

Case Series of Ruptured Jamaican Berry Aneurysms Four Decades Ago

MON Ibe

Consultant Neurosurgeon
Central Medicare
43 Okigwe Road
Imo State

All Correspondence:
Dr MON Ibe
Central Medicare
43 Okigwe Road Owerri
Imo State, Nigeria

E-mail:
Snowwhyte_hosp@yahoo.com

Abstract

Objective

From our personal experiences, it is our wish to examine the incidence of ruptured intracranial aneurysms about four decades ago in Jamaica, the West Indies, with regards to postoperative deterioration of neurological/psychological functions.

Materials and Method

The author, while working in Jamaica, the West Indies, from 1975 to 1977 (3 years), operated on 6 cases of ruptured intracranial aneurysms of 5 females and 1 male. The source of these data is from the author's surgery records.

Result

There were 4 aneurysms from the posterior communicating artery, and 1 each from the anterior communicating artery, and the middle cerebral artery. Their ages ranged from 31 to 64 years. They presented with diverse neurological disturbances. Their aneurysms, which were diagnosed through contrast carotid angiography, were all clipped through fronto-lateral craniotomy under general anaesthesia. The procedures were well tolerated by the patients. There were no complications and no gross additional neurological deficits postoperatively. The wounds had healed by primary intention. The patients were discharged home 12 days postoperatively, the stitches having been removed 2 days earlier. Follow-up checks started about 7 days after discharge. The neurological deficits had cleared about 3 months later.

Conclusion

Having bled, the aneurysms exposed themselves by causing sudden severe headaches, and various neurological problems, depending on their locations. We found more females than males. The posterior communicating artery aneurysm was predominant. There were no cerebro-vascular spasms postoperatively.

Greek --- ANEURYSMA (Ana= up or across, eurus = wide or broad)

Key Words: Subarachnoid Haemorrhage, Cerebro-Vascular Spasms.

Introduction

Intracranial aneurysms arise from defects in the media of the arterial wall, especially, at the branch points of large arteries. It is accepted that, because of the media defect, there is a weakness in the vessel wall, from which, as the years go by, through pulsation and blood pressure, an aneurysm develops¹. They could be as small as the head of a pin and up to the size of a cherry. They could start bleeding at any age. They are classified into saccular, berry or congenital aneurysms²; dolichoectatic, fusiform, or arteriosclerotic aneurysms of proximal arteries; infectious or mycotic aneurysms, situated peripherally. We also have neoplastic aneurysms and post-traumatic aneurysms. Microaneurysms do occur in small perforating vessels, as a result of hypertension. Cigarette smoking, illicit drug use, alcohol intake³, pregnancy⁴ may induce aneurysmal formation^{5,6}

Aneurysms, especially the berry type, frequently

rupture spontaneously into the subarachnoid space, causing subarachnoid haemorrhage. Rupture could also result in intraparenchymal, intraventricular or subdural haemorrhage. Giant aneurysms, usually larger than 25 mm in diameter, could also produce mass effects and distal thrombo-embolism.

Aneurysms could be single or multiple. Some rupture, others do not. The overall incidence of ruptured intracranial aneurysms has been estimated at 10.5 per 100,000 Individuals¹.

Symptoms, amongst others, of subarachnoid haemorrhage, are usually sudden severe headaches, alterations in the level of consciousness, meningeal irritation, autonomic disturbances and focal neurological deficits. Severe subarachnoid haemorrhage from ruptured intracranial aneurysms is catastrophic, leading either to death or permanent disabilities, in most cases. Survivors often have neuropsychological dysfunction. About 10% of cases die before reaching medical attention; about 25% die

within 24 hours and 40 to 49% die within 3 months¹. Diagnosis is usually confirmed with the assistance of Computerized Tomography Angiography Scan(CTAS)⁷⁻⁹, Magnetic Resonance Angiography Imaging (MRAI)⁹ and, very usefully, conventional 4-vessel angiography.

