CASE REPORT

Temporary Unilateral Amaurosis with Pneumosinus Dilatans of the Sphenoid Sinus

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Abstract—Pneumosinus dilatans (PSD), first described by Meyes in 1898, is an abnormal dilatation of one or more of the paranasal sinuses without bony erosion. The term sinus pneumocele indicates the presence of bony erosion. Review of the literature from 1968 to 1992 revealed 24 cases of idiopathic PSD and 17 cases of pneumocele of various paranasal sinuses. PSD occurred most commonly in the frontal sinus. Males were more often affected than females. The average age for males ranged from 16.5 for the maxillary sinus to 35.5 years for the sphenoid sinus. Pneumocèles occurred most commonly in the maxillary sinus. The average age for males ranged from 29 years for the frontal sinus and 47 years for the ethmoid sinus. Two patients (1 pneumocele and 1 PSD) had a temporary loss of vision. We report the case of a 37-year-old mountain climber who experienced temporary loss of vision in his left eye above 3000 m. Vision returned below 2000 m. High resolution computed tomography scan revealed pneumosinus dilatans of the sphenoid sinus with dehiscence of the optic canal on the left side. Endonasal microendoscopic resection of the anterior wall of the left sphenoid sinus was performed. The patient has remained symptom-free after 2 years of follow-up. Pneumosinus dilatans should always be considered in the differential diagnosis of sudden visual loss associated with atmospheric pressure changes. (*Skull Base Surgery*, 4(3):169–175, 1994)

Pneumosinus dilatans (PSD) is an abnormal dilatation of an otherwise normal paranasal sinus. This finding was first reported by Meyes in 1898¹ and classified by Benjamins in 1918.² Lombardi et al reviewed all patients with this rare abnormality and characterized its common features in 1968.³ Although the etiology of a PSD is not known, it has been described in association with many disorders: fibrous osseous dysplasia,^{4,5} brain anomalies such as hydrocephalus⁶ and arachnoid cysts,^{7–9} Klippel-Trenaunay (angio-osteohypertrophy),¹⁰ and Melnick-Needles syndrome (osteodysplasia-syndrome).¹¹ Several reports have postulated that a sphenoid PSD may be the first sign of a meningioma at the tuberculum sellae or the planum sphenoidale.^{12–18}

Clinically, a PSD can present with a variety of signs and symptoms ranging from facial swelling, especially

with frontal and maxillary sinus dilatation, to headaches or vertigo. Increasing loss of vision and reduction of visual fields is frequently found in association with PSD and meningiomas.^{12–18} In massive dilatation of the sinuses, proptosis¹⁹ or exophthalmus may be seen.^{19,20} If the hypophysis is compressed, there can be signs of endocrine disturbance.^{3,19} Pneumosinus dilatans may be found accidentally in asymptomatic individuals.^{21,22}

Benjamins² distinguished PSD from sinus pneumocele; he defined the latter as an enlarged sinus with a defect in the bony wall. Pneumocèles are most common in the maxillary sinus.^{23–35}

We report on the rare case of a patient with sudden reversible unilateral loss of vision and PSD of the sphenoid sinus of unknown etiology.

CASE REPORT

W.J. is a 37-year-old truck mechanic who experienced sudden blindness in the left eye and frontal headache in September 1990 while mountain climbing above 3000 m. Vision recovered completely after descending below 2000 m. An ophthalmological examination on the same day demonstrated no abnormality. A second climbing expedition again resulted in sudden loss of vision. A second ophthalmological and a neurological examination revealed no reason for the origin of the patient's complaints. A visit to an otolaryngologist demonstrated a normal examination except for a septal deviation. W.J. was referred to our department and a high resolution axial computed tomogram of the paranasal sinuses showed a PSD of the sphenoid sinus with a dehiscence of the optic canal (Fig. 1). Believing that the optic nerve was being compressed, we performed an endonasal microendoscopic resection³⁶ of the anterior wall of the left sphenoid sinus

combined with a septoplasty under general anesthesia. During surgery the left aspect of the sphenoid sinus was explored and found to be air filled, and an area of absent bone could be identified. We discharged W.J. after an uncomplicated postoperative course of 7 days. At an ambulatory follow-up 2 years later the patient reported no symptoms while climbing above 3000 m. Postoperative endoscopy showed a wide open, air filled sphenoid sinus. Vision was normal.

