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PREVALENCE OF CRITICAL ANATOMICAL VARIATIONS IN PARANASAL SINUSES PREDISPOSING TO SURGICAL COMPLICATIONS: A CT-BASED RETROSPECTIVE CROSS-SECTIONAL STUDY FROM EASTERN INDIA

V K Sundeep Nalamolu[1], Srilatha K[2], Deepali Singh[2], Ahammad Basha Shaik[3]

[1]*Department of Radiology, Manipal Tata Medical College, Manipal Academy of Higher Education, Manipal, Karnataka, India.*
[2]*Department of Otorhinolaryngology, Manipal Tata Medical College, Manipal Academy of Higher Education, Manipal, Karnataka, India.*
[3]*Department of Community Medicine, Manipal Tata Medical College, Manipal Academy of Higher Education, Manipal, Karnataka, India.*

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Corresponding Author:
Dr. Ahammad Basha Shaik,
Department of Community Medicine, Manipal Tata Medical College, Manipal Academy of Higher Education, Manipal, Karnataka, India.
Email: ahammad.basha@manipal.edu, ahammadbasha@gmail.com.

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ABSTRACT

Background: Anatomical variations in the paranasal sinuses (PNS) have important clinical implications, particularly during functional endoscopic sinus surgery (FESS). Variants involving the cribriform plate, lamina papyracea, Onodi cells, sphenoid sinus pneumatization, and the anterior ethmoid artery- collectively remembered as “CLOSE,” can increase the risk of intraoperative complications. Computed tomography (CT) remains the gold standard for visualizing sinonasal anatomy and guiding surgical planning. The objective is to determine the prevalence of anatomical variations of PNS that predispose to surgical complications using computed tomography (CT) among patients in Eastern India.

Materials and Methods: A retrospective cross-sectional analysis was conducted on CT scans of 100 patients (aged 20–70 years) with suspected sinusitis, evaluated between July and December 2024 at a tertiary care center in Jharkhand, India. Multiplanar CT reconstructions were reviewed to identify PNS variations. Statistical analysis was performed using RStudio (v2025.05.1+513), employing chi-square tests and prevalence ratios with 95% confidence intervals.

Results: Among the findings, 72% had Keros type I, 33% had Onodi cells, 52% had Haller cells, 82% exhibited sellar type sphenoid sinus pneumatization, and 60% had supraorbital cells. Additional variations included optic canal and carotid canal dehiscence or protrusion, as well as various forms of pneumatization.

Conclusion: Sellar-type sphenoid sinus pneumatization, Type I uncinate process attachment, Haller cells, and supraorbital cells were the most common variants associated with potential surgical risk. These findings emphasize the importance of individualized preoperative CT evaluation to identify anatomical variations, reduce intraoperative complications, and improve patient outcomes.

Keywords: Anatomical variations of Paranasal Sinuses; CT paranasal Sinuses; Surgical complications of Paranasal Sinuses; Functional Endoscopic Sinus Surgery (FESS).

INTRODUCTION

Anatomical variations are most frequent in the Paranasal sinuses (PNS), and the prevalence of these anatomical variations varies among ethnic groups.[1,2] ‘Functional endoscopic sinus surgery (FESS)’ plays a critical role in the management of rhinosinusitis; however, it carries the risk of significant surgical complications.[3] Computed tomography (CT) is the preferred imaging modality for evaluating the paranasal sinuses, as it allows for precise identification of anatomical variants and facilitates comprehensive preoperative planning for FESS.[1,3] Key anatomical variations associated with increased surgical risk primarily involve the cribriform plate, lamina papyracea, Onodi cells, sphenoid sinus pneumatization, and the anterior ethmoid artery, collectively remembered by the mnemonic “CLOSE”.[3] Despite the clinical importance of assessing these variations, detailed studies on their prevalence remain limited in the Indian population. This study aims to evaluate the prevalence of anatomical variants of paranasal sinuses that predispose to surgical complications in the study population of Jharkhand, which benefits the surgeons in the preoperative evaluation and contributes to existing epidemiological data in the literature.

MATERIALS AND METHODS

Study design and ethical clearance: The cross-sectional retrospective study was conducted in the Department of Radiology at Meherbai Tata Memorial Hospital (MTMH), Jamshedpur, Jharkhand. The Institutional Ethics Committee at MTMH provided ethical approval (MTMH/IEC/141/2024).

