The Journal of Laryngology & Otology (2017), ***131***, *620–626*.

doi:10.1017/S002221511700086X

MAIN ARTICLE

Influence of opacification in the frontal recess on frontal sinusitis

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Abstract

Objectives: This study aimed to radiologically evaluate the influence of inflammatory changes in frontal recess cells on frontal sinusitis.

Methods: A total of 93 patients (186 sides) who underwent primary sinonasal surgery at Hyogo College of Medicine were enrolled in 2015 and 2016. Opacification of agger nasi, fronto-ethmoidal, ethmoid bulla, suprabullar and frontal bulla cells was determined by pre-operative computed tomography and its influence on frontal sinusitis was investigated.

Results: In all, 42 per cent of 186 sides were affected by frontal sinusitis. Agger nasi, ethmoid bulla, fronto-ethmoidal, suprabullar and frontal bulla cells were identified in 99 per cent, 100 per cent, 38 per cent, 69 per cent, and 16 per cent of sides, respectively. The presence of frontal recess cells and frontal ostium size did not significantly influence frontal sinusitis development. However, opacification of agger nasi, type 1 fronto-ethmoidal and suprabullar cells significantly influenced frontal sinusitis development.

Conclusion: Frontal sinusitis is caused by inflammatory changes in frontal recess cells.

Key words: Frontal Sinus; Helical Computed Tomography; Paranasal Sinus Diseases; Eosinophil

Introduction

Chronic rhinosinusitis patients suffering from headaches due to repeated frontal sinusitis episodes are commonly seen in ENT practice. Acute inflammatory exacerbation of the frontal sinus is a risk factor for intracranial and/or intraorbital complications, such as brain abscess.[1] Inflamed, thickened mucosae and mucopurulent secretions in the frontal sinus and the presence of frontal recess cells narrow the frontal drainage pathway. This can consequently block drainage, thereby contributing to frontal sinusitis pathogenesis. Topical and/or systematic pharmacotherapy using antibiotics and corticosteroids is recommended to manage the inflammation. Surgery is indicated for patients with chronic rhinosinusitis who are refractory to topical and medical treatments. The best option is functional endoscopic sinus surgery to pneumatise and drain the frontal sinus, which involves enlarging the frontal sinus drainage pathway. Three procedures were described by Draf[2]: type I, simple drainage; types IIa and IIb, extended drainage; and type III, endonasal median drainage. The FESS procedure can be performed when the frontal sinus drainage pathway is difficult to identify due to osteoplastic obliteration, and has therefore expanded the range of indications for endonasal frontal sinus surgery. Where type III drainage is technically impossible or has failed, external surgery is indicated. The frontal sinus is one of the most anatomically complex and inaccessible parts of the sinonasal area.[3] Frontal sinus surgery must therefore be performed using angled endoscopes by proficient, experienced surgeons. However, there is always a risk of injury to adjacent tissues, such as the base of the skull, orbit and anterior ethmoidal artery, which may result in serious intracranial and intraorbital complications. To perform frontal sinus drainage safely, full anatomical knowledge of the sinonasal area, especially the frontal recess cells and the frontal sinus border area, is necessary.

The number of patients with eosinophil-dominant (i.e. eosinophilic) chronic rhinosinusitis is increasing.[4] The most effective therapeutic strategy is usually to combine topical and/or systemic corticosteroids and endoscopic sinus surgery. In such patients, computed tomography (CT) images show opacification of the posterior ethmoid sinus and the olfactory cleft at an early stage[5]; in contrast, the maxillary sinus predominates, extending to the anterior ethmoid sinus and frontal sinus, in non-eosinophilic chronic rhinosinusitis.[6] Therefore, differentiating eosinophilic from

Accepted for publication 18 February 2017 First published online 20 April 2017

non-eosinophilic chronic rhinosinusitis is critical to an analysis of pathogenesis.

This pre-operative radiological study aimed to assess the anatomy of frontal recess cells in chronic rhinosinusitis patients.[7] Cells within the frontal recess that strongly influence frontal sinusitis development were identified and associations of frontal sinusitis with eosinophilic *vs* non-eosinophilic chronic rhinosinusitis were determined.

