Case Report

Onodi Cell Mucocele-Associated Optic Neuropathy: A Rare Case Report and Review of the Literature

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Abstract

Purpose: To present a rare case report of Onodi cell-associated optic neuropathy, conducting a review of the literature.

Methods: A 36-year-old male presented with an 18-h history of acute deterioration of vision in his left eye (LE). Ophthalmic examination and Magnetic Resonance Imaging (MRI) were consistent with an Onodi cell-associated compressive optic neuropathy.

Results: Despite immediate, successful surgical decompression, severe optic nerve atrophy and permanent visual loss occurred during early postoperative period. The reported case gives rise to different hypotheses regarding pathophysiology that may lead to irreversible blindness. A systematic review of the respective literature is provided attempting to compare different approaches in the management of Onodi cell-associated compressive optic neuropathy and assess their efficacy in the final visual outcome. Poor initial visual acuity (VA) may represent a bad prognostic factor. Moreover, age and gender do not seem to significantly influence the outcome.

Conclusion: This report and associated literature review highlight the importance of the radiologic characteristics and early diagnosis in the final visual outcome of the Onodi cell-associated optic neuropathy. High level of suspicion is crucial for early diagnosis of mucoceles, which must be treated promptly by surgical and medical means to enhance visual recovery.

Keywords: Acute visual loss, Compressive optic neuropathy, Mucocele, Onodi cell

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Introduction

Mucocele is a benign and chronic epithelial-lined cystic lesion, arising at the expense of the paranasal sinus mucosa, usually containing sterile mucus. The vast majority of mucoceles arises from the frontal (65%) and ethmoidal sinuses (25%), as they are numerous with narrow ostia, in contrary to the sphenoid sinus (only 1–2%).[1-5]

The Onodi cell, initially described by Onodi in 1904,[6] is an anatomical variant, whereby during normal embryological development, the posterior ethmoid cell enlarges and pneumatises superolaterally into the sphenoid sinus, an area closely related to the optic canal, the optic nerve and the internal carotid artery.[7] It can, thus, represent a possible cause of retrobulbar optic neuropathy. Based on radiological findings, the incidence rate of the Onodi cell is calculated 8–24%, while in cadaveric studies, the prevalence rate has been reported to be up to 60%.[8,9]

Compressive optic neuropathy caused by an Onodi cell-related mucocele is an extremely rare complication with only a few reports in the literature. We herein present a rare case of unilateral, permanent visual loss, without any relevant ocular

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or nasal history, caused by compressive optic neuropathy due to an Onodi cell mucocele, despite urgent surgical intervention. This case highlights the importance of early diagnosis of the Onodi cell-associated optic neuropathy, based on clinical and radiologic findings, as well as prompt intervention with regards to final visual outcome.

Case Report

A 36-year-old male presented to our emergency department 18 hours after a sudden deterioration of vision in his left eye (LE). The patient had a clear ophthalmic and medical history, reporting no use of medication, tobacco, or alcohol. Upon presentation, best corrected visual acuity (BCVA) was measured 20/20 in the right eye (RE) and counting fingers (CF) in the left. A prominent relative afferent pupillary defect (RAPD) was noted on the left side. Ocular motility and main cranial nerves function were undisturbed, while slit-lamp biomicroscopy was unremarkable. Dilated fundoscopy was not diagnostic for optic disc edema or vein congestion, while the rest of the retina appeared normal [Figure 1a]. Requested Magnetic Resonance Imaging (MRI) of the brain and orbits revealed a hyper-dense cystic bilobed mass in the far posterior ethmoid cell. The lesion was located in the superior aspect of the sphenoid sinus and extended to the left orbital apex in close proximity to the left optic nerve [Figure 1b and c].

