Turkish Neurosurgery 2008, Vol: 18, No: 4, 366-373

Review of a Series with Abducens Nerve Palsy

Abducens Paralizi Hastaların Gözden Geçirilmesi

Giyas AYBERK[1]
Mehmet Faik ÖZVEREN[2]
Timur YILDIRIM[3]
Karabekir ERCAN[4]
Emine KALKAN ÇAY[5]
Ayşegül KOÇAK[6]

[1,2,3] Atatürk Eğitim ve Araştırma Hastanesi, 2. Nöroşirürji Kliniği, Ankara, Turkey
[4] Atatürk Eğitim ve Araştırma Hastanesi, Radyoloji Kliniği, Ankara, Turkey
[5,6] Atatürk Eğitim ve Araştırma Hastanesi, Oftalmoloji Kliniği, Ankara, Turkey

ABSTRACT

OBJECTIVE: In this report, we aimed to investigate the patients that presented at our clinic complaint with diplopia due to the abducens nerve palsy and neurosurgical disease.

METHODS: The study design was a retrospective review of ten cases with the abducens nerve palsy. The causes of the abducens nerve paralysis of our patients were as follows: two cases with head trauma, three cases with pituitary tumors, one case with sphenoid sinus mucocele, one case with greater superficial petrosal nerve cellular schwannoma at the petrous apex, one case with hypertensive intraventricular hemmorhage, one case with hydrocephalus, and one case with parotid tumor and skull base/brain stem invasion.

RESULTS: Depending on the location of the lesion, the symptoms due to nuclear damage showed no improvement as in our case with adenocarcinoma of the parotid gland. The lesions sited at the subarachnoid portion of the abducens nerve or in the cavernous sinus, the abducens nerve palsy improved or botilinum injection was performed during recovery period.

CONCLUSION: We presented abducens nerve palsy cases due to neruosurgical disorders. A botilinum injection was performed in three patients with the abducens palsy. Botilinum injection can help patients with sixth nerve palsy during the recovery period.

KEY WORDS: Abducens nerve paralysis, Botilinum toxin, Diplopia

ÖZ

AMAÇ: Bu çalışmada, kliniğimize çift görme yakınmasıyla başvuran ve nöroşirürjik bir hastalığa bağlı olarak altıncı sinir parezisi saptanan hastalar incelenmiştir.

METOD: Çalışma, abdusens siniri paralizisi saptanan on vakanın retrospektif olarak incelenmesiyle gerçekleştirilmiştir. hastalarımızın abdusens paralizisinin nedenleri şunlardır: İki hastada kafa travması, üç hastada hipofiz adenomu, bir hastada sfenoid sinus mukoseli, bir hastada petroz apekse yerleşmiş greater superficial petrozal sinir sellüler şıvannomu, bir hastada hipertansif intraventriküler kanama, bir vaka hidrosefali ve bir hastada da parotis bezi adenokarsinomuna bağlı olarak gelişen kafa kaidesi ve beyin sapı invazyonu.

BULGULAR: Lezyonun yerleşim yerine bağlı olarak, parotid tümörlü hastamızda olduğu gibi semptomlar nükleer harabiyete bağlı ise düzelme gözlenmemektedir. Abdusens sinirinin subaraknoid kısmında veya kavernöz sinusta yerleşen lezyonlarda, abdusens paralizisi düzelmiştir ya da iyileşme sürecinde botilinum injeksiyonu yapılmıştır.

SONUÇ: Çalışmamızda, nöroşirürjik hastalıklara bağlı olarak gelişen abdusens siniri paralizileri sunulmuştur. Abdusens paralizili üç hastaya botilinum injeksiyonu uygulanmıştır. İyileşme fazında botilinum injeksiyonu, 6. sinir paralizili hastalara yardımcı olabilir.