MATERIALS AND METHODS

We reviewed the literature from 1969 to 1992 using Medline. Pneumosinus dilatans and pneumocele were used as keywords. Additional articles were selected from references of the pertinent literature. A total of 47 articles were found, of which 3 were eliminated due to insufficient patient information (age, sex, location, etc) or because they were unnotable. We also eliminated publications in

Figure 1. Preoperative axial high resolution computerized tomograms of the paranasal sinuses of the patient W.J. A: Section below the optic canal showing the pneumosinus dilatans of the sphenoid sinus. B: Section at the level of the optic canal. The optic nerve is seen without bony cover in the sphenoid sinus (arrow).

which the same patients were reported on repeatedly. Thirty-eight publications remained, with a total of 75 patients (Table 1).

In reviewing the literature there seemed to be a difference between a pneumocele, which has a trap-valve mechanism and occurs more suddenly, and a PSD, which usually has a much longer history. Since the origin of a PSD is unknown and the distinction between a PSD and a pneumocele by a bony defect is very convenient, we decided to

use the original classification by Benjamins² and Jeans²⁰ to select cases of idiopathic PSD and pneumocele.

RESULTS

The literature review revealed 24 cases of an idiopathic PSD (Table 2). The frontal sinus was most affected

Table 1. Overview of Literature from 1969 to 1992 with Pneumocele and Pneumosinus Dilatans (PSD)

<i>Authors Year</i>	<i>Type</i>	<i>Sex</i>	<i>Age</i>	<i>Location</i>	<i>Bony Defect (Yes/No)</i>	<i>Symptoms</i>	<i>Origin</i>
Benedikt et al 1991	PSD	F	17	Ant.ethm L	Y	Nasal congestion, pain	Spont. drainage of mucocele
Borelli et al 1979	PSD	M	18	Sphenoid	N	Asymptomatic, epilepsy	Idiopathic
Bourdial 1970	PSD	M	45	Frontalis R	Not known	Rhinitis	Idiopathic
	PSD	M	20	Frontalis L	Not known	Swelling	Idiopathic
	PSD	F	70	Frontalis L	Not known	Meningitis	Idiopathic
Candan et al 1990	PSD	M	17	Frontalis R	N	Swelling, pain	Valve
Chan et al 1992	Pneumocele	M	33	Maxillar R	Y	Numbness, swelling	Valve
	Pneumocele	M	51	Maxillar R	Y	Swelling	Valve, radiation
Dhillon et al 1987	PSD	F	16	Maxillar L+R	N	Proptosis, loss of vision	Fibro-osseous disease
Dross et al 1992	PSD	F	35	Sphenoid L	N	Headaches, blurred vision	Arachnoid cyst
	PSD	M	37	Sphenoid L	N	Asymptomatic	Arachnoid cyst, adenoid CA
Gray et al 1978	Pneumocele*	F	44	Frontal L	Y	Proptosis, pain	Valve
Hirst et al 1979/82	PSD	M	18	Sphenoid	N	Loss of vision	Meningioma
	PSD	M	35	Sphenoid	N	Loss of vision	Meningioma
	PSD	F	14	Sphenoid	N	Loss of vision	Meningioma
Jarvis 1974	Pneumocele	M	18	Sphenoid, frontal R	Y	Proptosis, pain	Mucocele
Komori et al 1988	PSD	F	36	Maxillar R	N	Swelling	Idiopathic
Legent 1991	PSD	M	23	Frontal R	Not known	Swelling	Idiopathic
	PSD	M	16	Frontal L	Not known	Swelling	Idiopathic
	PSD	M	13	Maxillar R	Not known	Proptosis	Idiopathic
Leonardi et al 1976	PSD	M	47	Sphenoid	Not known	Loss of vision	Meningioma
	PSD	M	66	Sphenoid	Not known	Loss of vision	Meningioma
	PSD	F	66	Sphenoid	Not known	Loss of vision	Meningioma
	PSD	F	40	Sphenoid	Not known	Loss of vision	Meningioma
	PSD	F	22	Sphenoid	Not known	Loss of vision	Meningioma
	PSD	F	52	Sphenoid	Not known	Loss of vision	Meningioma
	PSD	F	35	Sphenoid	Not known	Loss of vision	Meningioma
	PSD	F	65	Sphenoid	Not known	Hemiplegia	Meningioma
Lloyd 1985	PSD	F	19	Ant ethmoid	N	Proptosis	Fibrous osseus disease
	PSD	M	24	Ant ethmoid	N	Proptosis	Ossifying fibroma
	PSD	F	15	Ant ethmoid	N	Proptosis	Meningioma
	PSD	F	17	Maxillar L+R	N	Proptosis	Fibrous dysplasia
	PSD	F	21	Maxillar L+R	N	Exophthalmus	Ossifying fibroma
	PSD	F	28	Ethmoid	N	Proptosis	Menigioma
Macialowicz 1969	PSD	F	76	Posterior ethmoid	N	Loss of vision	Idiopathic
Meyer-Breiting 1978	Pneumocele	M	47	Sphenoid Ethmoid R	Y	Chronic sinusitis Double vision	Spontaneous drainage of mucocele

