Junctional Scotoma in Giant Cerebral Aneurysm

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A brain lesion located at the lateral side of the sella turcica can produce a junctional scotoma by compressing the ipsilateral optic nerve and the contralateral inferonasal nerve fiber. This study reports a female patient with a junctional scotoma caused by a cerebral aneurysm. At the initial visit, she complained of visual disturbance in both eyes and the right optic disc was atrophied. The visual field showed right blindness and left superotemporal quadrantanopsia. A brain CT indicated an approximately 3 cm sized brain mass located superolateral to the sella turcica. The brain MRI showed the lesion to be more like an aneurysm than a pituitary adenoma. Therefore, 4 vessels angiography was done, and this lesion was confirmed to be a sellar variant of an aneurysm located at the right carotid siphon. Like a tumor of the optic chiasm, a cerebral aneurysm can cause visual disturbance and visual field defects. Therefore, an early differential diagnosis is important because the prognosis and treatment of an aneurysm differ.

Key words: cerebral aneurysm, junctional scotoma, optic chiasm, superotemporal quadrantanopsia, visual field defect

INTRODUCTION

A brain lesion located at the lateral side of the sella turcica produces a junctional scotoma by compressing the ipsilateral optic nerve and the contralateral inferonasal nerve fiber. The most frequent cause of the junctional scotoma is meningiomas, but in this case the junctional scotoma was manifested by a cerebral aneurysm. This study reports a case of a junctional scotoma caused by a cerebral aneurysm.

CASE REPORT

A 65 year-old female patient with a junctional scotoma visited the ophthalmologic department complaining of a visual disturbance of both eyes. Two years ago, she had surgery for a right eye cataract. At the initial visit, her visual acuity was finger counting at 30cm in her right eye and 0.2 in her left eye. Right optic atrophy was found by a fundus examination (Fig. 1). The IOP was normal in both eyes at 15mmHg. There was no limitation in the motion of the extraocular muscle and there was no relative afferent pupillary defect.

The left eye had lens opacity. Therefore, phacoemulsification and intraocular lens implantation were done in the left eye. After the cataract operation, the best-corrected visual acuity of the left eye had improved up to 0.8. Because right optic atrophy was found by the fundus examination, we recommended a visual field examination after the cataract operation. In the visual field, there was right total blindness and left superotemporal quadrantanopsia.
Fig. 1. Fundus photographs of case. The right eye shows optic disc atrophy (Top) and the left eye is normal (Bottom).

(Fig. 2, 3). In the follow-up visual field examination, the finding of the junctional scotoma was same as the previous result (Fig. 4). The brain CT was checked and a neurosurgical examination showed a space-occupying lesion of brain. The brain CT showed an approximately 3cm sized brain mass located superolateral to the sella turcica (Fig. 5). The brain MRI (Fig. 6) showed that this lesion was more like an aneurysm than a pituitary adenoma. MRI showed the mass lesion to be more laterally deviated and pedunculated at its base. Finally, in the enhanced view, the central portion of the lesion was highly enhanced, and this centrally enhanced lesion and outer high signal lesion were delineated by a ring-like low signal intensity. These findings(target sign) suggested an aneurysm rather than a tumorous

Fig. 2. A right total field defect was found in the visual field (Humphrey field analyzer) of the right eye.

Fig. 3. A left superotemporal quadrantanopsia was found in the visual field (Humphrey field analyzer) of the left eye.
Fig. 4. In a follow-up visual field (Humphrey field analyzer) of the left eye, the left superotemporal quadrantopsia was same as in Fig. 3.

Fig. 5. In the coronal view of brain CT, an approximately 3 cm sized mass lesion was found at the right side sellar and the suprasellar area.

Fig. 6. In the coronal (Top) and sagittal (Bottom) view of the brain MRI, an approximately 3 cm sized mass lesion was found at the right side sellar area and the suprasellar area. In this post-enhance view, the wall enhancement and central high signal were delineated by a ring-like, low-signal intensity (target sign).

condition in the brain. Therefore, the 4 vessels cerebral angiography was checked. The 4 vessels angiography (Fig. 7) indicated this mass-like lesion was a sellar variant of a cerebral aneurysm located at the right carotid siphon.

**DISCUSSION**

A junctional scotoma is a typical visual field defect that is found at the condition space-occupy-
Fig. 7. In the coronal (Top) and sagittal (Bottom) view of 4 vessels angiography, an approximately 3 cm sized aneurysmal lesion was located at the right side internal carotid artery (carotid siphon).

ing lesion of brain. It is located at the lateral side of the sella turcica and compresses the ipsilateral optic nerve and inferonasal fiber of the contralateral optic nerve. Typically, a central scotoma in an ipsilateral eye and a superotemporal quadrantopsia in the contralateral eye are found in the visual field. The cause of this visual field defect is most often a meningioma at the lateral side of the sella turcica, but also it can be caused by a cerebral aneurysm.

