BRAIN ARTERIOVENOUS MALFORMATIONS PRESENTING WITH HAEMORRHAGE

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M Med Neurosurgery

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DECLARATION

I, Ntethelelo Mjoli hereby declare that the work on which this dissertation/thesis is based is my original work (except where acknowledgements indicate otherwise) and that neither the whole work nor any part of it has been, is being, or is to be submitted for another degree in this or any other university.

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LIST OF ABBREVIATIONS

AVM	Arteriovenous malformation
CSF	Cerebrospinal fluid
CT	Computerised Tomography
CTA	Computerised Tomography Angiography
DMSO	dimethy-sulfocise
DRC	Departmental Research Committee
DSA	Digital Subtraction Angiogram
EVD	External Ventricular Drain
GCS	Glasgow Coma Score
ICH	Intracerebral Haemorrhage
IVH	Intraventricular Haemorrhage
MCA	Middle Cerebral artery
MRA	Magnetic Resonance Angiogram
MRI	Magnetic Resonance Imaging
MRV	Magnetic Resonance Venogram
NBCA	n-butyl-cyanoacrylate glue
PET	Positron Emission Tomography
PTET	Partial Targeted Embolization Therapy
SAH	Subarachnoid Haemorrhage
SM	Spetzler-Martin score
UCT	University of Cape Town

TOTAL WORD COUNT: 13934

PART A: STUDY PROTOCOL

WORD COUNT: 1088

Cerebral Arteriovenous Malformation Presenting With Haemorrhage.

Principal Investigator: Dr Ntethelelo Mjoli

Supervisors: Associate Professor Allan Taylor and Dr David Le Feuvre

INTRODUCTION

Cerebral arteriovenous malformations (AVMs) are a congenital disorder where there is a fistulous connection between arteries and veins without an intervening capillary bed. They can present with hemorrhage (50 %), epilepsy (40 %), focal neurological deficits or may be asymptomatic. In patients who present with a haemorrhage the incidence of AVM re-bleeding is increased from 2-4% / year [1] to 7-11% / year. [2]

Cerebral AVM can be diagnosed on a brain CT scan or MRI scan, brain CT angiogram or MRI angiogram but the gold standard investigation is a cerebral angiogram as this shows the angioarchitecture of the lesion.

Important factors in deciding whether an AVM is treated include clinical presentation, location, size and features such as venous drainage which determine the risk of complications. Treatment options include microsurgical excision, endovascular neurointervention, radiosurgery or observation. Attempts at cure particularly with larger lesions has been associated with a high rate of complications, (6) which has led to a new treatment approach where intervention is directed at specific areas in an attempt to reduce the associated morbidity.(5)

RATIONALE FOR THE STUDY

Patients who initially present with intracerebral haemorrhage may have an identifiable source of bleeding on angiogram which can be a treatment target. [3 – 4] Previous work suggests that the re-bleed rate may be lowered if a weak area is eliminated. [5]

HYPOTHESIS

Patients with cerebral AVM presenting with haemorrhage may have an angiographically identifiable bleeding source suitable for treatment. Where treatment is successful the future bleeding rate may be lowered.

METHODS

1. Study design

Retrospective folder review of patients who presented to Groote Schuur hospital and UCT Private Academic Hospital over a 6 year period from January 2005 to December 2010.

2. Subject identification or selection

Patients will be selected by the Principal Investigator from a neurovascular database that includes all patients who underwent cerebral angiography for various disorders. All patients with the diagnosis cerebral AVM will be selected and a folder review performed in order to identify those who presented with intracranial haemorrhage.

Brain CT scans will be reviewed by the Principal Investigator and the Neurovascular consultants (Professor Allan Taylor and Dr David Le Feuvre) to confirm the presence of haemorrhage i.e. intracerebral, post fossa haematoma, intraventricular, subarachnoid hemorrhage and acute subdural haematoma.

Cerebral angiograms of patients with a confirmed presentation with haemorrhage will be reviewed by the same team of investigators to confirm the presence of an AVM, identify potential sources of bleeding and determine what neurovascular therapy was performed.

Exclusion criteria include:

i. Any patient with no evidence of haemorrhage on CT Brain
ii. Patients that presented with haemorrhage but were found to have facial, dural and spinal AVM as a source of the hemorrhage
iii. Children under 11 years of age.

3. Data collection

Instruments:

An electronic data capture sheet was designed for collation of data from patient folders, Brain CT scans and Cerebral Angiograms.

List of variables:

Demographic data: gender and age.

Clinical data: date of admission, presentation, presenting clinical features (headache, vomiting, seizures, neurological deficit), duration of symptoms and Glasgow Coma Score.

Brain CT scan: date of imaging, site of hematoma, midline shift, cerebral swelling, status of the basal cisterns and hydrocephalus

Cerebral Angiogram: location of nidus, size of the AVM nidus, fistula supply, choroidal supply, nidus false aneurysm, bleeding point, flow related aneurysm, associated aneurysm, number of veins, vein names, deep venous drainage, venous pouch, venous stasis, intervention, bleeding site treated, avm cured, angiogram complications.

Intervention: surgery, neurovascular intervention, radiotherapy, complications.

Follow up: date of last follow up, headache, vomiting, seizure, neurological deficit, GCS, intervention and AVM cured.

ANALYSIS:

Collected data will be statistically analyzed.

ETHICS AND COMMUNICATION

1. Ethics

The study protocol will be presented to the Surgical Departmental Research Committee (DRC) for approval, following which it will be sent to the Ethics committee with a copy of the synopsis for Ethics approval.

The data collation will commence as soon as the protocol has been approved. The folders of patients that had an AVM on the database will be requested for analysis. The folders will be reviewed in the Records Department for safety and to maintain patient privacy.

The radiographic imaging will be requested from Radiology filing. This will be kept safe in a locked room that is only available to the research team. The imaging will only be requested for a short period to allow the analysis by the research team. The imaging will be sent back to records as soon as the review process is finished. The data collated will be entered into the electronic data sheet.

Patient confidentiality will be maintained by following a strict protocol. The patient data will be entered into a computer immediately. The computer and research data file will be secured by a password that is only know to the reviewers. A computerized data sheet will be used to prevent having multiple data cards that can be lost with patient information. The patients' names and confidential information will not be used for publication purposes.

2. Reporting and implementation

The results of the study will be submitted to a peer review journal for publication and will be the basis of an MMed thesis. It is also anticipated that the results will be presented at an international conference in November 2011.

LOGISTICS

This will be a retrospective study which will be done during working hours. A three month leave from clinical duties will be provided by the Department of Neurosurgery.

There is no funding allocation for the study.

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PART B: LITERATURE REVIEW

WORD COUNT: 5588

BRAIN ARTERIOVENOUS MALFORMATION (AVM)

Outline

1. AVM definition
2. The natural history of AVM
3. Investigations
4. Grading
5. Treatment

AVM Definition

A brain AVM is a fistulous (non-nutrient supplying) connection between the arteries and veins of the brain where instead of normal intervening capillaries there is a nidus. The lesion is charecterised by an arterial supply which is pial, transmedullary or choroidal. The venous drainage follows the normal deep or superficial drainage of the brain. The nidus is a collection of vascular channels that are not similar to capillaries, arteries or veins; these are located outside the brain parenchyma. The AVM nidus has a varying angiographic appearance and may be very fistulous or comprised of small interconnected channels. The size of the nidus is variable from a few millimetres to centimetres.

Arteriovenous shunts are classified by the length of time it takes for venous filling once arterial filling has occurred. High shunt lesions fill within 2 seconds whereas a low shunt nidus fills more than 2 seconds after arterial filling.[1]

THE NATURAL HISTORY OF AVM

The estimated incidence of AVM in post mortem and clinical studies is 1: 100000[2,3] with a prevalence of 18 per 100000. Fifteen per cent of patients with an AVM are asymptomatic and will not ever present for medical treatment.[2] The gender distribution ratio is 3:2 male: female. Of the patients who are symptomatic 50% will present with intracranial haemorrhage, 40% will develop epilepsy and about 10% will have a neurological deficit without a bleed. Headache may also be a prominent symptom in a small number of patients.

The headache that is associated with an AVM is non-characteristic. The fourth decade is the median of symptomatic presentation.[4-6]

Patients that present with seizures can have focal, generalized or a combination of seizures. Seizures tend to present in the younger age group. There are a number of factors that have been associated with seizure occurrence such as lesion size and supratentorial cortical location, feeders from the middle cerebral artery, cortical feeders, venous varix, the absence of intranidal aneurysms[7] and location in an arterial border zone.[8] From a physiological viewpoint however it is more likely to be as a result of cortical ischaemia with the motor and mesial temporal cortex being more susceptible. Although arterial steal is a postulated cause of ischaemia in the tissue surrounding an AVM because of the sumping effect of the fistula a venous cause is more likely. Venous congestion of cortex remote from the AVM has been noted in patients presenting with epilepsy.[9]

Neurological deficits can be as a direct result of the avm compressing eloquent brain tissue or as a complication of uncontrolled seizures or avm rupture with resultant haemorrhage and brain tissue damage. In some patients however deficits present without these preceding problems and this is again likely due to venous hypertension and resultant ischaemia. Patients can present with visual field defects, sensorimotor deficits or language deficits.

Most patients will present with a ruptured AVM with resultant intracerebral haemorrhage, intraventricular haemorrhage or subarachnoid haemorrhage. There may be patients that present with a combination of the above. The haemorrhage can extend into the meninges depending on the site of the rupture. Patients presenting with a ruptured AVM have the highest risk of morbidity and mortality and close to 80% of patients that present with a haemorrhage will have a neurological deficit. The resulting late morbidity and mortality however varies widely between studies.

The overall morbidity rate is 2 - 7% for non-ruptured AVM and 23% for patients with haemorrhage.[10] The mortality rate associated with a non-ruptured AVM is 4% while that of ruptured AVM is between 6 -16%, but this number can be as high as 29%. The risk of haemorrhage in a patient with unruptured AVM is between 2 – 4% per year but this is increased to 7 – 18% for patients with a previous haemorrhage.[1,11-17]

Haemorrhage carries a very high risk to the patient with an avm, so attempts have been made to identify factors in the AVM that are associated with a high risk for haemorrhage. There is no single factor that has been identified that can conclusively be associated with a haemorrhage risk. The following are some of the factors that have been identified by different authors and they are not in the order of importance.

Several factors have been identified on the venous end of the AVM. A deep venous drainage (ventricular drainage, brainstem drainage or Vein of Galen); a single venous exit, since, the number of venous exits is inversely proportional to the venous pressure. Venous stenosis that leads to increased pressure within the veins and nidus and retrograde flow in the veins especially the superior sagittal sinus.

On the arterial supply side of the AVM there are also multiple factors to consider. The presence of an aneurysm is considered to be high risk and there are a number of classifications for these. Flow related aneurysms are considered to be saccular aneurysms on the downstream feeding vessel of an AVM and these are further divided into proximal when close to the Circle of Willis and distal if they are closer to the AVM or on the branches of the main arterial supply. Intranidal aneurysms are dilatations within the nidus but their observation is subjective and based on the quality of angiography, the frame rate used, the speed of flow and observer susceptibility. A false aneurysm is associated with a ruptured AVM and represents an area of contrast or blood filling within a haematoma. False aneurysms are angiographically characterized by a smooth round wall and contrast stasis. They are usually seen within the nidus and contrast lingers in them after the rest of the nidus has emptied. An associated aneurysm is a saccular aneurysm that is seen on an artery that is not feeding the avm these represent a generalized vasculopathy.