Materials and methods

Patients

A case series study of 93 patients (186 sides) who underwent primary sinonasal surgery at Hyogo College of Medicine between April 2015 and March 2016 was performed. In all, 64 male and 29 female patients with a mean age of 49 years (range 13–83 years) were included. The presence of cells and their opacification (i.e. inflammation) status were investigated bilaterally in the frontal recesses of all patients. Patients with tumour-associated disease, trauma or history of any sinonasal surgery were excluded. The study conformed to the regulations of the ethics committee of Hyogo College of Medicine (approval number 1512).

Eosinophilic chronic rhinosinusitis was diagnosed according to the criteria of the Japanese Epidemiological Survey of Refractory Eosinophilic Chronic Rhinosinusitis.[4] A total score from four items of at least 11 points was necessary for diagnosis: bilateral lesions (3 points); nasal polyps (2 points); ethmoid sinus dominant or pansinusitis on CT (2 points); and the percentage of blood eosinophils – more than 2 per cent and up to 5 per cent (4 points), over 5 per cent and up to 10 per cent (8 points) or over 10 per cent (10 points).

Patients were divided into three groups: an eosinophilic chronic rhinosinusitis group ($n = 32$, 64 sides), a non-eosinophilic chronic rhinosinusitis group ($n = 49$, 98 sides) and a control group without sinusitis ($n = 12$, 24 sides). Patients in the eosinophilic and non-eosinophilic chronic rhinosinusitis groups underwent endoscopic sinus surgery, and those in the control group underwent septoplasty and inferior turbinate surgery under general anaesthesia.

Radiological analysis of the frontal recess and frontal sinusitis

Kuhn's classification of frontal recess cells[7] (Table I) was used by three rhinologists to determine the presence and degree of opacification of agger nasi, types 1–4 fronto-ethmoidal, ethmoidal bulla, suprabullar and frontal bulla cells on pre-operative sinonasal axial, coronal and sagittal CT images (Figure 1).

The severity of chronic rhinosinusitis was assessed on sinonasal CT images using the Lund and Mackay scoring system and a scoring system previously reported by the present authors.[8,9] Opacification (indicating inflammation) of the maxillary, frontal, anterior and posterior ethmoid and sphenoid sinuses, and olfactory clefts was scored as: 0, not opaque; 1, partially opaque; or 2, completely opaque. Opacification of the ostiomeatal complex was scored as: 0, not opaque; or 2, opaque. Thus, the maximum possible total CT score was 14 points per side. Frontal sinusitis was defined as partial (1 point) or complete (2 points) opacification of the frontal sinus.

Relationships between (1) the presence of frontal recess cells (anatomical factors) and frontal sinusitis development and (2) opacification of frontal recess cells (inflammation) and frontal sinusitis development were evaluated.

The relationship between frontal sinusitis and the anterior–posterior diameter of the frontal ostium was investigated (Figure 2a). For this, the anterior–posterior diameter was defined as the shortest distance between the most prominent portion of the frontal beak and the posterior table of the frontal sinus. Effects of laterality on the anterior–posterior diameter of the frontal ostium and the relationship between the anterior–posterior diameter and frontal sinusitis were investigated.

Statistical analysis

Associations of the presence and opacification status of frontal recess cells with frontal sinusitis were analysed using χ^2 tests. Results between groups were compared using the Mann–Whitney U-test. Data are presented as means ± standard deviation, unless otherwise indicated. All p values are two sided and p values of < 0.05 were considered statistically significant. All statistical analyses were performed using Stat Flex version 6.0 software (Osaka, Japan).