(Continued)

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<i>Authors Year</i>	<i>Type</i>	<i>Sex</i>	<i>Age</i>	<i>Location</i>	<i>Bony Defect (Yes/No)</i>	<i>Symptoms</i>	<i>Origin</i>
Meyers et al 1980	Pneumocele	M	25	Maxillar R	Y	Swelling	Valve
Morrison et al 1976	Pneumocele	M	45	Maxillar L	Y	Pain, fullness	Valve
Noyek et al 1974	Pneumocele	M	35	Maxillar R	Y	Pain, fullness	Valve
Prott 1977	PSD	M	24	Frontal	Not known	Pain	Idiopathic
	PSD	M	28	Frontal	Not known	Swelling	Idiopathic
Reicher et al 1986	PSD	M	14	Sphenoid	N	Proptosis, loss of vision	Idiopathic
	PSD	M	45	Sphenoid	N	Sinusitis, headaches	Idiopathic
	Pneumocele*	M	26	Sphenoid	Y	Sinus headaches	Idiopathic
	Pneumocele*	F	31	Sphenoid L	Y	Headaches, glactorrhea	Idiopathic
Schayk et al 1992	PSD	M	17	Sphenoid		Asymptomatic	Hydrocephalus
	PSD	M	16	Fron, eth, sph	N	Asymptomatic	Hydrocephalus
Seur et al 1976	PSD	M	15	Sphenoid R	N	Frontal headaches	Arachnoid cyst
	(other case no true PSD)						
Seutin et al 1979	PSD	M	33	Sphenoid	Not known	Eye pain	Meningioma
	PSD	F	60	Sphenoid	Not known	Unspecific	Meningioma
	PSD	M	33	Sphenoid	Not known	Psychiatric disorder	Meningioma
	PSD	M	33	Sphenoid	Not known	Anosmia	Meningioma
Smith et al 1987	PSD	M	21	Frontal L+R	N	Swelling	Idiopathic
Som et al 1983	Pneumocele	M	48	Sphenoid	Y	Temporary loss of vision	Valve
Spoor et al 1981	PSD	F	18	Frontal + sphenoid	N	Loss of vision	Klippel-Trenaunay syndrome, meningioma
Steinberg 1969	PSD	M	26	Frontal R	Not known	Swelling	Idiopathic
Stretch 1992	PSD	M	17	Pansinus	N	Loss of vision	Melnick-Needles syndrome, osteodysplasia
							Arachnoid cyst
Strottman et al 1992	PSD	F	58	Ethmoid	N	Vertigo	
Sugita et al 1977	PSD	M	26	Sphenoid	Not known	Temp. bilat. loss of vision	Idiopathic
Tovi et al 1991	PSD	M	20	Maxillar L	N	Swelling	Idiopathic
Urken et al 1987	PSD	F	80	Frontal L	N	Asymptomatic	Idiopathic
	PSD	M	45	Frontal L+R	N	Asymptomatic	Idiopathic
	PSD	M	37	Frontal L	N	Swelling	Idiopathic
	PSD	F	56	Frontal	N	Swelling	Idiopathic
	Pneumocele	M	33	Frontal R	Y	Pain, swelling	Idiopathic
	Pneumocele	M	36	Frontal	Y	Pain, swelling	Idiopathic
Vines et al 1976	Pneumocele	F	62	Maxillar R	Y	Proptosis	Valve
Wiggli et al	PSD	M	47	Sphenoid	N	Loss of vision, hyposmie	Meningioma
	PSD	M	56	Dorsal ethmoid	N	Loss of vision, hyposmie	Meningioma
	PSD	M	67	Sphenoid	N	Loss of vision	Meningioma
	PSD	F	49	Dorsal ethmoid	N	Loss of vision	Meningioma
	PSD	M	56	Sphenoid	N	Loss of vision, anosmie	Meningioma
Williams et al 1975	PSD	M	73	Sphenoid	N	Loss of vision	Idiopathic
Wolfensberger et al 1987	Pneumocele	M	15	Maxillar R	Y	Pain, exophthalmus	Valve
Zismor et al 1975	Pneumocele	F	13	Maxillar R	Y	Nasal obstruction	Valve
						Exophthalmus	

