



# Carotid canal and optic canal at sphenoid sinus

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## Abstract

In the present study, we investigated the relationship between sphenoid sinus, carotid canal, and optic canal on paranasal sinus computed tomography (PNSCT). This study was performed retrospectively. PNSCT images of 300 adult subjects (159 male, 141 female). Sphenoid sinus (pneumatisation, dominancy, septation, inter-sinus septa deviation), anterior clinoid process pneumatisation, Onodi cell, carotid and optic canals (width, dehiscence, classification) were measured. In males, type 3 pneumatised sphenoid sinus (in both sides) and in females type 2 pneumatised sphenoid sinus (right side) and type 3 pneumatised sphenoid sinus (left side) were detected more. Anterior clinoid pneumatisation was present 47.2% in males and 39.7% in females. In male group, more septation (i.e. 22.6%,  $\geq 3$  septa) in sphenoid sinus were detected. Onodi cell was present 26.6 and 19.1% in males and females, respectively. Carotid canal protrudation to the sphenoid sinus wall was present 23.9–32.1% in males and 35.5–36.2% in females. Dehiscence in carotid canal was detected more in females (34%) compared to males (22%). Optic canal protrudation was 33.3 and 30.5% in males and females. Type 4 optic canal was detected more in both gender. Optic canal dehiscence was detected 11.3 and 9.9% in males and females. Carotid and optic canal diameters were higher in males. In pneumatised sphenoid sinuses and in females, type 3 carotid canal (Protrudation to SS wall) (bilaterally) and type 1 optic canal type (No indentation) (ipsilateral side) were detected more. In elderly patients, carotid and optic canal width increased. When carotid canal protrudation was detected, there was no indentation in optic canals In pneumatised SS, carotid canal protrudation was observed with a greater risk in surgery. However, type 1 (non indentation) optic canal was present in highly pneumatised SS with lower risk for the surgery. In women, the risk of carotid canal protruding (about 1/3) is greater than that of males, and carotid canal dehiscence rates are also higher in females. Therefore, physicians should be very careful during the preparatory stages of the sphenoid sinus surgery. Otherwise, it may not be possible to prevent lethal carotid artery bleeds.

**Keywords** Sphenoid sinus · Carotid canal · Optic canal · Anterior clinoid process · Pneumatisation · Dehiscency · Onodi cell

This study is retrospective. Ethics committee approval was obtained from Adana City Training and Research Hospital, Clinical Researches Ethics Committee was also taken (Date: 27.03.2018, Number:184)

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## Introduction

Internal carotid artery or optic nerve injury is a serious complication of transsphenoidal surgery [1, 2]. To reduce surgical complications, sphenoid sinus and related regional anatomy should be known for transsphenoidal and functional endoscopic sinus surgery [3–7]. Pneumatisation of the sphenoid sinuses may be shown on a high-resolution CT scan at 2 years of age. Pneumatisation proceeds more down and posterolateral direction. The sinus reaches its mature size until the age of 14 [8]. The degree of pneumatisation of the sphenoid sinus is highly variable [9].

The pneumatisation of SS sometimes extends into “the vomer, palatine bone, ethmoid bone, occipital bone, anterior clinoid processes, the lesser wings, the greater wings, the pterygoid process and plates, posterior clinoid processes, and the clivus” [10, 11]. Depending on the degree of sinus pneumatisation, the bone covering the carotid arteries, optic nerves, maxillary nerves and vidian nerves may be thin or absent, making these structures susceptible to iatrogenic damage [7, 12].

Intranasal septa can have posterior insertion on the internal carotid artery canal, especially in the bulging variant, increasing the risk of damage in the vessel with dramatic consequences. The optic nerve and the internal carotid artery may present bulging and dehiscence in the sinus [13, 14].

In the present study, we investigated the relationship between sphenoid sinus, carotid canal and optic canal.

## Materials and methods

This retrospective study was conducted in Kırıkkale University Faculty of Medicine according to the principles of the Declaration of Helsinki. Paranasal Sinus Computed Tomography (PNSCT) images were obtained from database of the Kırıkkale University Faculty of Medicine Radiology Department. Ethics committee approval was obtained from Adana City Training and Research Hospital; Clinical Researches Ethics Committee was also taken (Date: 27.03.2018, Number:184).

## Subjects

This study was performed retrospectively. PNSCT images of 300 adult subjects (159 male, 141 female), between 18 and 86 years old, were randomly selected from a digital radiology database of all PNSCT in Kırıkkale University Faculty of Medicine Radiology Department. Mean ages of the males were  $33.7 \pm 13.7$  years; and of the females were  $33.4 \pm 14.7$  years. Most of the patients' PNSCT indication was the suspect of sinusitis or nasal septal deviation. However, the

PNSCT results showed no sinusitis. Nasal septal deviation positive patients were included into the study.