TABLE I
FRONTAL RECESS CELL TYPES IN THE ANTERIOR ETHMOID SINUS

Cell type	Location
Agger nasi	The most anterior cells lying above the insertion of the middle turbinate
Fronto-ethmoidal	Cells in close proximity to the frontal process of the maxilla
– Type 1	Single fronto-ethmoidal cells lying above agger nasi cells
– Type 2	Tier of fronto-ethmoidal cells lying above agger nasi cells
– Type 3	Fronto-ethmoidal cells that pneumatise cephalad into the frontal sinus through the frontal ostium, but not extending beyond 50% of the vertical height of that frontal sinus
– Type 4	Fronto-ethmoidal cells extending more than 50% of the vertical height of the frontal sinus
Ethmoidal bulla	Cells located between uncinate process and middle turbinate
Suprabullar	Cells above the ethmoidal bulla cells that do not enter the frontal sinus
Frontal bulla	Cells originating in the suprabullar region that pneumatise along the skull base into the frontal sinus along the posterior wall of the frontal sinus

https://doi.org/10.1017/S002221511700086X Published online by Cambridge University Press

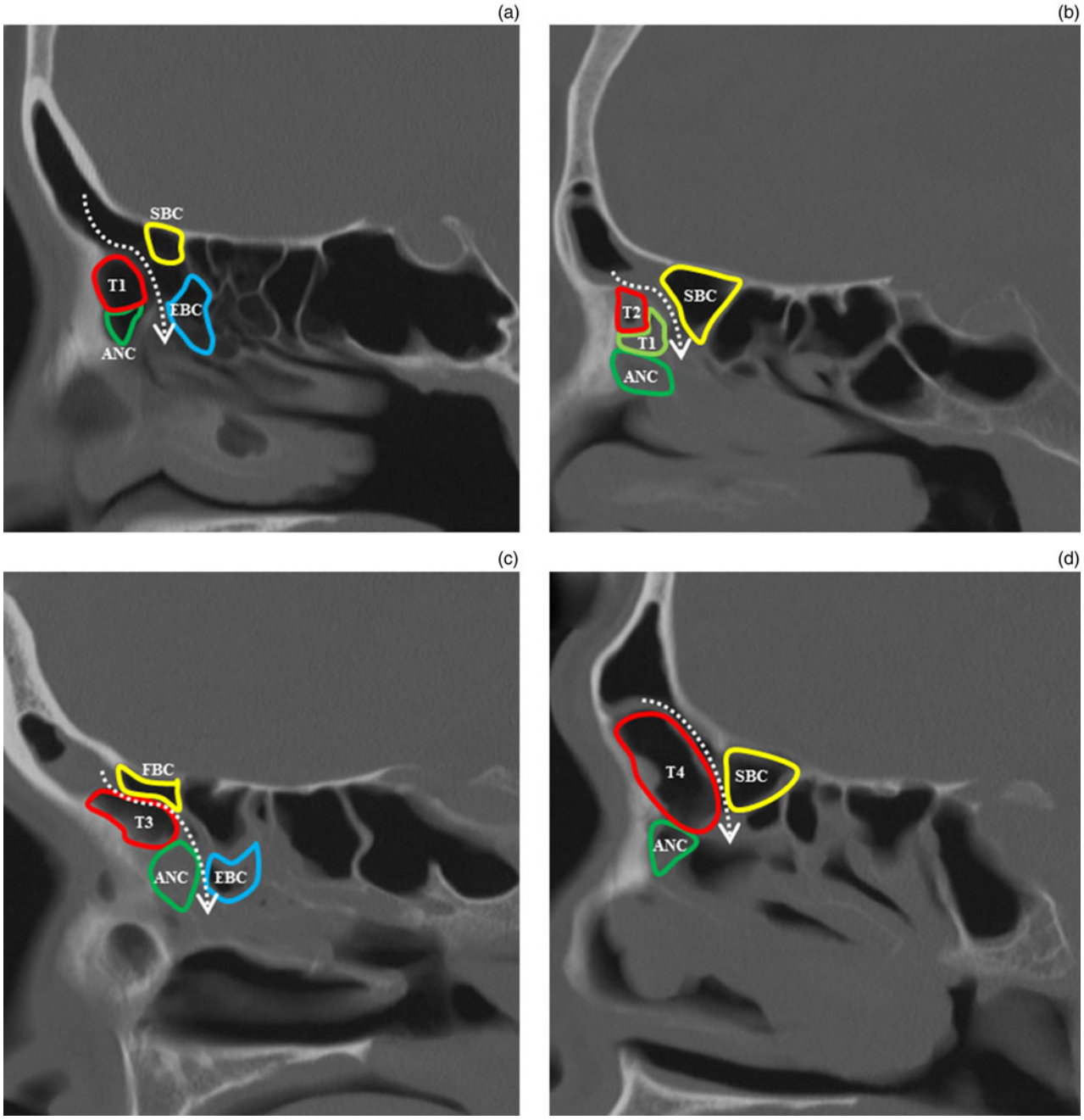

FIG. 1

Sagittal sinonasal computed tomography scans showing identification of frontal recess cells. Fronto-ethmoidal cells are classified as (a) type 1 (T1), (b) type 2 (T2), (c) type 3 (T3) and (d) type 4 (T4). Dotted arrows indicate the frontal sinus drainage pathway. ANC = agger nasi cell; EBC = ethmoidal bulla cell; SBC = suprabullar cell; FBC = frontal bulla cell