ANAHTAR SÖZCÜKLER: Abdusens sinir paralizisi, Botilinyum, Diplopi

Received : 12.02.2008
Accepted : 18.08.2008

Correspondence address:
Giyas AYBERK
E-mail: giyas67@hotmail.com

INTRODUCTION

Lesions causing abducens nerve palsy may be located in the brain stem, subarachnoid space, petroclival region, cavernous sinus or in the orbit along the course of the nerve (14, 23, 15, 22, 18). The abducens nucleus is located in the pontine tegmentum, just ventral to the fourth ventricle, and axons of the seventh cranial nerve loop around the abducens nucleus. Fascicles emerging from the nucleus course forward through the pontine tegmentum to emerge from the ventral surface of the brain stem (43). The subarachnoid portion of the sixth nerve lies in the prepontine cistern, and also inside the Dorello's canal (20, 24, 46). Dorello's canal was described as a short, osteofibrous channel between the petrous apex and Gruber's ligament in earlier strudies (28). The abducens nerve flattens on the lateral side of the internal carotid artery (ICA) where the anastomosis point of the nerve with the periarterial sympathetic plexus of the ICA, and then course on the inferolateral side of the ICA in the cavernous sinus to enter to the orbita through the annulus of Zinn (27, 31).

The abducens nerve can be compromised in any of the above locations, as well as a consequence of generalized intracranial pressure increase (16). In this report, we present ten cases with sixth nerve palsy due to neurosurgical disorders, and discuss the mechanisms of abducens nerve palsy together with the treatment methods.

MATERIALS and METHODS

Ten cases with diplopia due to abducens nerve palsy were admitted to our clinic during the years 2006-2007. These patients were examined retrospectively. The diagnosis of the patients, neurological examinations, computerized tomography (CT) and magnetic resonance imaging (MRI) findings are presented in (Table I).

RESULTS

The results of treatment and follow-up of the patients are presented in (Table I).

DISCUSSION

Abducens nerve palsy is the most common encountered extraocular muscle palsy and its incidence is 11.3 in 100.000 people (30). The nucleus of the abducens nerve has been involved in congenital lesions such as the Mobius syndrome and Duane retraction syndrome type I (36). The functional loss of the abducens nucleus and fascicles generally results from insufficient vascular supply due to an ischemic syndrome, demyelinating disease, and intra-axial tumors in the brain stem (12). Extra-axial tumors may also invade the brain stem, and cause dysfunction of the abducens nucleus and its fascicles. In case 10, concomitant loss of both the facial and the abducens nerve functions because of the close proximity of their nucleus indicates a nuclear lesion (12). Depending on the location of the lesion, the symptoms due to nuclear damage showed no improvement in our case.

The anterior pontine membrane constitutes the anterior wall of the prepontine cisterns, and along with the abducens nerve invaginates in the petroclival dura mater (20). The potential subdural and the subarachnoid space, which is the continuation of the prepontine or cerebellopontine cisterns, also lies between the nerve and the arachnoid membrane within Dorello's canal (29). The dural and arachnoid layers form a strong barrier around the nerve for extra-dural tumors and distort the nerve at the petrous apex.

Parasellar extension is demonstrated in 6-10% of hypophysis tumors, and the amount of the extension is related to tumor size (3, 11 ,17). However, 30% of normal individuals have lateral expansion of the pituitary gland reaching the cavernous sinus without encasement of the intracavernous internal carotid artery more than 25% (8). The medial wall of the cavernous sinus forms a thick dural wall limiting the pituitary fossa. Since the pituitary adenomas are slowly growing tumors, and there is no dural barrier at the internal side of the diaphragma sella, the first structure compressed is the chiasm (44). Although the sixth nerve is more adjacent to the pituitary gland, the third nerve is the most involved cranial nerve in pituitary tumor (32). Ocular motor nerve involvement due to pituitary tumors generally results from an apoplectic event in the literature (39, 2, 19, 34, 9) (Table II). An ocular nerve palsy resulting from encroachment of the cavernous sinus is less common, but has been reported in 1-6% of the patients with pituitary tumor, and generally develops at the end stage of a pituitary tumor (44). We operated three patients with pituitary adenoma, and one of them also had a left temporal lobe arteriovenous malformation (case 5) (Figure 2). We did not detect pituitary apoplexy in any patient. The sixth nerve palsy improved on the second day of the