The treatment of ruptured intracranial aneurysms is usually conservative (aimed at vasospasm), surgical¹¹, and/or use of endovascular techniques with platinum coils, etc.¹²⁻¹⁷, depending on the condition of the patient¹⁸ and the environment. Because of the risk of a rebleed, surgical or endovascular treatment is recommended. Complications after rupture include cerebral vasospasm¹⁹, recurrent haemorrhage^{19,20}, seizures, hydrocephalus, venous thrombo-embolism, neurological deficits, psychological disturbances, infections, etc; while, after clipping, there could be slipped clips as well.

Prognosis is usually dependent on age, neurological status, aneurysm location, how long between onset of symptoms and intervention, presence of vasospasm, presence and extent of intraparenchymal or intraventricular haemorrhage, the presence of hypertension and other diseases.

Patients and Method

From 1975 to 2011, the author operated on 6 (six) cases of ruptured intracranial aneurysms. All six were in Jamaica from 1975 to 1977. There were 5 females and 1 male (Table 1). Their ages ranged from 31 to 64 years (Table 1). They were all admitted on same day of rupture through the Accident and Emergency department. All patients had had sudden severe excruciating headaches, as if struck by lightning, brief loss of consciousness, neck-stiffness, photophobia, nausea and vomiting and blurred vision. The 4 patients with posterior communicating artery aneurysms had nervus oculomotorius pareses in addition. The patient with the right middle cerebral artery aneurysm had a slight left hemiparesis also (Table 2).

Past medical histories revealed no abnormalities. Routine laboratory investigations were carried out, as well as plain skull radiographs, which were within normal limits. Conventional carotid angiography by direct common carotid artery puncture under routine general anaesthesia, a day after admission, clearly demonstrated the aneurysms (Pictures 1,2,6) without evidence of vascular spasms. MRI was not carried out since the facilities were not available. Preoperative treatment was basically bed rest and routine analgesics. Operations were carried out about 7 days later on our routine operation days, under general anaesthesia, during which the aneurysms were clipped, through a fronto-lateral osteoplastic

craniotomy in each case. There was a slight yellowish discoloration of the brain around the middle cerebral artery aneurysm, due to the haemorrhage.

Stitches were removed on the tenth day postoperatively. The patients were sent home 2 days later, and referred to the out-patient's department for follow-up checks 7 days after discharge.

There were no particular measures or other treatment given postoperatively, apart from anticonvulsant prophylaxis with phenobarbitone sodium 30 mg twice daily for 2 years.

Results

Out of the 6 diagnosed cases, 4 aneurysms were located in the posterior communicating/internal carotid junction (all females), 2 on each side (Picture 1) and 1 each from the anterior communicating artery (female) (Picture 2) and the middle cerebral artery (male) (Table 3). The predominant age range was 40-50 years, and all posterior communicating artery aneurysms were in females.

Table 1: Ruptured Intracranial Aneurysms.

Age Ranges	Males	Females	Total
30 – 40	1	1	2
40 – 50	----	3	3
50 – 60	----	----	----
60 – 70	----	1	1
Total	1	5	6

Table 2:

Clinical Presentation	Ant. Comm. Artery	Middle Cerebral Art.	Post. Comm. Artery
Headaches	all	all	all
Brief loss of Consciousness	all	all	all
Neckstiffness	all	all	all
Photophobia	all	all	all
Nausea	all	all	all
Vomiting	all	all	all
Blurred Vision	all	all	all
Oculomotor Paresis	None	none	all
Hemiparesis	None	all	none

The patients tolerated the procedures very well and recovered from the anaesthesia without any additional neurological deficits. The wounds had healed by primary (1st) intention. Repeat contrast radiographs, postoperatively (Pictures 3-5), showed the clips in situ, without any aneurysms. All their

complaints and symptoms had completely disappeared and they were fully mobilized and integrated back into the society about 3 months after the operations.

