Aneurysmal Third Nerve Palsy

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To study the clinical characteristics, treatment and outcome of patients with aneurysmal third nerve palsy. Eleven patients with isolated third nerve palsy from an intracranial aneurysm from 1998 to 2002 at Ramathibodi Hospital were reviewed retrospectively. The average age was 60 years. Ipsilateral headache and/or retroorbital pain occured in 80 percent of the patients. Ten patients had unilateral disease, whereas one patient had bilateral involvement. The most common site of aneurysm was at the origin of the posterior communicating artery. Endovascular treatment with coil embolization provided successful occlusion as well as neurosurgical clipping. Recovery of third nerve function was found in all patients but had variable degrees. Patients who received early treatment, especially within 10 days after onset of oculomotor dysfunction appeared to have the best chance of recovery. Aberrant regeneration developed in 5 cases. The commonest sign was lid retraction during adduction and downward gaze.

Keywords : *Aneurysm, Third nerve palsy, Aberrant regeneration*

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Third cranial nerve palsy caused by internal carotid - posterior communicating aneurysm is one of the most challenging problems facing the ophthalmologist. This aneurysm is responsible for 13 - 30% of acquired oculomotor palsy[1]. Three clinical signs usually presented include ipsilateral facial, orbital or ocular pain, extraocular muscle and levator involvement and pupillary paresis. Urgent radiographic and neurosurgical evaluation is necessary because of the possibility of aneurysmal rupture and intracranial hemorrhage that carry a significant risk for morbidity and mortality.Treatment usually requires craniotomy and aneurysmal clipping and the recovery of neural function after surgery is well documented[2]. Recent reports indicated that endovascular management also produce effective neural recovery[3]. After damage to the third cranial nerve, regeneration may occur in a normal or an aberrant fashion. Some factors have been shown to be related with a degree of neural recovery such as duration of preoperative palsy and degree of preoperative deficit[1,4,5].

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This study reported the clinical characteristic of eleven cases of aneurysmal third nerve palsy during 5 years at Ramathibodi Hospital.

Patients and Method

The records of all patients who underwent operation for intracranial aneurysm over the past 5 years, from January 1998 through Deceber 2002, at Ramathibodi Hospital were reviewed in a search for individuals with isolated preoperative third nerve deficits for enrollment in this retrospective study.

A total of 12 patients were entered into the present study. The following information was abstracted from each patient' s chart: age; sex; underlying systemic and ocular disease; duration of illness before treatment; degree of third nerve palsy, ptosis, extraocular muscle (EOM) paresis, pupil size and reactivity; radiologic and angiographic findings; treatment and outcome; recovery of third nerve function and development of aberrant regeneration.

The authors defined EOM paresis as reduced ocular ductions of third nerve innervated striated muscles.The degree of EOM paresis was classified as either complete (duction completely abolished) or incomplete (duction incompletely abolished). The authors subdivided complete and incomplete palsy

into two patterns: diffuse, when all EOM were paretic and focal, when only some but not all the EOM were paretic.

Attempts were made to complete information by telephone contact with some patients that were lost to follow up.

Results

During the 5 year period, 12 eyes of 11 patients were diagnosed as having aneurysmal third nerve palsy. The study group consisted of 4 males and 7 females. The median age at first presentation was 60 years, ranging from 29 to 67 years. Five patients had underlying hypertension and 1 patient had hyperlipidemia with old cerebrovascular disease. The follow up period ranged from 8 to 43 months (average, 16 months).

Ten patients had unilateral disease with nine cases involving the right side and one involving the left. One patient had bilateral disease which initially involved the right side and subsequently involved the left side 9 years later. Ipsilateral headache and/ or retroorbital pain occurred in eighty percent of the patients. Five patients had third nerve palsy that coincided with the onset of pain. Four had head and eye pain for several days, ranging from 1 day to 2 weeks before ophthalmoparesis developed. Only two patients had no pain. The interval from onset of third nerve palsy to treatment ranged from 2 to 40 days with a median of 4 days.

At the time of initial presentation, ptosis with poor levator function was developed in all of patients. Severity of extraocular muscle dysfunction varied in each case. Complete diffuse third nerve palsy was found in 8 patients and diffuse incomplete palsy in 4 cases. Impairment of pupillary function, demonstrated by dilated, poorly responsive pupil was found in all the patients. The size of the pupil varied from 4 to 7 mm with an average of 4.9 mm.