Study population and sampling method: Convenience sampling was used to include the average monthly outpatient flow of patients undergoing CT for suspected sinusitis during six months (July 2024–December 2024), and the sample size of 100 patients was chosen based on the practical availability of cases in a tertiary care setting and the retrospective nature of the study. While convenience sampling facilitates timely data collection, it may limit the generalizability of the findings due to potential selection bias.

Inclusion and exclusion criteria:

This study included patients aged 20 to 70 years with symptomatic sinusitis, and other symptoms like headache, nasal block, and allergic rhinitis. Patients under 20 years old, those with sinonasal polyposis, fungal sinusitis, pansinusitis, a history of sinus surgery, sinonasal malignancy, and facial trauma were excluded from the study.

Radiological Evaluation: Computed tomographic (CT) scans of the nose & paranasal sinuses were performed using a 128-slice Siemens spiral CT scanner. Axial plane images were acquired with a slice thickness of 1.5 mm, followed by multiplanar reformatting. All images were stored and retrieved using the SYNGO platform via the “Picture Archiving and Communication System (PACS)”. The anatomical variations related to the drainage pathways of the paranasal sinuses were evaluated by a radiologist with over nine years of experience in cross-sectional imaging and an otolaryngologist (ENT surgeon) with more than nine years of clinical experience.

The following anatomical variations, with operational definitions as described, were studied in all the patients. The anatomical variants are grouped for convenience.

1. Cribriform plate and Olfactory fossa: The cribriform plate is a thin bony structure that forms the roof of the nasal cavity and separates it from the anterior cranial fossa. It also houses the olfactory bulbs within the olfactory fossa.[3]

A. Keros classification: Classifies the depth of the olfactory fossa or the lateral lamella length, as type I, II, and III with depth as less than or equal to 3mm, 4-7mm, and 7mm or greater, respectively.[4]

B. Asymmetric Keros: Asymmetry on both sides in the olfactory fossa depths.[3]

2. Lamina papyracea: A Thin ethmoid bone layer that forms the wall of the medial orbital.[3]

A. Lamina Papyracea Dehiscence (LPD) or the orbital prolapse into the ethmoid sinus: Defined as a prolapse of the orbital contents into the ethmoid sinus due to a defect in the medial orbital wall.[5]

B. Haller cell: Air cell along the floor of the orbit or at the roof of the maxillary sinus, which lies lateral to the lamina papyracea and inferior to the ethmoid bulla.[6,7]

C. Atelectatic uncinate process: The uncinate process's lateral displacement and apposition to the inferomedial orbital wall.[8]

D. Type I attachment of the uncinate process: Superior attachment of the uncinate process to the lamina papyracea.[9]

3. Onodi cell: The posterior ethmoid air cell, known as the Onodi cell, that extends posteriorly along & superior and lateral aspects of the sphenoid sinus.[3]

A. Presence or absence

B. Dehiscence of the optic nerve within the Onodi cell: The lack of the bony wall dividing the sinus and optic nerve bony canals is known as dehiscence.[10]

4. Sphenoid sinus pneumatisation

A. Pneumatization pattern: There are three types of sphenoid sinus pneumatization: conchal, presellar, and sellar. Pneumatization in the conchal type is restricted to the anterior part of the sphenoid body and does not extend to the anterior wall of the sella. It reaches the sella's anterior wall in the presellar form. Pneumatization extends inferiorly and posteriorly to the sella turcica in the sellar type.[11]

B. Extension of pneumatization to the skull base: The pterygoid process is said to be pneumatized if it extends over the horizontal plane that crosses the vidian canal, while the larger wing of the sphenoid is said to be pneumatized if it extends past the vertical plane that crosses the foramen rotundum.[12]

C. Extension of pneumatization to the lesser wing of the sphenoid: extension into the optic strut, the opticocarotid recess, which is formed inferiorly to the optic canal, and the pneumatized anterior clinoid process, which is formed far beyond the anterior clinoid process.[11,13]

D. Dehiscence and protrusion of the carotid canal and optic canal within the sphenoid sinus: The lack of the bony wall between the sphenoid sinus and the bone canal is known as dehiscence. The protuberance of the canal into the sphenoid sinus, which occupies more than half of its circumference, is referred to as the protrusion.[10]