Based on clinical and radiologic findings, an urgent surgical intervention was scheduled to facilitate decompression of the optic nerve. The Onodi cell mucocele was efficiently evacuated through a left transnasal endoscopic approach, without any iatrogenic damage to the optic nerve, while its content was sent for microbiology evaluation. Cultures revealed the growth of *E. Coli*, Enterobacter aerogenes and coagulase negative Staphylococcus. Postoperatively, the patient was set on intravenous steroids (500 mg Methyl-prednisolone I.V./day) and antibiotics (Ceftriaxone 1000 mg I.V./twice a day). Upon discharge, he was prescribed per oral steroids in tapering doses and antibiotics (Doxycycline and Ciprofloxacin) for 2 weeks.

BCVA of the LE was handmotion (HM) atday1, deteriorated to perception of light (LP) at day 5, and remained unchanged throughout later follow-up visits, with the last being 3 years thereafter. Fundoscopy and fundus photography at 4 weeks postoperatively showed progressively established paleness of the optic disc [Figure 1d], while MRI sequences confirmed the complete evacuation of the cystic lesion [Figure 1e and f].

Discussion

There are numerous etiologies that are associated with the formation of mucoceles. Primary causes, such as secretory duct blockage and obstruction of mucus drainage, as well as secondary causes, such as sinus surgery and trauma, result in progressive accumulation of mucus and subsequent dilation of the lesion.[10] All these may result to local bone destruction, deformation, and progressive remodeling of the surrounding osseous walls.[11,12]

The potential pathophysiological mechanisms, responsible for the mucocele-associated optic neuropathy and visual loss, are not yet completely understood.[13] The optic nerve inside the optic canal is not surrounded by fat or other soft tissues. Consequently, the mucocele pressure is directly transferred

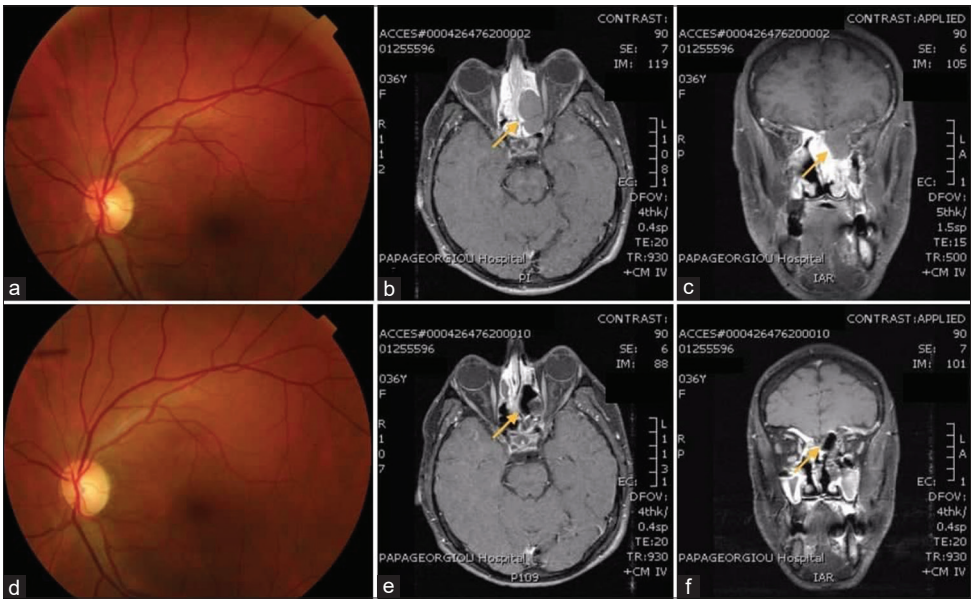

Figure 1: (a) Fundus photograph at baseline. No signs of optic nerve compression are noticed (b and c) magnetic resonance imaging (fat-saturated post contrast T1-weighted axial and coronal cuts, respectively) at presentation depicting an iso-dense bilobed cystic lesion lateral to the ethmoid sinuses, compressing the optic nerve inferomedially (orange arrow) (d) Fundus photograph at week 4 postoperatively. Progression of optic nerve pallor is noted (e and f) magnetic resonance imaging (fat-saturated post contrast T1-weighted axial and coronal cuts, respectively) at week 1 postoperatively. The area corresponding to the evacuated cyst is marked with the orange arrow