Anatomically, the structures surrounding the optic chiasm are a vascular system such as the anterior cerebral artery, the anterior communicating artery, the internal carotid artery and the middle cerebral artery (branch of internal carotid artery), and the cavernous sinus and nervous system such as the optic, oculomotor, trochlear nerve and the ophthalmic branch of the trigeminal nerve. When these structures are compressed by a tumors condition, an inflammation or an aneurysm, various symptoms can develop depending on what site is compressed. In addition, when the optic nerve is involved, typical visual field change can develop.

Shannon\(^1\) reviewed the traditional junctional scotoma in which cases of an ipsilateral central scotoma and a superotemporal quadrantopsia in the contralateral eye were found in a giant cerebral aneurysm. These findings were also presented in our case report.

Jefferson\(^2\) reviewed 53 cerebral aneurysm cases. In addition, he reported 6 cases in which the aneurysm directly compressed the optic chiasm. Jefferson\(^2\) also classified the typical visual field defect in the cerebral aneurysm into three groups. He described the first group as a nasal field defect in the ipsilateral eye and a temporal field defect in the contralateral eye. The second group was described as a nasal field defect in both eyes and the third group was described as a nasal hemianopsia in the ipsilateral eye. However, these field defects were not found in our case. This is because in our case, the giant aneurysm was located at the lateral side of the sella turcica. Therefore, the aneurysm compressed the optic chiasm from the outer side.

Walsh and Hoyt\(^3\) reported a patient with dolichoectasia in the right internal carotid artery. They reported that at the first visit, a complete inferior field defect in the ipsilateral eye was noted and a superotemporal visual field defect in the contralateral eye was also found. They described the cause of the visual field defect in the contralateral eye as a compression of Wilbrand’s knee in the distal portion of the right optic nerve, adjacent to the optic chiasm. In our case, at first visit, a central scotoma in the ipsilateral eye and a superotemporal quadrantopsia in the contralateral eye were found.

Many authors also reported ipsilateral nasal field defect\(^4\) as the first sign of a giant cerebral aneurysm. However, this nasal field defect was not found in our case. This was because the right optic nerve was so severely compressed that optic atrophy
had developed. Furthermore, the visual field examination was done at the later stages therefore this first sign of a nasal field defect was not found.

Meadows\textsuperscript{12} reported 6 cases of optic atrophy in 15 patients diagnosed with carotid artery aneurysm placed in the cavernous sinus. In our case, the visual acuity in the ipsilateral eye was only finger counting at 30cm. Furthermore, optic atrophy was found in the right eye (ipsilateral side as Meadows’ report) (Fig. 1). It is believed that this was due to the right optic nerve compression by the cerebral aneurysm.

Bird\textsuperscript{13} described the causes of chiasm compressive lesions in 8 cases; 5 cases due to meningiomas, one case to craniopharyngioma, one case to an aneurysm of the anterior communicating artery and one case to carotid artery aneurysm. In those cases he also reported the importance of a differential diagnosis in junctional compressive disease. In our case, the visual field revealed the typical findings of a junctional scotoma (Fig. 2, 3). Moreover, we were suspicious of the space-occupying lesion in brain. Therefore, the patient underwent a brain CT. The brain CT showed an approximately 3cm sized mass-like lesion at the superolateral side of the sella turcica (Fig. 5). The radiological findings showed that a well-circumscribed solid-like mass lesion, sized 2.5 x 2 cm, was located at the sella turcica and superolateral side of the sella turcica, and this lesion was enhanced well. The brain MRI (Fig. 6) showed the mass lesion to be more laterally deviated and pedunculated at its base. Finally the enhanced view showed the central portion of the lesion was highly enhanced, and this centrally enhanced lesion and outer high signal lesion were delineated by a ring-like low signal intensity. These findings (target sign) suggested an aneurysm more than a tumorous condition in the brain. Consequently, we checked the 4 vessels cerebral angiography, which confirmed this mass-like lesion was a sellar variant of a cerebral aneurysm located at the right carotid siphon (Fig. 7).

Salinas-Garcia and Smith\textsuperscript{14} reported that a nasal field defect in the bilateral nasal hemianopsia was mostly due to intraocular lesions. They suggested that intraocular lesions included ischemic optic neuropathy, optic disc drusen, glaucoma, sector retinitis pigmentosa, and a congenital optic pit. In addition, they also reported that chronic papilledema can cause a nasal field defect. They concluded that the previously mentioned lesions must be considered as the cause of the visual field defect in the first place. However, in our case, there was no intraocular lesion causing visual field defect.

This case shows that in patients with a visual field change such as a junctional scotoma, other intraocular lesions that can cause a visual field defect should first be ruled out, and that when other lesions are ruled out, a cerebral aneurysm like brain tumor should be considered as a possible cause of the junctional scotoma.

Because the treatments differ according to the cause of the junctional scotoma, a differential diagnosis should be made carefully. Furthermore, when a central scotoma in the ipsilateral eye and a supertemporal quadrantopsia in the contralateral eye are typically found as a junctional scotoma, a space-occupying lesion in brain, a cerebral aneurysm should be considered as one of the causes of the junctional scotoma.

REFERENCES