Results

Presence of frontal recess cells and frontal sinusitis

Frontal sinusitis was observed in 42 per cent of sides (78 out of 186; Table II). Computed tomography showed partial and complete opacification of 53 per cent (41 sides) and 47 per cent (37 sides) of frontal sinuses, respectively. Agger nasi cells were observed in 99 per cent of sides (184 out of 186). Fronto-ethmoidal cells were noted in 38 per cent of sides (71 out of 186): 20 per cent of cells were type 1 (38 out of 186), 1 per cent were type 2 (1 out of 186), 15 per cent were type 3 (28 out of 186) and 2 per cent were type 4 (4 out of 186). Ethmoid bulla cells, suprabullar cells, and frontal bulla cells were identified in 100 per cent (186 out of 186), 69 per cent (128 out of 186) and 16 per cent (29 out of 186) of sides, respectively. The presence of frontal recess cells was not significantly associated with frontal sinusitis development.

Anterior–posterior diameter of the frontal drainage pathway and frontal sinusitis

The anterior–posterior diameter of the frontal ostium was significantly larger on the left side (8.57 ±

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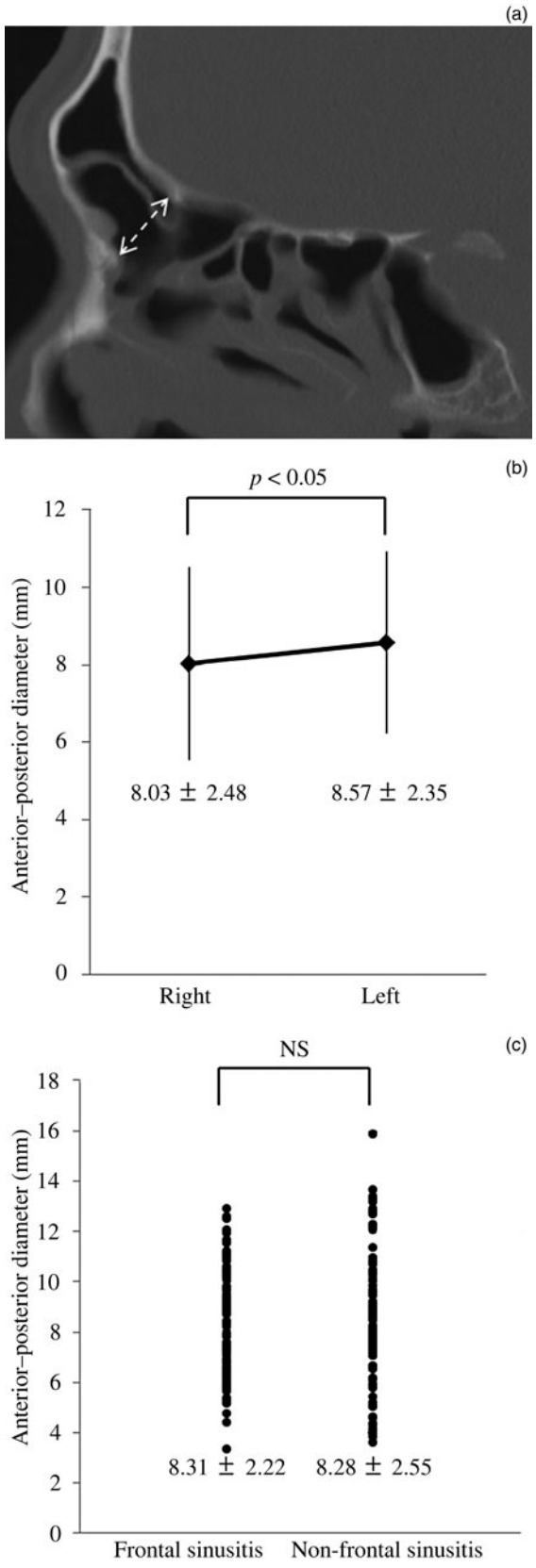

