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Spontaneous Occlusion of Giant ICA Aneurysm and Presentation with Ischemia: Treatment with Bypass

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Abstract: ***Background: Stroke caused by spontaneous thrombosis of an unruptured intracranial aneurysm is a rare event. Surgical management of such aneurysms arouses many complexities for the neurosurgeon and an analytical decision with regards to above is the need of the hour. Case Presentation: We report a case of a 54-year-old male who experienced recurrent ischemic attacks and cerebral infarctions due to spontaneous thrombosis of an unruptured left internal cerebral artery bifurcation aneurysm. Spurts of emboli into the ipsilateral ACA and MCA, resulting in ischemic infarction in both the vascular territories, was diagnosed by CT scanning, MRI, and cerebral angiography and was surgically successfully managed by left STA-MCA bypass technique. Conclusion: Thrombosis is one of the most significant events in the natural history of giant aneurysms, and partial thrombosis does not preclude the risk of rupture. Thrombosed aneurysms may display additional growth brought about by wall dissections or intramural hemorrhages. Surgical treatment of thrombosed aneurysms is the preferred treatment since it has been shown to alleviate TIAs and would reduce the risk of subarachnoid hemorrhage. Non-conventional surgical techniques are discussed in correlation to the microsurgical management of these aneurysms. Isolated bypass technique can be considered as a better alternative to direct clipping of the aneurysm in such cases.***

1. Introduction

Based on 6368 cases in a cooperative study, Locksley classified aneurysms that were 25 mm or greater as giant and observed a high rate of morbidity and mortality associated with these lesions. **[1]**

Although thrombosis in giant aneurysms is a relatively common phenomenon, complete thrombosis is uncommon, and usually spares the parent vessel. When such an event occurs nonetheless, it may either remain silent or cause compressive and ischemic symptoms. Its radiological characteristics may suggest the existence of a mass lesion.

Some authors **[15]** observed that the clinical presentation of giant aneurysms with spontaneous thrombosis, in the absence of subarachnoid hemorrhage (SAH), frequently includes paroxysmal neurological signs, such as seizures and ischemic events. Stroke and transient ischemic attack due to thromboembolism from aneurysmal thrombus are rare events, despite being consistently described in association with partially thrombosed aneurysms.

We herein present a case of a patient diagnosed with a giant aneurysm of the internal cerebral artery (ICA) that progressed to spontaneous thrombosis of the aneurysm and its parent artery and presented with recurrent ipsilateral MCA and ACA territory infarcts. A brief literature review on the subject related to the case and its surgical management is presented.

2. Case Report

A 54 year old male, with background history of hypertension, initially presented 20 years back to another hospital with headache. An MRI and DSA done revealed a giant ICA aneurysm incorporating the ACA and MCA origins. At that time, it was deemed to be unfavorable for endovascular management, and was advised high flow bypass with parent artery occlusion. However, the patient did not consent for surgery and was lost to follow-up.

In 2015, he presented with sudden onset right hemiparesis and had left MCA territory acute ischemic changes upon imaging. Although he presented within the window period endovascular reperfusion was attempted yet unsuccessful and was hence treated conservatively. Over the next 5 days, he gradually improved and was discharged in a stable state with antiplatelet medications. In 2016, upon follow-up he presented with multiple episodes of headache and the necessary imaging was done.

