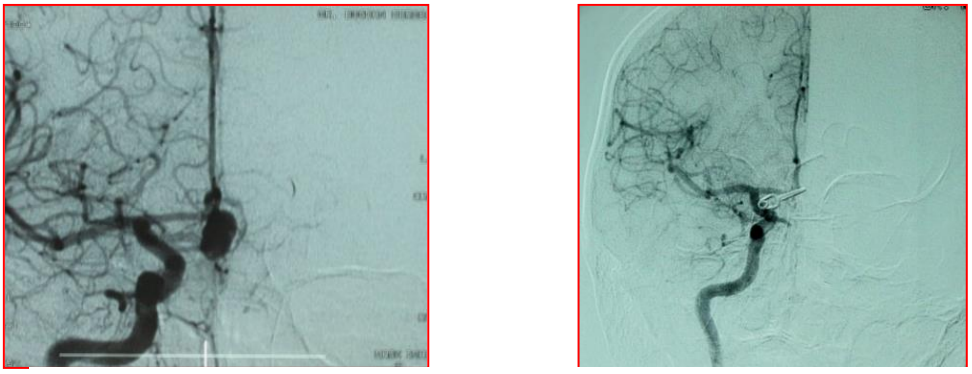

Figure 1. Clipped ACoA Aneurysm (with permission of A.V. Ciurea MD, PhD – personal archive)

Personal experience for ruptured IA

Authors present **the personal experience of 701 IA consecutive cases – Prof AV Ciurea MD, PhD, operated on using open microsurgical approach – Figure 1,** between 1 January 2000 and 1 January 2020 (20 years). Most cases (319 cases - 45,5%) were reported between 41 and 50 years old, with male preponderance - 476 cases (67,9%).
The symptoms were dominated by: headache 686 cases (97.86%), neck stiffness 658 cases (93.86%) focal neurologic deficits 498 cases (71.04%), seizures 364 (51.9%) etc., Hunt and Hess (H&H) grade II (359 cases – 51.21%) There were 54 (7.7%) difficult cases with H&H IV and no H&H V cases operated on. The associated pathology was: systemic arterial hypertension (526 cases, 75%) and obesity/ hypercholesterolemia - (238 cases, 34%), ischemic cardiopathy - (119 cases, 17%), diabetes mellitus - (112 cases, 16%), miscellanea (98 cases, 14% - anticoagulant therapy).
The common localization of intracranial aneurysms was the anterior communicating artery 170 cases (25%); the other locations were: medium cerebral artery - 105 cases (15%), posterior comunicating artery - 113 cases (16,11%), internal carotid artery - 280 cases, (40%), pericallossal artery – 7 cases (0.99%), basilar top artery - 21 cases (2,99%) and vertebral artery - 35 cases (10%). All cases were operated, as soon as possible after the subarachnoid haemorrhage (SAH) – Figure 1.
The results of this cohort: Glasgow Outcome Scale (GOS - extension) in our data (at 6 months postoperative) shows: Upper Good Recovery (GR) 199 cases (28.44 %); Lower GR 198 cases (28.24 %); Upper MD 129 cases (18.40 %); Lower MD 107 cases (15.26 %); Upper SD 20 cases (2.85%); Lower SD 14 cases (1.99%); Vegetative state 7 cases (0.99%); Death 27 cases (3.85%).

Conclusions

IA represents a neurosurgical emergency, a life threatening pathology due risk of rupture and vasospasm development. Subarachnoid haemorrhage (SAH) associated with IA rupture has an incidence of 6-26 cases/100.000 people, with a female male ratio of 1.6:1 and a peak between 40 and 60 years old. Rupture mortality reaches up to 40% while only 30% of patients can fully recover. Main treatment options are represented by neurosurgical clips and endovascular coils, the former becoming increasingly popular.

For ruptured IA, it can be stated that although short term results are promising for endovascular approach (coils), also proving to determine less perioperative complication, mortality and morbidity rate, in the long term those parameters are similar with the neurosurgical approach. However, clips still remains more efficient in terms of occlusion, rate of thrombosis and rebleeding prevention, especially on longer periods of time. Taking everything into account, further indications can be made regarding the appropriate choice of treatment. Clips are most suited for younger patients, IA with wide neck and located on MCA, when there is also a hematoma which needs to be evacuated, complex/unfavorable anatomy as well as sinuous arteries. Coils indications include H&H score > 2, cerebral edema, patient already receiving anticoagulant treatment as well as posteriorly located IA, dome: neck ratio > 2, fusiform and multiple IA.

Conflict of Interest

Authors have no conflict of interest to disclose

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