

A Non-Bleeding Complex Intracerebral Giant Aneurysm Case: Case Report

Kanamamış Kompleks İntraserebral Dev Anevrizma Olgusu: Olgu Sunumu

ABSTRACT

An intracranial aneurysm with a diameter larger than 25 mm is considered a giant aneurysm (GA), and represent about 3-5% of all aneurysms. They are divided into two forms, specifically saccular and fusiform. Fusiform aneurysms are rare, making up only 1% of all intracranial aneurysms. They frequently involve the internal carotid artery (ICA) or the basilar or vertebral arteries, and rarely bleed. Treatment of huge aneurysms that have not bled is still controversial. Unlike the saccular huge aneurysms that lead to death of 80% of the patients few years after diagnosis, fusiform huge aneurysms, particularly those presenting with mass effect, have a better prognosis. In this manuscript, we discuss the infrequently seen intracranial fusiform giant aneurysms in light of the pertinent literature.

KEY WORDS: Giant aneurysm, Intracerebral, Non-bleeding aneurysm, Treatment

ÖZ

İntrakranial anevrizmalarda en geniş çap 25 mm'den büyük ise dev anevrizma olarak adlandırılırlar. Dev anevrizmalar tüm anevrizmaların %3-5'ini oluşturur. Sakküler ve fuziform olarak iki tipi vardır. Fuziform anevrizmalar ise oldukça nadir görülürler ve tüm intrakranial anevrizmaların %1'ini oluşturur. Sıklıkla internal karotid arter, baziler veya vertebral arterleri tutar ve ender olarak kanarlar. Kanamamış dev anevrizmaların tedavisi günümüzde halen tartışmalı bir konudur. Tedavi edilmeyen hastaların %80'inin tanı konulduktan sonraki birkaç yıl içerisinde kaybedildiği sakküler dev anevrizmaların aksine, özellikle kitle etkisi ile bulgu veren fuziform yapıları dev anevrizmaların prognozu daha iyidir. Bu makalede, nadir görülen fuziform yapıları intrakranial dev anevrizmalar literatür eşliğinde tartışılmıştır.

ANAHTAR SÖZCÜKLER: Dev anevrizma, İntraserebral, Kanamamış anevrizma, Tedavi

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INTRODUCTION

Aneurysms, specifically those that are fusiform or dolicoectatic, are also considered atherosclerotic since they frequently progress in combination with prevalent atheromatous vascular disease. They normally involve the ICA, basilar or vertebral arteries (9,31). They cause clinical symptoms secondary to compression of brain tissue around the nerve roots with elongated, enlarged and irregularly shaped arteries. The fusiform aneurysm of the ICA generally develops in the intracavernous segment of the artery. Similar to other fusiform aneurysms, they rarely rupture and hardly bleed (9,32). In this paper, the clinical manifestation and radiological findings in an elderly patient with non-bleeding complicated giant aneurysm are presented and treatment alternatives are discussed in light of the relevant literature.

CASE REPORT

An 83-year-old male patient was admitted to the hospital with complaints of progressive headaches for 2 weeks and memory lapse. The Glasgow coma scale score was 15/15, and the Hunt-Hess classification grade was 1 for his neurological examination. A hyperdense mass was observed in the left frontal region on computerized tomography (CT), and a meningioma was considered in the differential diagnosis (Figure 1). A mass image possibly due to aneurysm on the left frontal area and measuring 52x40 mm at its maximal section, isointense with blood but showing artifacts due to flow inside, extending from the Sylvian cistern to basal ganglia and showing diffuse enlargement in its proximal middle cerebral artery (MCA) M1-segment was observed in the cranial magnetic resonance imaging (MRI) of the patient. Prominent vasogenic edema was observed around the mass. The left lateral ventricle was compressed by the mass and mild effacement of sulci was observed (Figure 2A,B,C,D). Cerebral angiography of the patient revealed a fusiform aneurysmatic dilatation and thrombosed aneurysm extending from the supracavernous segment of the left internal carotid artery to the MCA M2 segment. Insular branches of the left MCA had normal calibration, but the anterior cerebral artery showed weak filling beginning from the A1 segment (Figure 3). Surgical treatment or endovascular embolisation was not considered due to the patient's age and conservative management with patient follow-up was considered acceptable.



Figure 1: A hyperdense mass approximately 5x4 cm in diameter. For the differential diagnosis, meningioma was considered because a mass was observed in the left frontal region on CT examination in the axial plan.

DISCUSSION

GAs, initially reported by Hutchinson in 1875, have been found to be operable with an acceptable rate of morbidity and mortality in the wide-ranging series of Drake and Sundt between 1979 and 1991 (5,17,28). GAs are observed more often in females, and 60% become symptomatic between the ages of 50 and 60. Only 5% of female patients display symptoms below the age of 20 (7).