Table I: Clinical findings and results of our patients

Case No	Age (yrs)/ sex	Diagnosis	Neurologic examination	CT Findings	MRI findings	Causes of abducens nerve paralysis	Therapy	Follow-up
1	12, M	Head trauma	GCS 14, nuchal rigitidy, rt 6th nerve palsy	Diffuse SAH, and spheonooccipital suture diastasis (fig.1)	Mesencephalic contusion, epidural hematoma loacated at left anterior C1-C2 region, and arachnoid cyst at C7-T1	Spheooccipital suture diastasis	Medical	6th nerve palsy improved partially at sixth month
2	21, M	Head trauma	GCS 15, nuchal rigidity, lt 6th nerve palsy	Rt frontal contusion and frontoparietal acute subdural hematoma	-	Entrapment of the 6th nerve at entrance porus and petrous apex	Medical	6th nerve palsy did not improved at second month, and botulinum (Botox vial, Abdi İbrahim) injection was performed
3	36, M	Pit. adenoma	Lt 6th nerve palsy	-	Gr III E macroadenoma according to modified Hardy-Wilson classificaiton	Entrapment of the 6th nerve at occulomotor porus	Trsph surgery	6th nerve palsy improved at the second day of the operation
4	45, M	Pit. adenoma	Bilateral 3rd and rt 6th nerve palsy	-	Gr IVC macroadenoma according to modified Hardy-Wilson classification	Entrapment of the 6th nerve at occulomotor porus	Trsph surgery	Bilateral 3rd nerve palsy improved at the first postoperative day and rt 6th nerve palsy improved partially at fourt month of operation
5	55, M	Pit. adenoma and lt temporal AVM	Lt 6th nerve palsy	-	Gr IVE macroadenoma according to modified Hardy-Wilson classification, and GrIV AVM according to Spetzler-Martin classification (fig.2)	Entrapment of the 6th nerve at occulomotor porus	Transcranial adenoma, and AVM excision	6th nerve palsy did not improved at first mont of the operation
6	25, M	Sphenoid sinus mucocele	Rt 3rd and 6th nerve palsy	Sphenoid sinus mucocele	Sphenoid sinus mucocele (fig.3)	Spreading tromboflebitis toward inferior petrousal dinus	Trsph mucocele excision	3rd nerve palsy improved at the fifth day of operation, and rt 6th nerve palsy did not improved at sixth month, and botilinum injection was performed (fig. 6A, B).
7	16, F	Rt GSPN cellular schwannoma	Rt 6th nerve palsy, and xerophthalmia	Erosion in rt petrous apex	Cystic mass located at rt petrous apex and displacement of ICA medially. (fig. 4)	Entrapment of the 6th nerve at petorus apex	Subtemporal extradural tumor excision	Rt 6th nerve palsy did not improved at the second month of operation and botilinum injection was performed
8	46, M	IV hemorrha-ge	Rt 6th nerve palsy	IV hemorrhage, and hydrocephalus	-	Increased ICP	IV drainage, medical treatment	Died
9	43, F	Aqueduct stenosis	Rt 6th nerve palsy	Triventricular hydrocephalus	Triventricular hydrocephalus, and aqueduct stenosis	Increased ICP	V-P shunt revision	Rt 6th nerve palsy improved at the first week of operation
10	42, F	Matastasis of rt parotid adeno-carcinoma to the skull base and prepontine region	Rt peripheral 7th, 6th and lower cranial nerves palsies.	Bone erosion at the skull base	Tumor metastasis to the prepontin/pontocerebellar region and rt parietal region	Entrappment of the 6th nerve at prepontin cistern	Surgical excision of rt parietal and preopontin/pontocerebellar mass	Rt parietal and pontocerebellar mass partially excised, but the patient died as a result of tumor progression. Neurologic findings did not improved