E. Sinus septation inserting onto carotid canal: Insertion of the sphenoid intersinus septum onto the carotid canal.[11]

5. Anterior ethmoid artery notch: A bony canal arises medially from the lamina papyracea, located near the fovea ethmoidalis or the lateral lamella, & contains the anterior ethmoid artery.[11]

A. Anterior ethmoidal notch dehiscence: The anterior ethmoidal artery is encased in a bone canal when the anterior ethmoidal notch makes contact with the lateral lamella or the fovea ethmoidalis. The artery is hanging inside the sinus, supported solely by the mucosa, if this canal dehisces or is absent.[3,11]

B. Presence of supraorbital pneumatisation: Air cells that extend superiorly and laterally over the orbital plate of the frontal bone.[11]

Statistical analysis: - Microsoft Excel was used to enter the data, and RStudio (v2025.05.1+513), was used to perform statistical analyses. Frequencies and percentages were used to describe categorical variables, whereas means with standard deviations (SD) were used to report continuous variables. The chi-square test was used to assess the relationship between gender and anatomical differences. The threshold for statistical significance was $p < 0.05$.

RESULTS

Out of 100 patients, males (51.0%) were more predominant than females (49.0%) in this study. 28.0% of patients were aged between 20 to 30 years, 19.0% of patients aged between 31-40 years, 20.0% of patients aged between 41-50 years, 15.0% of patients aged between 51-60 years, and 18.0% of patients were aged between 61-70 years, respectively. The association between age group and sex was shown to be statistically not significant ($p = 0.245$). The overall mean±SD age was 42.90±15.48 years with a range of 20 to 70 years, respectively. The proportional distribution of males and females across age groups.

A comparison of critical anatomical variations in paranasal sinuses predisposing to surgical complications between male and female participants is presented in Graph 1. The most prevalent variants observed in the overall sample included Type I uncinate process (85.0%), Keros type I (72.0%), and sphenoid sinus (SS) sellar configuration (82.0%).

Statistically significant sex differences were observed in the distribution of Keros type I and Keros type II variants. Keros type I was significantly more prevalent in females (81.6%) compared to males (62.8%) (PR = 0.77, 95% CI: 0.60, 0.99, $p = 0.04$), indicating that the prevalence of Keros type I was 23% lower in males than in females. In contrast, Keros type II was more common in males (52.9%) than in females (28.6%) (PR = 1.85, 95% CI: 1.11, 3.09, $p = 0.01$), suggesting that males were 85% more likely to have this variant.

Insignificant differences were found between males and females for Keros type III, though it was observed only in males (5.9%) ($p = 0.09$). Similarly, asymmetry of the olfactory fossa was more prevalent in males (21.6%) than in females (10.2%), but this difference did not reach statistical significance (PR = 2.11, 95% CI: 0.79, 5.64, $p = 0.39$).

Variants such as LPD, Haller cells, Onodi cells, and optic canal protrusion showed varying prevalence across sexes, but none of these differences were statistically significant. For example, Haller cells were present in 56.9% of males and 46.9% of females (PR = 1.21, 95% CI: 0.93, 1.77, $p = 0.32$).

Several less common findings, including atelectatic uncinate process (PR = 1.92, 95% CI: 0.18, 20.52, $p = 0.58$), carotid canal protrusion (PR = 1.92, 95% CI: 0.51, 7.26, $p = 0.32$), and pneumatized anterior clinoid process (PR = 1.20, 95% CI: 0.52, 2.79, $p = 0.67$), also did not show significant sex-based differences, likely due to low overall prevalence and limited power to detect small effects. No cases of sphenoid sinus hypoplasia, conchal types, or optic nerve dehiscence were observed in either group.

Although most anatomical variants did not differ significantly between sexes, the notable differences in Keros classifications highlight the importance of considering sex as a variable in preoperative imaging evaluation, particularly given the surgical relevance of olfactory fossa depth and adjacent structures.