Table 3:

Location	Males	Females	Total
Anterior Communicating Artery.	----	1	1
Middle Cerebral Artery.	1	----	1
Posterior Communicating Artery.	----	4	4
Total	1	5	6

Figure 1: Post. Comm. Art. Aneurysm - before Clipping.



Figure 2: Ant. Comm. Art. Aneurysm - before Clipping.



Figure 6: Giant Bilat. Ophthalmic Art. Aneurysm.



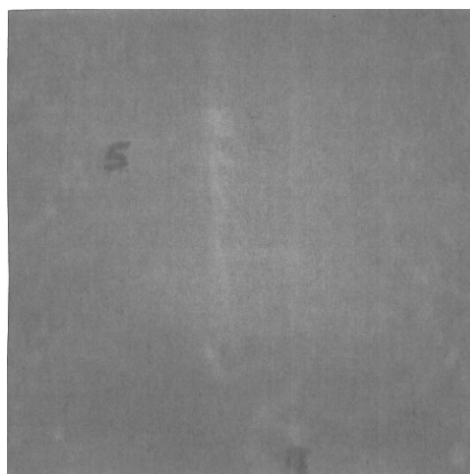
Figure 3: Middle Cerebral Art. Aneurysm - after Clipping.



Figure 4: Ant. Comm. Art. Aneurysm - after Clipping.



Figure 5: Post. Comm. Art. Aneurysm - after Clipping.



Discussion

None of our cases had any indications, clinically or radiographically, of vasospasms, as widely reported, post-rupture and postoperatively. Could this be racially/genetically dependent? The age range, in our series, of 40-50 years, did not agree with those reported by others of 55-60 years¹.

Ruptured berry or congenital aneurysms constitute about 90% of all cerebral aneurysms, we had 100%. This might be due to our small series.

Berry aneurysms are situated in the anterior circulation in 85 to 95% of cases¹, which is in agreement with our findings. In all cerebral aneurysms, female to male ratio is estimated at 1.6 to 1¹, we had 5 females to 1 male. That the junction of the internal carotid artery with the posterior communicating artery is the most common site for saccular aneurysms in women was also supported by our findings. That the prognosis of aneurysmal subarachnoid haemorrhage is worse for women¹ was however, not observed from our cases. In our series, 66.7% were from the posterior communicating artery junction with the internal carotid artery, as against 23% published.

A larger series might indicate otherwise. Unruptured aneurysms could present themselves by causing neurological deficits and/or psychological problems, as evidenced by one of our cases (Picture 6) with ophthalmological and psychological dysfunctions.

Conclusion

From our experience, ruptured berry aneurysms were more in females than in males, and, posterior communicating artery aneurysms were predominant. They were most prevalent in the age range of 40-50 years and none in the 50-60 years range and after the age of 70 years. There were no resultant vasospasm pre- or postoperatively. The different findings could have been due to genetics/genetical mutations, changes in life style, environment, etc. over the years. No definite conclusions could be deduced from our findings. Further research might throw more light on these.

Acknowledgement

We are very grateful to my then chief at the Kingston Regional Hospital, Mr. John McHardy, for his assistance and encouragement. We thank Prof. Charles A. Attah, Provost, College of Health Sciences, Ebonyi State University, Ebonyi State, Nigeria, for proof-reading this article, and my dear wife, Lolo Jennifer Annette Ibe, for her encouragement. A lot of thanks to the typist, my

daughter, Miss Nkechinyere S-A. N. Ibe. Dr. John A. S. Hall, Consultant Physician/Neurologist, Dr. Christian Alele, Consultant Physician/Cardiologist/Nuclear Radiologist, and Dr. Ken. Dennison, Consultant Ophthalmologist, all of the Kingston Regional Hospital, Kingston, Jamaica, the West Indies, referred the various cases.