Table II: Cranial nerve involvement in pituitary apoplexy and adenoma cases

Author	Number of patients	Involved cranial nerve palsies	Type of treatment	Follow-up and outcome
Agrawal D, Mahapatra AK. Surg Neurol 2005;63:42-46	23	IInd nerve involvement in 8 patients III, IV and VI involvement, bilateral in three, unilateral in two patients, isolated sixth nerve palsy in two patients	Transsphenoidal surgery	Range 3-15 months, return of vision in all patients, not mentioned about III, IV and VIth nerve
Lubina A, Olchovsky D, Berezin M, Ram Z, Hadani M, Shimon I. Acta Neurochir (Wien) 2005;147:151-57	40	IInd nerve involvement in 31 patients, and ocular paresis in 16 patients	34 patients underwent transsphenoidal surgery	Range 4-4.5 years, visual improvement appeared following surgery or medical intervention, visual field and ophthalmoplegia improved in 81% and 71%, respectively. Six patients treated conservatively and visuel field defect imroved in three, two of six patients had ocular paresis and improved in one
Bills DC, Meyer FB, Laws ER, et al. Neurosurgery 1993;33:602-609	37	2nd nerve involvement 64%, 3rd nerve involvement 57%, 4th nerve involvement 13%, 6th nerve involvement 30%	36 patients underwent transsphenoidal surgery	Degree of improvement for 2nd nerve releated with timing of surgery, ocular paresis improved 100% and not releated with timing of surgery
Yen MY, Liu JH, Jaw SJ. British J Ophtalmol 1990;74:188-91	3	3rd nerve involvement only	2 patients undervent transsphenoidal surgery, 1 patient underwent craniotomy	All third nerve palsies improved at follow-up

operation in case 3 and partially in case 4 at the fourth month after surgery. In case 5, the clinical findings did not improve at the first postoperative month.

Several mechanisms have been proposed for ocular motor nerve paralysis in hypophysis adenoma; transmission of the intrasellar pressure to the wall of the cavernous sinus, breaking of the wall of the cavernous sinus and direct compression of the nerve or its blood supply (40, 34). There are many entrapment points during the course of the sixth nerve. The first one is the dural entrance porus and the petrous apex, which acts as a fulcrum and may cause the sixth nerve injury (1, 27, 38). The second is the anastomosis between the sympathetic plexus on the lateral wall of the ICA and the abducens nerve (26, 27). In an autopsy study of patients who had died of severe head trauma, the abducens nerve was found to be significantly damaged at its anastomosis site with the sympathetic plexus and at the dura mater entrance porus in the petroclival area (33). Involvement of the periarterial sympathetic plexus around the ICA appears with the development of partial Horner's syndrome and accompanies the sixth nerve palsy (27, 37). Therefore, anastomosis of the abducens nerve with the periarterial sympathetic plexus may be responsible for the diplopia in our pituitary adenoma cases. Decompressing the pituitary gland may lead to removal of the trapping effect on the abducens nerve, and increase the expectation of resolution of the diplopia. In addition to the mechanisms described above, we

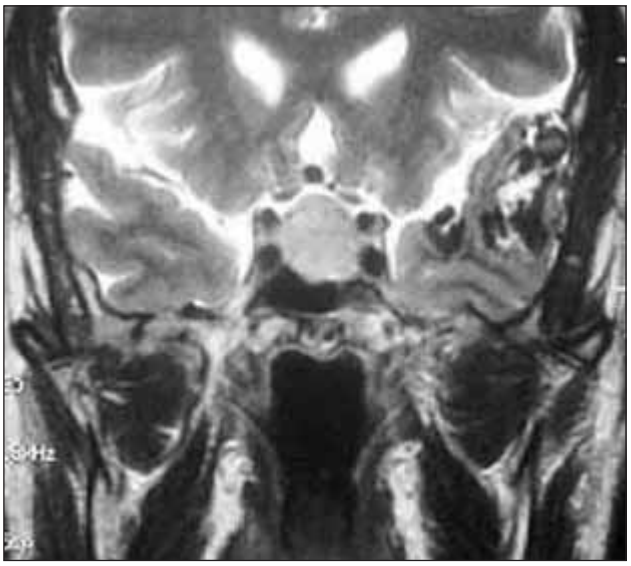

Figure 2: *T2W MRI of the patient shows the anterior communicating artery aneurysm, left temporal arteriovenous malformation, and Gr IV E hypophysis adenoma (case 5).*