Subjects with previous trauma or surgery history, sinonasal tumour, acute and chronic rhinosinusitis, sinonasal polyposis, CSF leak, and marked facial deformity were not included to the study.

## CT imaging and analysis

All of the scans were obtained with routine PNS-computed tomography imaging in the supine position, with no contrast or sedation being used for the procedures. The images were acquired using a 64-slice CT (MSCT; Brilliance 64, Philips Medical System, Best, the Netherlands). All of the scans were obtained using the following parameters: tube voltage = 120 kV, effective mAs = 350, slice thickness = 1.00 mm, field of view (FOV) = 180 mm and image matrix =  $768 \times 768$ . The images were transferred to a commercially available workstation, and the raw data was reconstructed using bone algorithms. After scanning, the coronal, axial, and sagittal images were reconstructed with a slice thickness of 1.00 mm. The coronal and axial plan was often preferred.

The following measurements were performed:

1. Sphenoid sinus pneumatisation (SSP): it was classified as types 1 to 3 at coronal plane (Fig. 1) [15]:

Type 1: SSP extends medially to the Vidian canal.

Type 2: SSP extends medially to the foramen rotundum.

Type 3: SSP extends laterally to the foramen rotundum.

2. Anterior clinoid process pneumatisation (ACPP): it was classified at coronal plane as 0: absent, 1: right, 2: left, 3: bilateral (Fig. 2).
3. Sphenoid sinus dominancy: it was classified as 0: none/or not available due to multiple septation, 1: right dominancy, 2: left dominancy.
4. Sphenoid sinus septation: it was classified as 0: no septation, 1: incomplete septa, 2: one complete septa (Fig. 3), 3: one complete + one incomplete septa, 4: double septa, 5:  $\geq 3$  septa (Fig. 2) [16].
5. Sphenoid sinus inter-sinus septa deviation: it was classified as type 1: no deviation (Fig. 4), type 2: deviation to the right, type 3: deviation to the left, type 4: “S” shaped deviation.
6. Onodi cell: it was evaluated at coronal slice as 0: absent, 1: present (Fig. 5).
7. Optic canal width: at coronal plane, both optical canals were measured as mm at the widest site internally without including bone structure (Fig. 3).
8. Optic canal dehiscence: it was classified at coronal plane as 0: absent, 1: right, 2: left, 3: bilateral (Fig. 6).

9. Optic canal classification: both optical canals were classified according to the relationship with the sphenoid sinus wall (Fig. 1) [17, 18]:

Type 1: No indentation.

Type 2: Indentation to SS wall.

Type 3: Protrusion to SS wall.

Type 4: Extends laterally to the sphenoid sinus and posterior ethmoid cell (Fig. 7a).

10. Carotid canal width: at coronal plane, both carotid canals were measured at the level associated with the sphenoid sinus wall as mm at the widest site internally without including bone structure (Fig. 4).
11. Carotid canal dehiscence: it was classified at coronal plane as 0: absent, 1: right, 2: left, 3: bilateral (Fig. 8).
12. Carotid canal classification: both carotid canals were classified according to the relationship with the sphenoid sinus wall (Figs. 8 and 9):

Type 1: No indentation.

Type 2: Indentation to SS wall.

Type 3: Protrusion to SS wall (Fig. 7b).

## Statistical analysis

SPSS for Windows 16.0 (SPSS, INC, an IBM Company, Chicago, Illinois), Mann-Whitney *U* test, Chi-square test, independent sample *t* test and Pearson correlation test were used.

*p* value < 0.05 was considered as statistically significant.

## Results

Sphenoid sinus pneumatisation and septation, anterior clinoid process pneumatisation, values in males and females are shown on Table 1:

SS pneumatisation: In males, type 3 (37.7%) and, in females, type 2 pneumatisation (41.8%) were detected more on the right; and type 3 pneumatisation (39.0% in males and 48.9% in females) was detected on the left ( $p > 0.05$ ) (Table 1). Anterior clinoid pneumatisation was present 47.2% in males and 39.7% in females. In both males and females, bilateral pneumatisation was detected ( $p > 0.05$ ) (Table 1).

Anterior clinoid process pneumatisation: Anterior clinoid pneumatisation was present 47.2% in males and 39.7% in females. In both males and females, bilateral pneumatisation was present in 25.7 and 22.7%, respectively ( $p > 0.05$ ) (Table 1).