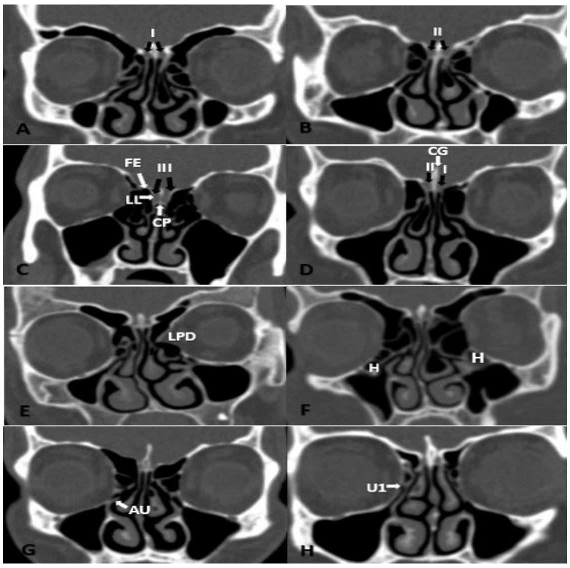
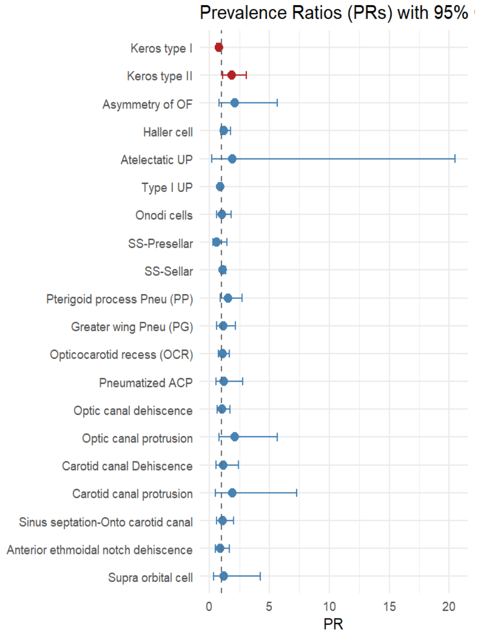


Figure 1: Plain CT of PNS coronal section: A) Kero's type I olfactory fossa (I). B) Kero's type II olfactory fossa (II). C) Kero's type III olfactory fossa (III). D) Asymmetry of olfactory fossa, type II on right side and type I on left side. Cribriform plate (CP), Fovea ethmoidalis (FE), Lateral lamella (LL), Cristagalli (CG). E) Dehiscence of lamina papyracea (LPD). F) Haller cell (H). G) Atelectatic uncinate process (AU). H) Type I attachment of uncinate process to the lamina papyracea (U1).

Variant	Male	Female	Prev. (%)	PR [95% CI]	p-value
Keros type I	32 (62.8%)	40 (81.6%)	72.0%	0.77 [0.60–0.99]	0.04
Keros type II	27 (52.9%)	14 (28.6%)	41.0%	1.85 [1.11–3.09]	0.01
Asymmetry of OF	11 (21.6%)	5 (10.2%)	16.0%	2.11 [0.79–5.64]	0.12
Haller cell	29 (56.9%)	23 (46.9%)	52.0%	1.21 [0.93–1.77]	0.32
Atelectatic UP	2 (3.9%)	1 (2.0%)	3.0%	1.92 [0.18–20.52]	0.58
Type I UP	41 (80.4%)	44 (89.8%)	85.0%	0.90 [0.76–1.06]	0.19
Onodi cells	17 (33.3%)	16 (32.7%)	33.0%	1.02 [0.58–1.79]	0.94
SS-Presellar	7 (13.7%)	11 (22.5%)	18.0%	0.61 [0.26–1.45]	0.26
SS-Sellar	44 (86.3%)	38 (77.6%)	82.0%	1.11 [0.92–1.34]	0.26
Pterigoid process Pneu (PP)	21 (41.2%)	13 (26.5%)	34.0%	1.55 [0.88–2.74]	0.12
Greater wing Pneu (PG)	14 (27.5%)	12 (24.5%)	26.0%	1.12 [0.58–2.18]	0.74
Opticocarotid recess (OCR)	25 (49.0%)	22 (44.9%)	47.0%	1.09 [0.72–1.66]	0.68
Pneumatized ACP	10 (19.6%)	8 (16.3%)	18.0%	1.20 [0.52–2.79]	0.67
Optic canal dehiscence	21 (41.2%)	19 (38.8%)	40.0%	1.06 [0.66–1.72]	0.81
Optic canal protrusion	11 (21.6%)	5 (10.2%)	16.0%	2.11 [0.79–5.64]	0.12
Carotid canal Dehiscence	12 (23.5%)	10 (20.4%)	22.0%	1.15 [0.55–2.42]	0.71
Carotid canal protrusion	6 (11.8%)	3 (6.1%)	9.0%	1.92 [0.51–7.26]	0.32
Sinus septation-Onto carotid canal	16 (31.4%)	14 (28.6%)	30.0%	1.10 [0.60–2.00]	0.76
Anterior ethmoidal notch dehiscence	14 (27.5%)	15 (30.6%)	29.0%	0.90 [0.49–1.66]	0.73
Supra orbital cell	27 (52.9%)	33 (67.4%)	60.0%	1.20 [0.34–4.21]	0.14