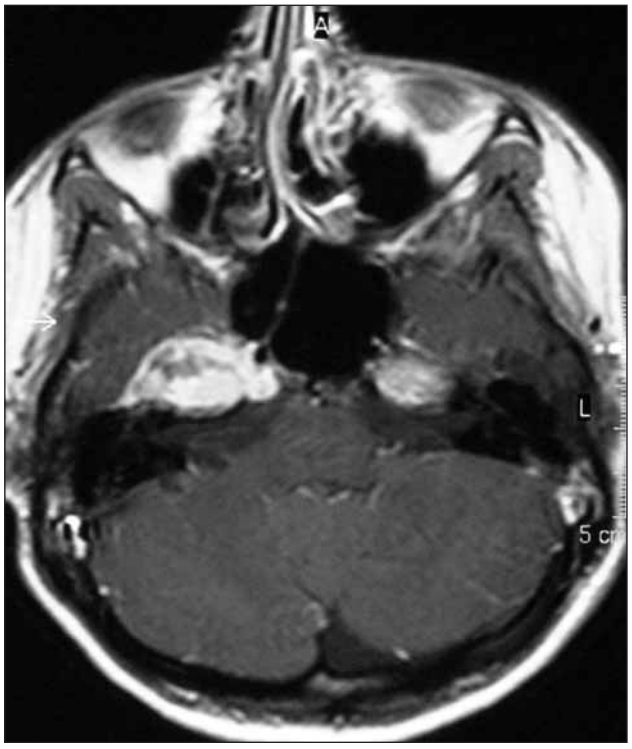

Figure 4: *T1W MRI of the right greater superficial petrosal nerve cellular schwannoma compressing the right abducens nerve at Dorello's canal (case 7).*

hypothesized two mechanisms that may cause the sixth nerve palsy. The first one is the distortion of the nerve by the pulsations of the ICA against the petrous apex. The second one is related to the anatomical spaces of the cavernous sinus. Anatomically, the cavernous sinus has three main venous spaces: medial, anteroinferior and posterosuperior. The hypophysis tumor that expands to the lateral side of the sella turcica reaches more easily toward the posterosuperior region of the cavernous sinus due to its anatomical peculiarity. The third nerve can be trapped at the posterosuperior side of the cavernous sinus where the oculomotor porus is located. Improvement of third nerve palsy immediately the day after the operation may be explained by compression of the nerve at the oculomotor porus.

In contrast to the pituitary tumor case, where the mass distorted the nerve from medial to lateral, the compression was lateral to medial in our case 7 (Figure 4). The sixth nerve was compressed by the tumor at the petrous apex. Petroclival dural entrance of the abducens nerve has a medial or lateral position on the clivus (28). It is assumed that the nerve becomes more susceptible to the lesions on the petrous apex in the laterally positioned porus (47).

Entrapment of the sixth nerve was reported around the dura mater entrance porus in the petroclival area and petrous apex in patients with severe cranial and cervical trauma (1, 27, 38). In Case 2, cranial fracture was not seen on CT and the cause of the sixth nerve palsy was entrapment of the nerve around entrance porus and petrous apex. The other trauma patient (case 1) was 12 years old and the sphenooccipital synchondrosis had not fused yet (Figure 1). Due to the close relation of the nerve with the clivus, sixth nerve palsy is seen frequently with clivus fracture. Clivus fractures can be seen in children as a sphenooccipital diastasis (13). Athough the patient had no evidence of diastasis, momentary diastasis during impact may have caused the sixth nerve palsy due to stretching of the nerve.

The sphenoid sinus is surrounded by the pituitary gland, the middle cranial fossa, and the optic nerves and chiasm superiorly; the cavernous sinus, the ICA, and the cranial nerves III to VI on both sides; and the nasopharynx, the pterygoid canals and nerves, and the pterygopalatine ganglion and artery anteriorly (41). The most common complications of sphenoid sinusitis are orbital and intracranial infection (45). Spread of infections to the orbit or cranium may be through congenital or acquired bony defects or through a thrombophlebitic phenomenon (45). Predisposing factors that cause