to the optic nerve, blood supply is thereby compromised, and subsequent optic atrophy may appear. Another possible explanation of the visual disturbances consistent with an Onodi cell mucocele is optic neuritis which can be caused by the respective inflammatory reaction. Microvascular changes and inflammatory factors are more frequently related to an acute onset and rapid progression of visual disturbances, rather than mechanical compression, which is mainly characterized by a gradual appearance of clinical symptoms.[13,14]

Clinical manifestations of paranasal mucoceles are variable and depend not only on the size of the mucocele but also on its location and direction of expansion. The most common ocular findings of fronto-ethmoidal mucoceles include diplopia, globe displacement, and increased intraocular pressure, due to the compression exerted on the eye.[10,13] On the other hand, Onodi cell and sphenoidal mucoceles appear as a more common cause of retrobulbar optic neuropathy, cranial nerve palsies, and acute visual loss due to their close anatomical relation and increased pressure exerted on cranial nerves.[15,16] Moreover, stretching of the dura and paranasal sinus mucosa may result in trigeminal nerve-mediated periorbital pain, which is very frequently reported by patients.[13,17]

Except for clinical symptoms and signs, imaging techniques, such as CT and MRI, play an essential role in the diagnosis of Onodi cell mucoceles, aiding in the differential diagnosis of similar clinical entities as well as optimal surgical planning.[18-20] Regarding MRI scans, Onodi cell mucoceles are optimally identified on axial images, where the track of the optic nerve in relation to the sphenoid sinus and the posterior ethmoid, can be better assessed.[19] Mucocele appearance varies on MRI and it depends on protein concentration, which alters over time. The initial high content of water results in hypointense T1 and hyperintense T2-weighted images, while the gradual rise of protein content may lead to a reverse intensity.[20]

In the literature, there are several reports of Onodi cell mucoceles that resulted in optic neuropathy. Hereby we provide an up-to-date review of the literature regarding this entity. Eligible articles were identified by a search of the bibliographic database in PubMed using the following combination of search terms: "Onodi cell-associated optic neuropathy" OR "Onodi cell AND optic neuropathy" OR "mucocele AND compressive optic neuropathy". The end date of the search period was June 3, 2019. We also checked all the references of relevant reviews and eligible articles that our search retrieved. Language restrictions were not used, and data were extracted from each eligible study by 3 investigators working independently (A.T., P.R., and A.D.). For each of the eligible studies, the following data were collected: lead investigator name; year of publication; journal name; demographic characteristics of the population being studied; symptoms at presentation; initial visual acuity; medical history; final visual outcome; final imaging outcome.

Table 1 summarizes all published relevant cases, presented in chronological order, providing additional information on the course of each case. Twenty-four cases (our case included) were identified according to the aforementioned inclusion criteria and were further analyzed. Patients' age at the time of presentation varied from 28 to 79 years with a mean age of 51.6 years. Nine cases were reported in female patients (37.5%) and 15 in males (62.5%), yielding no statistically significant difference in the mean age or final visual outcome between genders ($P > 0.05$, Mann-Whitney test). All visual acuities were converted to decimal system in order to facilitate comparison between cases.

In most of the reported cases, despite an initial visual decrease, a certain amount of visual recovery was noted after immediate surgical intervention and evacuation of the compressing mucocele.[2,5,17,21-28,31,36,37] However, it is of note that 37.5% of the reported cases in the literature (9/24, 7 males, 2 females, $P > 0.05$, Chi-square test) resulted in very poor visual acuity despite medical or/and surgical treatment.[3,15,29,30,32-35] In 6 of those cases, final VA was no LP, in two cases LP, and in one case 0.1 (decimal VA). In all the above-mentioned patients, initial VA upon presentation was extremely poor (≤ CF). On the other hand, the rest (62.5%) of the reported cases (15/24) demonstrated a complete (10/15) or fair (5/15) recovery of vision with or without peripheral visual field defects. In those cases, initial VA varied significantly between LP and 0.8 (decimal VA).