Graph 1: Forest plot of prevalence of critical anatomical variations in paranasal sinuses predisposing to surgical complications

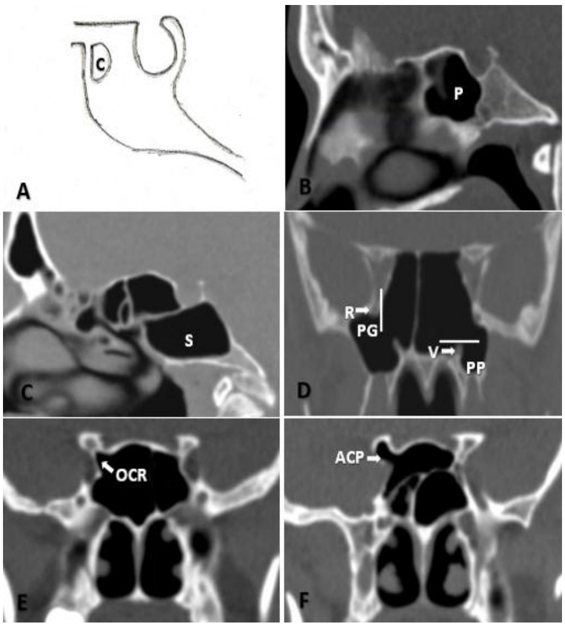


Figure 2: A) Line diagram of conchal type (C) of sphenoid sinus pneumatization. Plain CT PNS B) Sagittal section shows presellar type (P) of sphenoid pneumatization. C) sellar type (S) of sphenoid pneumatization. D) Coronal section shows pneumatization pterygoid process (PP) extending inferior to the horizontal plane below the vidian canal (V) and pneumatization of greater wing of sphenoid (PG) extending lateral to the vertical plane adjacent to the foramen rotundum (R). E) Opticocarotid recess (OCR). F) Anterior clinoid process pneumatization (ACP).