Six patients initially were investigated by conventional computerized tomography (CT) scan and one case by magnetic resonance imaging (MRI) but no aneurysm was detected. Subsequent cerebral angiogram was performed at Ramathibodi Hospital in these cases and revealed positive results. An aneurysm was detected in the first investigation with MRI plus magnetic resonance angiography (MRA).

The site of the aneurysm was at the origin of the posterior communicating artery in 7 patients. In the remaining 4 patients, 3 were located at the supraclinoid portion of the internal carotid artery before bifurcation and 1 at the anterior choroidal artery.

Subarachnoid hemorrhage, that was detected by neuroimaging or xanthochromic cerebrospinal fluid after lumbar puncture, occurred during the development of oculomotor palsy in 6 patients.

Aneurysmal clipping was done in 7 patients and endovascular management with coil embolization was done in 4 patients with successful occlusion. After treatment all third nerve palsies improved. Seven patients (63%) had full recovery of levator function but mild residual ptosis was found in 4 cases. Three patients had a record of complete recovery of extraocular muscle involvement while the majority of patients had different degrees of residual ophthalmoparesis. Spontaneous partial recovery of ptosis and ophthalmoparesis with aberrant regeneration was found in 1 patient before treatment.

In the group of patients with incomplete recovery of third nerve function, there were only 5 cases who could be contacted by telephone. All of these patients developed aberrant regeneration. The most common signs was lid retraction during adduction and downward gaze. One patient was found to have miosis during adduction.

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Discussion

Third nerve palsy may be the only presenting sign of an intracranial aneurysm. About 13-30% of all third nerve palsy are due to aneurysms and posterior communicating artery aneurysm are implicated most commonly[1,6]. Patients with isolated third nerve palsy with pupillary involvement should be considered to be indicative of aneurysm until proven otherwise. Oculomotor paresis may result from compression of a nonruptured, expanding aneurysm or damage to the nerve at the time of aneurysm rupture. It is important to correctly diagnose these patients because the treatment of a symptomatic, nonruptured aneurysm provides the best chance for a favorable neurologic outcome. Missing a cerebral aneurysm that subsequently ruptures carries a significant risk for morbidity and mortality[7].

Various types of studies have documented relationships between the patients' age and gender and the risk of aneurysm formation. Most clinical series have a peak incidence of intracranial aneurysm in the fifth and sixth decade of life[8,9]. Consistent with previous reports, the disease occured in the presented patients with a median age of 60 years and has a female preponderance (females outnumbered males by 2.6:1). The majority of autopsy studies have documented an association between hypertension and aneurysm

formation[10,11]. From this study, The authors found hypertension in slightly more than half of the overall patients but up to 80 percent in the subarachnoid hemorrhage group. Systemic hypertension factor may be an important risk of aneurysmal bleeding.

Pain in the ipsilateral orbit or forehead is a prominent feature in the majority of aneurysmal third nerve palsy (80% in the present report) and is presumably related to involvement of the trigeminal fibers on the third nerve[7]. Severe headache, neck stiffness, or other meningeal signs raise the suspicion of an aneurysm in third nerve palsy patients. However, absence of pain does not exclude the diagnosis of aneurysm.

The diagnosis of an aneurysm must be made in a timely manner because the average time from the onset of third nerve palsy due to unruptured posterior communicating aneurysm to major subarachnoid hemorrhage is 29 days[12] Emergent neuroimaging should be performed in all suspected patients. Conventional CT scan and MRI are not the procedures of choice for detection of an aneurysm because of the high false negative rate ranging from 12-40%[7], as shown in some patients of the present study. Recent developments in neuroimaging studies have improved the investigation of intracranial aneurysms. Two evolving noninvasive technologies are magnetic resonance angiography and computed tomography angiography (CTA). The overall sensitivity for detection of an aneurysm in both MRA and CTA has been reported up to 97%. False negative cases were attributed to small aneurysms and unusual locations. The size limit for aneurysm detection is usually less than 5 mm. In the setting of negative neuroimaging (MRI/MRA or CTA) and high risk of aneurysm, consideration for conventional catheter angiography must be performed.