*Defined as a PSD with a bony defect by author, reclassified as pneumocele.

Table 2. Patients with Idiopathic Pneumosinus Dilatans from the Literature 1969 to 1992 Inclusive Case Report

<i>Location</i>	<i>Male</i>	<i>Average Age (Years)</i>	<i>Female</i>	<i>Average Age (Years)</i>	<i>Total</i>
Frontal PSD	10	28.5	3	68.7	13
Maxillary PSD	2	16.5	1	36	3
Ethmoid PSD	—	—	1	76	1
Sphenoid PSD	6	35.5	1	76	7
Total	18	29.5	6	65.7	24
	Range = 13–73		Range = 36–80		
	Median = 25.0		Median = 73.0		

(54.2%), then the sphenoid sinus (29.2%). The ratio of females to males was 1:3 (Table 2).

Pneumoceles were most common in the maxillary sinus in males (35.3%). The ratio of females to males was 1:3.3 (Table 3).

DISCUSSION

Since the initial description of PSD there has been some controversy about defining this entity. Benjamins² first reviewed 5 cases of PSD (initially reported by Meyes in 1898¹) in the literature and added 1 case of his own, in which he differentiated PSD from pneumocele. Pneumosinus dilatans was defined as a large, air-containing sinus cavity that enlarges slowly. The bony walls are intact and there is no air outside the bony cavity or in soft tissue. There is no change in pressure. A sinus pneumocele or pneumatocoele was described as an enlarged sinus with air in soft tissues communicating through a hole in the bony wall of the sinus. There is air present in soft tissue on forced expiration. The symptoms have a sudden onset.^{2,20} In a recent publication Urken et al investigated 100 normal