FIG. 2

(a) Sagittal sinonasal computed tomography image showing the anterior–posterior diameter of the frontal ostium (indicated by the dotted arrow). (b) Graph showing the mean anterior–posterior diameters for the right and left sides (error bars represent standard deviation (SD)). (c) Graph showing individual and mean ± SD anterior–posterior diameter values in frontal sinusitis and non-frontal sinusitis patients. NS = not significant

2.35 mm) than on the right side (8.03 ± 2.48 mm; Figure 2b). There was no significant difference in anterior–posterior diameter between patients with (8.31 ± 2.22 mm, 78 sides) and without (8.28 ± 2.55 mm, 108 sides) frontal sinusitis (Figure 2c).

Comparisons among patient groups

Frontal sinusitis was significantly more common in the eosinophilic chronic rhinosinusitis group (81 per cent, 52 out of 64 sides) than in the non-eosinophilic chronic rhinosinusitis (27 per cent, 26 out of 72 sides) and control groups (0 per cent; Figure 3a). The frontal sinus score was significantly higher in the eosinophilic chronic rhinosinusitis group (1.22 ± 0.75, $n = 64$) than in the non-eosinophilic chronic rhinosinusitis group (0.38 ± 0.68, $n = 98$). The total CT score was also significantly higher in the eosinophilic chronic rhinosinusitis group (8.8 ± 3.1) than in the non-eosinophilic chronic rhinosinusitis (2.9 ± 3.1) and control (0.0 ± 0.2) groups (Figure 3b).

Opacification of frontal recess cells and frontal sinusitis

In the eosinophilic chronic rhinosinusitis group, the proportion of agger nasi, type 1 fronto-ethmoidal, and suprabullar cells showing opacification was 96 per cent (50 out of 52 sides), 100 per cent (8 out of 8), and 95 per cent (35 out of 37), respectively. The presence of opacification was significantly associated with frontal sinusitis development in this patient group (Table III). In the non-eosinophilic chronic rhinosinusitis group, the proportion of ethmoid bulla, agger nasi, type 1 fronto-ethmoidal and suprabullar cells showing opacification was 85 per cent (22 out of 26), 92 per cent (24 out of 26), 100 per cent (7 out of 7) and 73 per cent (11 out of 15). The presence of opacification was also significantly associated with frontal sinusitis in this patient group (Table IV).

Discussion

A radiological investigation into the presence of frontal recess cells in the region of the frontal sinus drainage pathway and their percentage opacification in preoperative CT scans of patients who underwent sinonasal surgery was performed. Relationships between these measures and frontal sinusitis development were assessed.

