

Giant Fusiform Aneurysm of Supraclinoid Internal Carotid Artery

SUPRAKLİNOİD INTERNAL KAROTİS ARTERİNİN DEV EÜZİFORM ANEVİZMASI

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SUMMARY

A 25 years old normotensive male applied due to unilateral visual loss, impotence and who had Foster Kennedy syndrome is shown in this article. The giant fusiform aneurysm of supraclinoid internal carotid artery imitating pituitary tumor is successfully cured by trapping.

KeyWords: Giant aneurysm, Fusiform aneurysm, ICA aneurysm.

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Giant aneurysms most of which are saccular form the 5% of all intracranial aneurysms.

Giant fusiform aneurysms is described as concentric dilatation at the 2.5 cm or longer segment of cerebral arteries. Commonly it holds the ICA and M1 segment of MCA (5).

It's well known that giant fusiform aneurysm of internal carotid artery causes visual symptoms. In the literature it was seen that the giant aneurysms which imitate the pituitary tumor are in a saccular form (6).

CASE REPORT

A 25 years old male patient applied to the clinic due to seven years period of progressive visual

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ÖZET

Bu makalede unilateral görme kaybı ve empotens yakınmaları ile başvuran ve Foster Kennedy sendromu gösteren 25 yaşında normotansif bir olgu sunulmaktadır. Olgumuzda hipofiz tümörünü taklit eden supraklinoid internal karotid dev fusiform anevrizması "trapping" ile başarılı bir şekilde tedavi edilmiştir.

Anahtar Kelimeler: Giant aneurysm, Fusiform aneurysm, ICA aneurysm.

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loss at the right eye and one year period of impotence. Conscious of the normotensive patient was clear, cooperated and oriented according to the results of neurologic examination.

Primary optic atrophy existed at the right eye and papilledema existed at the left eye. Visual acuity was normal at the left and 0.2 at the right, the visual field was normal at the left and peripheral concentric constriction existed at the right. There was no deficit in the other cranial nerves, motor systems and sensory systems.

In the plain skull radiography the sella was larger and it was destructed (Fig.1). The blood sample of the patient who is thought to have pituitary tumor was sent to the laboratory for endocrine testing.

Then giant fusiform aneurysm was determined at the supraclinoid ICA at the right carotid angiogram (Fig.2-3). The M1 segment at the right was in an enlargement form. Left carotid angiogram was



Figure 1. X-Ray film, marked sellar destruction.



Figure 2. Right carotid angiogram. Fusiform aneurysm in the internal carotid artery.

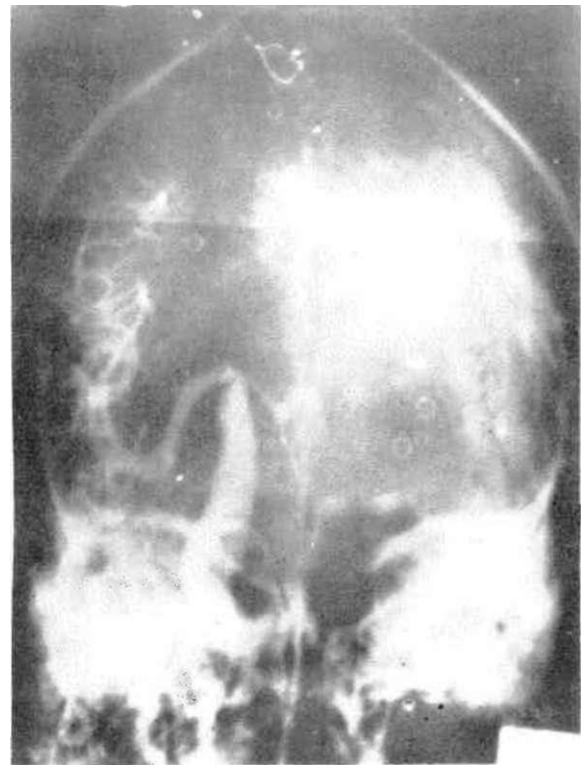


Figure 3. Right carotid angiogram. Fusiform aneurysm in the internal carotid artery.