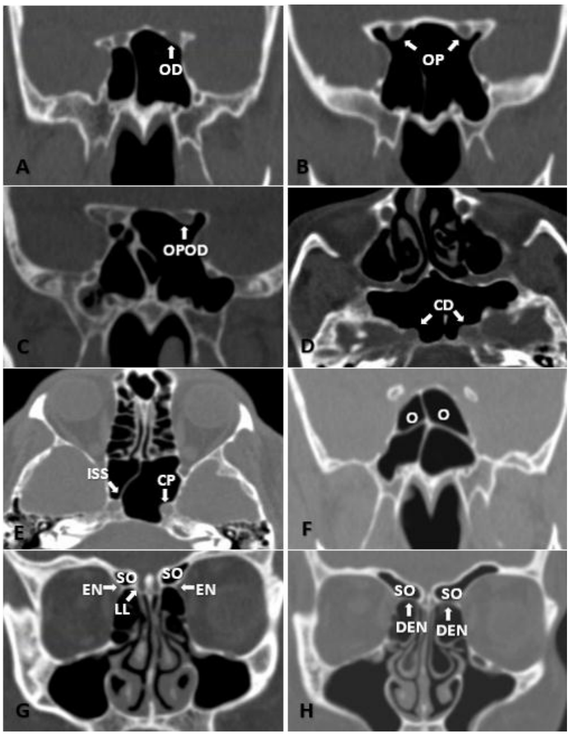


Figure 3: Plain CT PNS A) Coronal section shows optic nerve dehiscence (OD) in sphenoid sinus on left side. B) Optic nerve protrusion (OP) in sphenoid sinus on both sides. C) Optic nerve protrusion in sphenoid sinus with dehiscence (OPOD) on left side. D) Axial section shows carotid canal dehiscence (CD) in sphenoid sinus. E) Carotid canal protrusion (CP) in sphenoid sinus on left side and inter sinus sphenoid septation (ISS) inserting onto carotid canal on right side. F) Onodi cell (O). G) Coronal section shows normal anterior ethmoidal notch (EN) abutting the lateral lamella (LL) on both sides. H) Dehiscence of anterior ethmoidal notch (DEN) on both sides with supraorbital cells (SO).

DISCUSSION

The Cribriform plate, Lamina papyracea, Onodi cells, Sphenoidal sinus pneumatization, and anterior ethmoid artery (which can be remembered by the acronym "CLOSE") are the primary critical anatomical variables that can lead to surgical difficulties.[3] The olfactory fossa has cribriform plate, fovea ethmoidalis, lateral lamella, and cristagalli as the inferior, supero-lateral, lateral, and medial boundaries, respectively [Figures 1C & 1D]. The lateral lamella is the thinnest bone, more vulnerable to injury during FESS with CSF leak, meningitis, pseudo meningocele, and meningoencephalocele as possible complications.[3,14] The risk of injury increases from Keros I to III and has the highest incidence in Keros type III, and asymmetry of the olfactory fossa.[14] [Figure 1A,1B,1C]. The reported prevalence of Keros typess I, II, and III in the literature was 25.4%, 58.3%, and 7.3%, respectively.[15] The prevalence of asymmetry of the olfactory fossa was 39% (Figure 1D).[16] This study found Keros types I, II, and III in 72.0%, 41.0%, and 3.0% of patients, and asymmetry of the olfactory fossa in 16.0% of patients.

In LPD, most commonly the orbital fat and rarely the orbital muscle (medial rectus) may herniate into the ethmoidal sinuses [Figure 1E].[2,5] It is important to avoid misdiagnosis of LPD as ethmoid sinus septation to prevent diplopia, intraorbital hemorrhage, blindness, orbital emphysema, and orbital cellulitis as potential complications during FESS.[3,17] The reported prevalence of the LPD in the literature was in the range of 0.3% to 6%.[2] Our study found LPD in 2.0% of patients.

Haller cell, if present, may predispose to an increased orbital injury risk during ethmoidectomy [Figure 1F].[2] The reported prevalence of Haller cells in the literature ranges from 8% to 57%.[2] Our study found Haller cells in 52.0% of patients.

The atelectatic uncinate process is seen in association with silent sinus syndrome due to obstruction of the ethmoid infundibulum [Figure 1G].[8] It improved orbital injury risk during FESS.[3,11] The reported prevalence of atelectatic uncinate process in the literature ranges from 0.5% to 9%.[2] Our study found an atelectatic uncinate process in 3.0% of patients.

A Type I attachment of the uncinate process to the lamina papyracea is associated with an increased risk of lamina papyracea injury during uncinectomy [Figure 1H].[7,9] The reported prevalence of this variant in the literature ranges from 55 % to 82 %.[18] Our study found Type I attachment of the uncinate process in 85.0% of patients.

The Onodi cell or sphenoethmoid cells can be identified by horizontal or cruciform septation in the air cells superior to choanae, indicating the horizontal displacement of the anterior wall of the sphenoid sinus by a more superior sphenoethmoid cell [Figure 3F].[7] The Onodi cell increases the risk of injury to the optic nerve during posterior ethmoidectomy due to its close relation at the superolateral aspect, with a thin bone separating both.[3] The stated prevalence of Onodi cells in the literature ranges from 2% to 50%.[2] Our study found sphenoethmoid cells in 33.0% of patients.