**Fig. 1** On coronal PNSCT, bilateral anterior clinoid process pneumatisation (\*), protrusion of the bilateral optic canal to the sphenoid sinus wall (type 3) (white thin arrow) and bilateral sphenoid sinus pneumatisation extends to foramen rotundum lateral (type 3) (white thick arrow) are shown

SS septation: In males, one complete septa (34.6%) and  $\geq 3$  septa (22.6%) in males and one complete septa (41.8%) and 1 complete + 1 incomplete septa (31.2%) were detected in females ( $p = 0.002$ ,  $\chi^2 = 19,121$ ) (Table 1).



**Fig. 2** On coronal PNSCT, bilateral anterior clinoid process pneumatisation (\*) and multiple intersinus septa are shown



**Fig. 3** On coronal PNSCT, measurement of bilateral optic canal (white arrow) and one complete septa of the the sphenoid sinus (\*) are demonstrated

SS inter-sinus septa deviation: In males, deviation to the left (48.1%) and deviation to the right (45.4%) were detected in both groups ( $p > 0.05$ ) (Table 1).

SS dominance: Left SS dominance was detected in both males and females ( $p > 0.05$ ) (Table 1).

Onodi cell: Onodi cell was detected in 26.6% of the males and 19.1% of the females ( $p > 0.05$ ) (Table 1).



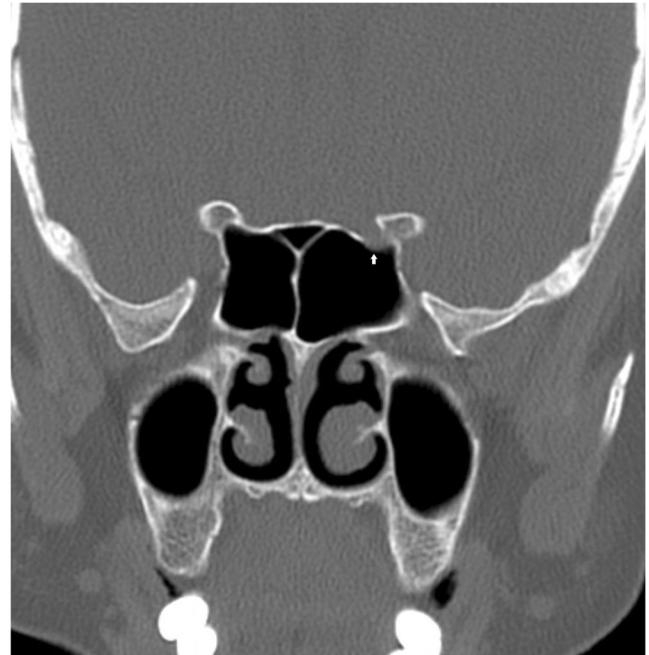
**Fig. 4** On coronal PNSCT, measurement of bilateral carotid canal (white arrow) and no deviation of the sphenoid sinus inter-sinus septa (\*) are demonstrated



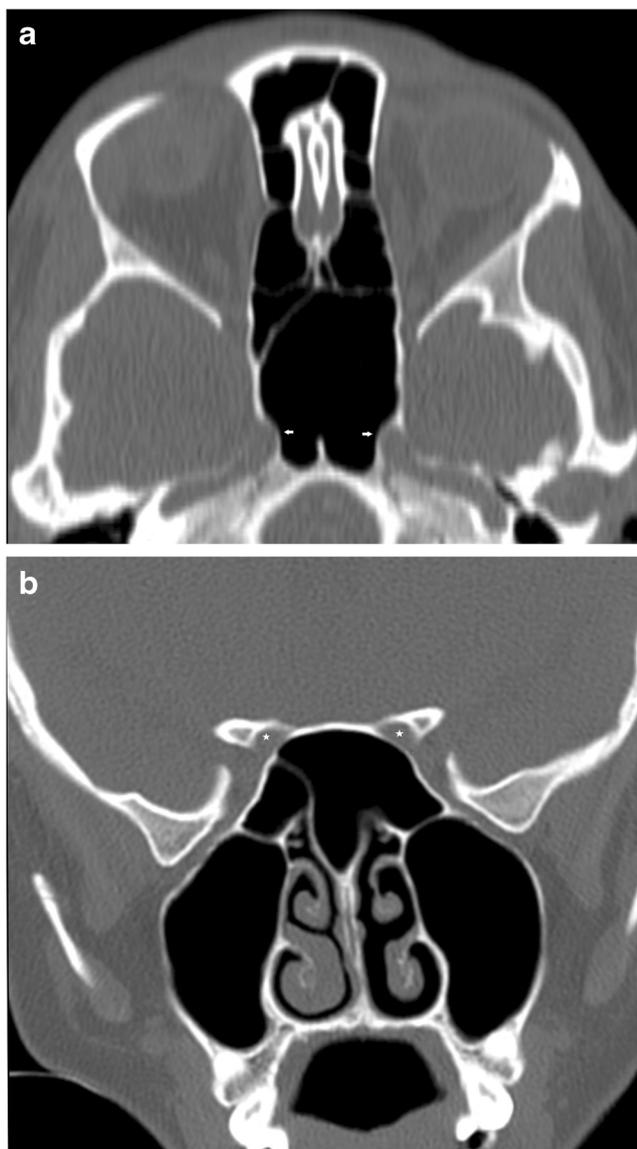
**Fig. 5** On coronal PNSCT, bilateral Onodi cells (\*) and bilateral anterior clinoid process pneumatisation are demonstrated