In our case, vision worsened rapidly, and despite immediate and efficient optic nerve decompression along with rapid administration of I.V. steroids and antibiotics, no visual improvement was reported. This condition led to unilateral visual loss along with established optic nerve atrophy. The rapid and painless visual loss may be suggestive of microcirculatory and inflammatory mechanisms involved in the optic neuropathy seen in our case. Moreover, the normal fundus findings at presentation, only 18 hours after acute visual loss was noticed, give additional evidence for minor mechanical optic nerve compression. One could also assume that an intraoperative, direct optic nerve trauma may have led to permanent visual loss. However, endoscopic videos of the operation were thoroughly reviewed and were not found to be suggestive of any iatrogenic optic nerve violation.

Apart from visual disturbance, the second most common symptom of Onodi cell mucocele was pain (13/24; 54.2%) appearing either as dull headache or as periorbital pain. Interestingly, RAPD was reported only in half of the cases (12/24; 50%), given the fact that almost all cases presented with visual acuity reduction possibly due to compressive optic neuropathy. Other less frequent symptoms were visual field constriction and double vision, which were reported in 7/24 (29.2%) and 3/24 (12.5%) cases, respectively. Interestingly in one case, reported by Fleissig *et al*.,[32] the patient complained not only about loss of vision, pain and diplopia, but also about eyelid edema and paresthesia in the region of V1 and V2 cranial nerves.

The mainstay of treatment in Onodi cell-associated optic neuropathy is urgent surgical decompression. Regarding

Table 1: Epidemiology and clinical characteristics of published cases

	Author, year	Age, sex	Presenting symptoms	Initial VA	Medical history	Course	Final VA
1	Ogata *et al*., 1998[17]	63, male	Recurrent optic neuropathy with visual loss Inferior VF defect	0.8		Improvement with steroids temporarily After surgery (ESS): VA=1.0, No VF defect (1 week later) Follow-up (6 months later): Stable	1.0
2	Klink *et al*., 2000[21]	41, male	Sudden VA loss RAPD Central scotoma of 20°	HM		After 1st surgery (ESS): VA=0.67, Small paracentral scotoma (9d later) Recurrence of mucocele, VA and VF: stable (3 months later) After 2nd surgery (ESS): VA=1.0, No paracentral scotoma (1 year later)	1.0
3	Kitagawa *et al*., 2003[22]	73, male	Headache Decreased vision RAPD Central scotoma	CF	Cataract surgery - phacoemulsification (1 week before)	After surgery (ESS) (7 days delay): VA=1.0 (1 month later) Follow-up (6 months later): Stable	1.0
4	Yoshida *et al*., 2004[23]	53, female	Retroorbital dull pain Blurred vision Inferomedial VF defect	CF		After surgery (pterional craniotomy) (3 weeks delay): Immediate VA recovery, VA=1.0, Normal VF (2 weeks later),	1.0
5	Yoon *et al*., [24] 2006	43, female	Sudden visual loss over 2 days RAPD Central scotoma	HM		After immediate surgery (ESS) and intravenous Methylprednisolone: VA=0.67, Mild temporal VF defect (6 months later)	0.67
6	Fukuda *et al*., 2006[5]	79, male	Bilateral visual loss Frontal headache Nausea	LP in both eyes	Glaucoma	Methylprednisolone: Visual Improvement (0.6 RE, 0.4 LE) After surgery (ESS): 0.9 RE, 0.8 LE Follow-up (5 years later): No recurrence	0.9 RE 0.8 LE
7	Toh and Lee 2007[2]	61, male	Sudden blurring of vision Pale optic nerve disc	0.2	Nasopharyngeal Ca treated with radiotherapy Polyps treated with endoscopic surgery	Amoxycillin-Clavulanate-Dexamethasone: VA=0.5 (3 days later) After surgery (ESS) (1 week delay): VA=0.67 (1 week later) Follow-up (1 year later): Stable - no further improvement	0.67
8	Toh and Lee 2007[2]	40, female	Headache Blurring of vision over 2 weeks RAPD Pale optic nerve disc Mild red desaturation	0.5		Amoxycillin-Clavulanate-Prednisolone (10 days course): No improvement (10 days later) After surgery (ESS): VA=0.8 (1 day later), 1.0 (2 week later), Normal color vision Follow-up (1 year later): Stable	1.0
9	Nonaka *et al*., 2007[3]	41, male	Pain VA decrease Optic disc edema	LP		Steroids: No improvement (5 days later) After surgery (ESS) (2 week delay): No VA improvement Follow-up (6 months later): Stable	LP
10	Lim *et al*., 2008[25]	60, male	Sudden VA loss Color vision decreased Pain RAPD Temporal VF defect	0.2	Nasopharyngeal Ca treated with Radiotherapy	After immediate surgery (ESS): VA=1.0, Normal color vision, Normal VF (Content: Purulent material)	1.0
11	Loo *et al*., 2008[15]	53, male	*First visit* Intermittent blurring Headache *Second visit* (5 months *later*) Loss of vision RAPD Proptosis Decreased motility	NLP	Endoscopic sinus surgery	After immediate surgery (ESS) and intravenous steroids-antibiotics: VA=NLP, Optic nerve atrophy (2 months later) (Content: Abscess)	NLP