Agger nasi cells and ethmoid bulla cells were detected in more than 98 per cent of patients, and the proportions of fronto-ethmoidal (38 per cent) and frontal bulla (16 per cent) cells were similar to those previously reported.[10–16] The most commonly identified cells were fronto-ethmoidal (71 sides), type 1 (54 per cent, 38 out of 71) and type 3 (39 per cent, 28 out of 71) cells, whereas type 2 (1 per cent, 1 out of 71) and type 4 (6 per cent, 4 out of 71) fronto-ethmoidal cells were rarely seen. The proportion of images showing suprabullar cells (68 per cent) was higher in the present study than in previous studies

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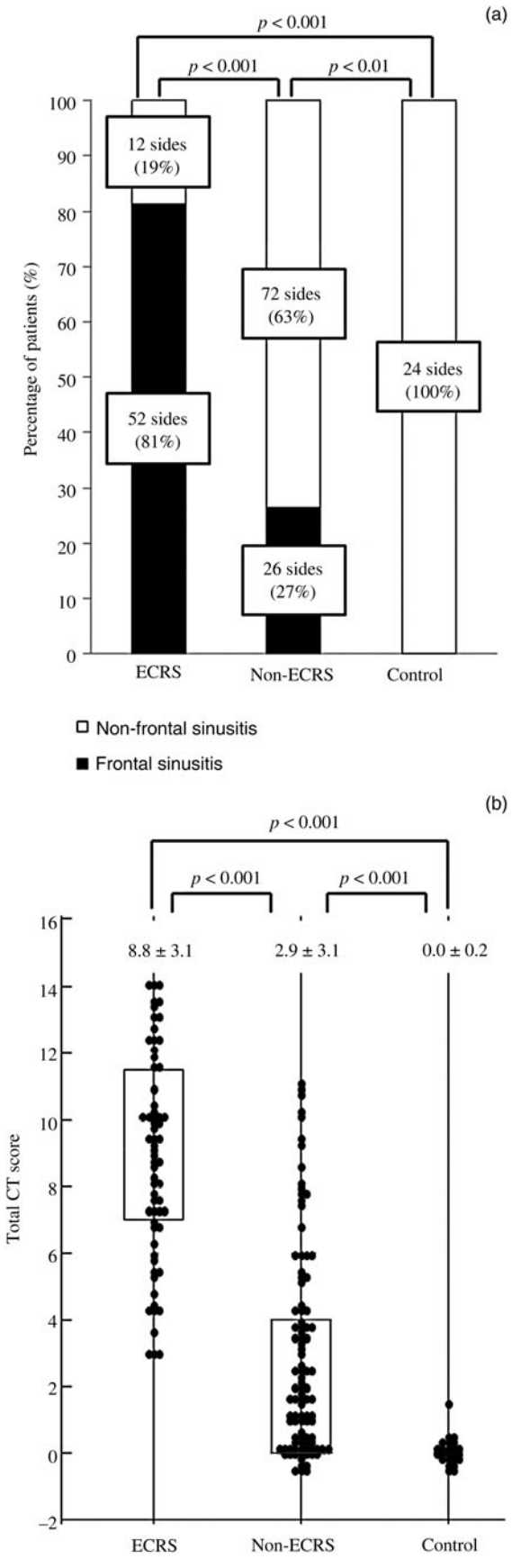

FIG. 3

(a) Graph showing the proportions of each patient group with frontal sinusitis. (b) Box and whisker plot showing computed tomography (CT) scores in each patient group. Mean ± standard deviation values are shown. ECRS = eosinophilic chronic rhinosinusitis

(range 11–40 per cent).[10–14] The presence of fronto-ethmoidal (types 3–4), suprabullar and frontal bulla cells is reported to significantly influence frontal sinusitis development.[17,18] As frontal bulla and types 3 and 4 fronto-ethmoidal cells grow into the frontal sinus and narrow the frontal sinus drainage pathway, these cells may physically block passage through the frontal ostium. In contrast, DelGaudio *et al.* and Eweiss *et al.* reported that fronto-ethmoidal cells do not influence frontal sinusitis development.[19,20] The present study similarly found that the presence of frontal recess cells is not a significant influence on frontal sinusitis development. It is possible that frontal sinus pneumatisation is maintained when the frontal sinus drainage pathway is narrowed and is only prevented by complete obliteration of the pathway. To investigate this possibility, frontal sinus drainage should be studied at the cellular level, for example by assessing ciliary function.