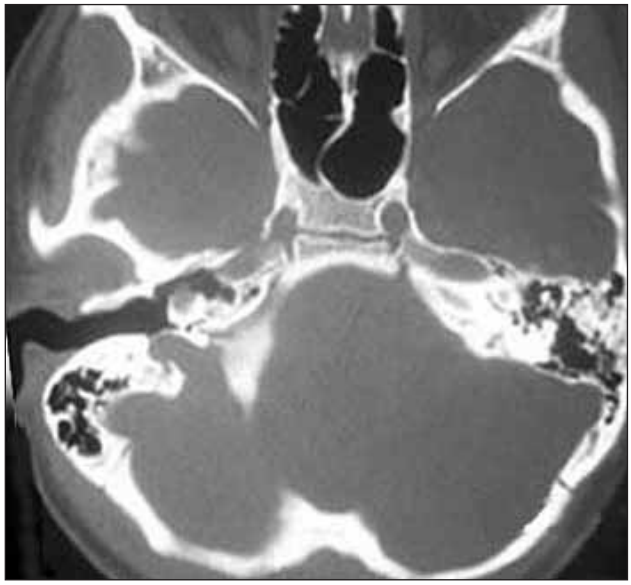

Figure 1: *CT of the patient reveals sphenooccipital suture diastasis (case 1).*

sphenoid sinusitis are well defined in the literature (7, 25). However, none of these factors could be found in our patient's history (Figure 3). The sixth nerve is the most frequently affected probably due to its medial location in the cavernous sinus (35). Progressive trombophlebitis affecting the inferior petrosal sinus may also lead to the involvement of the sixth nerve (7). The predominant pathogen is staphylococcus but a variety of organisms are detected (42). In isolated sphenoid sinusitis with VIth nerve palsy, first line treatment consists of conservative antibiotic therapy but we believe that surgical intervention is indicated when sphenoiditis is persistent even if the abducens palsy recovers. In this way, the focus of infection is effectively eradicated and recurrences are avoided.

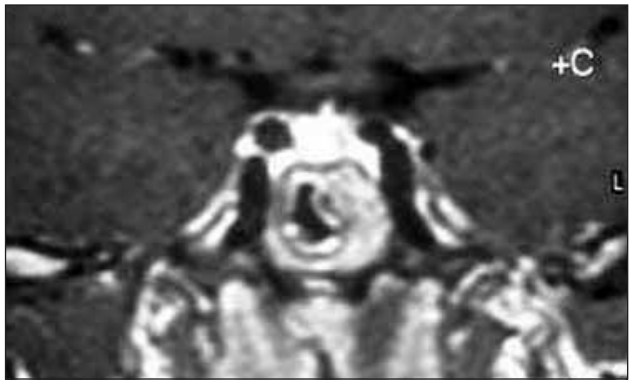

Figure 3: *Contrast enhanced MRI of the patient with sphenoid sinus mucocele shows debris in the sphenoid sinus (case 6).*

The botilinum neurotoxin has been advocated to weak the medial rectus muscle in patients with sixth nerve palsy from trauma, ischemia, inflamation, or tumors (10). Results have been reported as better than expected spontaneous recovery rate of 12-54% in the literature (10). When treated with botilinum toxin less than six months from the onset, recovery has been reported in 38-70% of cases, with a significant number achieving binocular fusion (6). The toxin is most commonly used in patients with lateral rectus muscle paresis in both its acute and chronic forms (4). It has been suggested that the botilinum toxin also has a role in preventing development of contracture in acute lateral rectus paresis (21). We injected botulinum (Botox® Vial, 100 units, Abdi İbrahim) into the medial rectus of our patients to prevent the development of contracture and to achieve binocular fusion (Figure 5). The diplopia disappeared completely on the 7th day. The effect of botulinum was fully evident after 3 months of follow-up. The function can be recovered by formation of new synaptic contacts, and this usually takes two to three months (21).

Figure 5: A*: The right lateral gaze palsy of the patient with sphenoid sinus mucocele (case 6).*
B*: The same patient after botilinum toxin injection..*

REFERENCES

1. Advani RM, Baumann MR: Bilateral sixth nerve palsy after head trauma. Ann Emerg Med 41:27-31, 2003
2. Agrawal D, Mahapatra AK: Visual outcome of blind eyes in pituitary apoplexy after transsphenoidal surgery: a series of 14 eyes. Surg Neurol 63:42-46, 2005
3. Ahmadi J, North CM, Segall HD, Zee CS, Weiss MH: Cavernous sinus invasion by pituitary adenomas. AJNR 6:893-898, 1985
4. Bentley C: Botilinum neurotoxin A in ophthalmology. Ophthalmic Physiol Opt 16:9-14, 1996
5. Bills DC, Meyer FB, Laws ER Jr, Davis DH, Ebersold MJ, Scheithauer BW, Ilstrup DM, Abboud CF: A retrospective analysis of pituitary apoplexy. Neurosurgery 33:602-609, 1993