Contd...

Table 1: Contd...

	Author, year	Age, sex	Presenting symptoms	Initial VA	Medical history	Course	Final VA
12	Chee and Looi 2009[26]	63, female	Acute horizontal diplopia - 6th nerve palsy Sudden VA loss RAPD Painless proptosis Decreased corneal sensation	CF		After surgery (lateral orbitotomy): VA 1.0, full recovery with antibiotics (Content: Abscess)	1.0
13	Fukuda *et al*., 2010[27]	45, female	Gradual visual loss over 1 year	0.67		After surgery (pterional craniotomy): VA Improvement (3 days later)	1.0
14	Wu *et al*., 2010[28]	28, male	VA loss Ocular pain and Headache RAPD Constricted VF on confrontation Pale optic nerve disc	0.2		Initial improvement with intravenous Methylprednisolone-Ceftriaxone After surgery (ESS) and intravenous Methylprednisolone-Ceftriaxone (5 weeks delay): VA=1.0, Clear VF (3 weeks later) Follow-up (1 year later): Stable (Content: Purulent fluid)	1.0
15	Nickerson *et al*., 2010[29]	51, female	Complete visual loss Diagnosis 5w later Optic nerve atrophy	NLP	Orbital fracture 38 years ago	After surgery (ESS) and steroids (7 weeks delay): Anatomical success, No VA improvement	NLP
16	Victores *et al*., 2012[30]	46, male	Blurring over 3 days Sudden VA loss	NLP	Chronic sinusitis Previous endoscopic sinus surgery	After surgery (ESS): No VA improvement (1 month later) Recurrence (eye pain and headache) (6 year later) (Content: Purulent and mucoid)	NLP
17	Taflan *et al*., 2013[31]	61, female	Acute visual loss Pain Optic nerve edema Macular star	0.1		After surgery (ESS) and intravenous antibiotics: VA=0.5 (10 days later)	0.5
18	Fleissig *et al*., 2014[32]	53, female	Sudden VA loss Pain in ocular movements RAPD Eyelid edema Diplopia Paresthesia V1 and V2 Eye movement limitation	CF	Chronic rhinosinusitis treated with endoscopic sinus surgery	After surgery (ESS) and methylprednisolone: NLP, Normal eye movements	NLP
19	Cheon *et al*., 2014[33]	60, male	Headache Visual loss	NLP	Brain infraction for 5 year	After surgery (ESS): No headaches, No visual improvement (Content: Fungal ball - Aspergilloma)	NLP
20	Rueping *et al*., 2014[34]	39, male	Acute visual loss RAPD Dull orbital headache	NLP	Endoscopic surgery 20 year a. for chronic sinusitis	After surgery (ESS): VA=NLP	NLP
21	Yen Nee See *et al*., 2016[35]	50, male	Acute painless loss of vision RAPD Pale optic nerve disc	CF		After surgery (ESS) and steroids (6 weeks delay): VA=0.1, No RAPD (5 months later)	0.1
22	Lee and Au 2016[36]	39, female	Progressive visual loss Retro-orbital pain Optic disc edema Decrease in color vision	0.08	Endoscopic surgery 10 years before for chronic sinusitis	After surgery (ESS): VA=0.67, Normal color vision, No optic disc edema Follow-up (6 months later): Stable	0.67
23	Kwon *et al*., 2019[37]	62, male	Double vision Trochlear nerve palsy Pathological ocular motility test	1.0		After surgery (ESS) and intravenous Dexamethasone: Orthotropia, Improvement of ocular motility (4 months later)	1.0