Although the pathogenesis of giant fusiform aneurysms is not clearly understood, they are believed to develop secondary to atherosclerotic changes in a particular segment of the vessel wall. Intracranial fusiform aneurysms can be divided into 2 clinically different subtypes; acute dissecting aneurysms and chronic fusiform or dolichoectatic aneurysms. Histological examination of chronic fusiform aneurysms are progressive lesions: [1] internal elastic lamina fragmentation and intimal hyperplasia; [2] neoangiogenesis in the thickened intima; [3] intramural hemorrhage and luminal thrombosis; and [4] recanalizing vessel formation in the thrombus (18). There is no aneurysmal neck in

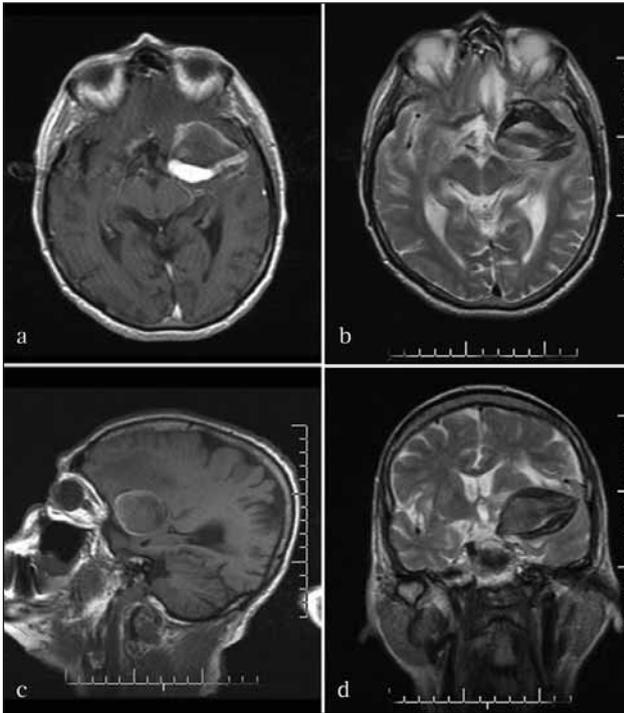


Figure 2: A mass image possibly due to aneurysm on the left frontal area measuring 52x40 mm at its maximal section, isointense with blood but showing artifacts due to flow inside, extending from the Sylvian cistern to basal ganglia and showing diffuse enlargement in its proximal MCA-M1 segment is observed in the cranial magnetic resonance imaging of the patient: (A) transverse T1 sequence, (B) transverse T2 sequence, (C) sagittal T1 sequence and (D) coronal T2 sequences. Prominent vasogenic edema is observed around the mass. The left lateral ventricle is compressed by the mass and mild effacement of the sulci is observed.

fusiform GA, and the blood vessel wall completely enlarges. Some collagen diseases, such as “Ehler-Danlos” and “pseudoxantoma elasticum”, may lead to fusiform aneurysms (13).

Carotid bifurcation aneurysms are observed at a rate of 5% among intracranial aneurysms and present with symptoms caused by mass effect simply when they become large. Our patient experienced headaches and memory lapse; however, the development of bitemporal hemianopsia due to pressure on the optic tracts and chiasma as well as the development of hemiparesis, epilepsy, and homonym hemianopsia as a consequence of the extension of the GA towards MCA in the GAs located in the ICA bifurcation have been reported in the literature. Symptoms such as hypopituitarism, hemiathetosis, Parkinsonism, personality change, dementia, cortical irritation and epilepsy attacks can also rarely develop due to a mass effect (22,30).



Figure 3: Cerebral angiography of the patient reveals a fusiform aneurysmatic dilatation and thrombosed aneurysm extending from supracavernous segment of the left internal carotid artery to the MCA M2 segment. Insular branches of the left MCA have normal calibration, but ACA shows weak filling beginning from the A1 segment.

Only 3-5% of the symptoms of all GAs are related to thromboembolism. Generally, a piece of the thrombus breaks off and embolizes in the distal beds of the circulation. This situation is usually seen in the carotid artery and the saccular GAs of the MCA. Similar to our case, progression of atherosclerotic plaques with aneurysmal neck location towards the parental artery, narrowing of the blood vessels over time or the development of ischemic complications as a result of total occlusion of perforating branches emerging from this region were reported in carotid bifurcation fusiform aneurysms (3,16).

Although GAs are believed to bleed due to their relatively thick wall because of the thrombus, several studies have demonstrated that this may not be the case (10,20). In an extensive series of 309 cases published in France, the symptoms of patients were examined upon admittance. Bleeding, mass effect and thromboembolism were reported at rates of 48%, 47% and 5%, respectively (14). Completely thrombosed GAs have a severe risk of bleeding (29). Bleeding generally develops in the form of subarachnoid hemorrhage in GAs; however, intracranial hemorrhage can also develop. Similar to our case, these forms of hemorrhage have been reported to be extremely rare for fusiform GAs (12).