6. Chuenkongkaew W: Botilinum toxin treatment for upper lid retraction of dystrophism. J Med Assoc Thai 86:1051-1054, 2003
7. Deans JA, Welch AR: Acute isolated sphenoid sinusitis: a disease with complications J Laryngol Otol105:1072-1174, 1991
8. Destrieux C, Kakou MK, Velut S, LeFranco T, Jan M: Microanatomy of the hypophyseal fossa boundaries. J Neurosurg 88:743-752, 1988
9. Douglas B, Fredric M, Edward L, Dudley D, Michael E, Bernd S, Duane I, Charles A: A retrospective analysis of pituitary apoplexy. Neurosurgery 33:602-609, 1993
10. Dutton JJ, Fowler AM: Botilinum toxin in ophthalmolgy. Surv Ophthalmol 52:13-31, 2007
11. Fahlbusch R, Buchfelder M: Transsphenoidal surgery of parasellar pituitary adenomas. Acta Neurochir (Wien). 92:93-99, 1988
12. Goodwin D: Differential diagnosis and management of acquired sixth cranial nerve palsy. Optometry 77:534-539, 2006
13. Guillaume D, Menezes AH: Retroclival hematoma in the pediatric population: report of two cases and review of the literature. J Neurosurg (4 suppl Pediatrics) 105:321-325, 2006
14. Heo JH, Bang OY, Choi SA: Pure motor hemiplegia with conjugate lateral gaze palsy in pontine lacunar infarction. Yonsei Med J 37:86-88, 1996
15. Kawase T, Shiobara R, Toya S: Anterior transpetrosal-transtentorial approach for sphenopetroclival meningiomas: surgical methods and results in ten patients. Neurosurgery 28:869-876, 1991
16. Keane JR: Bilateral involvement of a single cranial nerve: analysis of 578 cases. Neurology 65:950-952, 2005
17. Knosp E, Erich S, Klaus K, Christian M: Pituitary adenomas with invasion of the cavernous sinus space: A magnetic resonance imaging classification compared with surgical findings. Neurosurgery 34(4):610-618, 1993
18. Knox DL, Clark DB, Schuster FF: Benign VI. Nerve palsies in children. Pediatrics 40:560-564, 1967
19. Lubina A, Olchovsky D, Berezin M, Ram Z, Hadani M, Shimon I: Management of pituitary apoplexy: clinical experience with 40 patients. Acta Neurochir (Wien) 147:151-157, 2005
20. Matsuno H, Rhoton AL Jr, Peace D.: Microsurgical anatomy of the posterior fossa cistern. Neurosurgery 23:58-80, 1988
21. Metz HS, Dickey C: Treatment of unilateral acute sixth nerve palsy with botilinum toxin. Am J Ophthalmol 112:381-384, 1991
22. Nakagawa T, Uchida K, Ozveren MF, Kawase T: Abducens schwannoma inside the cavernous sinus proper: case report. Surg Neurol 61:559-563, 2004
23. Okada Y, Shima T, Nishida M, Okita S: Large sixth nerve neuroma involving the prepontine region: case report. Neurosurgery 40:608-610, 1997
24. Ono K, Arai H, Endo T, Tsunoda A, Sato K, Sakai T, Makita J: Detailed MR imaging anatomy of the abducens nerve: evagination of CSF into Dorello's canal. AJNR Am J Neuroradiol 25:623-626, 2004
25. Oruçkaptan HH, Akdemir P, Ozgen T: Isolated sphenoid sinus abscess: clinical and radiological failure in preoperatvive diagnosis. Case report and review of the literature. Surg Neurol 53:174-177, 2000
26. von Overbeeke JJ, Dujovny M, Dragovic L, Ausman JI: Anatomy of the sympathetic pathways in the carotid canal. Neurosurgery 29(6):838-844, 1991
27. Ozveren MF, Uchida K, Erol FS, Tifitkci MT, Cobanoglu B, Kawase T: Isolated abducens nerve paresis associated with incomplete Horner's syndrome caused by petrous apex fracture. Neurol Med Chir (Tokyo) 41:494-496, 2001