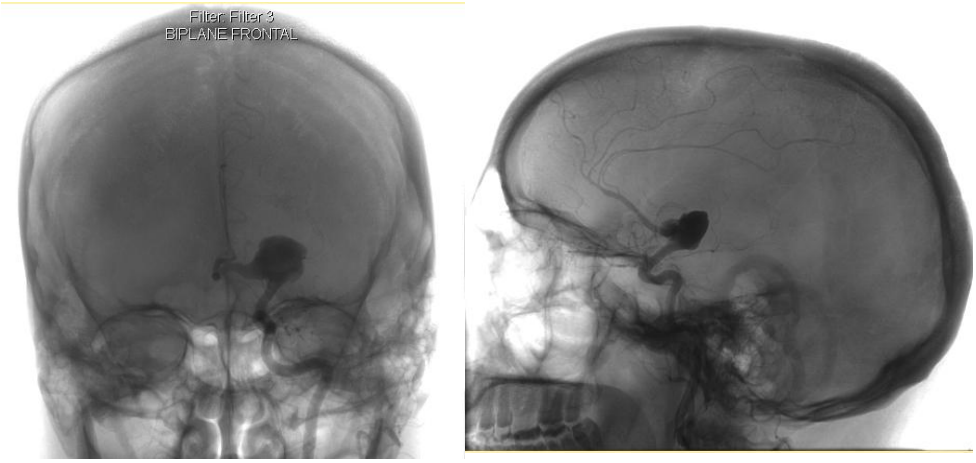


Figure 1

DSA (**Figure 1.)** showed a partially thrombosed saccular aneurysm (1.6cm (AP) x 2.1cm (ML) x 2.0 cm (CC)) with 8mm neck incorporating left A1 and M1 leading to occlusion with lenticulostriate collaterals seen in this region reforming the M2 branches and the cortical branches via ACA and PCA collaterals.

Based on the imaging reports, he was advised for endovascular flow diverter insertion for the management of the aneurysm, however the patient did not consent for the same.

In July 2019, he had a recurrent acute ischemic attack and presented with right sided hemiparesis and speech disturbances and was noted to have acute left ACA territory ischemic stroke.

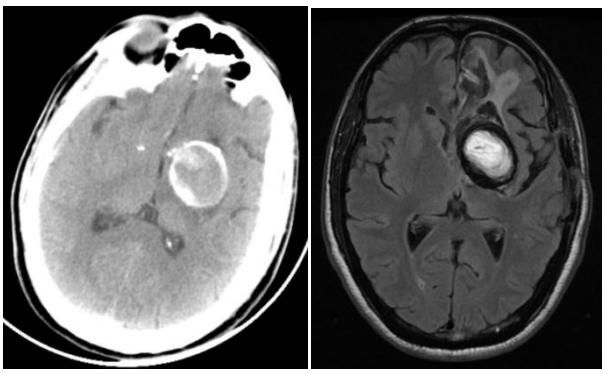


Figure 2

The CT Head (Plain and Angiogram) and MRI Brain (**Figure 2**) showed a giant aneurysm arising from the left ICA bifurcation measuring about 3.4cm (AP) x 3.2cm (ML) x 3.7cm (CC) directed posterosuperiorly into the left capsuloganglionic region with no obvious contrast filling on angiography suggestive of thrombosed aneurysm and was also noted to have diffusion restriction involving left anterior cingulate cortex suggestive of hyperacute partial left ACA territory ischemic stroke. Surrounding the aneurysm were encephalomalacic changes in the left capsuloganglionic and adjacent corona radiata due to chronic left M1 territory MCA infarct.

DSA (**Figure 3**) showed the Left ICA was thrombosed at the level of the anterior choroidal artery and the aneurysm could not be visualized because of the thrombosis with paucity of vessels in the ACA and MCA territory being supplied by the collaterals from the left PCA which was inadequate.