The CT characteristics of GAs, initially defined by Pinto, are divided into three forms, specifically non-thrombosed, partially thrombosed and completely thrombosed (23). Non-thrombosed GAs are mildly hyperdense. After injection of the contrast agent, they become intensely and homogeneously stained. Complete thrombosis may be observed as either hypodense or hyperdense lesions on CT, according to the age of the coagulation in the aneurysm. Because of these CT characteristics, they can be confused with basal meningioma, craniopharyngioma and hypophyseal adenomas. Similar to our case, CT images can be altered as a result of the extent of thrombosis in the partially thrombosed group, which is most frequently observed. They may also be confused with intracranial masses (15). The rate of GAs on CT was reported as 66% (24).

MRI yields important information such as the actual magnitude of the aneurysm, quantity and age of the thrombosis as well as the three-dimensional anatomy of the aneurysm and its close proximity to other neural structures. It is quite valuable for differentiating masses (19,21).

A diagnostic method for GA is cerebral angiography. Generally, their magnitude and mass effects and aneurysm necks cannot be revealed clearly on angiography. In an extensive association study, the rate of demonstrating GA by cerebral angiography was reported to be 14% (26). Moreover, completely thrombosed GAs cannot be demonstrated in angiographies. 3D BT-angiography and 3D MR-angiography, which have been used in recent years, can be significant with respect to the three-dimensional evaluation of the aneurysm (2,4,21,23).

GAs pose serious surgical difficulties, not only because of their magnitude, but also because of their wide and generally calcified necks, proximity to the cranial nerves and vital neural structures and inherent thrombosis (8,20). The treatment of non-bleeding aneurysms is controversial (25). Criteria for surgical indication have not been standardized; as a result, the decision should be made according to the age and the neurological and medical status of the patient in addition to the localization of the aneurysm and angiographic features (8,20). Surgical treatment is suggested in young patients with non-bleeding GAs, who have a family history of aneurysmal hemorrhage. Alternatively, a

conservative approach is suggested for asymptomatic cases of non-bleeding GAs due to old age, serious medical problems and expected short duration of life (25).

Presently, closure of the aneurysm neck by clip placement and the protection of blood flow in the parent artery is considered the most appropriate treatment method for GAs (1,14,20). Ligation and trapping can be performed in GAs with a fusiform structure that are not suitable for clipping; however, these techniques always bear a risk of ischemia in the early and late periods unless they are supported by revascularization (25,26). Fusiform aneurysms are usually large in size if they produce symptoms. They have no neck and almost always contain thrombi. These aneurysms recanalize, and a central channel forms through the thrombus. These anatomic features require special procedures for surgical treatment (20,21,27).

Preliminary diagnostic tests are essential to evaluate the fusiform aneurysm. The relationship of the fusiform segment to the normal parent artery and the relationship of the short branches to the fusiform segment must be determined individually. Their careful use of distal occlusion during low volume pressure injection allows the surgeon to evaluate perforating channels that may arise from the fusiform segment. This method must be used with caution because pressure can lead to progression of the aneurysm or rupture (6,20,32).

Both the parent artery proximal to the fusiform segment and the distal portion must be seen in order to preserve flow. An extracranial-intracranial bypass or an end-to-end anastomosis between the proximal and distal segment after excision of the fusiform section may be considered. Circulation in the short branches must also be considered. Fenestrated clips may be used to form a new lumen, providing the intraluminal thrombus can be handled effectively. Monitoring cerebral blood flow is important in determining outcome after arterial clipping of major feeding vessels (20,26,27,32).

Endovascular operations for fusiform aneurysms also require special techniques. Endovascular techniques do not permit reconstruction of fusiform aneurysms, and the endovascular approach is always deconstructive because the parent artery must be sacrificed. As in giant aneurysms, the placement of the balloon is a critical feature of the

deconstructive procedure. The balloon should be placed proximally in the fusiform aneurysm. This procedure permits shrinkage of the lesion and preserves penetrating arteries that are required for vital perfusion (11,20,24).

The prognosis is better in GAs with fusiform structure, which display symptoms with mass effect and low rupture and bleeding rates (12,15,17). This is contrary to saccular GAs with bad prognosis, as 80% of untreatable patients die within a few years after the diagnosis.

CONCLUSION

Fusiform GAs are rare. Symptoms from this form of aneurysm arise because of increasing pressure around brain tissue and nerve roots. Although surgical clipping in the treatment of GAs is still the most secure and reliable method, conservative treatment should be performed in fusiform GA cases with low risk of rupture and bleeding. Angiographic images should be supported by MRI or CT and the true dimensions of the aneurysm should be ascertained in suspected GA cases.

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