Other factors that are related to an increased risk of bleeding are a small sized avm that is less than 3cm; advancing age of the patient and patients older than 60 years old who have less elasticity of their arteries, which leads to less compliance of the blood vessels. Periventricular and transcallosal location of the avm nidus are also risk factors for haemorrhage but these lesions are also drained by the deep venous system. This illustrates the difficulty of retrospective assessment of angiographic features in patients who have bled in that observed features may be a consequence and not a cause. Posterior fossa location of the lesion, male gender and previous presentation with haemorrhage increase the risk of further haemorrhage.[4,13,18-25]

IMAGING (INVESTIGATIONS)

Computerised Tomography of the brain (CTB)

For most patients presenting with focal neurological symptoms, the initial radiological investigation is CTB. This is usually the first investigation that alerts the clinician to the possible presence of an AVM.

A CTB with no contrast will show a hyperdense lesion in the brain if there is associated haemorrhage. The hyperdensity may or may not be associated with a mass effect. The lesion is usually in an atypical site for a hypertensive haematoma. Localised calcification in the brain parenchyma may be the only sign of an AVM. The AVM may appear as a localised area of hypodensity with no visible associated vessels.

A contrast CT brain will show a mass of vessels that are not typical for that area of the brain. The nidus can show as a hyperdensity with the contrast media. The AVM will show as a ball of blood vessels.

The advantage of a CTB is that it is easily accessible, fast and non-invasive unless contrast media has to be administered. The CTB will show associated conditions such as hydrocephalus, haematoma, cerebral swelling, ischaemic changes and cerebral oedema.

Computerised Tomography Angiography (CTA)

A high resolution CTA requires a contrast medium to show the vasculature of the brain. An AVM will appear as a tangle of vessels and supplying arteries and draining veins will be seen running from the nidus. A high resolution CTA is comparable to a Digital Subtraction angiogram in diagnosing an AVM but not as good at resolving angioarchitecture because arterial and venous phases are overlapped on CTA.

An advantage of CTA is that it can be done immediately if there is a suspicion of an AVM in a CTB. This investigation can also show any flow or associated aneurysms when present. The brain can be subtracted for better visualization of the vasculature.(although this) A

source of the haemorrhage can be suggested depending on whether there is predominantly subarachnoid blood, related to aneurysm rupture, or a parenchymal haematoma adjacent to the nidus.

The CTA is highly dependent on the software and the speed of the scanner. It requires a contrast medium and is contraindicated in patients that are allergic to contrast medium. The presence of hematoma can mask the presence of a nidus. The CTA is not good for lesions that are peripherally located in the cortex.

Magnetic Resonance Imaging

MRI has the advantage of giving good brain parenchymal resolution and the AVM relation to gyral, sulcal and deep structures. It will show the AVM as a flow void on T1 and T2 images. If there is a bleed the age of the hematoma can be learned from the characteristics seen on the MRI. A gradient Echo T2 MRI may show haemosiderin staining which is suggestive of old haemorrhage that may have been subclinical. An MRA and MRV can be done to clearly show the vasculature of the brain. A further advantage of MRI is the ability to demonstrate associated thrombosis in the AVM or water changes in the surrounding brain as a consequence of venous hypertension.

The limitations of MRI are that it requires the patient to be co-operative because they have to lie still for an elongated period of time to get good images without motion artefact. Claustrophobic patients may find it difficult to tolerate MRI

Digital Subtraction Angiogram (DSA)

This is the gold standard of investigation; it is the best in showing the architecture of the AVM. The entire vasculature of the brain can be assessed which will bring to light any other vascular lesions that may not have been detected on another investigation. Because of the fast frame rate possible on DSA the temporal resolution of flow from arterial to venous structures can be separated and weaknesses in the lesion seen. DSA also allows clear visualization of brain arterial supply and venous drainage allowing the identification of en-passage vessels and shared lesion and brain venous drainage.

The diagnostic procedure can be done under local anaesthesia, but endovascular intervention

is performed under general anaesthesia. The test is safe with a complication incidence of 1:1000. DSA offers the ability to move directly from diagnosis to a treating intervention procedure.

AVM GRADING

There are a number of grading systems that have been developed to help determine the treatment risk for the management that is planned for the lesion.[26-33] There are no histological grading systems that have been developed. The most common grading system will be discussed.

Spetzler-Martin Grading[34]

This grading system was published in 1986 and it has been the most cited grading system ever since.[35,36] The grading system was designed for risk assessment when considering surgical intervention. The authors conducted a retrospective study to grade an avm and associate the grade with patient outcomes. Three elements were looked at that were found to be of significance for surgical resection of the AVM's, AVM size, location in eloquent cortex and the presence of deep venous drainage. The authors proposed a 5 -grade system.

Spetzler-Martin Grading score:

		Points
Size of lesion	*< 3cm*	*1*
	3 – 6 cm	*2*
	> 6 cm	*3*
Venous drainage	*Superficial*	*0*
	Deep	*1*
Location	*Non-eloquent*	*0*
	Eloquent	*1*

The size of the avm nidus received the most number of points because it is the most significant factor when excising an AVM. The size of the nidus is calculated from the angiogram, CTB or MRI. The largest diameter of the lesion is considered. A large avm nidus

is difficult to remove because it may span several eloquent areas therefore the risk of damage to the cortex is high. Small avm excision was associated with no morbidity. The authors propose that the size of the nidus can be indirectly correlated with the number of feeding vessels, amount of flow and degree of steal in the lesion. Assessing the nidus size requires experience as it is easy to include arterial supply and venous drainage which will falsely enlarge the lesion.

The second factor was the location of the avm nidus in relation to the areas that are considered to be eloquent. The areas that are considered to be eloquent were those areas that leave the patient with a severe focal deficit if injured during the surgery. The authors considered the following to be eloquent areas, language centres, sensory and motor cortex, visual cortex, internal capsule, thalamus, hypothalamus, brainstem and cerebellar peduncles. The proximity to eloquent areas can be judged on radiological imaging, there is no confirmatory test (Wada test, functional MRI and Positron emission tomography PET scan or brain mapping) that is required to prove the function of the area.

The last factor considered was the draining vessels of the AVM nidus. The drainage system was considered to be superficial or deep. The superficial drainage veins were those that drain the cerebral cortex including the superior and inferior sagittal sinuses, in the posterior fossa it is all the veins that drain the cerebellar cortex directly into the lateral or straight sinus. The deep venous systems are all the veins that drain the ventricles and the brainstem i.e. internal cerebral veins and vein of Galen. Deep venous drainage received a point because these veins are difficult to ligate and they may retract leading to severe blood loss.

The grades are allocating according to the total number of points accumulated by the AVM.

GRADE	POINTS
I	1
II	2
III	3
IV	4
V	5

A grade VI AVM is described as an AVM nidus that is large with a deep venous drainage that cannot be excised. A grade 1 lesion is a lesion less than 3 cm that has superficial venous drainage and is not associated with eloquent cortex. A grade V lesion is a lesion larger than 6 cm with deep venous drainage and close to an eloquent area. Morbidity and mortality is directly proportional to increasing grade of a lesion.

Spetzler at al believed that the gold standard of treatment for AVM was surgical excision to reduce the risk of further haemorrhage; they believed that incomplete surgical ligation does not reduce the future risk of haemorrhage. They also believed that the surgery should only be performed if it can improve the natural history of the disease.

A number of studies have supported the findings of this grading system indicating that it is a suitable grading system when surgical excision of an AVM is the treatment goal.[37-40]

Disadvantages.

A weakness of the scale is that there can be variability with cortical eloquence and anatomical imaging may not accurately define an area of eloquence. There are also areas of functional importance that are not classified as eloquent in the SM score. Cortical injury in a non-dominant parietal lesion will lead to visiospatial deficits that can be disabling.

The grading system makes no mention of the arterial supply to the lesion. A lesion with a high flow fistula can lead to very severe oedema after excision of the nidus. The number of arterial feeders and the areas of supply are also important in planning the excision of the lesion. Posterior fossa AVMs even if in a superficial location have a higher treatment complication rate because of hydrocephalus and the confined access space during surgery. This is a further omission in the SM grading.

Because the grading system is so widely used it is often used out of context in many publications. The SM score has little relevance when a patient is to undergo radiotherapy or endovascular intervention. It is also of little use when considering the natural history of these lesions. The classification does not predict the haemorrhage risk for an individual patient. The factors that are associated with an increased risk of haemorrhage are not taken into

account. Lastly it does not further the scientific understanding of the disease because it is only an anatomical grading system.[16]

ANGIOARCHITECTURE

An AVM has three components that have to be considered when looking at the architecture, the arterial supply, nidus and venous drainage. The Angioarchitecture is best assessed on the digital subtraction angiogram.

The dominant feeding artery is identified by having a large diameter than would be expected or when compared to similar region vessels. A feeding artery may have multiple smaller feeding arteries close to the nidus, which are minute in size, these may appear as a puff of smoke which should not be confused with Moyamoya disease. An enlarged and elongated artery identifies it as a high flow artery. The feeding artery may have a saccular aneurysm or a pseudo aneurysm (a localized irregular dilation of the artery wall). The feeding artery supplies the nidus but it may also supply brain tissue as well (en-passage vessel).

The nidus can have a variable appearance and can range from compact to diffuse. An intranidal false aneurysm is possible after a haemorrhage has occurred and is identified by a focal, smooth appearance within the nidus with contrast lingering into the venous phase and after the rest of the nidus contrast has cleared. A true nidal aneurysm might be detected when no haemorrhage has occurred and this would differ from a false aneurysm in that it has an intact wall. True nidal aneurysms are harder to define, as it is dependent on the quality of angiography and the size threshold that the observer applies when making this diagnosis. A false aneurysm clearly represents a weak area with only clot preventing further bleeding whereas the significance of a nidal aneurysm is not as clear. The feeding artery and the draining vessels can localize the nidus. The size can be worked out during the contrast injection. The flow through the nidus is defined as a high flow fistula if contrast leaves the nidus within two seconds, it is low a low fistula if the flow through the nidus is more than two seconds.[1]

The venous drainage of the nidus will use the normal veins of the area where the nidus is localized. A draining vein should only be counted as such if it can be seen to be directly exiting the nidus, as opposed to veins that contribute to AVM drainage through reflux from the draining vein. True exit veins and reflux veins may be difficult to identify if the nidus is

close to a venous sinus and the veins are short. The draining vein may have a varix or venous pouch. The venous drainage can be from a single or multiple vessels. The vessels can be superficial, deep or a combination. The venous drainage with localized stenosis is easily identified by contrast stasis in the vessel.

TREATMENT OPTIONS AND DECISION-MAKING

Haemorrhage is the risk that all patients and treating doctors would like to avoid in the treatment of an AVM, as it is associated with the highest morbidity and mortality. The only certain way to prevent haemorrhage is AVM cure. As haemorrhage risk remains fairly constant at 2-4% per annum the patient age at the time of presentation is a crucial factor. If a patient presents at age 65 years their risk of haemorrhage over the next 15 years is 45% with an overall morbidity and mortality risk of 7%. Curative treatment is likely to have a higher morbidity at this age and therefore conservative treatment would be a better option.[5,41]

In younger patients with a higher lifetime bleeding risk the decision to obtain a cure is largely based on the AVM size. This is irrespective of treatment choice because surgery, radiotherapy and endovascular cure are more likely to be achieved in smaller AVM's.

In patients who have an AVM that is too large to consider cure palliative embolisation is an option. Any patient presenting with haemorrhage has a higher bleeding risk for at least the next 2 years. If an angioarchitectural weakness could be closed in these patients the bleeding risk may be reduced to the baseline 2-4% per year again.

A patient that presents with seizures can be commenced on medical treatment and expectant management. Medication will be adjusted according to patient response. If the patient is well controlled on medication then the patient can be followed up.

Patients that present with headaches and non-specific symptoms can be managed symptomatically. If the symptoms improve then the patient can be followed up. If the symptoms do not improve but they are not disabling the patient can be followed up closely.