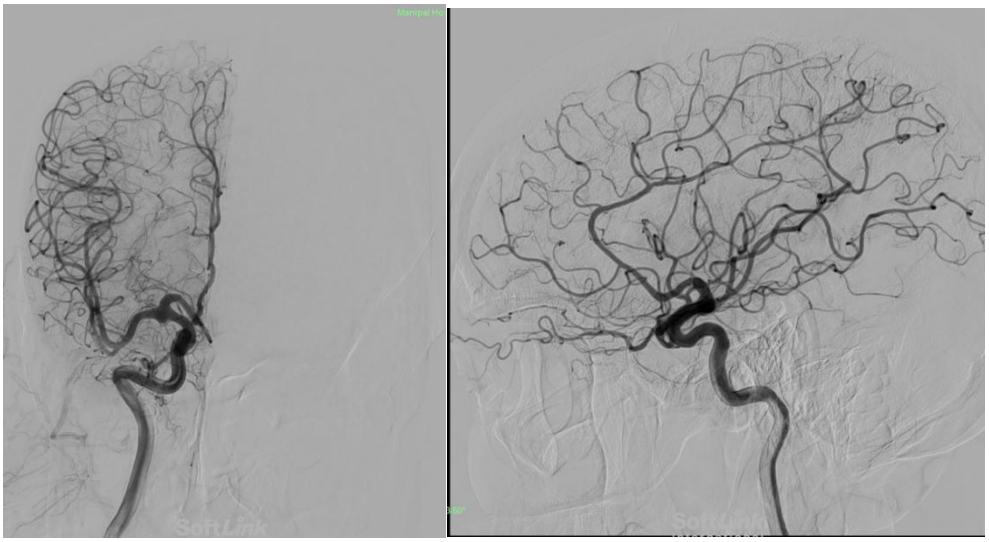


Figure 3

Since the parent artery was completely occluded with partial filling of distal ACA and MCA branches through collaterals and no retrograde filling up of aneurysm, the plan for surgical revascularisation was decided upon and he underwent left STA-MCA bypass with encephaloduromyosynangiosis under General Anaesthesia with no intraoperative complications.

The patient had an uneventful postoperative course with strict neuromonitoring and neurorehabilitative care over the next 5 days and discharged in a neurologically and hemodynamically stable condition, with improvement in power of the right peripheries, on a single antiplatelet medication.

Over the course of the following 3 months, he was noted to have complete recovery of his right upper and lower limb power with improvement in dysphasia and was observed to be functionally entirely independent.

He underwent imaging in November, 2019 on a follow-up basis as follows:

MRI Brain (**Figure 4**) showed the thrombosed giant aneurysm (3.1cm (AP) x 3.0cm (ML) x 3.5cm (CC)), significantly reduced in size as compared to the previous scans, with surrounding areas of encephalomalacia noted and no acute diffusion restriction.

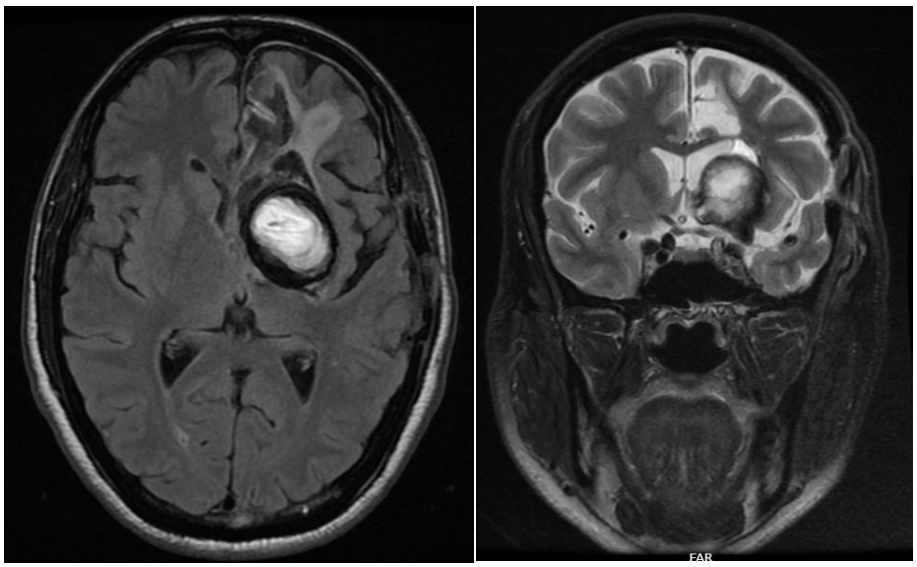
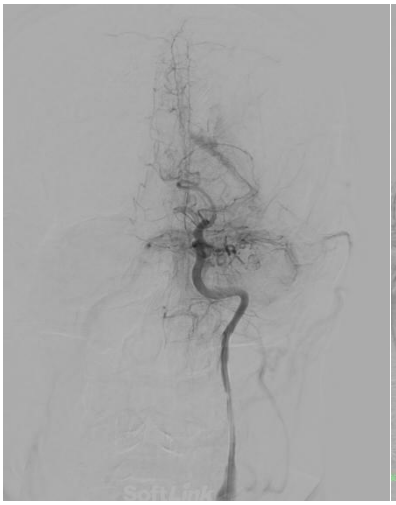
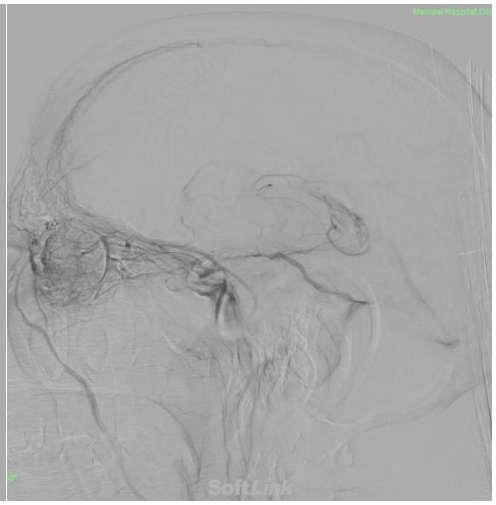