Carotid canal classification and dehiscence values in males and females are shown on Table 2. Carotid and optic canal diameter values are shown on Table 3.

Carotid canal classification: In males, type 3 carotid canal (protrudation to SS wall) was observed 32.1% on the right side and 23.9% on the left side. In females, type 3 carotid



**Fig. 6** On coronal PNSCT, dehiscency in left optic canal (white arrow) is shown



**Fig. 7** In a female patient, **a** on axial PNSCT, protrusion of the bilateral carotid canal to the sphenoid sinus wall (type 3) is shown. **b** In the same patient, on coronal PNSCT, optic canals were seen on course, lateral to the sphenoid sinus wall and indentation or protrusion is not observed (type 4)

canal was observed 35.5% on the right side and 36.2% on the left side ( $p > 0.05$ ) (Table 2).

Carotid canal dehiscence was detected in 22% of the males and 34% of the females ( $P = 0.038$ ,  $\chi^2 = 8.451$ ) (Table 2). Bilateral dehiscence was present in 5.0% of the males and 9.9% of the females (Table 2).

Carotid canal diameter: In males, carotid canal diameter was higher than those in females ( $p < 0.05$ ) (Table 3).

Optic canal classification and dehiscence values in males and females are shown on Table 4:

Optic canal classification: On the right side, in both males and females, type 4 (extends lateral to the sphenoid sinus and posterior ethmoid cell) was detected more. In males, 45.9%



**Fig. 8** On coronal PNSCT, protrusion of the bilateral carotid canal to the sphenoid sinus wall (white arrow) and bilateral dehiscency on carotid canals are observed

and, in females, 34.8% type 4 classification were observed ( $P = 0.048$ ,  $\chi^2 = 7.893$ ) (Table 4).

Optic canal dehiscence was detected in 11.3% of the males and 9.9% of the females ( $p > 0.05$ ) (Table 3).

Optic canal diameter: In males, optic canal diameter was higher than those in females ( $p < 0.05$ ) (Table 3).

Pearson correlation test results are shown on Table 5:

Age: In older patients, carotid canal width (right) and optic canal width (right and left) increased ( $p < 0.05$ ) (Table 5).



**Fig. 9** On axial PNSCT, protrusion of the bilateral carotid canal to the sphenoid sinus wall (type 3) (white arrow) is shown

**Table 1** Sphenoid sinus pneumatisation and septation, anterior clinoid process pneumatisation values in males and females

			Male (n = 159)		Female (n = 141)		P*
			n	%	n	%	
Type of the SS pneumatisation	R	Type 1	45	28,3	29	20,6	P = 0.218 $\chi^2 = 3.045$
		Type 2	54	34,0	59	41,8	
		Type 3	60	37,7	53	37,6	
	L	Type 1	37	23,3	20	14,2	P = 0.084 $\chi^2 = 4.953$
		Type 2	60	37,7	52	36,9	
		Type 3	62	39,0	69	48,9	
Anterior clinoid process pneumatisation	Absent		84	52,8	85	60,3	P = 0.574 $\chi^2 = 1.991$
	Right		13	8,2	10	7,1	
	Left		22	13,8	14	9,9	
	Bilateral		40	25,2	32	22,7	
SS septation	No septation		11	6,9	2	1,4	P = 0.002 $\chi^2 = 19,121$
	Incomplete septa		2	1,3	1	0,7	
	1 complete septa		55	34,6	59	41,8	
	1 complete + 1 incomplete septa		27	17,0	44	31,2	
	Double septa		28	17,6	14	9,9	
	≥ 3 septa		36	22,6	21	14,9	
SS inter-sinus septa deviation**	Type1: no deviation		17	10,9	16	11,3	P = 0.540 $\chi^2 = 2.160$
	Type2: deviation to the R		59	37,8	64	45,4	
	Type3: deviation to the L		75	48,1	58	41,1	
	Type 4: “S” shaped deviation		5	3,2	3	2,1	
SS dominancy	None/or not available due to multiple septation		20	12,6	18	12,8	P = 0.990 $\chi^2 = 0.021$
	Right dominancy		61	38,4	55	39,0	
	Left dominancy		78	49,1	68	48,2	
Onodi cell	Absent		116	73,4	114	80,9	P = 0.128 $\chi^2 = 2.319$
	Present		42	26,6	27	19,1	