28. Ozveren MF, Uchida K, Aiso S, Kawase T: meningo-venous structure of the petroclival region: clinical impotance for surgery and intravascular surgery. Neurosurgery 50 (4):829-836, 2002
29. Ozveren MF, Erol FS, Aklan A, Onal C, Türe U: Microanatomical architecture of Dorello's canal and its clinical implications. Neurosurgery 2007;60(2 Suppl 1):ONS1-7; discussion ONS7-8
30. Patel SV, Mutyala S, Leske DA, Hodge DO, Holmes JM: Incidence, associations, and evaluation of sixth nerve palsy using population-based method. Ophthalmology 111:369-375, 2004
31. Rhoton AL: The cavernous sinus, the cavernous venous plexus, and the carotid collar. Neurosurgery (4 Suppl) 51:375-410, 2002
32. Robert CM Jr, Feigenbaum JA, Stern WE: Ocular palsy occuring with pituitary tumors. J Neurosurg 38: 17-19, 1973
33. Sam B, Ozveren MF, Akdemir I, Topsakal C, Cobanoglu B, Baydar CL, Ulukan O: The mechanism of injury of the abducens nerve in severe head trauma: a postmortem study. Forensic Sci Int 140:25-32, 2004
34. Saul RF, Hilliker JK: Third nerve palsy: the presenting sign of a pituitary adenoma in five patients and the only neurologic sign in four patients. J Clin Neuroophthalmol 5 (3):185-193, 1985
35. Sethi DS: Isolated sphenoid lesions: diagnosis and management. Otolaryngol Head Neck Surg 120:730-736, 1999
36. Traboulsi EI: Congenital abnormalities of cranial nerve development: Overwiev, molecular mechanisms, and further evidence of heterogeneitiy and complexity of syndromes with congenital limitation of eye movement. Trans Am Ophthalmol Soc 102:373, 2004
37. Türe U, Seker A, Kurtkaya O, Pamir MN: Internal carotid plexus schwannoma of the cavernous sinus: case report. Neurosurgery 52:435-438, 2003
38. Uzan M, Hancı M, Sarıoğlu AÇ, Kaynar MY, Bozkuş H: Bilateral traumatic abducens nerve paralysis with cervical spine flexion injury. Eur Spine J 5:275-277, 1996
39. Verrees M, Arafah BM, Selman WR: Pituitary tumor apoplexy: Characteristics, treatment, and outcomes. Neurosurg Focus (serial online). April 2004,16:Article 6. Available from: AANS.ORG. Free to public since 1996
40. Walsh FB: Bilateral total ophthalmoplegia with adenoma of the pituitary gland: Report of two cases; an anatomic study. Arch Ophthalmol 42:646-654, 1949
41. Wang ZM, Kanoh N, Dai CF, Kutler DI, Xu R, Chi FL, Tian X: Isolated sphenoid sinus disease:an analysis of 122 cases Ann Otol Rhinol Laryngol 111:323-327, 2002
42. Watkins LM, Pasternack MS, Banks M, Kousoubris P, Rubin PAD: Bilateral cavernous sinus thromboses and intraorbital abscess secondary to streptococcus milleri. Ophthalmology 110:569-574, 2003
43. Wilson-Pauwels L, Akesson E J, Stewart PA. Cranial Nerves, Anatomy and Clinical Comments. Philadelphia: B.C. Decker Inc, Toronto, 1988, 72-79
44. Yen MY, Liu JH, Jaw SJ: Ptosis as early manifestation of pituitary tumor. British J Ophthalmol 74:188-191, 1990

45. Younis RZ, Anand VK, Childress C: Sinusitis complicated by meningitis:Current managemet. Laryngoscope 111:1338-1342, 2001

46. Yousry I, Camello S, Wiesman M, Schmid UD, Moriggl B, Bruckman H, Yousry AT.: Detailed magnetic resonance imaging anatomy of the cisternal segment of the abducens nerve: Dorello's canal and neurovascular relationship and landsmark. J Neurosurg 91:276-283, 1999

47. Ziyal IM, Bozkurt G, Bilginer B, Gülsen S, Özcan OE: Abducens nerve palsy in a patient with a parasagittal meningioma, case report. Neurol Med Chir (Tokyo) 46, 2006