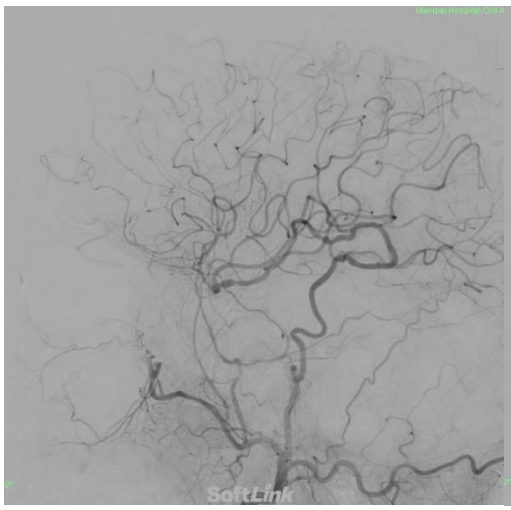
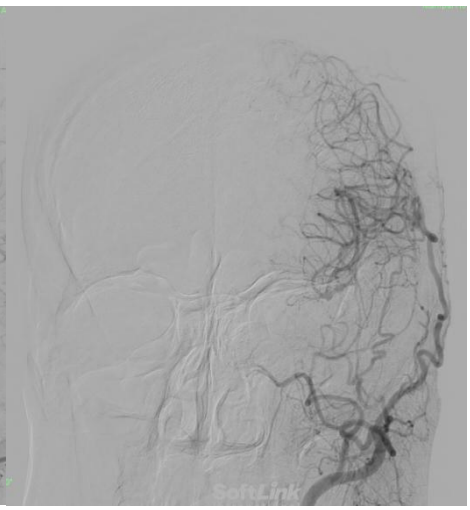
Figure 4

DSA imaging (**Figure 5**) revealed that the left ICA supraclinoid aneurysm was completely thrombosed and good flow was achieved through the left distal MCA across the STA-MCA graft with adequate blood supply and no retrograde flow into the aneurysmal sac.