In the present study, the mean anterior–posterior diameter of the left frontal sinus was larger compared with the right frontal sinus, as previously reported.[21] This is because the right hemisphere of the human brain continues developing until a later growth stage compared with the left, thus reducing the final size of right frontal sinus. There was no significant difference in frontal ostium size between patients with and without frontal sinusitis in this study. Therefore, frontal sinusitis may be caused by inflammatory changes in frontal recess cells rather than changes in frontal ostium size.

Inflammatory opacification of agger nasi, suprabullar and type 1 fronto-ethmoidal cells significantly influenced frontal sinusitis development in both eosinophilic and non-eosinophilic chronic rhinosinusitis patients (Tables III and IV). Consistent with this finding, DelGaudio *et al.* proposed that mucosal inflammation is a major contributory factor in the pathogenesis of frontal sinusitis.[19] Most cases of non-eosinophilic chronic rhinosinusitis (primarily affecting the maxillary sinus) show initial inflammatory changes and impaired ventilation in the anterior sinonasal area.[15] In contrast, most cases of eosinophilic chronic rhinosinusitis (primarily affecting the ethmoid sinus) show initial inflammatory changes in the posterior sinonasal area, such as the posterior ethmoid sinus and olfactory clefts.[5] Bilateral pansinusitis was predominant in eosinophilic chronic rhinosinusitis patients, whereas partial sinus opacification was predominant in non-eosinophilic chronic rhinosinusitis patients. Although the mean total CT score was significantly lower in the non-eosinophilic chronic rhinosinusitis group than in the eosinophilic chronic rhinosinusitis group, ethmoid bulla cell opacification in the non-eosinophilic chronic rhinosinusitis group significantly influenced frontal sinusitis development. These data suggest that thickened mucosae and secretions due to inflammation of frontal recess cells (agger nasi, type 1 fronto-ethmoidal, ethmoid bulla

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TABLE II
PRESENCE OF FRONTAL RECESS CELL AND FRONTAL SINUSITIS

Cell type	Frontal sinusitis (78 sides), *n* (%)	Non-frontal sinusitis (108 sides)	Odds ratio (95% CI)	*p* value
ANC	78 (100)	106 (98)	–	0.51
FEC	31 (40)	40 (37)		
– Type 1	15 (19)	23 (21)	0.88 (0.43–1.82)	0.73
– Type 2	0 (0)	1 (1)	–	1.00
– Type 3	15 (19)	13 (12)	1.74 (0.78–3.88)	0.18
– Type 4	1 (1)	3 (3)	0.45 (0.05–4.21)	0.64
EBC	78 (100)	108 (100)	–	1.00
SBC	52 (67)	76 (70)	0.84 (0.45–1.58)	0.59
FBC	13 (17)	16 (15)	1.15 (0.52–2.55)	0.73

CI = confidence interval; ANC = agger nasi cells; FEC = fronto-ethmoidal cells; EBC = ethmoidal bulla cells; SBC = suprabullar cells; FBC = frontal bulla cells

TABLE III
FRONTAL RECESS CELL OPACIFICATION AND FRONTAL SINUSITIS IN THE EOSINOPHILIC CHRONIC RHINOSINUSITIS GROUP

Cell type	Frontal sinusitis (52 sides)*	Non-frontal sinusitis (12 sides)*	Odds ratio (95% CI)	*p* value
ANC	50/52 (96)	8/12 (67)	12.5 (2.61–59.91)	< 0.01
FEC	14/20 (70)	4/9 (44)		
– Type 1	8/8 (100)	1/4 (25)	–	< 0.05
– Type 2	0/0 (0)	0/0 (0)	–	1.00
– Type 3	6/12 (50)	2/4 (50)	1.0	1.00
– Type 4	0/0 (0)	1/1 (100)	–	1.00
EBC	52/52 (100)	11/12 (92)	–	0.19
SBC	35/37 (95)	2/4 (50)	17.5 (2.45–124.81)	< 0.05
FBC	5/8 (62)	1/3 (33)	3.33 (0.22–50.97)	0.55