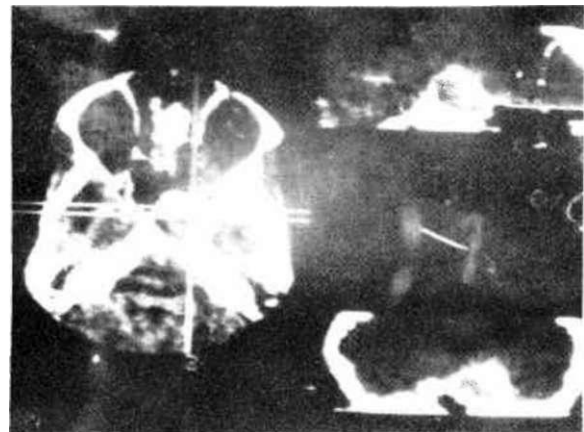


Figure 4. Axial CT scan. A circular hyperdense area in the right parasellar region is seen on the enhanced CT.

not performed. A circular hyperdense area was determined in the right parasellar region by CT (Fig 4-5-6-7). Partially thrombosed part of aneurysmal lumen was seen on the enhanced scan. There wasn't peripheral edema, but partially calcified giant aneurysm was clearly visible on the CT.

After the patient had tolerated the Matas test, he was operated and poppen clamp was placed to the right common carotid artery. Post-operative first day the poppen clamp entirely closed and neither kind of neurological deficit developed.

When was explored by right pterional craniotomy it was seen that right optic nerve was pushed from downwards and side, towards upwards and inwards by the aneurysm. Trapping was made by applying a Yasargil clip in the postoperative period. As a result of preoperative endocrine testing, PRL 50 mIU and FSH 31.4 mIU was found to be high (Fig.8).

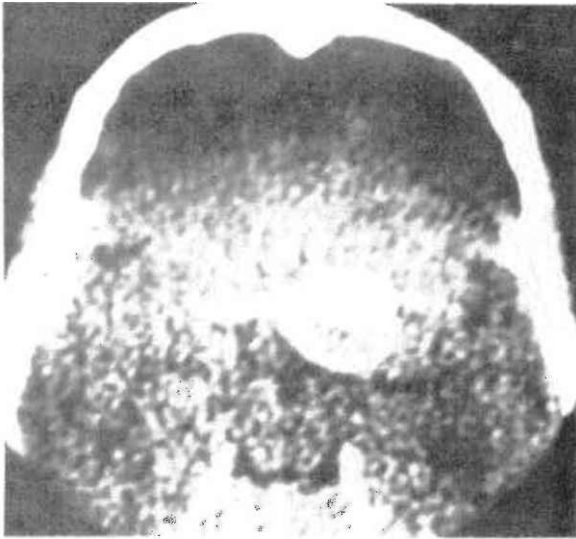


Figure 5. Partially Trombosed aneurysmal lumen is seen on the enhanced CT.

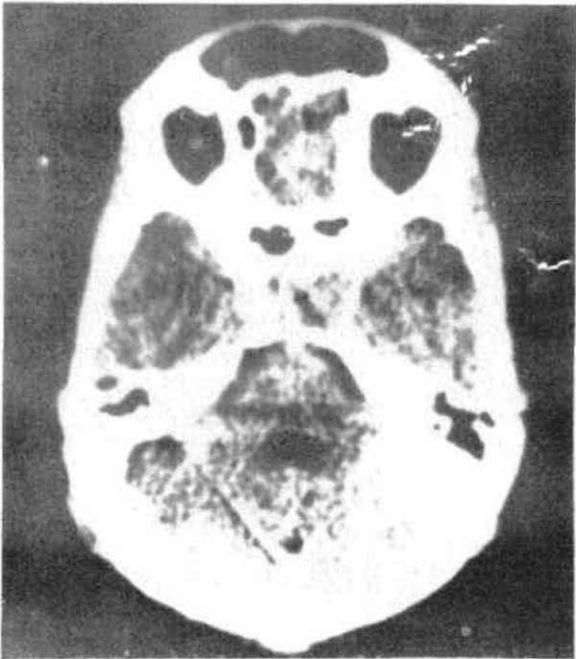


Figure 6. Partially Trombosed aneurysmal lumen is seen on the enhanced CT.