Figure 5:

Left ICA (Coronal view) **Left ICA (Sagittal view)**

Left ECA (Coronal view) **Left ECA (Sagittal view)**

3. Discussion

Giant intracranial aneurysms have, by definition, a maximal diameter of at least 25mm. **[1]** Theyrepresent 2% to 5% of all intracranial aneurysms. **[3]**In general, 34% to 67% of giant intracranial aneurysms are associated with the ICA, 11% to 40% with the anteriorcerebral artery (ACA) and middle cerebral artery (MCA), and 13% to 56% with the vertebral and basilar arteries. **[4]**

Spontaneous partial thrombosis of giant aneurysms is a fairly common finding, occurring in up to 60% of such lesions. [**7, 8, 9]** Occlusion of an aneurysm due to spontaneous thrombosis was first reported in 1955. **[2]** Vasconcellos et al. **[19]** proposed that 25% (five cases) of 20 cases showed courses of spontaneous occlusion during observation of giant carotid cavernous aneurysms of the internal carotid artery with a relatively low risk of bleeding. The possibility of spontaneous occlusion was relatively high in cases of improvement of initial symptoms, such as retrobulbar pain or migraine during courses of carotid giant aneurysms.

A factor to analogize spontaneous occlusion of an unruptured aneurysm is the volume-to-orifice ratio. **[20]** Through analysis of the results for the ratio of the size (mm2) of the aneurysm neck to the aneurysm volume (mm3) from studies on 21 aneurysms, a value greater than 25 mm indicates the potential for thrombosis.

Thus, it is assumed that as the volume-to-orifice ratio grows larger, thrombosis appears more frequently by inducement of red blood cell aggregation thrombosis due to blood stasis in the aneurysm. Accordingly, the pathophysiology of thrombosis is not clear, however, it has received the most support. The volume-to-orifice ratio calculated in our case was 40 mm.

Other biophysiological parameters such as the age of the aneurysm, hemodymanics in the parent artery, direct distortion of the parent artery by the aneurysmal sac, endothelial damage due to intrasaccular turbulent flow, increased coagulability and the angiographic procedure itself have been also proposed.

Associated concomitant thrombosis of a giant aneurysm and their parent vessel has been observed in aneurysms of the ICA, middle cerebral artery, and posterior cerebral artery. However, progression from aneurysm thrombosis to parent vessel occlusion has been documented only in one previous communication [**21**].

Cerebral infarcts can be the first manifestation of spontaneous thrombosis of large or giant UIAs. Two mechanisms have been proposed for the parent vessel occlusion in the presence of a giant thrombosed carotid artery aneurysm. Whitle et al. [**22]** suggest that occlusion is due to the direct distortion and compression of the parent artery by the aneurysm. The other possibility is the retrograde development of a thrombus originating from the aneurysm [**23]**.

According to the Antonio et al., [**24]** their study presented three patients with spontaneous thrombosis of an UIA and embolic ischemic stroke. In all three cases, the imaging features of the infarcts were suggestive of embolism, alternative etiologies were ruled out, and the presence of aneurysmal thrombus was confirmed. These features suggest a causal relationship between the aneurysm and the infarct. All patients had large aneurysms. Black and German **[25]** found that the larger the volume of the aneurysmal sac in relation to the cross-sectional area of the neck, the more sluggish the flow within the fundus, which increased the possibility of thrombosis.

Our patient suffered a stroke 15 years after diagnosis of the aneurysm. Head MR imaging revealed enlargement and thrombosis of the aneurysm, and the cause of the stroke was considered as embolism due to this thrombosis in the aneurysm sac. However, further medical treatment with dual antiplatelet therapy could not prevent a subsequent stroke which he suffered in 2019, and hence surgical treatment was considered necessary.

A similar case was reported by Hiedeaki et al. [**26]** wherein a 76-year-old woman was diagnosed with a large intracavernous internal carotid artery (ICA) aneurysm at the bifurcation with the primitive trigeminal artery (PTA) which caused repeated embolic strokes after enlargement and thrombosis of the aneurysm. The strokes recurred in spite of medical treatment but were successfully prevented by high-flow bypass using radial artery graft (RAG) following coil embolization of the PTA.