A patient that presents with focal neurological signs has to be investigated to establish the cause of the deficit. If the deficit is due to an avm compressing brain then a decision has to

be as to how disabling is the deficit. Patients with a good Rankin score have to be observed. If the oedema is the cause then medical therapy can be instituted to improve the oedema.

A patient presenting with haemorrhage will need treatment for any urgent conditions for example hydrocephalus, mass effect or herniation. The AVM can be managed electively once these urgent conditions have been treated. Three options are available for the AVM, these will be discussed briefly but emphasis will be on endovascular treatment.

Surgery

Surgery offers patients the highest chance of cure when appropriate patients are selected. As surgery carries incremental morbidity and mortality with the increasing grade of the AVM it is most suitable for low SM grade lesions. Surgical management of SM Grade I and II is associated with a morbidity rate of 6 – 8%.[40]

Radiotherapy

Radiosurgery is a good option for small deep AVM's as it offers a good cure rate with low morbidity. Radiosurgery is done under local anaesthetic with no possible complication of a general anaesthetic. The procedure is done in one day. The disadvantage is that it takes up to two years before the AVM reduces in size. Radiosurgery is not a good choice in patients who have presented with haemorrhage because of the increased bleeding risk. Patients with bleeding may benefit from targeted embolisation prior to radiosurgery. Lesions that are above 12ml in volume are not suitable for radiotherapy as they carry a high morbidity and mortality due to radiation spread into normal tissue.[42]

Endovascular treatment

Neuroendovascular treatment can be performed for complete occlusion of the avm, partial targeted occlusion of the areas that are considered to be weak area and preoperative embolisation to reduce the size of the avm before surgery or radiotherapy is instituted.

The role of curative embolisation is to obliterate the AVM and restore the normal arterial supply and venous drainage of the brain. The right setting is an AVM less than 3cm, less than 3 feeding arteries and the nidus must be accessible with a catheter. Procedure related complications are low when the procedure is done for the right indication.[43]

Preoperative embolisation is used to reduce the size of the nidus and decrease the amount of blood loss during surgery. It is carries 2 - 4% mortality rate and 9% permanent neurological deficit. Le Feuvre et al in their prospective study found a 1.2% death rate and a 6.5% permanent neurological deficit.[9]

Partial targeted embolisation therapy (PTET) is used to occlude and reduce the size of the AVM with minimal morbidity. The advantage of PTET is that it can be used for any size of the AVM nidus. Partial embolisation leads to total obliteration of a flow associated aneurysm in 8% of cases where the nidus is reduced by 50%. The morbidity associated with PTET is between 4.5 - 13% and the mortality is 1 - 3.5%.[44,45]

There are multiple forms of embolisation, n-butyl-cyanoacrylate glue (NBCA), Onyx and silk sutures.

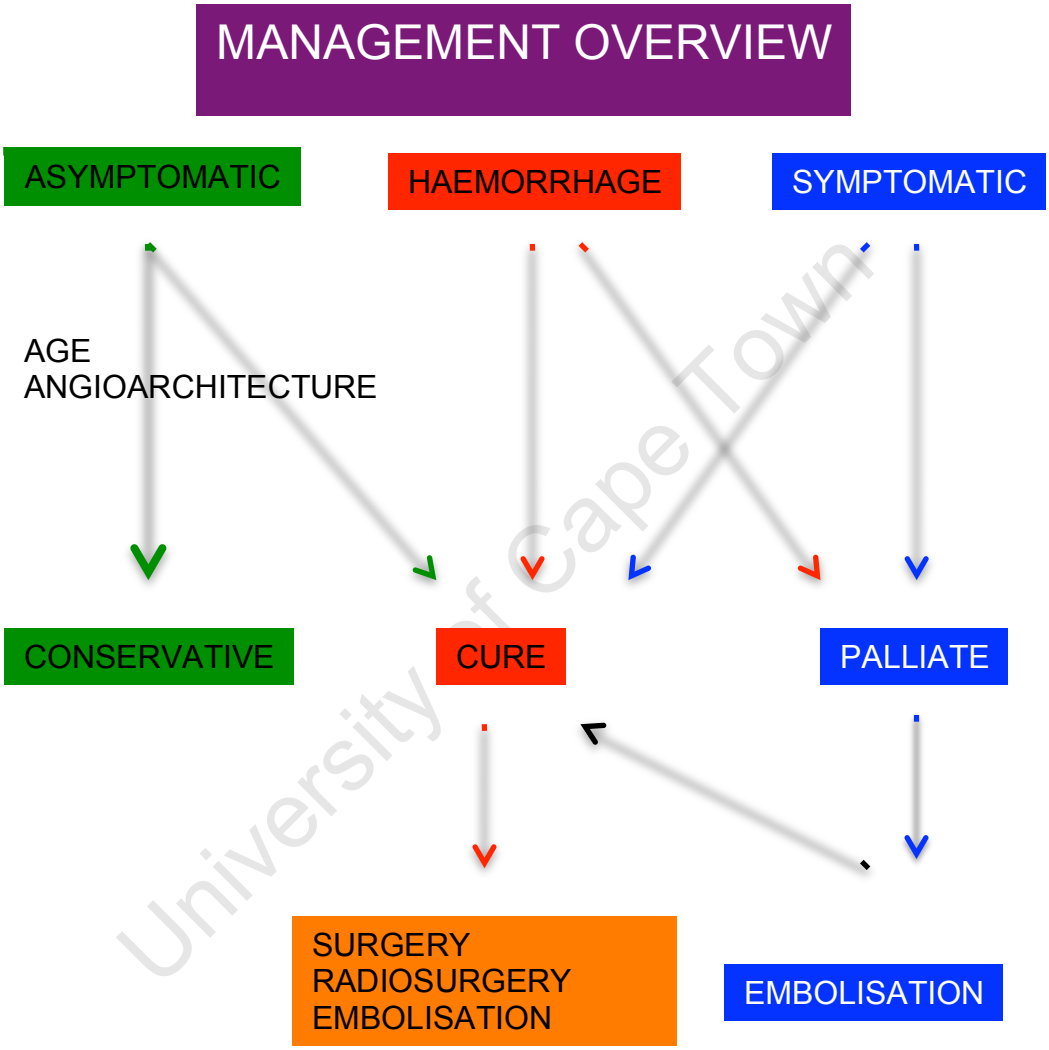
NBCA glue is injected through a small lumen micro-catheter that can reach the distal location of the AVM. The material polymerizes quickly. The procedure requires an experienced operator as proximal occlusion may occur and the microcatheter may glue against a vessel wall. If the procedure is not correctly performed there may be revascularization of the nidus with small arteries that cannot be embolised. There is a limited amount of glue that can be used per catheter therefore multiple catheters may have to used to get occlusion of the nidus.[46]

Onyx is an embolic material made of ethyl-vinyl copolymer dissolved in dimethy-sulfocise (DMSO) with tantalum powder added for radio-opacity. The solidification process is slower than with NBCA. Onyx is associated with 3.7% morbidity and 1.4% mortality per session embolisation. Complete occlusion of the AVM with Onyx is associated with 8% morbidity and 3% mortality on all patients.[45,47]

Embolization with silk sutures involves the use of pieces of 6-0 silk sutures. Silk sutures are injected into the feeding pedicles leading to occlusion of the artery. This is associated with inflammation of the artery and occlusion of the artery. Studies have failed to show an increased incidence of vasculitis with silk sutures.[48-50] The use of silk is however limited as occlusion tends to be proximal leaving the nidus intact.

The complications of embolisation include the rupture of feeding pedicles resulting in intracerebral haemorrhage. Post embolisation haemorrhage may also be as a result of venous

side contamination with glue leading to venous thrombosis and nidus congestion. The micro-catheter may get stuck against a blood vessel, with the risk of later thromboembolic complications. Onyx extravasations can occur if too much pressure is applied during injection.



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PART C: MANUSCRIPT

WORD COUNT: 3785

Brain Arteriovenous Malformation Presenting With Haemorrhage.

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ABSTRACT

Objective:

AVM patients who initially present with intracerebral haemorrhage may have an identifiable source of bleeding on angiogram, which can be a treatment target. Previous work suggests that the re-bleed rate may be lowered if a weak area is eliminated.

Methods:

A retrospective cohort study was conducted of patients that presented over a 6-year period with a bled AVM. We were looking for the source of the haemorrhage by applying a strict criteria looking at radiological imaging (CT brain, CT angiogram, Digital subtraction angiograms and MRI). Neuroendovascular notes were reviewed to identify the treatment targets.

Results:

One hundred patients presented with a brain AVM with a 1.7:1 male: female ratio, 41 patients had an initial presentation of haemorrhage. The source of haemorrhage was identified in 18 subjects with 11 intranidal false aneurysms, 5 flow related aneurysms, 2 associated aneurysms and 1 venous pouch. The location of haemorrhage on presenting scan significantly correlated with the identified bleeding source using Chi-square analysis (chi-square 16.25, correlation 0.52 and P-value 0.039). Partial targeted embolization was used successfully in 90% with 9% related morbidity with no mortality. Long-term follow-up data was 1074 months with annual haemorrhage rate of 0.7%.

Conclusion:

Aneurysms can be identified as a source of haemorrhage in half the patients with AVM. Patients are likely to have subarachnoid haemorrhage than intracerebral haemorrhage. The weak point is a good target for partial endovascular treatment and it may reduce the re-bleeding rate over the next 2 years.

KEYWORDS: Arteriovenous malformation Intranidal aneurysm
Partial targeted embolisation

INTRODUCTION

A brain arteriovenous malformation (AVMs) is a fistulous connection between arteries and veins without an intervening capillary bed. They can present with hemorrhage (50%), epilepsy (40%), and focal neurological deficits or may be asymptomatic. In patients who present with a haemorrhage the incidence of AVM re-bleeding is increased from 2-4% / year to 6-18% / year.[1-6]

The ideal treatment of an AVM is complete obliteration of the lesion which removes the risk of further haemorrhage and reduces the seizure risk. This is however not always possible at an acceptable risk.

Important factors in deciding whether an AVM is treated include clinical presentation, location, size and features such as venous drainage, which determine the risk of complications. Treatment options include microsurgical excision, endovascular neuro-intervention, radiosurgery or observation. Attempts at cure particularly with larger lesions has been associated with a high rate of complications,[7-14] which leaves an alternative approach where endovascular intervention is directed at specific angiographic target areas in an attempt to reduce the associated morbidity.[3,15,16] In this study we sought to find a clear correlation between presenting with a haemorrhage and having an identifiable angiographic bleeding source. Although we may be finding something that is a consequence rather than a cause of bleeding it allows more certain correlation between bleed and lesion. As AVM haemorrhage rate is significantly increased after a bleed it also offers an opportunity to lower this bleed rate to baseline or below.

SUBJECTS AND METHODS

The Neuro-endovascular database is an ongoing prospective project collecting clinical and neurovascular notes of patients admitted to Groote Schuur and UCT Private Academic Hospital. All new AVM patients are captured as well as any subsequent symptomatic events that patients may present with.

Subjects were selected from the database if they were presenting for the first time and had an arteriovenous malformation presenting with haemorrhage. Patients identified as having a dural AVM, facial AVM, spinal AVM or aneurysms with no associated AVM were all excluded. A retrospective folder review was conducted to find the demographic and clinical data of the subjects. All subjects had undergone a CT brain scan that identified the hemorrhage. A consensus review of all the scans was conducted by the authors to identify the location of the hemorrhage (intracerebral, subarachnoid or intraventricular) and associated findings such as, cerebral oedema, mass effect and hydrocephalus. The haemorrhage site was then compared to the digital subtraction angiogram to identify the source of the bleeding.