At the 3rd postoperative month, right primary optic atrophy was going on when the left papilledema returned to a normal. Visual acuity was 0.1 at the right and normal at the left. Visual field was a little better when it's compared with the preoperative period at the right. At the control CT of the patient in spite of the artefact bifurcation, both

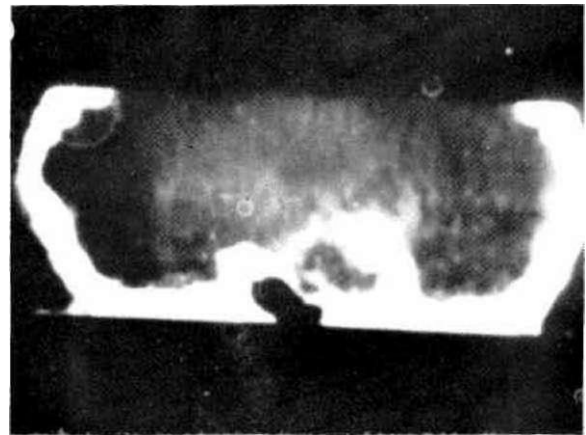


Figure 7. Partially Trombosed aneurysmal lumen is seen on the enhanced CT.

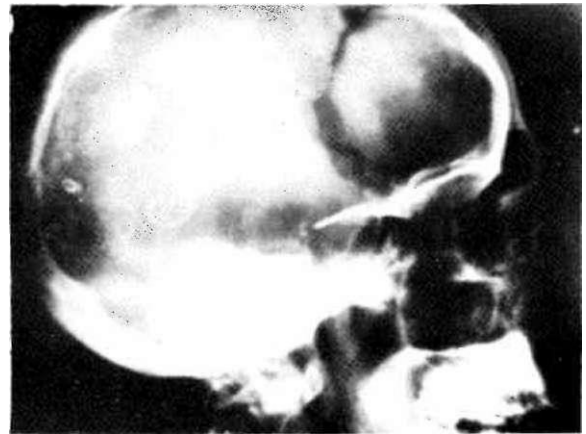


Figure 8. Post operative X-Ray film. A Clip just under the internal carotid bifurcation.

before and after contrast enhancement neither testing of the patient, whose impotence complaint continued, PRL was 270 mIU/ml and FSH was 34.3 mIU/ml.

The patient only had impotence complaint during the controls taking place in post-operative 1.5 years. At the examination right optic atrophy was, still continuing. Visual acuity was normal at the right and 0.2 at the left. The concentric constriction in the visual field was far more better. At the endocrine testing PRL was found 293 mIU/ml and testosterone was found 12 ng/ml. Spermogram contained 4.8 million/ec sperm. After the urological consultation the medical treatment was put in order.

Cerebral ischemia is rarely seen in giant fusiform aneurysms. Two possible occurring mechanism are thought of, first one is embolization

from the thrombus which is in aneurysm, secondly the decreasing of blood flow by thrombus. The youngest age reported in the literature about ICA giant fusiform aneurysm is 9 years. Others have been seen on hypertensive people who are in elder ages. In elder ages dementia and disturbance of gait are the additional problems to symptoms. That's why our patient's being 25 years old and normotensive is so interesting.

It's natural that the giant aneurysms are congenital. Both clinical and pathological differences make these kind of aneurysms to be evaluated differently from saccular aneurysms. Because in the examination of the wall of aneurysm, secondary degeneration in intima layer is linked to hypoplasia which is in muscular layer (2,4,5,6,11).