Contd...

Table 1: Contd...

	Author, year	Age, sex	Presenting symptoms	Initial VA	Medical history	Course	Final VA
24	Tzamalis A. 2019	34, male	Sudden VA loss RAPD	CF		After surgery (ESS) and intravenous antibiotics/steroids: VA=HM, optic nerve paleness Follow-up (3 years later): VA=LP, Stable (Content: Purulent fluid)	LP

VF: Visual field; VA: Visual accuity measure in decimal system; RAPD: Relative afferent pupil defect; HM: Hand motion; CF: Counting fingers; LP: Light perception; NLP: No light perception; ESS: Endoscopic sinus surgery; RE: Right eye; LE: Left eye

surgical techniques, endoscopic transnasal approach is the most commonly used due to its less invasive nature related to lower complication rates and quicker patient recovery.[27] In our review, the majority of patients (21/24; 87.5%) underwent endoscopic sinus surgery (ESS), 2 patients (8.3%) underwent pterional craniotomy, and just one patient (4.2%) was treated with lateral orbitotomy and drainage of the abscess.

A very important issue that needs to be further investigated is whether, and to which extent, delay of the surgical decompression could determine the final visual outcome. Kitagawa *et al*.[22] and Yoshida *et al*.[23] reported a complete visual recovery despite the fact that decompression surgery was delayed approximately 1 and 3 weeks after initial diagnosis, respectively. Moreover, Wu *et al*.[28] reported a postoperative visual acuity of 20/20 in their patient despite a 5 week delay of surgery. On the other hand, there are some reports with no visual recovery, despite early surgical decompression and intravenous treatment with steroids and/or antibiotics.

Furthermore, the role of corticosteroids and antibiotics in visual rehabilitation remains unclear, and future studies may investigate whether their use is beneficial in Onodi cell-associated optic neuropathy. Our systematic review has not identified any strong evidence for the efficacy of intravenous treatment with steroids or antibiotics with regards to the final visual outcome, which also may differ in cases of purulent mucoceles. In 7 cases with purulent content reported in the literature, only 3 of them (42.9%) achieved a complete visual acuity restoration, despite appropriate medical and surgical treatment.[25,26,28]

All the aforementioned reports give rise to further questions regarding pathophysiology of Onodi cell mucoceles that may lead to irreversible blindness despite early surgical intervention. Poor initial VA may represent a factor associated with poor prognosis. Moreover, age and gender do not seem to significantly influence the outcome. Further work needs to be carried out to establish the usefulness of steroids and antibiotic treatment regarding the final visual outcome. In conclusion, a high level of suspicion is crucial for early diagnosis of mucoceles, which must be promptly treated in order to enhance visual recovery.