Treatment of a posterior communicating aneurysm should not be delayed. Treatment by neurosurgical clipping is widely advocated. Endovascular embolization and carotid occlusion previously offered alternatives to those patients unable to undergo surgical repair. Recent advances in endovascular approaches, particularly coil embolization, have resulted in a higher success rate[13] and more rapid recovery[3]. A lower success rate is still encountered with giant aneurysms, particularly those with broad necks[13,14]. In the present study, endovascular treatment provided a successful result as well as neurosurgical clipping. The recovery of neural function after treatment of an aneurysm is well documented in both groups.The return of third nerve function follows a predictable course: ptosis is frequently the first ocular sign to subside, with recovery beginning within the first month after surgery, and full recovery taking several months. Resolution of ptosis is usually complete, whereas extraocular muscle function frequently remains impaired[2-4]. However, the authors did not compare the result of both techniques because of limitation of the sample size and retrospective study.

The duration of preoperative palsy has been shown to be related with the degree of neural recovery. The authors found that 3 patients who were operated on within 10 days of onset of signs of third nerve palsy experienced complete recovery of neural function. The authors did not find an association between some factors such as preoperative deficit, present or absent of subarachnoid hemorrhage and degree of neural recovery. Recovery may occur spontaneously or after treatment of the aneurysm

Any recovery that is not complete within several months will be associated with secondary oculomotor nerve synkinesis[2,15]. This aberrant regeneration of the third nerve may indicate a long standing compressive lesion. The commonest signs in the present study were lid retraction during downward gaze (pseudo-Graefe sign) and lid retraction during adduction.

Conclusion

Aneurysmal third nerve palsy remains a diagnostic challenge for the ophthalmologist. MRA or CTA is a noninvasive, highly sensitive diagnostic tool in these patients. Neurosurgical or interventional neuroradiologic consultation will be needed if an aneurysm is discovered. Endovascular treatment provides a successful result as well as neurosurgical clipping. Patients who receive early treatment, within 10 days after onset of oculomotor dysfunction appear to have the best chance for recovery of third nerve function.

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อัมพาตของเส้นประสาทสมองคู่ที่ 3 จากหลอดเลือดโป่งพองในสมอง

พิศิษฐ์ ปรีชาวัฒน์, พลกฤษณ์ สุขขะวัชรินทร์, อนุชิต ปุญญทลังค์, อาภัทรสา เล็กสกุล

เพื่อศึกษาลักษณะทางคลินิก วิธีและผลการรักษาในผู้ป่วยที่มีอัมพาตของเส้นประสาทสมองคู่ที่ 3 จากหลอดเลือดโป่งพองในสมอง ได้ทำการศึกษาย้อนหลังในผู้ป่วยจำนวน 11 ราย ผลการศึกษาพบว่าอายุโดยเฉลี่ย 60 ปี 80 เปอร์เซนต์ของผู้ป่วยจะมีอาการปวดศีรษะหรือ ปวดด้านหลังลูกตาร่วมด้วย ผู้ป่วยจำนวน 10 ราย ป่วยเป็นโรคนี้เพียงข้างเดียวแต่มีผู้ป่วย 1 รายที่เป็นทั้งสองข้าง ตำแหน่งของหลอดเลือดโป่งพองที่พบได้บ่อยที่สุด คือบริเวณจุดกำเนิดของหลอดเลือด posterior communicating การรักษาโดยการผ่าตัดหนีบหลอดเลือด หรือ ใช้ขดลวดพบว่าได้ผลดีในการอุดหลอดเลือดที่โป่งพอง และผู้ป่วยทุกรายมีการทำงานของเส้นประสาทสมองคู่ที่ 3 ดีขึ้น แต่ในระดับที่แตกต่างกัน พบว่าผู้ป่วยที่ได้รับการผ่าตัดรักษาภายในระยะเวลา 10 วันหลังจากเริ่มมีอาการจะมีโอกาสฟื้นตัวของเส้นประสาทดีที่สุด มีผู้ป่วยจำนวน 5 รายที่พบว่าเกิดการฟื้นตัวของเส้นประสาทผิดทิศทาง โดยอาการแสดงที่พบได้บ่อยที่สุด คือ การที่มีหนังตาบนยกขึ้นในขณะที่ผู้ป่วยมองเข้าในหรือมองลง