SS sphenoid sinus

\*p value shows the results of Chi-square test

\*\*In three cases, there was no intersinus septa

**Table 2** Carotid canal classification and dehiscence values in males and females

			Male (n = 159)		Female (n = 141)		P*
			n	%	n	%	
Carotid canal classification at SS wall	R	Type 1: no indentation	68	42,8	60	42,6	P = 0.751 $\chi^2 = 0.573$
		Type 2: indentation to SS wall	40	25,2	31	22,0	
		Type 3: protrudation to SS wall	51	32,1	50	35,5	
	L	Type 1: no indentation	73	45,9	50	35,5	P = 0.053 $\chi^2 = 5868$
		Type 2: indentation to SS wall	48	30,2	40	28,4	
		Type 3: protrudation to SS wall	38	23,9	51	36,2	
Caroid canal dehiscence	Absent		124	78,0	93	66,0	P = 0.038 $\chi^2 = 8.451$
	Right		16	10,1	13	9,2	
	Left		11	6,9	21	14,9	
	Bilateral		8	5,0	14	9,9	

SS sphenoid sinus

\*p value shows the results of Chi-square test

**Table 3** Carotid canal and optic canal diameters in the males and females

		Male			Female			P*
		Mean	Median	Std. Dev.	Mean	Median	Std. Dev.	
Carotid canal diameter (mm)	R	5.74	5.76	0.75	5.07	5.11	0.69	0.000
	L	5.87	5.89	0.69	5.34	5.31	0.67	0.000
Optic canal diameter (mm)	R	4.05	4.05	0.53	3.85	3.89	0.51	0.001
	L	4.06	4.06	0.51	3.86	3.84	0.52	0.001

\**p* value shows the results of the independent sample *t* test

Gender: In females, carotid canal width and optic canal width decreased on the right and left ( $p < 0.05$ ). In females, carotid canal type (left) was detected as type III (protrusion to SS wall) and optic canal type (right and left) was detected as type I (no indentation) ( $p < 0.05$ ) (Table 5).

Carotid canal (type and width) and optic canal (type and width): A positive correlation was present between the carotid canal types and width on the right and left sides ( $p < 0.05$ ).

There was a positive correlation between carotid and optic canal width ( $p < 0.05$ ).

There was a negative correlation between carotid canal type and optic canal type and optic canal width ( $p < 0.05$ ) (Table 5) (Fig. 7a, b).

SS pneumatization: In pneumatized sphenoid sinuses, type 3 carotid canal (protrusion to SS wall) (bilaterally) and type 1 optic canal type (no indentation) (ipsilateral side) were detected more ( $p < 0.05$ ) (Table 5) (7a-b). In less pneumatized sphenoid sinuses, type 1 carotid canal (no indentation) (bilaterally) and type 4 optic canal type (extends laterally to the sphenoid sinus and posterior ethmoid cell) (ipsilateral side) were detected more ( $p < 0.05$ ) (Table 5). In pneumatized SS, ipsilateral optic canal width decreased ( $p < 0.05$ ) (Table 5).

## Discussion

The sphenoid sinuses, a pair structure in the body of the sphenoid bone, with drainage through the sphenoidal recess into the nasopharynx, is located in the infero-medial region compared to the posterior ethmoid. This aspect is particularly important in sphenoidotomies: after the posterior ethmoidectomy, the dissection area is infero-medial, outside this area being a risk of penetration into the anterior cranial fossa or the orbit [19].

The trans-sphenoid pathway appears to be the standard approach in the surgery of pituitary adenomas [20]. Different routes to sella: “transethmoid, transnasal, transseptal, microscopic or endoscopic” and eventually pass through the sphenoid sinus to reach the sella. For this reason, anatomical variations of the sphenoid sinus have a great influence on the probability of surgical entry and complications [9]. Preoperative evaluation of the sphenoid sinus anatomy with computed tomography (CT) is a routine procedure and may lead to a surgical decision [9]. CT shows bone/air visualization at the sphenoid sinus, therefore superior to the MR in this region. Additionally, MR