Informed consent

The study was performed with informed consent and following all the guidelines for experimental investigations required by the Institutional Review Board of Ethics Committee of which all authors are affiliated.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given his consent for his images and other clinical information to be reported in the journal. The patient understand that name and initials will not be published and due efforts will be made to conceal identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

References

1. Devars du Mayne M, Moya-Plana A, Malinvaud D, Laccourreye O, Bonfils P. Sinus mucocele: Natural history and long-term recurrence rate. Eur Ann Otorhinolaryngol Head Neck Dis 2012;129:125-30.
2. Toh ST, Lee JC. Onodi cell mucocele: Rare cause of optic compressive neuropathy. Arch Otolaryngol Head Neck Surg 2007;133:1153-6.
3. Nonaka M, Fukumoto A, Nonaka R, Ozu C, Baba S. A case of a mucocele in an Onodi cell. J Nippon Med Sch 2007;74:325-8.
4. Kösling S, Hintner M, Brandt S, Schulz T, Bloching M. Mucoceles of the sphenoid sinus. Eur J Radiol 2004;51:1-5.
5. Fukuda Y, Chikamatsu K, Ninomiya H, Yasuoka Y, Miyashita M, Furuya N. Mucocele in an onodi cell with simultaneous bilateral visual disturbance. Auris Nasus Larynx 2006;33:199-202.
6. Onodi A. Die Sehstörungen und Erblindungnasalen Ursprunges, bedingt durch Erkrankungender hinteren Nebenhöhlen. Z Augenheilkd 1904;12:23-46.
7. Lang J. Clinical Anatomy of the Nose, Nasal Cavity and Paranasal Sinuses. New York: Thieme; 1989. p. 88-9.
8. Meybodi AT, Vigo V, Benet A. The onodi cell: An anatomic illustration. World Neurosurg 2017;103:950.e5-10.
9. Thanaviratananich S, Chaisiwamongkol K, Kraitrakul S, Tangsawad W. The prevalence of an onodi cell in adult Thai cadavers. Ear Nose Throat J 2003;82:200-4.
10. Kim YS, Kim K, Lee JG, Yoon JH, Kim CH. Paranasal sinus mucoceles with ophthalmologic manifestations: A 17-year review of 96 cases. Am J Rhinol Allergy 2011;25:272-5.
11. Kass ES, Fabian RL, Montgomery WW. Manometric study of paranasal sinus mucoceles. Ann Otol Rhinol Laryngol 1999;108:63-6.
12. Terranova P, Karligkiotis A, Digilio E, Basilico F, Bernardini E, Pistochini A, *et al*. Bone regeneration after sinonasal mucocele marsupialization: What really happens over time? Laryngoscope 2015;125:1568-72.
13. Tseng CC, Ho CY, Kao SC. Ophthalmic manifestations of paranasal sinus mucoceles. J Chin Med Assoc 2005;68:260-4.