frontal sinus films and calculated a 99th percentile for the “normal” expansion of a frontal sinus.³⁷ They suggested a new definition of “hyperpneumatization” or “hypersinus” for frontal sinuses which have developed beyond the upper limits of normal. These sinuses were aerated and their walls were normal. If the sinus walls were outwardly displaced, Urken et al defined it as PSD. If a bone defect was present it was described as a pneumocele. The authors postulated that the three forms of abnormally enlarged sinuses may be the same entity with different degrees of manifestation.²¹ Tovi et al³⁸ proposed the label “air cyst” to avoid confusion between different etiologies of abnormally large paranasal sinuses. Because PSD or pneumocele is a radiological diagnosis, Reicher et al¹⁹ and Dross et al⁸ favor the term PSD to describe a dilated sinus regardless of etiology or association. In 1968 Lombardi et al reported on 51 patients with pneumosinus dilatans. Thirty-nine of the cases involved the frontal sinus, 5 involved the anterior ethmoid sinus, 5 the posterior ethmoid and the sphenoid sinus, and 2 the maxillary sinus. They postulated that certain areas of the paranasal sinuses have a predilection for developing pneumosinus dilatans, such as the lateral recess of the frontal sinus, the superior recess of the maxillary sinus, the region of the sella turcica at the ethmoid-sphenoid junction, and the anterior ethmoids. In their series of PSD there were more males than females and patients ranged in age from 20 to 40 years. In our literature review of PSD a similar age and sex distribution was found. Males with PSD ranged in age from 13 to 73, with an average age of 29.5 years (Table 2). Pneumosinus dilatans was even more rare in females. The average age of the 6 female patients is 65.6 years. Because of the small sample size it is not clear if this represents a true difference in age distribution between males and females. The apparent rarity of documented ethmoid PSD (only one case reported³⁹) may be because of the difficulty of defining a PSD in the highly variable ethmoid sinus. Pneumocoeles seems to occur slightly more in males, although the average ages between the sexes are very similar.

The first case of a patient with unilateral temporary amaurosis in a sphenoid sinus pneumocele was described by Som et al.³² The patient reported blurred vision in his left eye during flying. Our patient reported no complaints when flying, probably due to adequate maintenance of cabin pressure. In a report by Som et al³² the computerized tomography revealed that the bone forming the sphenoid sinus wall was replaced by a thin soft-tissue membranous structure. In our case we were unable to demonstrate a loss of bone of the sphenoid sinus wall, except for the area we found along the optic nerve. However, Renn et al⁴⁰ reported in an anatomical study of 50 adult sellae that 4% of optic canals had bone defects exposing the optic nerve in the sphenoid sinus.

In our patient with PSD, ventilation of the sinus seemed to be disturbed. Two explanations are possible:

1. Air is temporarily trapped within the sphenoid sinus due to a valvelike mechanism at the os-

Table 3. Patients with a Pneumocele from the Literature 1969 to 1992

<i>Location</i>	<i>Male</i>	<i>Average Age (Years)</i>	<i>Female</i>	<i>Average Age (Years)</i>	<i>Total</i>
Frontal pneumocele	3	29	1	44	4
Maxillary pneumocele	6	34	2	37.5	8
Ethmoid pneumocele	1	47	—	—	1
Sphenoid pneumocele	3	30.7	1	31	4
Total	13	33.1	4	37.5	17
	Range = 15–51		Range = 13–62		
	Median = 33.0		Median = 37.5		

tium. A subsequent decrease in ambient pressure (as with high altitude) results in a relative increase in intrasinus pressure that compresses the optic nerve and its blood supply.

2. There is a decrease in pressure in all of the paranasal sinuses resulting in a suction effect on the optic nerve and its blood vessels. It appears that temporary blindness occurs *only* when the optic canal is dehiscence. This would also explain the extremely rare occurrence of such an event.

The first theory is favored by Sugita et al,⁴¹ who described a similar patient with bilateral transient amaurosis. They proved the postulated mechanism by placing the patient in a hyperbaric chamber and lowering the atmospheric pressure. We did not test our patient in a hyperbaric chamber because we saw the exposed optic nerve on the computed tomogram. Sugita et al did not state whether they believed that the symptoms resulted from excess or low pressure in the sphenoid sinus. As with our patient, Som et al³² used a transnasal approach to remove the anterior wall of the sphenoid sinus, and the patient remained free of symptoms. This procedure seems to establish a pressure equilibrium in our and other patients, but which mechanism was the cause of the optic nerve dysfunction is still speculative. In future patients with this rare entity, testing in a hyperbaric chamber while optic function is monitored may be helpful in clarifying the precise mechanism that causes the change in function of the optic nerve.