The source of bleeding was identified by looking for the presence of an aneurysm or a venous pouch. A flow related aneurysm was defined as a downstream saccular aneurysm in a vessel supplying the nidus and an intranidal false aneurysm was identified as a defect with a smooth wall that was larger than the nidus channels with contrast lingering within the defect into the late venous phase. Nidus irregularities that did not demonstrate all these features were ignored as the identification of a nidal aneurysm other than a false aneurysm is subjective relying on DSA quality, timing and observer opinion. An associated aneurysm was defined as a saccular aneurysm located away from the vessels supplying the nidus. A venous pouch was a localised eccentric dilation of a vein draining the nidus.

The angiograms were also assessed for location and size of the nidus, fistulous and choroidal supply, the number of draining veins and deep venous drainage. The angiograms and angiogram notes were used to assess the neuro-intervention for technical success and complication occurrence.

Follow up data was collected from folders. A telephonic interview was also conducted to assess the clinical status of the subjects with particular interest on the: current level of function, presence of headaches, seizures, residual focal deficits and history of rebleeding.

RESULTS

In the review period 100 new subjects presented with a cerebral AVM. The group that presented with hemorrhage was comprised of 41 subjects who collectively had 44 episodes of hemorrhage, rebleeding will be discussed later in the results.

The demographic profile of the patients is analyzed in Table 1. Subjects presented with a sudden severe headache in 61% of cases and 10 subjects (22%) presented with a generalized or focal seizure as a result of their haemorrhage. There were 21 (47%) patients that presented with focal neurological deficits ranging from dysphasia, memory loss, and long tract signs. The average age of the subjects was 36,2 years with a distribution of 11 to 62 years. The median Glasgow Coma Score at presentation was 13 and there were no patients that died on arrival or during admission.

The location of haemorrhage on the presenting scans, intracerebral haemorrhage, intraventricular hemorrhage or subarachnoid hemorrhage, significantly correlated with the identified bleeding source using Chi-square analysis (Table 2). Nine patients presented with SAH and of these 5 had bleeding from an aneurysm distant from the nidus, only 2 patients with nidal rupture developed significant SAH. Twenty-eight patients had a parenchymal haemorrhage associated with the nidus and in 17 of these patients no bleeding point was visualized, however of the 11 where a weakness was found, 8 had an intranidal false aneurysm.

The source of bleeding (Table 3) was identified in 18 subjects with 19 episodes of hemorrhage. Eleven of the patients had a clear intranidal rupture with a false aneurysm visible on DSA done shortly after presentation. Eight patients did not bleed from the nidus, 5 bled from a flow related aneurysm, 2 from an associated aneurysm and 1 from a venous pouch. The patient that presented with a repeat bleed, had a right parietal AVM that initially presented with SAH and a ruptured flow related aneurysm was identified on the Anterior Communicating Artery, the aneurysm was wide necked and it was not amenable to endovascular treatment, he represented a month later with intracerebral hemorrhage and no

source of the bleeding could be found, so he was managed conservatively, he presented after 4 years with another intracerebral hematoma, an intranidal aneurysm was identified and treated, he has not bled since.

All the subjects with an identifiable source of hemorrhage were managed with targeted endovascular treatment and this was successful in 89% of the cases. There was a 10,5% procedure related technical complication rate, one patient had glue reflux with no clinical consequences and the other had thrombi-embolic occlusion of the left MCA branch with transient dysphasia, there was no long-term morbidity related to the procedure. The procedure had to repeated on some patients in order to achieve the targeted result. There was no mortality related to the procedure.

Surgical intervention was performed in 8 (18%) of the subjects following hemorrhage; this was for mass effect and hydrocephalus. There were two subjects that had an evacuation of the intracebral hematoma and excision of the residual AVM. Only one patient had evacuation of intracerebral hematoma and the AVM was left insitu. The remaining 5 patients were surgically treated for hydrocephalus, 4 patients had an external ventricular drain on admission and it was later removed when the hydrocephalus improved, only one patient had an EVD done on admission and later had an Endoscopic Third Ventriculostomy for permanent CSF diversion. There were no recorded adverse effect related to the surgery and there was no mortality as a result of the surgery.

Long-term follow-up data was only available for 29 patients as some patients were referred from far away and were not contactable. 1074 months of patient follow up were completed for this cohort (mean of 34 months). Only one patient had rebleeding during this period and bled on 2 separate occasions giving an annual haemorrhage rate of 0.7%

DISCUSSION

Arteriovenous malformations present with haemorrhage in 50% of cases, this accounts for 2% of all patients that present with stroke.[17,18] There is an equal gender distribution of all patients that have an avm, but there are slightly more male patients than females who present with a haematoma.[19] Multiple factors have been found to be associated with an increased risk of haemorrhage such as a single draining vein, deep seated avm location, small size avm, deep venous drainage, advanced age and aneurysms associated with an avm.[20-28] The

annual risk of a haemorrhage in a patient with an unruptured avm is 2-5% / year with annual morbidity and mortality rate of 2,7%% and 1% respectively.[1-3,19] The ruptured avm rebleeding risk is 6%-18% within the first year and it lowers to 2% per year for the next 20 years.[29] This is most likely because the rupture point remains weak and susceptible to re-haemorrhage until vascular repair is complete.

For AVM's presenting with haemorrhage the primary treatment objective remains exclusion and cure of the AVM and associated vascular weaknesses. This is however not possible in all cases as sometimes the AVM is too large or the lesion is inaccessible. Even in patients where cure is possible by surgery or radiosurgery, exclusion of the recent bleed site is advantageous. In the case of surgery it makes haematoma drainage possible without the need to urgently exclude the AVM, which can be operated on electively. In patients who require radiosurgery it could reduce the high bleed risk that the patient faces in the next year or two to a safer level.

Partial targeted embolisation is a safe alternative to complete excision or occlusion of an avm and a useful adjunct even when these treatments are planed. In patients that present with haemorrhage, a localised area of weakness representing the rupture point can be identified in some patients[9] had a cohort of nine patients that had intranidal aneurysms, which were a treatment target. The one-year follow-up of these patients showed a lower rebleeding rate than expected.[15] In this study we sought to analyse in more detail the group of AVM patients presenting with haemorrhage.

We had one hundred patients that presented with a brain avm over a 6-year period and only 41 patients initially presented with haemorrhage. These numbers are small but the distribution of the patients that presented with haemorrhage is consistent with the natural history so this sample is likely representative of a larger AVM population.

It has been previously shown that the location of hemorrhage could not be used to predict the presence of an aneurysm.[29] In our cohort, patients that presented with a subarachnoid hemorrhage were more likely to have bled from a flow related or associated aneurysm and patients presenting with intracerebral bleeding more likely to have a nidal false aneurysm. Based on this finding AVM patients who present with SAH should be carefully investigated, to look for an aneurysmal source of bleeding.

We were unable to conclusively identify the source of haemorrhage in 25 out of 44 bleeding episodes (57%) which is disappointing. We did however apply strict criteria when reviewing the presenting scans and angiograms. There had to be good correlation between the haemorrhage site and the angiographic weakness and we only identified intranidal false aneurysms as a nidal bleeding source. Although other nidal irregularities were observed, and some were even called nidal aneurysms in procedure notes, we ignored these lesions because they could not definitely be identified as the bleeding site. Nidal aneurysms have been associated with a higher haemorrhage risk.[6,7,25] However there is currently no clear evidence that it is the nidal aneurysm that ruptures when this patient group presents with haemorrhage.

Other authors have identified flow related and associated AVM aneurysms as a sign of vessel fragility, as this group of patients has a higher haemorrhage rate, although the source of bleeding was not felt to be aneurysmal.[5,6,15] In our series, five episodes of bleeding out of 44 were identified as aneurysmal in origin. Given that only 7 patients in this study were identified as having flow/associated aneurysms our findings offer a different perspective and perhaps avm related aneurysms should be considered as a bleeding source.

We use Neuroendovascular embolisation as our first line of treatment for patients that have an identified angioarchitectural target. All patients that presented with haemorrhage were embolized with NBCA, one patient was treated with Onyx and there were no patients that were treated with particles or silk sutures. NBCA has been shown to effectively occlude avm feeders and the avm nidus.[7,30-34] Only one patient had a craniotomy to evacuate the haemorrhage because of mass effect, but one patient had an avm excised after embolisation, this was a small avm associated with a large hematoma.

Partial targeted embolisation was our treatment of choice for all those patients that had an intranidal false aneurysm. All of patients with an intranidal aneurysm could have a catheter passed up to or into the nidus and the false aneurysm was occluded successfully. This was done in one session for all this group of patients. The flow related aneurysms were all easily accessible on the avm feeding artery and all patients were treated using bare platinum coils except for one patient who had a wide necked anterior communicating artery aneurysm that could not be embolized. This aneurysm was thought to be the source of bleeding in this patient and was the only patient where the target could not be treated.. In this patient an attempt was made to reduce the flow in the AVM by reducing the size of the nidus to below

50%. This patient was the only one to rebleed in the series with an identifiable cause of bleeding..

Although follow-up data was not complete for all patients the rebleed rate of 0.7% was significantly lower than would be expected for patients presenting with haemorrhage.

CONCLUSION

Flow related and associated aneurysms in patients with brain AVM can cause haemorrhage. These patients are more likely to have SAH than intracerebral haemorrhage. In just under a half of patients with AVM bleeding a source of haemorrhage can be identified on DSA. This weak point is a good target for partial endovascular treatment is usually accessible and may reduce the higher haemorrhage rate expected over the next year.

Table 1

Gender Distribution

Avm	Male	Female
Total	63	37
Bled	31	10
Identified bleeding source	16	3

Table 2

Location of the haematoma vs. source of bleeding for the 44 bleeding events

Haemorrhage	Flow related aneurysm	Intranidal aneurysm	Associated aneurysm	Venous pouch	Source not identified
SAH	4	2	1	0	2
IVH	0	1	0	0	6
ICH	1	8	1	1	17

Chi-square 16.25

Correlation 0.52

P-value 0.039

Table 3

Bleeding source vs. partial targeted neuro-endovascular treatment

Source of bleeding	Number of patients (%)	Bleeding point treated	Failed
Flow related aneurysm	5 (11,3)	4	1
Intranidal false aneurysm	11 (25)	11	0
Associated aneurysm	2 (4,5)	1	1
Venous pouch	1(2,2)	1	0

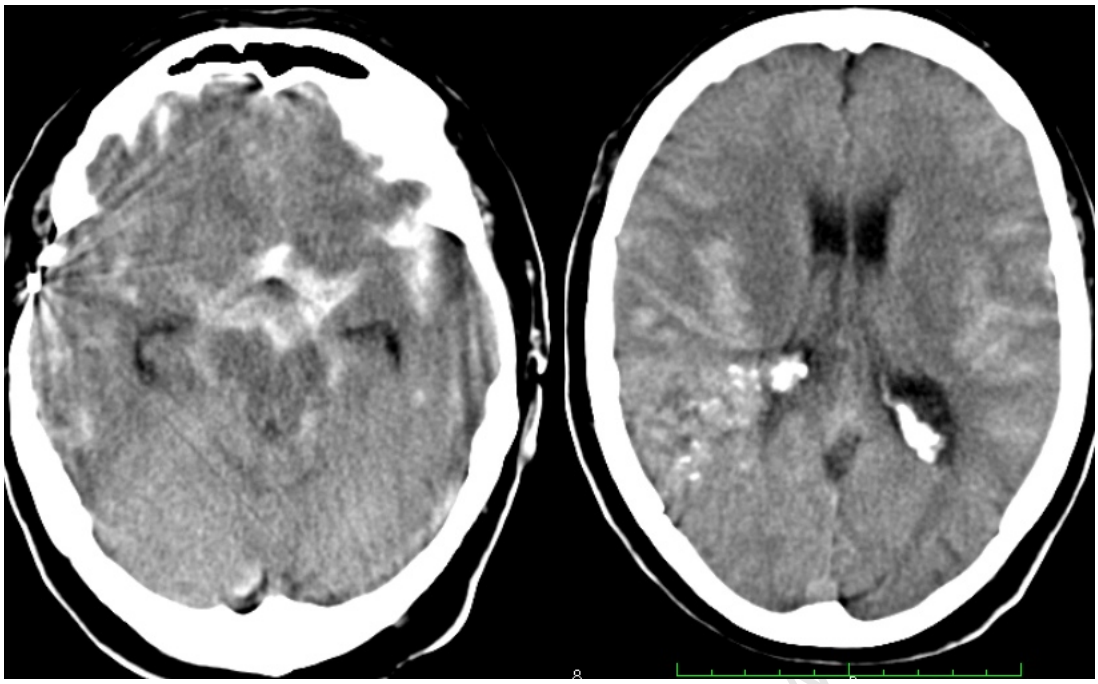

Fig 1

Patient 1. A 60 year old patient presenting with SAH 20 years after failed surgery for a large right parietal AVM. Haemorrhage is mostly in the supra-sellar cisterns and peri-mesencaphalic cisterns. There is hemispheral cistern blood but no parenchymal blood that would indicate nidus haemorrhage.