Sphenoid sinus pneumatization patterns are classified as conchal, presellar, and sellar types concerning the sella and clivus [Figure 2A, 2B, and 2C].[3,11] Sellar type of pneumatization is associated with a high risk of penetration during the endoscopic transsphenoidal surgery (ETSS) and may cause a CSF leak.[2] Sphenoid sinus hypoplasia and conchal type of pneumatization are relative contraindications for ETSS.[11,13] The reported prevalence of conchal, presellar, and sellar types of pneumatization ranges from 0% to 9%, 10%-38%, and 53% to 89 %, respectively, and the prevalence of the sphenoid sinus hypoplasia ranges from 0.9% to 14.5%.[2] Our study found the presellar, sellar types of pneumatization in 18.0%, 82.0%, and none of the patients had conchal and sphenoid sinus hypoplasia.

The sphenoid sinus pneumatization may extend to skull base, causing pneumatization of Pterigoid Process (PP) and pnematization of greater wing of sphenoid (PG) and may extend to lesser wing of sphenoid resulting the opticocarotic recess (OCR) and anterior clnoid process pneumatization (ACP) [Figure 2D, 2E and 2F].[11-13] Bony dehiscence and protrusion of neurovascular structures adjacent to the sphenoid sinus tend to increase proportionally with the degree of sphenoid sinus pneumatization.[13] The optic nerve and internal carotid artery are in close relation to the OCR and ACP, and the maxillary and vidian nerves are near PP and PG with increased risk of injury during ETSS.[7,13] The reported prevalence of the OCR, ACP, PP, and PG in the literature ranges from 29% to 34%,10% to 29.3%,29% to 43.6%, and 10% to 47%, respectively.[12,19] Our study found OCR, ACP, PP, and PG in 47.0%, 18.0%, 34.0%, and 26.0%, respectively.

The Internal Carotid Artery (ICA) passes through the posterior and lateral sides of the sphenoid sinus, whereas the optic nerve canal passes through the superior aspect. The optic and ICA canals may dehisce and protrude as a result of the hyperpneumatization of the sphenoid sinuses [Figure 3A,3B,3C,3D, and 3E].[10] The anatomic variants that predispose to optic nerve injury during the FESS or ETSS are Delanos type II and type III configurations, dehiscence of the optic nerve, and pneumatized anterior clinoid process.[20] The reported prevalence of Optic Canal Dehiscence (OCD) and Optic Canal Protrusion (OCP) in the literature is 4.0% and 7-35%, respectively.[2,11] Our study found the OCD and OCP in 40.0% and 16.0% of patients. The sphenoid intersinus septum may be deviated and may have an insertion onto the ICA canal or the optic canal [Figure 3E].[11] ICA canal dehiscence, ICA canal protrusion, and sphenoid intersinus septum insertion onto the ICA canal increased the injury risk to the ICA during FESS or ETSS and may result in catastrophic hemorrhage.[3] The reported prevalence of ICA canal dehiscence, ICA canal protrusion, and insertion of the sphenoid intersinus septum onto the ICA canal in the literature ranges from 2% to 23%,5.2% to 67%, and 4.7 %, respectively.[2] Our study found the ICA canal dehiscence, ICA canal protrusion, and insertion of the intersinus bony septum onto the carotid canal in 40.0%, 16.0%, and 30.0% of patients.

Anterior ethmoidal notch dehiscence or absence of the bony canal predisposes to the injury of the anterior ethmoid artery during FESS, resulting in devastating intraorbital hemorrhage. [Figure 3G, 3H].[3,11,21] The prevalence of the anterior ethmoid notch dehiscence in literature ranges from 65 to 66%.[22] Our study found the anterior ethmoidal notch dehiscence in 29.0% of patients.

The presence of supraorbital cells above the anterior ethmoidal notch increases the risk of anterior ethmoid artery injury during the FESS [Figure 3G,3H].[3] The reported prevalence of the supraorbital cells in the literature ranges from 6% to 42.4%.[2] Our study found supraorbital cells in 60.0% of patients.

Limitations of the study

This study's main drawbacks include its single-center design and limited sample size, which could limit the findings' external validity and generalizability. To overcome these limitations and provide more comprehensive evidence, future research should incorporate larger, more diverse populations across multiple centers and regions.

CONCLUSION

Significant anatomical variability in the paranasal sinuses underscores the need for individualized preoperative CT evaluation to minimize surgical risks during FESS. Key variants, including the sellar type of sphenoid pneumatization, type I uncinate process attachment, Haller cells, and supraorbital cells—must be carefully assessed to optimize outcomes.

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