The characteristic plain radiographic findings in giant aneurysms include calcification and bony erosion. The calcification seen in approximately 18 percent of proven giant aneurysms may be either curvilinear, ringlike or even shell like. 18 percent of cases the plain films will show bone erosion.

Angiography can be insufficient in showing the real dimension of aneurysm due to intramural thrombus. However it will be able to show the localization of aneurysm and its relation with adjacent cerebral vessels (10).

The CT findings of giant aneurysm will vary depending whether the lumen of the aneurysm is totally patent, partially thrombosed or completely thrombosed. In the completely patent giant aneurysm the nonenhanced CT scan will show around or spherical mass of slightly increased density.

According to the correlation between CT and angiography, there are 3 varying types.

- 1- Nonthrombosed aneurysms with thin wall.
- 2- Partially thrombosed aneurysms.
- 3- Completely thrombosed aneurysms.

Peripheral edema is not seen and curvilinear wall calcification is seen in partially and completely thrombosed aneurysms.

Coronal reformation is useful since it shows the real dimension of lesion (7).

DISCUSSION

Giant aneurysms are mostly seen in suprasellar and parasellar regions and qualified as pituitary tumor since it's together with hypopituitarism (8,10).

The 14% of whole aneurysms imitate pituitary tumors (10). Commonly these are ACoA or supraclinoid ICA originated saccular aneurysms (9,12). The first case about the giant ICA aneurysms which imitates pituitary tumor is reported by Mitchell in 1889. White and Ballantine report that the aneurysms couldn't be differentiated clinically from chromophobe adenoma and craniopharyngiomas if aneurysm sac was thrombosed (12). Raymond and Tew published two ICA aneurysms which are suprasellar localized and which imitate pituitary tumor (9). But endocrinal dysfunction wasn't reported in these cases. No giant fusiform aneurysm of internal carotid artery which imitate pituitary tumor is seen in the literature. Commonly giant fusiform aneurysms give characterized clinical syndrome by pressing optic pathway by aneurysm reported by Jefferson (3).

Visual loss and optic atrophy by pressing optic nerve at the patients with giant fusiform aneurysm is reported. A few cases this arterial lesion cause Foster Kennedy syndrome (5).

Mitts and Me Queen research fusiform dilatation of intracranial segment of ICA with visual loss in 4 cases. They determined Foster Kennedy syndrome 3 cases out of 14. But at these cases unilateral visual loss and headache were present but not pituitary symptoms (6).

The occurrence of impotence and both extreme enlarging and destruction of the sella are thought that aneurysm presses sellar region in our case. Visual field defect mostly occurs in the superior quadrant but it also occurs as concentric constriction seen in our case.

In the patient with giant fusiform aneurysm headache is always seen and it's commonly ipsilateral but it's interesting our case didn't have a headache (5).

Since giant saccular aneurysms cause subarachnoid hemorrhage more than fusiform aneurysms, giant fusiform aneurysms rarely cause subarachnoid hemorrhage, intracerebral hemorrhage and cerebrospinal fluid circulatory problems (5).

Microsurgery provides great useful to aneurysm surgery interventions have to many problems. Only CCA occlusion is not enough for treatment of these aneurysms. But even in this state the aneurysm may become large.

The patient which good cross circulation is provided by preoperative angiography can tolerate the acute occlusion of unilateral carotid artery 890 percent.

ICA ligation can create unestimated ischemia and cerebral infarction. Also opening of carotid artery just after the occurring ischemia sign cannot cure whole neurological deficit. The long time ischemia sign cannot cure whole neurological deficit. The long time following shows us that the risk ischemia does not only contain short time, but also it can occur after a long period. The important thing is about stump pressure. It must be over 30 mmHg after ligation. Because EC-IC bypass can not prevent the ischemia which will occur.

Little et al (5). Published 6 b CA giant fusiform aneurysms two of which were non operated died, two cases received medical treatment and their statements were good, one case received ICA ligation, one case received trapping and thrombectomy. Both of them were in a good statement in the post operative period.

On our case visual acuity and visual field get better and CT sign shows that aneurysm is treated in a successfully way.

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