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Original Article

Regulatory function of TH17/Treg ratios in the children suffering from adenoidal hypertrophy with complications

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Abstract: Objective: To investigate the differences between the TH17 (cells)/Treg (regulatory Treg cells) ratios and the correlated factors IL-17, IL-10, TGF-β levels in the children suffering from Adenoidal hypertrophy (AH) complicated with/or without allergic rhinitis (AR) and/or secretory otitis media (SOM). Methods: 175 children were divided into five groups: the control group was Group1 (Degree I or II of Adenoidal hypertrophy without complications, 22 cases); Group 2 (the AH alone group, 55 cases), Group 3 (the AH + SOM group, 37 cases), Group 4 (the AH + AR group, 30 cases), Group 5 (AH + SOM + AR group, 31 cases). The flow cytometer was applied to determine the Th17, Treg levels in the peripheral blood and local adenoid tissue; the ELISA method was performed to determine the IL-17. IL-10, TGF-β levels and the RT-PCR was used to determine the Foxp3 and RORγt levels. Results: The Th17/Treg ratios were upregulated in both the peripheral blood and local adenoid tissues of the patients in Group 3 but decreased in Group 2. There were no significant differences in the deviation of the Th17/Treg ratios between Group 5 and Group 2, but the ratios in Group 3 were unregulated. The RORγt mRNA levels in Group 2 (39.53±7.73) increased significantly compared with those in Group 1 (5.37±3.19). However, the RORγt mRNA levels in Group 3 (27.31±3.65) were lower than Group 1 but higher than Group 2 and the difference was statistically significant. The RORγt mRNA levels were higher in Group 5 than in Group 3 and Group 4, while the Foxp3 mRNA levels were lower in Group 3 and Group 4. The TGF-β levels in the peripheral blood and adenoid tissue samples changed significantly in all the groups. The IL-10 and IL-17 levels were different between the Group 2 and the control group, but there was no significant difference in Group 3. Conclusion: AR, SOMand AH might realize the systemic immuno-regulation thorough Treg cells.

Keywords: Adenoid hypertrophy, secretory otitis media, allergic rhinitis, treg cell

Introduction

Adenoid tissue is a member of lymphoid tissues (MALT) associated with the upper airway mucosa, and T cells play an important role in the local immune response. Adenoid hypertrophy (AH) is the main cause of obstructive sleep apnea hypopnea syndrome in children. Studies found [1] that the deviated ratio of Th17 to Treg cell subsets in local adenoid tissue and the peripheral blood of children with AH is associated with the development of diseases.

Secretory otitis media (SOM) is a non-suppurative inflammatory disease in the middle ear characterized by middle-ear tympanic effusions (including serous and mucous or pulp mucus) and hearing loss in the middle ear. It is one of the most frequent diseases in children. Many studies have been focused on OME caused by AH. Mucosal epithelium in the middle ear contains a large number of mucosa-related lymphoid tissues. The occurrence of mucosa-specific immunity is closely related to MALT. Therefore, the onset of exudative otitis media complicated by AH is also associated with the autoimmunity of adenoid in addition to the Eustachian tube drainage blocked by AH. It has been confirmed that adenoid [2] is involved in the local immune response of otitis media with effusion. In this immune response, however, the role of Treg cells and their interaction with effector T cells is unknown. Allergic rhinitis is prevalent in children. Many children diagnosed with obstructive sleep apnea syndrome

(OSAHS) have been complicated with allergic rhinitis. In recent years, some relevant studies [3] believe that regulatory T cells and Th17 ones play a pivotal role in the pathogenesis of allergic rhinitis (AR). Th17 and Treg cells showed significant changes in children with AH. The Vitamin A acid orphan nuclear receptorγ (RORγt) and the fork-head or winged helix transcription factor (Foxp3) are specific transcription factors regulating the differentiation of Treg cells. Whether the changes of Treg and Th17 ratios are different from those of RORγT and Foxp3 transcription factors complicated with allergic rhinitis remains to be elucidated.

Clinically, high-rated complications of AR and/or OME have been found in children with AH. Treg and Th17 cells play an crucial role in the pathogenesis of the three kinds of diseases, so the relationship between them is worth studying. In this study, we investigated the differences between the TH17 (cells)/Treg (regulatory Treg cells) ratios and IL-17, IL-10, TGF-β levels in the children suffering from Adenoidal hypertrophy (AH) with complications .

Materials and methods

General information

Between September 2013 and September, 2015, 175 cases, including 81 males and 72 females (mean, 4.97±2.11 years), were treated in our hospital. The patients with cardiovascular, endocrinal, urinary, metabolic or neuromuscular diseases were excluded from the study based on their self-reported medical history. None of the included patients had chemical history of aspirin specific reaction, bronchial dilation or autoimmune diseases, nor did they have upper respiratory tract infection within 2 weeks. The allergens were determined using the Skin prick test and a fibrous nasopharyngoscopy test was performed on adenoid tissues. An acoustic immittance examination and the polysomnography (PSG) were also performed. The peripheral blood and adenoid tissue were collected from all the included patients. The study was approved by the Ethics Committee of the hospital and carried out with the written informed consent of all the children and their custodians.