Indirect aneurysm occlusion (proximal occlusion, distal occlusion, or trapping) with or without a bypass has become a more acceptable alternative. This strategy has important advantages. The bypass can be performed with predictable ischemia times, cerebroprotection, and relatively low complication rates. The dangers of direct attack are avoided, including perforator dissection and preservation around clips. **[15]**

Various bypass techniques have been described for both the anterior and posterior circulations. Sometimes, altering hemodynamics with a bypass alone may result in aneurysm thrombosis and resolution. **[16, 17]**The superficial temporal arteries and occipital arteries have been used to supply blood to branches of the MCA, posterior cerebral artery, and superior cerebellar artery. In our case, we have used a pedicled left STA parietal branch and anastomosed with the distal M3 branch of the left MCA. Good flow was noted across the graft intraoperatively using indocyanine green fluoresence dye and its patency was confirmed by a DSA done 3 months post-operatively. With these techniques, we must be aware of mismatches in blood flow because some bypass techniques are limited by the small caliber of donor vessels. In such cases, high-flow bypasses can be created using interposition saphenous vein grafts, or “double-barrel” bypasses can connect the anterior and posterior branches of the STA to two recipient vessels. **[17, 18]**Likewise, interposition grafts such as radial artery graft and petrous-supraclinoid vein grafts can be used to bypass diseased segments of large-caliber vessels of the ICA. [**18]**

Finally, blood flow distal to the trapped aneurysm can be augmented with a side-to-side anastomosis from the contralateral circulation popularly known as in-situ bypass. The anterior temporal artery branch of the MCA can sometimes be mobilized to bypass diseased segments just distal to it. **[17, 18]**

4. Conclusion

Completely thrombosed GIAs with parent vessel thrombosis are rare lesions that are dynamic and unstable, being prone to spontaneous recanalization and additional growth. There is no consensus about the ideal management of such lesions. A noninterventionist management approach is possible but requires strict follow-up as rare complications like an acute ischemic stroke, as was observed in our case, might be highly debilitating if not managed accordingly. Our case was managed with an alternative surgical option, to the widely reported superlative direct clipping technique, which involved an isolated revascularisation procedure (low-flow STA-MCA bypass without aneurysm occlusion). The patient did not have any fresh neurological deficits post-surgery and showed complete recovery from his disability on follow-up. Hence it can be concluded that bypass procedures are important contingency strategies with giant aneurysms which present with spontaneous occlusion of parent vessel causing ischemia and neurological deficits, however dependent upon the morphology, location and complexities of the aneurysm also taking into account, the nature of disability of the patient upon presentation, in order for a good outcome.

References

[1] Locksley HB. Natural history of subarachnoid hemorrhage, intracranial aneurysms and arteriovenous malformations: based on 6368 cases in the cooperative study. **J Neurosurg**.1966; 25: 219–239.
[2] De Lange SA. [A case of spontaneous thrombosis of a cerebral arteriovenous aneurysm] Ned Tijdschr Geneeskd.1955 Mar; 99 (13): 944–946. Dutch.
[3] Peerless SJ, Wallace MC, Drake CG. Giant intracranial aneurysms. Youmans JR. **Neurological Surgery: A Comprehensive Reference Guide to the Diagnosis and Management of Neurological Problems**. WB Saunders: Philadelphia; 1990: 1742–1763.
[4] Atkinson JLD, Piepgras DG. Giant aneurysms: supratentorial. Carter LP, Spetzler RF. **Neurovascular Surgery**. McGraw-Hill: New York; 1995: 815–828.
[5] Khurana VG, Wijdicks EF, Parisi JE, et al. Acute deterioration from thrombosis and rerupture of a giant intracranial aneurysm. **Neurology**.1999; 52: 1697–1699.