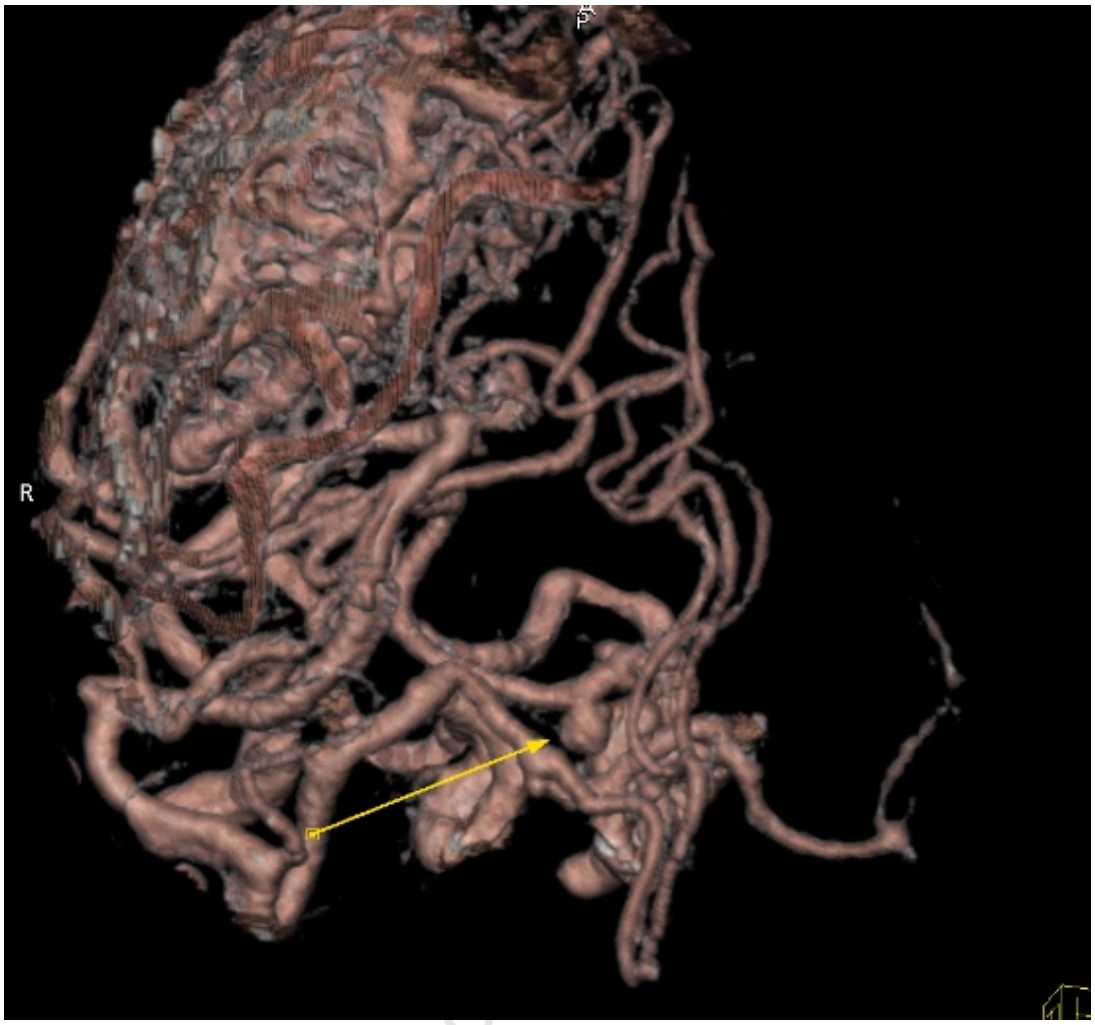

Fig 2.

Patient 1. A CT angiogram revealed a right P1 proximal flow related aneurysm corresponding to the SAH localization.

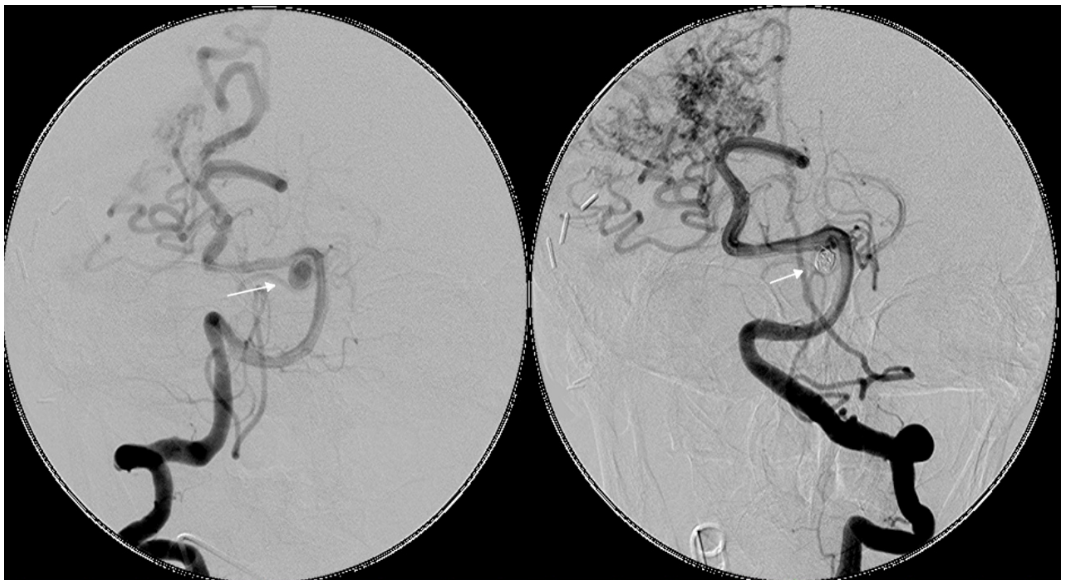

Fig 3.

Patient 1. Vertebral angiography demonstrating the P1 aneurysm before and after coiling.

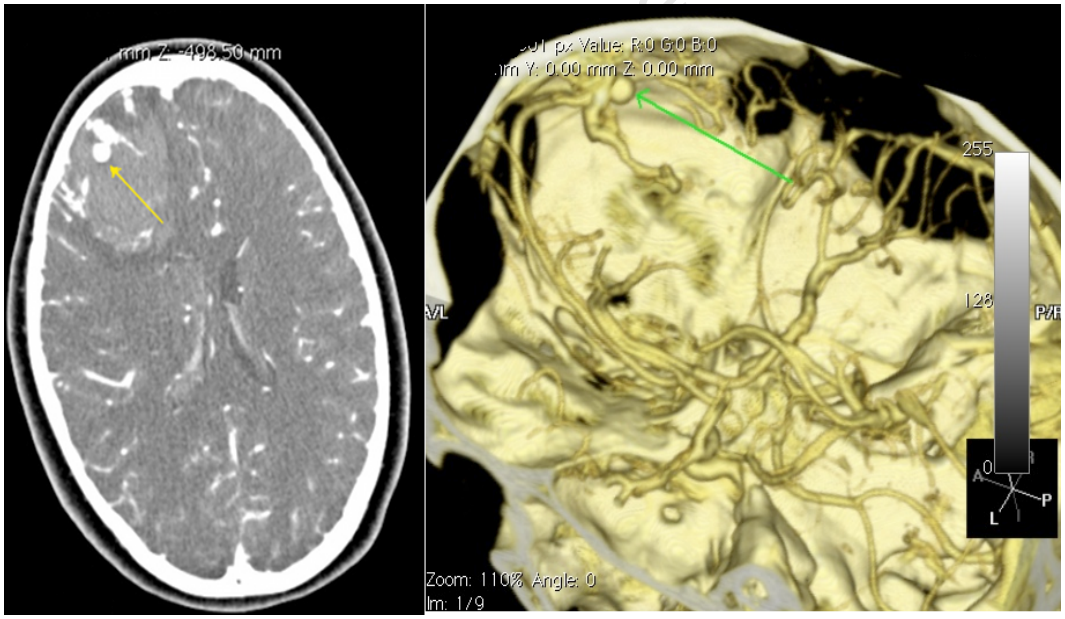

Fig 4.

Patient 2. A 14 years old female presenting with sudden onset of headache. A CT angiogram demonstrates a right frontal parenchymal haemorrhage with an associated intranidal false aneurysm.

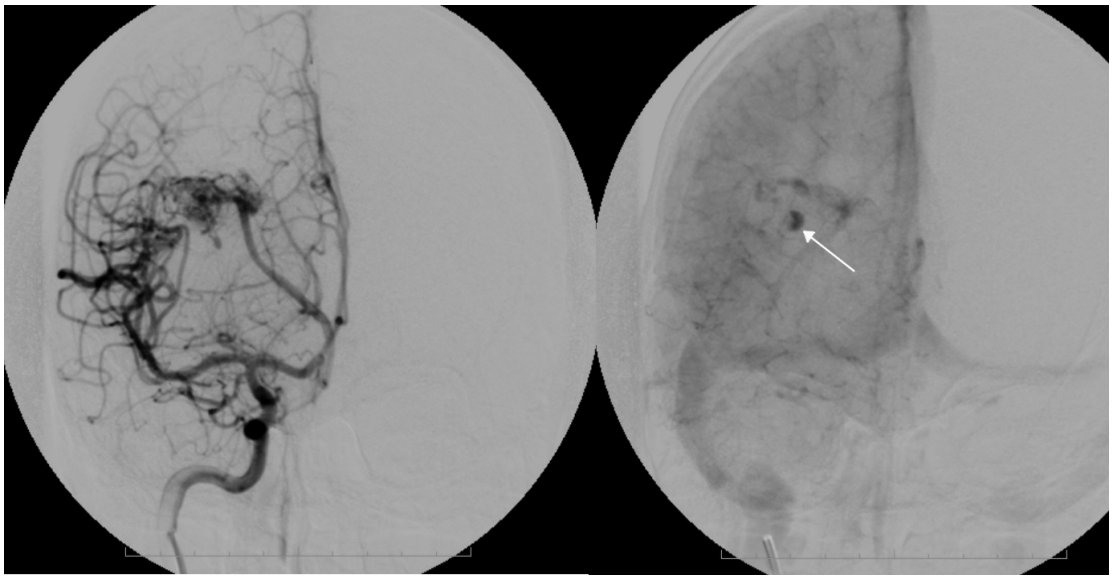

Fig 5.

Patient 2. DSA demonstrates the typical features of an intranidal false aneurysm.