**Table 4** Optic canal classification and dehiscence values in males and females

		Male (n = 159)		Female (n = 141)		P*	
		n	%	n	%		
Optic canal classification at SS wall	R	Type 1: no indentation	6	3.8	10	7.1	$P = 0.048$ $\chi^2 = 7.893$
		Type 2: indentation to SS wall	27	17.0	39	27.7	
		Type 3: protrusion to SS wall	53	33.3	43	30.5	
		Type 4: extends lateral to the sphenoid sinus and posterior ethmoid cell	73	45.9	49	34.8	
	L	Type 1: no indentation	7	4.4	13	9.2	$P = 0.202$ $\chi^2 = 4.613$
		Type 2: indentation to SS wall	25	15.7	29	20.6	
		Type 3: protrusion to SS wall	60	37.7	49	34.8	
		Type 4: extends laterally to the sphenoid sinus and posterior ethmoid cell	67	42.1	50	35.5	
Optic canal dehiscence	Absent	141	88.7	127	90.1	$P = 0.686$ $\chi^2 = 1.445$	
	Right	7	4.4	3	2.1		
	Left	7	4.4	6	4.3		
	Bilateral	4	2.5	5	3.5		

\**p* value shows the results of Chi-square test

might be routine for adenomas and visualisation of the para-/sellar region.

In the present study, we investigated the relationship between sphenoid sinus, carotid canal, and optic canal. In males, type 3 pneumatized sphenoid sinus was detected in both right and left sides. In females, on the right side, type 2 and, on the left side, type 3 pneumatized sphenoid sinuses were detected. In Gibelli et al. [21] study in a sample of 300 CT of the patients, pneumatization of the sphenoid sinuses was reported as pneumatized pterygoid processes (39.6%), dorsum sellae (32.9%), and clinoid processes (20.3%). A combined pneumatization of these three structures was detected in 26.3% of patients. The most frequent type was the combination pterygoid processes-dorsum sellae (11.3%) and in 9.3% of the patients, all the three sphenoid structures were affected. According to the Dal Secchi et al. study [22] on 90 patients' CT scans, there was the association between the lateral extension of pneumatization of the SS and the protrusion of the parasellar segments of the internal carotid artery (psICA) ( $p = 0.014$ ). The presence of the posterior extension of pneumatization of the SS and protrusion of the pcICA occurred in 46% of the cases.

Anterior clinoid pneumatization was present 47.2% in males and 39.7% in females. In both males and females, bilateral pneumatization was present. Anterior clinoid process pneumatization was detected as 4% (De Lano et al.) [23], 13% (Bolger et al.) [24], 15.3% in Hewaidi and Omami's study [7] and 29.3% (Birsan et al.) [25] in different studies.

In the present study, in male group, more septation (i.e. 22.6%,  $\geq 3$  septa) in sphenoid sinus were detected. Hamid et al. [9] reported that there was no intersphenoid sinus septum in 32 patients, a single intersphenoid septum in 212 patients, and an accessory septum in 32 patients. Multiple intersphenoid septa were found in 20 patients (6.8%) [9]. Ahmadipour et al. [26] evaluated the CT scans of the 120 patients diagnosed with pituitary adenomas (study group) and 125 patients receiving a spinal trauma (control group). They reported that a septum of the sphenoid sinus located in the midline 23%. SSS was directed into the bony shield of the internal carotid artery in 28% of underlying tumours and in 37% of the control group. Dal Secchi, et al. [22] reported that deviation of the sphenoid septum in the direction of the pcICA was present in 14% and dehiscence of the paraclival segments of the internal carotid artery (pcICA) was seen in 3.6% of the 90 cases.

In our study, left SS dominancy was detected in both males and females. Onodi cell was present 26.6 and 19.1% in males and females, respectively.

In our study, carotid canal protrusion to the sphenoid sinus wall was present 23.9–32.1% in males and 35.5–36.2% in females. Dehiscence in carotid canal was detected more in females (34%) compared to males (22%). Hewaidi and Omami [7] reported that internal carotid artery protrusion into the sphenoid was found in 41% of the patients, and in

30%, there was a dehiscence of the artery. Fuji et al. study [27] on 25 cadaveric sphenoidal bones showed dehiscent carotid arteries (8%) in the lateral sphenoid. In Kennedy et al. study [28], 25% of patients had internal carotid dehiscence on the bony walls. In our study, dehiscence of the carotid artery was observed more in females compared to Fuji et al. [27] and Kennedy et al. [28] rates. If the surgeon is unaware of dehiscence or protrusion of the artery, it may even be fatal bleeding because it is not possible to control the bleeding of an injured internal carotid artery within the sphenoid sinus. Moreover, neurological sequelae are inevitable. Infection of the sphenoid sinus can make the internal carotid artery vulnerable to degenerative or protruded arterial injury [29].