[6] Khurana VG, Piepgras DG, Whisnant JP. Ruptured giant intracranial aneurysms. Part I. A study of rebleeding. **J Neurosurg**.1998; 88: 425–429.
[7] LeRoux PD, Winn HR, Newell DW. **Management of cerebral aneurysms**. Philadelphia, Pa: Saunders, 2004.
[8] Choi IS, David C. Giant intracranial aneurysms: development, clinical presentation and treatment. **Eur J Radiol** 2003; 46 (3): 178–194.
[9] Forsting M. **Intracranial vascular malformations and aneurysms**. Berlin, Germany: Springer, 2004.
[10] Unruptured intracranial aneurysms: risk of rupture and risks of surgical intervention. International Study of Unruptured Intracranial Aneurysms Investigators. **N Engl J Med** 1998; 339 (24): 1725–1733. [Published correction appears in N Engl J Med 1999; 340 (9): 744.]
[11] Wiebers DO, Whisnant JP, Huston J et al. . Unruptured intracranial aneurysms: natural history, clinical outcome, and risks of surgical and endovascular treatment. **Lancet** 2003; 362 (9378): 103–110.
[12] Ertl L, Hottmannspotter M, Patzig M, Bruckmann H, Fesl G. Use of flow-diverting devices in fusiform vertebrobasilar giant aneurysms; a report on periprocedural course and long-term follow up. **AJNR** 2014; 35: 1346-52.
[13] Lawton MT, Spetzler RF. Management strategies for giant intracranial aneurysms. **Contemp Neurosurg** 1994; 16: 17
[14] Awad IA, Barrow DL. Giant intracranial aneurysms. Rolling Meadows, Ill: American Association of Neurological Surgeons, 1995.
[15] Sughrue ME, Saloner D, Rayz VL, Lawton MT. Giant intracranial aneurysms: Evolution of management in a contemporary surgical series. **Neurosurgery**.2011; 69: 1261–1270.
[16] Zabramski JM, Kiris T, Sankhla SK, et al. Orbitozygomatic craniotomy. Technical note. **J Neurosurg**.1998; 89: 336–341.
[17] Cantore G, Santoro A, Da Pian R. Spontaneous occlusion of supraclinoid aneurysms after the creation of extra-intracranial bypasses using long grafts: report of two cases. **Neurosurgery**.1999; 44: 216–219
[18] LawtonMT, SpetzlerRF. Surgicalstrategiesforgiantintracranialaneurysms. **Neurosurg Clin N Am**.1998; 9: 725–742.
[19] Vasconcellos LP, Flores JAC, Conti MLM, Veiga JCE, Lancellotti CLP. Spontaneous thrombosis of internal carotid artery: a natural history of giant carotid cavernous aneurysms. **Arq Neuropsiquiatr**.2009 Jun; 67 (2A): 278–283.
[20] Wiebers DO, Whisnant JP, Huston J, 3rd, Meissner I, Brown RD, Jr, Piepgras DG, et al. Unruptured intracranial aneurysms: natural history, clinical outcome, and risks of surgical and endovascular treatment. Lancet.2003 Jul; 362 (9378): 103–110.
[21] Sato K, Fujiwara S, Yoshimoto T, Onuma T (1990) Two cases of spontaneous internal carotid artery occlusion due to giant intracranial carotid artery aneurysm. **Stroke** 21: 1506–1509
[22] Whittle IR, Williams DB, Halmagyi GM, Besser M (1982) Spontaneous thrombosis of a giant intracranial aneurysm and ipsilateral internal carotid artery. **J Neurosurg** 56: 287–289
[23] Ray A, Leach P, Vafifidis J (2002) Spontaneous thrombosis of a giant internal carotid aneurysm in a patient who presented with hypopituitarism. **J Neurosurg** 16: 590–592
[24] Arauz A, Patino-Rodriguez HM, Chavarria-Medina M, Becerril M, Merino JG, Zenteno M. Embolic stroke secondary to spontaneous thrombosis of unruptured intracranial aneurysm: Report of three cases. **Interv Neuroradiol**.2016; 22 (2): 196-200.
[25] Black SP, German WJ. Observations on the relationship between the volume and the size of the orifice of experimental aneurysms. **J Neurosurg** 1960; 17: 984–990.
[26] Ono H, Inoue T, Iijima A, Tanishima T, Tamura A, Saito I. Recurrent embolic stroke caused by large cavernous carotid aneurysm: Stroke recurrence prevention with high-flow bypass followed by proximal occlusion. **Asian J Neurosurg** 2018; 13: 901-5