*Data show opacification / presence (%) for each cell type. CI = confidence interval; ANC = agger nasi cells; FEC = fronto-ethmoidal cells; EBC = ethmoidal bulla cells; SBC = suprabullar cells; FBC = frontal bulla cells

TABLE IV
FRONTAL RECESS CELL OPACIFICATION AND FRONTAL SINUSITIS IN THE NON-EOSINOPHILIC CHRONIC RHINOSINUSITIS GROUP

Cell type	Frontal sinusitis (26 sides)*	Non-frontal sinusitis (72 sides)*	Odds ratio (95% CI)	*p* value
ANC	24/26 (92)	25/71 (35)	22.08 (6.53–74.60)	< 0.001
FEC	9/11 (82)	6/22 (27)		
– Type 1	7/7 (100)	3/14 (21)	–	< 0.01
– Type 2	0/0 (0)	0/0 (0)	–	1.00
– Type 3	2/3 (67)	1/6 (17)	10.0 (0.49–202.60)	0.46
– Type 4	0/1 (0)	2/2 (100)	–	0.33
EBC	22/26 (85)	21/72 (29)	13.36 (4.72–37.80)	< 0.001
SBC	11/15 (73)	16/52 (31)	6.19 (1.85–20.68)	< 0.05
FBC	2/5 (40)	3/10 (30)	1.56 (0.17–14.55)	1.00

*Data show opacification / presence (%) for each cell type. CI = confidence interval; ANC = agger nasi cells; FEC = fronto-ethmoidal cells; EBC = ethmoidal bulla cells; SBC = suprabullar cells; FBC = frontal bulla cells

and suprabullar cells) may block the frontal sinus drainage pathway and consequently influence frontal sinusitis development, even if the sinonasal area is only partially inflamed. For surgical management of frontal sinusitis, it is particularly important to remove these inflammatory cells to enlarge the frontal sinus drainage pathway.

There is a need to plan and perform safe and adequate sinus surgery for frontal sinusitis. This is difficult because the frontal sinus is one of the most anatomically complex and inaccessible parts of the sinonasal region for FESS.[3] The present radiological study identified critical sites for FESS in the frontal sinus. A comprehensive understanding of the anatomy of the frontal sinus drainage pathway based on CT could help pre-operative planning. However, as frontal sinusitis is caused by various factors, including anatomical variation and mucous membrane

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inflammation, larger multidisciplinary studies aimed at defining the factors influencing this disease are required.

- **Acute inflammatory exacerbations in the frontal sinus are a risk factor for intracranial and/or intraorbital complications**
- **Inflamed, thickened mucosae and mucopurulent secretions in the frontal sinus and frontal recess cells narrow the frontal drainage pathway**
- **This study analysed frontal recess cells in pre-operative radiological images of chronic rhinosinusitis patients**
- **The presence of frontal recess cells did not significantly influence frontal sinusitis development**
- **Opacification of agger nasi, suprabullar and type 1 fronto-ethmoidal cells significantly influenced frontal sinusitis development in both eosinophilic and non-eosinophilic chronic rhinosinusitis patients**
- **A full understanding of the frontal sinus drainage pathway is required for surgical management of frontal sinusitis**

Conclusion

Frontal sinusitis is caused by inflammatory changes in frontal recess cells. Complete removal of inflamed agger nasi, type 1 fronto-ethmoidal, ethmoid bulla and suprabullar cells is important for the surgical management of this condition. A full understanding of the anatomy of the frontal sinus drainage pathway is also required.

Acknowledgements

The authors gratefully acknowledge the technical assistance of Ms Y Kida and Mrs M Tanide. This work was supported by the Japan Society for the Promotion of Science Grants-in-Aid for Scientific Research (KAKENHI; numbers JP25462671 and JP16K11220), the Practical Research Project for Rare/Intractable Diseases from the Japan Agency for Medical Research and Development and Grants-in-Aid for Researchers, Hyogo College of Medicine, for 2015 (to KT) and 2016 (to KH).

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Dr K Tsuzuki takes responsibility for the integrity of the content of the paper
Competing interests: None declared