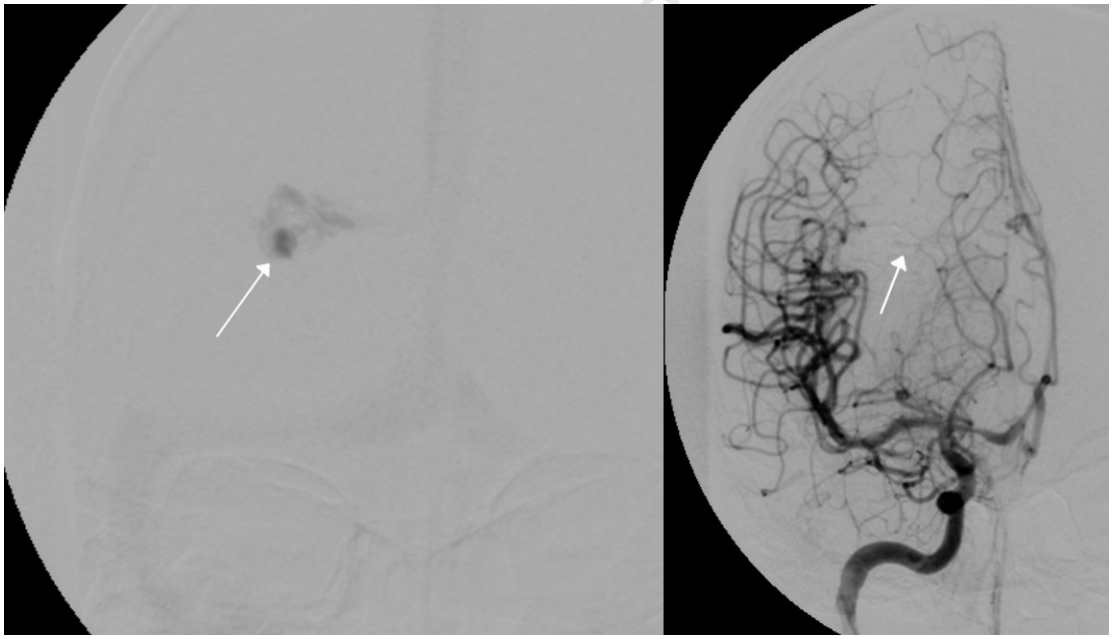

Fig 6.

Patient 2. An intranidal microcatheter injection shows filling of the targeted false aneurysm which was closed with an NBCA glue injection. Post embolisation angiography shows no filling of the false aneurysm.

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PART D: APPENDIX

Departmental Research Committee Approval Letter

UNIVERSITY OF CAPE TOWN

Department of Surgery

Departmental Research Committee
Professor Anwar Suleman Mall
J-45 Room Old Main Building, Groote Schuur Hospital,
Observatory 7925, South Africa
Tel (021) 406 6168/6232/6227 **Fax** (021) 448 6461
Email: Anwar.Mall@uct.ac.za

15th March 2011

Dr N Mjoli
Department of Surgery
Division of Neurosurgery
Groote Schuur Hospital
University of Cape Town

Dear Dr Mjoli

RE: PROJECT 2011/021

PROJECT TITLE: Cerebral arteriovenous malformation presenting with haemorrhage

The above proposal was reviewed by the Department of Surgery Research Committee and I am pleased to inform you that the committee approved the study.

Please use the above project number in all future correspondence.

Yours sincerely

PROFESSOR ANWAR S MALL
CHAIRMAN: RESEARCH COMMITTEE

"OUR MISSION is to be an outstanding teaching and research university, educating for life and addressing the challenges facing our society."

Ethics Committee Approval Letter

UNIVERSITY OF CAPE TOWN

Faculty of Health Sciences
Human Research Ethics Committee
Room E52-24 Groote Schuur Hospital Old Main Building
Observatory 7925
Telephone [021] 406 6626 • Facsimile [021] 406 6411
e-mail: shuretta.thomas@uct.ac.za

11 April 2011 **Sent via Internal mail & Email**

HREC REF: 191/2011

Dr N MJOLI,
Neurosurgery
Department of Surgery
OMB

Dear Dr MJOLI,

PROJECT TITLE: CEREBRAL ARTERIOVENOUS MALFORMATION PRESENTING WITH HAEMORRHAGE.

Thank you for submitting your new study to the Faculty of Health Sciences Human Research Ethics Committee

It is a pleasure to inform you that the Ethics Committee has formally approved the above-mentioned study.

Approval is granted until 15 April 2011

Please submit an annual progress report (FHS016) if the research continues beyond the expiry date. Please submit a brief summary of findings if you complete the study within the approval period so that we can close our file.

Please note that the ongoing ethical conduct of the study remains the responsibility of the principal investigator.

Please quote the HREC. REF in all your correspondence.

Yours sincerely

pp **A/PROF MARC BLOCKMAN**
CHAIRPERSON, FHS HUMAN ETHICS

Federal Wide Assurance Number: FWA00001637.
Institutional Review Board (IRB) number: IRB00001938

This serves to confirm that the University of Cape Town Human Research Ethics Committee complies to the Ethics Standards for Clinical Research with a new drug in patients, based on the Medical Research Council (MRC-SA), Food and Drug Administration (FDA-USA), International Convention on Harmonisation Good Clinical Practice (ICH GCP) and Declaration of Helsinki guidelines.

The Human Research Ethics Committee granting this approval is in compliance with the ICH Harmonised Tripartite Guidelines E6: Note for Guidance on Good Clinical Practice (CPMP/ICH/135/95) and FDA Code Federal Regulation Part 50, 56 and 312.

INSTRUCTIONS FOR AUTHORS First formulated in November, 1995 • Revised on January 26, 2010

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   Part of book if number of authors is more than two:
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Electronic Data Sheets

All AVM Clinical Table

Sex	Age	DOA	Presentation	Site	Headache	Vomiting	Seizure	Neuro Deficit	GCS	Symptom Duration	Surgery	Radiotherapy
M	33	24 Apr 2007	Headaches	Left Temporal								
		04 Nov 2004	Proptosis	Rt Frontal AVM								
M	59	04 Jan 2006	SAH	R Parietal	Yes	Yes	No	No	13	0	No	
M		19 Feb 2006	Rebleed		Yes	Yes	No		14		No	No
F	66	12 Oct 2010	Seizures	L Post Temporal								
M	26	09 Oct 2007	Seizures	Right Frontal 1cm								
F	45	05 Sep 2006	Seizures	Left Posterior								

Sex	Age	DOA	Presentation	Site	Headache	Vomiting	Seizure	Neuro Deficit	GCS	Symptom Duration	Surgery	Radiotherapy
				Temporal								
M		20 Jul 2010	Seizures And Transient Hemianopia	Lt Occipital								
F	60	10 Jun 2008	Haemorrhage, 20 Yrs Ago	Left Temporal 1cm								
M	42	08 Dec 2005	Seizures	Left Temporal 2								
	42	31 Oct 2005	Headache	Superior Vermis								
	14	03 Apr 2005	Rt Hemiparesis And Sensory Loss, Dysphasia	Rt Parietal Interhemispheric								

Sex	Age	DOA	Presentation	Site	Headache	Vomiting	Seizure	Neuro Deficit	GCS	Symptom Duration	Surgery	Radiotherapy
M	55	02 Mar 2010	Seizures	Right Sylvian Fissure 1								
F	11	06 Sep 2006	SAH	Pontomedullary Fissure	Yes	No	No	No	15	6	No	No
	58	11 May 2009	Hemianopia	Right Occipital								
	51	21 Aug 2007	Seizures	Capsula Externa 3								
M	17	27 Aug 2005	Hematoma	Right Frontal	Yes	No	No	Dysphasia	1	O	Craniotomy For ICH And AVM Excision	No
M	57	17 Feb 2009	Rebleed 20 Yrs Post Sx	Right Frontal	No	Yes	Yes	Left Hemiparesis	14	1	No	No

Sex	Age	DOA	Presentation	Site	Headache	Vomiting	Seizure	Neuro Deficit	GCS	Symptom Duration	Surgery	Radiotherapy
M	19	17 Jan 2006	Haemorrhage	Right Parietal 6								
M	63	09 Aug 2005	Haemorrhage	Vermian Post Fossa	Yes	No	No		10t	1	EVD And ETV, Tracheostomy	No
M	30	30 Nov 2010	Seizures	L Medial Temporal 3								
F		30 Jun 2008	Hemianopia	Left Occipito Parietal 8cm								
F	25	15 Feb 2010	Blackout	Right Motor Strip 4								
M	34	08 Nov 2008	Haemorrhage	Right Post Fossa AVM	Yes	Yes	No	0	14	2	No	No

Sex	Age	DOA	Presentation	Site	Headache	Vomiting	Seizure	Neuro Deficit	GCS	Symptom Duration	Surgery	Radiotherapy
F	34		Seizures	Right Frontal 3								
M	43	15 Jun 2010	ICH	Lenticulo Striate Vessels	No	No	No	Right Hemiparesis	9a	0	No	No
F		16 Jul 2007	Headache And Dizziness	Left Sylvian / Temporal AVM 2-3								
	50	06 Apr 2010	Haemorrhage	Left Parietal 6cm								
M	28	12 Feb 2006	Seizures	Left Parietal Interhemispheric 6 Mm								
			Hemiparesis	Right Proliferative Hemisphere								
M	42	02 Aug	Headache	R Parieto Occipital								

Sex	Age	DOA	Presentation	Site	Headache	Vomiting	Seizure	Neuro Deficit	GCS	Symptom Duration	Surgery	Radiotherapy
		2005										
M	17	19 Jan 2009	Seizures	Temporal Tip 3								
F	25	20 Jun 2006	Post Radiosurgery	Calcarine								
F	42	29 May 2006	Follow Up	Right Occipito Temporal								
M	55	09 Feb 2010	Haemorrhage	R Parietal								
F	47	22 Feb 2005	Headache	Right Parieto Occipital								
M	47	18 Oct 2007	Haemorrhage	Left Occipital 3	No	No	Yes	Right Heminomous Hemianopia	13	1	1	No

Sex	Age	DOA	Presentation	Site	Headache	Vomiting	Seizure	Neuro Deficit	GCS	Symptom Duration	Surgery	Radiotherapy
F	25	14 Mar 2007	Haemorrhage	Right Centrum Semiovale 2	No	No	Yes	Pupils Unreactive	4t	0	EVD	No
M	12	11 Aug 2007	Haemorrhage	Left Frontal 3	Yes	No	No	Right Hemiparesis	10	21	No	No
F	45	13 Mar 2008	Haemorrhage	Right Cerebellar 2	Yes	No	No	Right Hemiparesis And Right Cerebellar Signs	15	3	No	No
	26	20 Mar 2005	Seizures	L Parietal								
M	40	29 Sep 2008	Seizures	Right Parietal 2								
F	19	21 May	Visual Problems	Right Occipital								

Sex	Age	DOA	Presentation	Site	Headache	Vomiting	Seizure	Neuro Deficit	GCS	Symptom Duration	Surgery	Radiotherapy
		2007										
F		11 Jul 2005	Headache And Bruit	L Parieto Occipital								
M	46	13 Mar 2007	Haemorrhage	Right Occipital	No	No	No	Left Hemiparesis	9t	0	No	No
M	26	08 Dec 2008	Haemorrhage Prior To Admission	Left Subcortical Parietal 1.5cm	No	No	No	0	15	6 Month	No	No
M	22	16 Nov 2005	Haemorrhage	Left Temporal 3	Yes	No	Yes	Memory Loss, Expressive Dysphasia	15	0	No	No
F	22	17 Jun 2009	IVH	Left Choroidal AVM	No	No	No	Right Hemiparesis	13	2	No	No
F	23	15	Seizures	R Parieto								

Sex	Age	DOA	Presentation	Site	Headache	Vomiting	Seizure	Neuro Deficit	GCS	Symptom Duration	Surgery	Radiotherapy
		Nov 2005		Temporal								
F	56	03 Oct 2006	Hemifacial Spasm	Right CP Angle								
F	41	21 Apr 2005	Seizures	Left Inferior Temporal								
M	30	18 May 2008	Haemorrhage	Right Choroidal	Yes	No	No	Left Hemiplegia And Aphasia	14	O	No	No
M			Athetoid Movement	Left Thalamic								
M	24	14 May 2008	Haemorrhage	Right Choroidal	No	No	No	0	9t		No	No
M	26	21 Apr 2008	Haemorrhage	Left Choroidal AVM	No	No	Yes	Right Hemiplegia	13		No	No