14. Chagla AS, Bhaganagare A, Kansal R, Tyagi D. Complete recovery of visual loss following surgical treatment of mucopyocele of the anterior clinoid process. J Clin Neurosci 2010;17:670-2.
15. Loo JL, Looi AL, Seah LL. Visual outcomes in patients with paranasal mucoceles. Ophthalmic Plast Reconstr Surg 2009;25:126-9.
16. Moriyama H, Hesaka H, Tachibana T, Honda Y. Mucoceles of ethmoid and sphenoid sinus with visual disturbance. Arch Otolaryngol Head Neck Surg 1992;118:142-6.
17. Ogata Y, Okinaka Y, Takahashi M. Optic neuropathy caused by an isolated mucocele in an Onodi cell. ORL 1998;60:349-52.
18. Driben JS, Bolger WE, Robles HA, Cable B, Zinreich SJ. The reliability of computerized tomographic detection of the Onodi (Sphenoethmoid) cell. Am J Rhinol 1998;12:105-11.
19. Kantarci M, Karasen RM, Alper F, Onbas O, Okur A, Karaman A. Remarkable anatomic variations in paranasal sinus region and their clinical importance. Eur J Radiol 2004;50:296-302.
20. Lim CC, Dillon WP, McDermott MW. Mucocele involving the anterior clinoid process: MR and CT findings. AJNR Am J Neuroradiol 1999;20:287-90.
21. Klink T, Pahnke J, Hoppe F, Lieb W. Acute visual loss by an Onodi cell. Br J Ophthalmol 2000;84:801-2.
22. Kitagawa K, Hayasaka S, Shimizu K, Nagaki Y. Optic neuropathy produced by a compressed mucocele in an Onodi cell. Am J Ophthalmol 2003;135:253-4.
23. Yoshida K, Wataya T, Yamagata S. Mucocele in an onodi cell responsible for acute optic neuropathy. Br J Neurosurg 2005;19:55-6.
24. Yoon KC, Park YG, Kim HD, Lim SC. Optic neuropathy caused by a mucocele in an onodi cell. Jpn J Ophthalmol 2006;50:296-8.
25. Lim SA, Sitoh YY, Lim TC, Lee JC. Clinics in diagnostic imaging (120). Singap Med J 2008;49:84-8.
26. Chee E, Looi A. Onodi sinusitis presenting with orbital apex syndrome. Orbit 2009;28:422-4.
27. Fukuda H, Fukumitsu R, Andoh M, Suzuki T, Yamana N, Kataoka H, *et al*. Small onodi cell mucocele causing chronic optic neuropathy: Case report. Neurol Med Chir (Tokyo) 2010;50:953-5.
28. Wu W, Sun MT, Cannon PS, Jianbo S, Selva D. Recovery of visual function in a patient with an onodi cell mucocele compressive optic neuropathy who had a 5-week interval between onset and surgical intervention: A case report. J Ophthalmol 2010;2010:483056.
29. Nickerson JP, Lane AP, Subramanian PS, Izbudak I. Onodi cell mucocele causing acute vision loss: Radiological and surgical correlation. Clin Neuroradiol 2011;21:245-8.
30. Victores A, Foroozan R, Takashima M. Recurrent onodi cell mucocele: Rare cause of 2 different ophthalmic complications. Otolaryngol Head Neck Surg 2012;146:338-9.
31. Taflan T, Gūngōr I, Cengel Kurnaz S, Gūngōr L. Optıc neuropathy secondary to inflammation of sphenoidal sinuses and onodi cell polyps: A case report. Ocul Immunol Inflamm 2013;21:247-50.
32. Fleissig E, Spierer O, Koren I, Leibovitch I. Blinding orbital apex syndrome due to onodi cell mucocele. Case Rep Ophthalmol Med 2014;2014:453789.
33. Cheon YI, Hong SL, Roh HJ, Cho KS. Fungal ball within onodi cell mucocele causing visual loss. J Craniofac Surg 2014;25:512-4.
34. Rueping J, Kramer M, Kook D, Kampik A, Ulbig M, Haritoglou C. Young patient with acute monolateral amaurosis. Ophthalmologe 2014;111:664-6.
35. Yen Nee See W, Sumugam K, Subrayan V. Compressive optic neuropathy due to a large onodi air cell: A case report and literature review. Allergy Rhinol (Providence) 2016;7:223-6.
36. Lee JM, Au M. Onodi cell mucocele: Case report and review of the literature. Ear Nose Throat J 2016;95:E4-8.
37. Kwon KW, Oh JS, Kim JW, Yoo RE, Kang JG. Onodi cell mucocele causing isolated trochlear nerve palsy: A case report. Medicine (Baltimore) 2019;98:e15475.