Pneumosinus dilatans can be classified as primary (idiopathic)^{19,21,22,39,42-49} or secondary.⁴⁻¹⁸ Secondary PSD can occur in fibrous-osseous disease^{4,5} and in congenital syndromes like Melnick-Needles¹¹ or Klippel-Trenaunay-Weber.¹⁰ Pneumosinus dilatans is frequently seen in patients with brain anomalies and long-term shunting, according to van Schayck and Niedeggen.⁶ They postulate that decreased intracranial pressure results in less compression of the surrounding skull bone and allows for more expansion of the sinuses.

Optic nerve meningioma with associated PSD must be considered in the differential diagnosis, especially when vision decreases slowly.¹²⁻¹⁸ This could be excluded in our patient by high resolution computed tomography. Meyer-Breiting reported a patient with transient decrease in vision, double vision, and proptosis with chronic polypoid maxillary and ethmoid sinusitis and PSD. He noted a spontaneous drainage of a mucocele as the etiology of the PSD.²⁷ Benedikt et al⁵⁰ also discussed spontaneous drainage of ethmoid sinus mucocoele as a possible cause of PSD. In contrast, our patient had no signs of inflammation.

Creation of a wide communication between the sinus and the nasal cavity was curative in all patients with PSD and pneumocele.^{32,41} In our patient a microendoscopic resection of the anterior wall allowed proper pressure equilibrium in the sphenoid sinus and prevented permanent optic nerve damage. In patients with disturbing cos-

metic deformity due to bulging in the frontal and maxillary sinuses, resection of part of the enlarged sinus has been shown to improve cosmetic appearance.^{39,42,44-48}

CONCLUSION

Temporary blindness associated with dilatation of the sphenoid sinus is extremely rare. According to the literature available to us, our case is only the third one. The patient is a 37-year-old male with PSD of unknown etiology; however, a valvular mechanism was postulated. In addition, the optic nerve was not covered by bone within the sphenoid sinus. A similar case described by Sugita et al⁴¹ presented with bilateral amaurosis. In patients with unclear recurrent loss of vision, dilatation of the sinuses, pneumosinus dilatans, or pneumocele should be considered in the differential diagnosis. Intracranial masses such as arachnoidal cysts or a meningioma should be ruled out by appropriate imaging studies.

In patients with a PSD of the sphenoid sinus and transient amaurosis, endonasal microendoscopic resection of the anterior wall of the sphenoid sinus establishes permanent pressure equilibrium and prevents permanent damage of the optic nerve.

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REVIEWER'S COMMENTS

The authors report a relatively unique case of apparent pneumosinus dilatans affecting the sphenoid sinus such that there was lack of bone along the medial wall of the left optic canal. The patient experienced two episodes of transient loss of vision in the left eye during mountain climbing. Following endoscopic surgery to remove the anterior wall of the sphenoid sinus, the patient had no further episodes of visual loss. The authors postulate a ball valve effect of trapped air at normal atmospheric pressure maintaining pressure against the intracanalicular portion of the optic nerve as transferred through the area of absent bone while the rest of the nerve is under reduced atmospheric pressure. Although it is unclear to me whether or not this is the explanation, it would seem clear from this case as well as several others reviewed by the authors that pneumosinus dilatans may produce both permanent and transient visual loss when the process affects the sphenoid region and that such visual loss can occur in the absence of any other associated lesion (eg, a meningioma). Furthermore, decompression of the sphenoid sinus seems to be effective in eliminating the episodes of transient visual loss and should be considered in all such cases. Finally, the authors have done an excellent job in reviewing cases of PSD in the literature and of describing their location, associated lesions, clinical manifestations, and treatment. This article is a welcome addition to the literature on this most interesting syndrome.