References

1. Liebeskin DS. Cerebral Aneurysms : eMedicine Specialties Neurology Neurovascular Diseases, 2006.
2. Ohaegbulam SC, Dujovny M, Ausman JI. Ethnic distribution of intracranial aneurysms. *Acta Neurochir(Wien)*. 1990; 106: 132-135.
3. McEvoy AW, Kitchen ND, Thomas DGT. Intracerebral haemorrhage in young adults, the emerging importance of drug misuse; *BMJ*. 2000 320:1322-1324.
4. Zak IT, Dulai HS, Kish KK. Imaging of neurologic disorders associated with pregnancy and the post-partum period; *Radiographics*. 2007; 27: 95-108.
5. Juvela S, Poussa K, Porras M. Factors affecting formation and growth of intracranial aneurysms: a long term follow-up study; *Stroke*. 2001; 32 (2): 485-91.
6. Schievink WI. Genetics and aneurysm formation; *Neurosurg Clin N Am*. 1998; 9 (3)485-95.
7. Velthuis BK, Van Leeuwen MS, Witkamp TD. Computerized tomography angiography in patients with subarachnoid hemorrhage: from aneurysm detection to treatment without conventional angiography. *J Neurosurg*. 1999; 91 (5), 761-7.
8. Hashimoto H, Lida J, Hironaka Y. Use of spiral computerized tomography angiography in patients with subarachnoid hemorrhage in whom subtraction angiography did not reveal cerebral aneurysms. *J Neurosurg*. 2000; 92(2); 278-83.
9. Adams WM, Laitt RD, Jackson A. The role of MR angiography in the pretreatment assessment of intracranial aneurysms: comparative study. *AJNR Am J Neuroradiol*. 2000; 21 (9), 1618-28.
10. Broderick JP. Coiling, clipping, or medical management of unruptured intracranial aneurysms: time to randomize? *Ann Neurol*. 2000; 48 (1): 5-6.
11. Benndorf G, Klucznik RP, Meyer D. "Crossover" technique for horizontal stenting of an internal carotid bifurcation aneurysm: using a new self-expandable stent: technical case report. *Neurosurgery*. 2006; 58 (1 Suppl): ONS-E172.

12. Brilstra EH, Rinkel GJ, van der Graaf Y. Treatment of intracranial aneurysms by embolization with coils: a systematic review; *Stroke*. 1999; 30 (2): 470-6.
13. Karmonik C, Strother CM, Chen X. Stent-assisted coiling of intracranial aneurysms aided by virtual parent artery reconstruction; *AJNR Am JNeuroradiol*. 2005; 26 (9): 2368-70.
14. Lylyk P, Ferrario A, Pasbon B. Buenos Aires experience with the Neuroform self-expanding stent for the treatment of intracranial aneurysms; *JNeurosurg*. 2005; 102 (2): 235-41.
15. Niimi Y, Song J, Madrid M. Endosaccular treatment of intracranial aneurysms using matrix coils: early experience and midterm follow-up. *Stroke*. 2006; 37 (4): 1028-32.
16. Parra A, Kreiter KT, Williams S. Effect of prior statin use on functional outcome and delayed vasospasm after acute aneurysmal subarachnoid hemorrhage: a matched controlled cohort study. *Neurosurgery*. 2005; 56 (3): 476-84.
17. Rosen DS, Macdonald RL. Subarachnoid hemorrhage grading scales: a systematic review. *Neurocrit Care* 2005; 2 (2): 110-8.
18. Mayberg MR. Cerebral vasospasm *Neurosurg Clin NAm*. 1998; 9 (3): 615-27.
19. Sluzewski M, van Rooij WJ. Early rebleeding after coiling of ruptured cerebral aneurysms: incidence, morbidity, and risk factors. *AJNR Am JNeuroradiol*. 2005; 26(7): 1739-43.
20. Sluzewski M, van Rooij WJ, Beute GN. Late rebleeding of ruptured intracranial aneurysms treated with detachable coils. *AJNR. Am J Neuroradiol*. 2005; 26 (10): 2542-9.