Sex	Age	DOA	Presentation	Site	Headache	Vomiting	Seizure	Neuro Deficit	GCS	Symptom Duration	Surgery	Radiotherapy
	54	10 Oct 2006	Haemorrhage 1984, Seizure And Headache	Left Sylvian / Temporal AVM								
M	28	10 Aug 2007	Haemorrhage	Right Frontal 4	Yes	Yes	No	Left Hemiparesis	14	0	No	No
M	38	2005-0724	Haemorrhage	Left Thalamic	No	No	No	Memory Loss And Visual Impairment	14	15	No	No
M	19	28 Apr 2007	Haemorrhage	Right ICH And IVH	No	No	Yes	Right Facial Weakness And Left Hemiparesis	12	1	EVD	No
M	51	10 Oct 2005	Haemorrhage	Right Frontal	Yes	No	No	0	14	7	No	No
M	29	24 Jun	Seizures	L Frontal 4								

Sex	Age	DOA	Presentation	Site	Headache	Vomiting	Seizure	Neuro Deficit	GCS	Symptom Duration	Surgery	Radiotherapy
		2006										
F	51	21 Apr 2005	Seizures And Headaches	Left Frontal								
M	24	16 Jun 2006	Post Fossa Bleed	L Cerebellar AVM	Yes	Yes	Yes	Cerebellar Sings	15	0	EVD	No
M	52	09 Mar 2010	Headache	R Parietal 2								
M	15	24 Oct 2007	Haemorrhage Into Ventricles	Right Temporal 3	Yes	No	No	0	15	20	No	No
F	45	06 Aug 2009	Seizures	Right MCA 4								
M	27	29 May 2010	Haemorrhage	L Parietal	Yes	No	No	0	15	1	No	No

Sex	Age	DOA	Presentation	Site	Headache	Vomiting	Seizure	Neuro Deficit	GCS	Symptom Duration	Surgery	Radiotherapy
F	50	08 May 2007	Haemorrhage	Right Temporal Tip	Yes	Yes	No	0	14	2	No	No
F	50	10 Jul 2008	IVH		Yes	Yes	Yes	Right UL Monoparesis	14	0	No	No
M	28		Seizure & Headaches	Right Occipital								
M			No Haemorrhage	L Aica								
F	54	24 Apr 2009	Haemorrhage	Right Frontal	No	No	No	Left Hemiparesis	14		Craniotomy For ICH And AVM Excision	No
F	35	18	Seizures	Right								

Sex	Age	DOA	Presentation	Site	Headache	Vomiting	Seizure	Neuro Deficit	GCS	Symptom Duration	Surgery	Radiotherapy
		May 2009		Temporal 4								
F	29	27 Mar 2006	Headache And Seizures	Left Occipital 4								
M	63		Cerebellar Signs Unbled	Right Cerebellar 2								
F	31	05 Jun 2007	Incidental	Left Parietal 2								
M	34	10 Aug 2006	SAH And IVH	Velum Interpositum Choroidal & Left 5mm PCOM	Yes	Yes	Right Focal Seizures	Neck Stiffness	14	1	No	No
F	41	10 May 2005	Right Homonymous Hemia0pia	Left Occipital								

Sex	Age	DOA	Presentation	Site	Headache	Vomiting	Seizure	Neuro Deficit	GCS	Symptom Duration	Surgery	Radiotherapy
M	39	28 May 2007	Seizures	Left Occipital 4								
F		27 Aug 2009	Hemia0pia	Left occipital Temporal								
F	40	19 Jan 2010	Incidental	Left Motor Cortex 1								
M	41	02 Dec 2010	Haemorrhage	Pica Aneurysm	Yes	Yes	No	0	15	7	No	No
M	38	06 Aug 2007	Haemorrhage	Right Cerebellar 3	No	No	No	Left Hemiparesis	15	5 Month	No	No
F		30 May 2007	Seizures	Right Temporal 3								
F	61	16 Jan	Haemorrhag	Right	Yes	Yes	No	0	14	3	No	No

Sex	Age	DOA	Presentation	Site	Headache	Vomiting	Seizure	Neuro Deficit	GCS	Symptom Duration	Surgery	Radiotherapy
		2007	e	Occipital 7 Cm								
M	19	18 Sep 2007	Seizures	Right Temporo-frontal 4								
M	40	20 Nov 2006	Haemorrhage And Left Hemi	Right Basal Ganglia								
M	26	22 May 2006	Haemorrhage	Left Calcarine 2	Yes	Yes	No	Right Hemianopia	15	7	No	1
M	20	16 Nov 2005	Haemorrhage	Post Fossa Vermis 4	Yes	No		0	15	0	No	No
M	39	18 Jun 2009	Haemorrhage	Right Parietal 3								
M	51	29 Mar	Seizures And Bleed	L Frontal	No	No	Yes	Left Upper Limb		0	No	No

Sex	Age	DOA	Presentation	Site	Headache	Vomiting	Seizure	Neuro Deficit	GCS	Symptom Duration	Surgery	Radiotherapy
		2010						Monoparesis				
M	22	30 Dec 2007	Post Radiosurgery	Left Parietal 3								
F	35	13 Sep 2005	Interhemispheric Haemorrhage	Left Frontal	Yes	Yes	No	0	15	3	No	No
M	29	17 May 2007	Proptosis, Papilledema And Tinnitus	Left Cerebellar	Yes5							
M	20	15 Nov 2007	Haemorrhage A Few Months Before	Right Sylvian	Yes	Yes	No	Left Hemiparesis	15	2 Month	No	No
M	42	04 Dec	Incidental	Right Frontal 4								

Sex	Age	DOA	Presentation	Site	Headache	Vomiting	Seizure	Neuro Deficit	GCS	Symptom Duration	Surgery	Radiotherapy
		2006										
F	62	24 Apr 2005	Haemorrhage	Right Frontal 2	Yes	Yes		0	15	1	1	No
F	59	30 Nov 2010	Headache	L Medial Occipital 5cm								
M	15	18 Aug 2009	Seizure	Right Frontal Interhemispheric								
M	35	02 Feb 2009	Haemorrhage	Right MCA	Yes	No	Yes	0	15	0	No	No
F	51	27 Feb 2007	Dysphagia	Left Motor Cortex 3								

Bled AVM Angio Table

Date	Site	Size of nidus	Fistula supply	Choroid plexus supply	Nidus false aneurysm	Bleeding point	Flow related aneurysm	Associated aneurysm	Vein number	Vein names	Deep venous drainage	Venous pouch	Venous stasis	Intervention	Bleeding site treated	AVM cured	Angio complication
	Rt Parietal	6	Yes	No	No	No	No	No		Multiple	No		No	No			No
	Rt Parietal	6	Yes	No	Yes	Yes	No	No		Multiple	No		No	Glue	Yes		No
04 Jan 2006	Rt Parietal	6	Yes	No	No	Yes	Yes	No	> 3	Multiple	No	No	No	Glue	No		No
14 Sep 2006	Left Pontomedullary Fissure	1	No	No	Yes	Yes	No	No	1	Vermian vein	Yes	No	No	Glue	Yes		No
27 A	Rt Frontal	1	No	No	No	No	No	No	2	Cortical veins	No	No	No	Glue	No		No

Date	Site	Size of nidus	Fistula supply	Choroid plexus supply	Nidus false aneurysm	Bleeding point	Flow related aneurysm	Associated aneurysm	Vein number	Vein names	Deep venous drainage	Venous pouch	Venous stasis	Intervention	Bleeding site treated	AVM cured	Angio complication
ug 2005																	
23 Mar 2009	Rt Frontal	1	No	No	No	No	No	No	No venous exit		No		No	No blush seen	No		
15 Aug 2005	Cerebellar Vermis	3	No	No	False Aneurysm end of SCA	Yes	No	No	2	Vermian veins	Yes	No	No	Glue	Yes		
10 Nov	Rt Cerebellar	4	No	No	Yes	Yes	No	No	Multiple	Superficial cerebell	No	No	No	Angio	Yes	No	No

Date	Site	Size of nidus	Fistula supply	Choroid plexus supply	Nidus false aneurysm	Bleeding point	Flow related aneurysm	Associated aneurysm	Vein number	Vein names	Deep venous drainage	Venous pouch	Venous stasis	Intervention	Bleeding site treated	AVM cured	Angio complication
2008	Hemisphere									ar							
17 Jun 2010	Left External Capsule	3	No	No	Yes	Yes	No	No	1	Sylvian vein	No	Yes	No	Glue	Yes	No	No
06 Apr 2010	L Parietal	6	MCA	No	No	Venous Pouch	No	No	1	Trolard into	No	Yes	No	Glue	Yes	No	No
55	R Parietal	2	MCA	No	No	No	No	No	2		No	No	No	No	No	No	No
06 Mar	Frontal AVM	1	No	No	Yes	Yes	No	No	2	Cortical veins	No	No	No	Glue	Yes		No

Date	Site	Size of nidus	Fistula supply	Choroid plexus supply	Nidus false aneurysm	Bleeding point	Flow related aneurysm	Associated aneurysm	Vein number	Vein names	Deep venous drainage	Venous pouch	Venous stasis	Intervention	Bleeding site treated	AVM cured	Angio complication
2006																	
26 Jan 2009	Left Occipital	3	No	No	No	No	No	No	Multiple		No	No	Yes	Glue and surgery	No	Yes	No
17 Aug 2007	Centrum Semiovale	1	No	No	No	No	No	No	1	Transcerebral vein	Yes	No	No	Glue	No	No	No
11 Feb 2008	Left Frontal Cortical	2	No	No	No	No	No	No	Two	Cortical veins	No	No	Yes	Glue	No	Yes	No

Date	Site	Size of nidus	Fistula supply	Choroid plexus supply	Nidus false aneurysm	Bleeding point	Flow related aneurysm	Associated aneurysm	Vein number	Vein names	Deep venous drainage	Venous pouch	Venous stasis	Intervention	Bleeding site treated	AVM cured	Angio complication
		1	Yes	No	Yes	No	No	COIL	1		No	Yes					
27 Mar 2008	Vermis AVM	2	No	No	No	No	No	No	Multiple into lat sinus	Transverse sinus	No	No	No	Glue	No	Yes	No
21 Mar 2007	Rt Occipital	4	Yes	No	No	Right P1	Yes	No	2	Cortical	No	No	No	Glue	No	No	No
08 Dec 2008	Left Parietal	1.5	No	No	No	No	No	No	Single	Sylvian vein	No	No	No	Glue	No	No	No
28	Left	3	No	Anter	Yes	Yes		No	Single	Superfi	No	No	No	Glue	Yes		No

Date	Site	Size of nidus	Fistula supply	Choroid plexus supply	Nidus false aneurysm	Bleeding point	Flow related aneurysm	Associated aneurysm	Vein number	Vein names	Deep venous drainage	Venous pouch	Venous stasis	Intervention	Bleeding site treated	AVM cured	Angio complication
Nov 2005	Temporal			ior Choroidal						cial Sylvian							
19 Jun 2009	Left Intraventricular Choroidal	3	No	Ant & Post Lat Choroidal	No	No	No	No	1	Internal cerebral vein	Yes	No	No	Glue	No	No	No
26 May 2008	Rt Choroidal	6	Yes	Posterior Choroidal	No	No	BASILAR TIP	No	Multiple		Yes	No	No	Glue	No	No	No
18 A	Corpus Callosum	5	Yes	Yes	Yes	Yes	No	No	2		Yes	No	No	Onyx	Yes	No	No