In our study, optic canal protrusion was 33.3 and 30.5% on the right side and 37.7 and 34.8% on the left side of the males and females, respectively. Type 4 optic canal was detected more in both gender. Optic canal dehiscence was detected 11.3 and 9.9% in males and females. Carotid and optic canal diameters were higher in males. Hewaidi and Omami [7] reported that the protrusion of the optic nerve was found in 35.6%, and dehiscence of the optic nerve was observed in 30.6% of the patients. Our study was similar to their study for optic canal protrusion, whereas optic canal dehiscence was lower in our study. Our optic canal dehiscence rates were approximately 10%; their rates were approximately 30% with the 3-fold increase compared to our rates. Fuji et al. reported that 4% of the optic nerves were dehiscent on the lateral sphenoidal bone [27]. Optic nerve injury in the event of protrusion or dehiscence can occur as a complication of surgical trauma or sinus disease. If the surgeon injures the nerve within the sinus, the risk of blindness is high [13]. In addition, vision deficits may be caused by a sphenoid sinus infection or by an optic canal or sinus compression mucocele. Compression of the optic nerve can cause nerve ischemia and venous congestion of the nerve. In addition, the optic canal is susceptible to injury when least nourished [30].

In pneumatized sphenoid sinuses and in females, type 3 carotid canal (protrusion to SS wall) (bilaterally) and type 1 optic canal type (no indentation) (ipsilateral side) were detected more. In elderly patients, carotid and optic canal width increased. Age-related intracranial signs of atherosclerosis along the course of the ICA [31, 32], hypertension and osteoporosis may cause these increased carotid and optic canal widths in elderly patients. When carotid canal protrusion was detected, there was no indentation in optic canals. Carotid and optic canal diameters increased together. Sirikci et al. [29] reported that the pneumatized sphenoid sinus could disrupt the anatomic configuration and weaken the bony in the lateral wall, creating a greater risk of optic nerve and carotid artery disease [29]. Excessive pneumatization of the sphenoid sinuses with protrusion of both internal carotid arteries was also reported by Baldea and Sandu [19]. Maniglia et al. [14] reported that in 75% of the cases, in the well-pneumatized

**Table 5** Pearson correlation test results

	Age	Gender (code 1: male, code 2: female)	Carotid canal				Optic canal				Onodi cell (code 0: absent, code 1: present)		SS pneumatisation			
			Type-R		Type-L		Type-R		Type-L		Width-R		Width-L		R	L
			Type-R	Type-L	Width-R	Width-L	Type-R	Type-L	Width-R	Width-L	Type-R	Type-L	Width-R	Width-L	R	L
Age	r	-0.011	-0.066	-0.085	0.113	0.137	0.002	0.010	0.292	0.316	0.042	0.042	-0.086	-0.197		
	p	0.851	0.257	0.144	0.051	0.017	0.974	0.859	0.000	0.000	0.473	0.473	0.138	0.001		
Gender (code 1: male, code 2: female)	r	-0.011	0.021	0.136	-0.417	-0.361	-0.156	-0.116	-0.187	-0.194	-0.093	-0.093	0.049	0.126		
	p	0.851	0.721	0.018	0.000	0.000	0.007	0.045	0.001	0.001	0.107	0.107	0.401	0.029		
Carotid canal	Type R	r	-0.066	0.021	0.136	-0.417	-0.361	-0.156	-0.187	-0.194	-0.093	-0.093	0.049	0.126		
	p	0.257	0.721	0.018	0.000	0.000	0.007	0.045	0.001	0.001	0.107	0.107	0.401	0.029		
Type L	r	-0.085	0.000	0.381	0.000	0.424	0.894	0.035	0.000	0.003	0.932	0.932	0.387	0.355		
	p	0.144	0.018	0.000	0.381	0.000	0.000	0.000	0.000	0.003	0.000	0.000	0.000	0.000		
Width R	r	-0.113	0.051	0.003	0.003	-0.055	-0.063	-0.160	-0.183	-0.189	-0.047	-0.047	0.290	0.385		
	p	0.051	0.003	0.955	0.955	0.344	0.280	0.006	0.001	0.001	0.420	0.420	0.000	0.000		
Width L	r	0.137	-0.361	-0.046	-0.055	0.697	0.059	0.077	0.204	0.197	0.074	0.074	-0.013	-0.093		
	p	0.017	0.000	0.424	0.344	0.000	0.311	0.186	0.000	0.001	0.203	0.203	0.817	0.107		
Optic canal	Type R	r	0.002	-0.156	-0.008	-0.063	0.059	0.033	0.033	0.175	0.187	-0.025	-0.042	-0.094		
	p	0.974	0.007	0.894	0.280	0.311	0.571	0.571	0.000	0.002	0.001	0.668	0.466	0.105		
Type L	r	0.010	-0.116	-0.122	-0.160	0.077	0.033	0.722	0.107	0.119	0.107	0.107	-0.132	-0.111		
	p	0.859	0.045	0.035	0.006	0.186	0.575	0.000	0.064	0.040	0.064	0.064	0.022	0.054		
Width R	r	0.292	-0.187	-0.209	-0.183	0.204	0.175	0.129	0.107	0.756	0.047	0.047	-0.243	-0.217		
	p	0.000	0.001	0.000	0.001	0.000	0.002	0.026	0.064	0.000	0.414	0.414	0.000	0.000		
Width L	r	0.316	-0.194	-0.169	-0.189	0.197	0.187	0.096	0.119	0.756	0.028	0.028	-0.193	-0.227		
	p	0.000	0.001	0.003	0.001	0.001	0.001	0.096	0.040	0.000	0.635	0.635	0.001	0.000		
Onodi cell (code 0: absent, code 1: present)	r	0.042	-0.093	0.005	-0.047	0.074	-0.025	0.020	0.107	0.047	0.028	0.028	-0.120	-0.072		
	p	0.473	0.107	0.932	0.420	0.203	0.668	0.728	0.064	0.414	0.635	0.635	0.038	0.213		
SS pneumatisation	R	r	-0.086	0.049	0.387	0.290	-0.013	-0.042	-0.132	-0.084	-0.243	-0.193	0.628	0.628		
	p	0.138	0.401	0.000	0.000	0.817	0.466	0.022	0.144	0.000	0.001	0.038	0.000			
L	r	-0.197	0.126	0.355	0.385	-0.093	-0.094	-0.111	-0.120	-0.217	-0.227	-0.072	0.628			
	p	0.001	0.029	0.000	0.000	0.107	0.105	0.054	0.038	0.000	0.000	0.213	0.000			