Date	Site	Size of nidus	Fistula supply	Choroid plexus supply	Nidus false aneurysm	Bleeding point	Flow related aneurysm	Associated aneurysm	Vein number	Vein names	Deep venous drainage	Venous pouch	Venous stasis	Intervention	Bleeding site treated	AVM cured	Angio complication
ug 2008																	
12 May 2008	Left Choroidal	4	No	Anterior Choroidal	No	No	No	No	1	Internal cerebral vein	Yes	No	No	Glue	Failed	No	No
16 Aug 2007	Rt Frontal	5	No	No	No	No	No	No	3	Deep & sup	Yes	No	No	Glue	No	No	No
28 Jul 20	Left Choroid Plexus	1	No	Lateral Post Chor	No	No	No	No	Single	0	Yes	No	No	Glue	No	Yes	No

Date	Site	Size of nidus	Fistula supply	Choroid plexus supply	Nidus false aneurysm	Bleeding point	Flow related aneurysm	Associated aneurysm	Vein number	Vein names	Deep venous drainage	Venous pouch	Venous stasis	Intervention	Bleeding site treated	AVM cured	Angio complication
05				oidal													
22 Sep 2008	Rt Frontal	1	No	No	No	No	No	No	1	Cortical veins	No	No	No	Glue	No		No
10 Oct 2005	Rt Frontal	5	Yes	No	No	Yes	ACOM	No	3	Cortical	No	Yes	No	Glue	Yes	No	Thrombo embolic occlusion on left mca branch dysphasia
24	Left CP	2	No	No	No	No	No	No	1	Cortica	No	No	No	Glue	No		Facial

Date	Site	Size of nidus	Fistula supply	Choroid plexus supply	Nidus false aneurysm	Bleeding point	Flow related aneurysm	Associated aneurysm	Vein number	Vein names	Deep venous drainage	Venous pouch	Venous stasis	Intervention	Bleeding site treated	AVM cured	Angio complication
Oct 2006	Angle									l veins							Palsy - Aica Occlusion
12 Nov 2007	Rt Medial Post Temporal	4	No	Yes	No	No	No	No	Multiple		Yes	No	Yes	Glue	No	No	
07 Jun 2010	Left Parietal	########	No	No	Yes	Yes	No	No	MULTIPLE	0	Yes	No	No	Glue	Yes	No	No
21 May	Rt Temporal Tip	1	No	No	No	No	No	No	1	Basal vein of Rosent	Yes	No	No	Glue	No	No	No

Date	Site	Size of nidus	Fistula supply	Choroid plexus supply	Nidus false aneurysm	Bleeding point	Flow related aneurysm	Associated aneurysm	Vein number	Vein names	Deep venous drainage	Venous pouch	Venous stasis	Intervention	Bleeding site treated	AVM cured	Angio complication
2007										hal							
10 Jul 2008	Rt Temporal Tip	1	No	No	No	No	No	No	1	Basal vein of Rosenthal	Yes	No	No	No	No	No	No
17 May 2009	Rt Frontal	6	No	No	No	No	No	No	1	Cortical veins	No	No	No	Surgery	No		No
	Post Corpus Callosum Avm	4	No	Yes	No	Yes	No	Yes	1	Internal cerebral vein	Yes	No	No	Coil and glue	Yes		No
02	4th	1	No	Yes	Yes	Yes	No	Yes	1	Superfi	No	No	No	Coil	Yes		No

Date	Site	Size of nidus	Fistula supply	Choroid plexus supply	Nidus false aneurysm	Bleeding point	Flow related aneurysm	Associated aneurysm	Vein number	Vein names	Deep venous drainage	Venous pouch	Venous stasis	Intervention	Bleeding site treated	AVM cured	Angio complication
Dec 2010	Ventricle									cial cerebellar							
11 Feb 2008	Rt Cerebellar	2	No	No	No	No	No	No	1	Vermian vein	Yes	No	No	Glue	No	No	No
19 Jan 2007	Rt Parietal	6	Yes	No	No	Yes	P Yes	No	Multiple		No	No	No	Glue	Coiled Aneurysm	No	No
20 Nov	Rt Basal Ganglia	4	No	Yes	No	No	No	No	1	Internal cerebral vein	Yes	Yes	No	Glue	No	No	No

Date	Site	Size of nidus	Fistula supply	Choroid plexus supply	Nidus false aneurysm	Bleeding point	Flow related aneurysm	Associated aneurysm	Vein number	Vein names	Deep venous drainage	Venous pouch	Venous stasis	Intervention	Bleeding site treated	AVM cured	Angio complication
2006	And Choroid Plexus																
29 May 2006	Left Calcarine	1	No	No	Yes	Yes	No	No	1	Cortical	No	No	No	Glue	Yes	No	No
04 Apr 2006	Cerebellar Vermis	3	No	No	No	No	No	No	2	Vermian veins	Yes	No	No	Glue	No		No
	Rt Parietal	3	No	No	No	No	No	No	2		No	No	No	Glue	No	No	No
16 Se	Left Frontal	5	No	No	No	Yes	Yes	No	1	Superficial	No	Yes	No	Glue	Yes		Reflux No

Date	Site	Size of nidus	Fistula supply	Choroid plexus supply	Nidus false aneurysm	Bleeding point	Flow related aneurysm	Associated aneurysm	Vein number	Vein names	Deep venous drainage	Venous pouch	Venous stasis	Intervention	Bleeding site treated	AVM cured	Angio complication
p 2005										through sagittal sinus							Clin Consequence
29 Dec 2008	Rt Sylvian	2	No	No	No	No	No	No	1	Superficial Sylvian	No	No	No	Glue	No	Yes	No
05 Feb 2009	Rt Frontal	1	No	No	Yes	Yes	No	No	Single	Cortical vein	No	No	No	Glue	Yes	Yes	No

Bled AVM CT Brain

Site Of Haemorrhage	Midline Shift	Swelling	Basal Cisterns	Hydrocephalus
Interhemispheric SAH	Yes	Yes	Patent	No
Right IVH	Yes	Yes	Patent	Yes
Right Parieto Occipital	No	Yes	Patent	No
Crural Cistern SAH	No	Yes	Patent	Yes
Right Frontal	Yes	Yes	Effaced	No
Right Thalamus With IVH	Yes	No	Patent	No
Left Cerebellar And Vermis	No	Yes	Effaced	Yes
Right CP Angle	No	Yes	Patent	Yes
Left Deep Frontal	Yes	Yes	Patent	No
Left Parietal	No	Yes	Patent	No
Right Parietal	No	Yes	Patent	No
Left Occipital ICH & L F-T-P ASDH, IVH	Yes	Yes	Effaced	No
IVH	No	Yes	Effaced	Yes
Left Fronto Parietal With IVH	No	No	Patent	No
Right Cerebellar	No	No	Patent	No
SAH Midbrain Into Left Cerebral Peduncle	No	No	Patent	No

Site Of Haemorrhage	Midline Shift	Swelling	Basal Cisterns	Hydrocephalus
Left Parietal With IVH	Yes	Yes	Effaced	No
Left Temporal Lobe	Yes	Yes	Effaced	No
Left IVH	No	Yes	No	No
Right Basal Ganglia	No	No	Patent	No
IVH	No	Yes	Effaced	Yes
Left Thalamus And IVH	No	Yes	Patent	Yes
Right Frontal With IVH	Yes	Yes	Patent	Yes
Left Lateral Ventricle Haemorrhage	No	Yes	Effaced	Yes
Right Frontal With IVH	Yes	Yes	Effaced	Yes
Interhemispheric SAH	No	Yes	Patent	No
Left Cerebellar Hemisphere	No	Yes	Effaced	No
Right Occipital Horn	No	No	Patent	No
Left Post Temporo Parietal	No	No	Patent	No
Right Medial Temporal Lobe	No	Yes	Patent	No
IVH	No	Yes	Patent	No
Right Frontal Lobar	Yes	Yes	Patent	No
Prepontine	No	No	Patent	No
Right Ambient Cistern SAH	No	No	Patent	No

Site Of Haemorrhage	Midline Shift	Swelling	Basal Cisterns	Hydrocephalus
Prepontine SAH	No	Yes	Patent	Yes
Left Occipital	No	No	Patent	No
Left Cerebellar Vermin	No	No	Effaced	No
Right Basal Ganglia	No	No	No	No
Right Parietal	No	No	Patent	No
Left Frontal	Yes	Yes	Effaced	No
SAH Right Sylvian Fissure	No	No	Patent	No
Right Frontal	Yes	Yes	Effaced	No
Right Frontal	No	Yes	No	No

Follow up bleed AVM

	Date	Proteins	Rebleed	Headache	Seizure	Vomiting	Neuro Deficit	GCS	Intervention	AVM Cured
06 Jan 2006	15 Dec 2010	59 M	2	No	Yes	No	Left Hemiparesis	15	Rebleed 2006 And 2010	2010 False Nidal Aneurysm Closed With Glue
06 Sep 2006	10 Jun 2007	9 M		No	No	No	No	15	Angio	1
27 Aug 2005	19 Jul 2006	11 M		No	No	No	Personality Changes	15	0	1
17 Feb 2009	22 Mar 2009	1 M	0	No	No	No	Left Hemiparesis	15	0	0
09 Aug 2005	17 Mar 2011	67 M	0	No	No	No	Right Hemiparesis Resolved	15	0	
08 Nov 2008	12 Jun 2010	17 M	0	Yes	No	No	No	15	1	0
18 Oct 2007	23 Mar 2011	41	0	No	No	No	No	15	Nil	0
14 Mar 2007	13 Dec	45 M	0	No	No	No	No			

	Date	Proteins	Reblee d	Headache	Seizure	Vomiting	Neuro Deficit	GCS	Intervention	AVM Cured
	2010									
13 Mar 2007	2011	45 M	0	No	No	No	Hemiparesis	15	Glue	0
16 Nov 2005	30 Jan 2006	2 M		No	No	No	No	15	0	Small Residual
18 May 2008	14 Mar 2011	34 M	0	No	No	No	Hemiparesis	15	0	Tel
21 Apr 2008	17 Feb 2010	23 M	0	Yes	No	No	Left Hemiparesis		0	0
24 Jul 2005	03 Nov 2005	4 M	0	No	No	No	Memory And Visual Impairment	15	Glue	
28 Apr 2007	21 Sep 2008	17 M	0	Yes	Yes	No	No	15		1
10 Oct 2005	25 Jan 2011	51 M	0	No	No	No	Dysphasia	15	Embo X3	0
24 Oct 2007	14 Mar	41 M	0	No	No	No	No	15	Tel	

	Date	Proteins	Reblee d	Headache	Seizure	Vomiting	Neuro Deficit	GCS	Intervention	AVM Cured
	2011									
29 May 2010	17 Mar 2011	10 M	0	Yes	No	No	No	15		
	10 Jul 2008	14 M	1	Yes	Yes	No	Left Arm Monoparesis	14	Await Surgery	0
24 Apr 2009	15 Nov 2010	19 M	0	Yes	Yes	No	Left Hemiparesis	15	0	1
16 Jan 2007	2010	36 M	0	No	No		No	15		0
20 Nov 2006	14 Mar 2011	43 M	0	No	No	No	No	15	For Radiotherapy	0
22 May 2006	2010	48 M	0	No	No	No	No	15	Radiotherapy	1
16 Nov 2005	17 Mar 2011	65 M		No	No	No	No	15	Angio	
13 Sep 2005	17 Mar	66 M		Yes	No	No	No	15	2nd	1

	Date	Proteins	Rebleed	Headache	Seizure	Vomiting	Neuro Deficit	GCS	Intervention	AVM Cured
	2011									
24 Apr 2005	07 Mar 2006			No	No	No	Hearing Loss Right Ear	15	1	1
02 Feb 2009	14 Mar 2011	25 M	0	No	No	No	No	15	0	