sinuses, there is optic nerve impression on the lateral sinus wall. Baldea and Sandu [19] reported that it can be visualized endoscopically and represents an orientation landmark to be avoided. In our study, we showed that carotid canal protrusion and greater risk in surgery were present in pneumatized SS similar to Sirikci et al. [29], Baldea and Sandu [19] and Maniglia et al. [14] studies. However, type 1 (no indentation) optic canal was present in highly pneumatized SS with lower risk for the surgery. This result of our study is different from Sirikci et al. [29] study.

To prevent morbid consequences at the time of surgery, clinicians have to determine the location and size of the sphenoid sinus walls and their relationship to adjacent vital structures [9].

## Conclusion

When pneumatized SS is seen, careful handling of functional endoscopic sinus surgery is necessary, as carotid canal protrusion may be a risk. In women, the risk of carotid canal protruding (about 1/3) is greater than that of males, and carotid canal dehiscence rates are also higher in females. Therefore, physicians should be very careful during the preparatory stages of the sphenoid sinus surgery. Otherwise, it may not be possible to prevent lethal carotid artery bleeds. Different from the carotid canal, optic canal classification is type 1 (no indentation) in patients with highly pneumatized sphenoid sinus and surgical trauma risks to the optic canal is lower in these patients. Our study provides detailed measurements in the sphenoid sinus, carotid canal and optic canal and our recommendations to the surgeons to avoid complications.

**Author contributions** Neşe ASAL: Planning, designing, data collection, literature survey.

Nuray Bayar Mülük: Planning, designing, literature survey, statistical analysis, writing.

Mikail İNAL: Planning, designing, literature survey.

Mehmet Hamdi ŞAHAN: Planning, designing, literature survey.

Adil Doğan: Planning, designing, literature survey.

Osman Kürşat Arkan: Planning, designing, literature survey.

## Compliance with ethical standards

This retrospective study was conducted in Kırıkkale University Faculty of Medicine according to the principles of the Declaration of Helsinki. Ethics committee approval was obtained from Adana City Training and Research Hospital; Clinical Researches Ethics Committee was also taken (Date: 27.03.2018, Number:184).

**Conflict of interest** The author Neşe ASAL declares that he has no conflict of interest.

The author Nuray BAYAR MÜLÜK declares that she has no conflict of interest.

The author Mikail İNAL declares that he has no conflict of interest.

The author Mehmet Hamdi ŞAHAN declares that he has no conflict of interest.

The author Adil Doğan declares that he has no conflict of interest.

The author Osman Kürşat Arkan declares that he has no conflict of interest.

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