All the enrolled children received fibrous nasopharyngoscopy and were divided into groups in terms of the severity of obstructive posterior nares by adenoids. The range of 0-25% was presented as Degree I adenoid, 26%-50% as Degree II adenoid, 51%-75% as Degree III adenoid and 76%-100% as Degree IV adenoid [4]. Children presenting Degree III and Degree IV adenoid were diagnosed as having AH; the ones having Type-B tympanogram or without elicited stapedial reflex were diagnosed as having OME; the ones having symptoms of AR and positive skin prick test were diagnosed as having allergic rhinitis. In accordance with *Children with obstructive sleep apnea syndrome treatment guidelines* released in 2007, obstructive apnea index (AOI > 1/h) or obstructive apnea hypopnea index(AHI > 5-fold/h) and the lowest oxygen saturation <92% could be diagnosed as OSAHS.

Grouping

175 cases were divided into five groups. The control group was Group 1 (22 cases of children with Degree I/II adenoid but without OME/AR); Group 2 (the AH group) included 55 cases of children with AH alone; Group 3 (the AH + OME) included 37 cases of children having AH complicated with OME; Group 4 (the AH + AR) included 20 cases having AH complicated with AR and Group 5 (the AH + OME + AR) contains 31 cases of children having AH complicated with OME and AR.

Experimental methods

Determination of Th17 and Treg cells in peripheral blood

Preparation of peripheral blood Mononuclear cells (PBMC): Venous peripheral blood (3 ml) was collected in two heparin anticoagulation tubes. Within 4 h, the blood was diluted by supplementing with equivalent HBSS (Hank balanced saline buffer, pH 7.2-7.4); then lymphocytes separating solution (Ficollsolution) equivalent with the diluted blood in size was poured into a centrifuge tube. Next, the diluted blood was carefully added onto the lymphocytes separating solution between which a clear interface should be kept. The solution was centrifuged at room temperature for 20 m (2000 r/min). Mononuclear cells were sucked out gently by a capillary pipet and evenly mixed with HBSS (5 ml) at 1500 r/min in a tube. The mixture was centrifuged for 10 min and then the supernatant was discarded. The residue was washed

Table 1. Primer sequence

Gene	Primer	Product length (bp)	Tm (°C)
GAPDH	5-GGCTGTGGGCAAGGTCATCCCTG-3	101	67.62
	5-GACGGCAGGTCAGGTCCACCACTG-3		68.96
FOXP3	5-GGCAGCCAAGGCCCTGTCGTCC-3	91	70.07
	5-GGCTACCCCACAGGTGCCTCCGG-3		70.87
RORC	5-GTGCCCACCACCTCACCGAGGCC-3	148	71.51
	5-TAGGCCCGGCACATCCTAACCAGC-3		68.46

Table 2. The basic data of each group of children

Group	Case	Age (year)	Weight (kg/m^2)
Group 1	22	5.32±2.56	15.6±3.62
Group 2	55	5.62±3.16	15.8±4.12
Group 3	37	5.87±2.86	15.4±3.72
Group 4	30	5.48±2.74	15.7±4.21
Group 5	31	5.51±3.04	15.5±4.52
χ^2 value		18.691	20.426
P value		0.143	0.185

once and then again the supernatant was discarded. Mononuclear cells were resuspended in RPMI1640 and the living cells accounted for more than 95%, counted by the trypan blue staining. The cells' concentration was regulated to 2×10^6 cell/ml.

Cell culture

The PBMC suspensions (2*10^6/mL) were poured into T25 culture flasks (3 ml per flask) and added with PMA (50 ng/ml, bought from American Sigma Corporation), ionomycin (Ionomycincaicium salt) (1 μg/ml, American Sigma Corporation) and BFA (10 μg/ml) followed by stimulation and culture in an incubator for 5 h in a humidified atmosphere of 5% CO_2 at 37°C.

The flow cytometry assay was performed to sort human Th17/Treg cells (the experiment was performed in accordance with BD's (560762) instructions).

Determination of Th17 and Treg cells in adenoid tissues

Adenoid grinding and the preparation of mononuclear cell suspensions of adenoids: Surgically removed adenoids were washed with 75% alcohol solution for 10 s, or/and sterilized tissues were cleaned in PBS + 10% dual resistant and then washed in incomplete culture solution (RPMI-1640) for 10 s. The peripheral tissues were dissected under aseptic conditions and then the adenoid parenchyma was minced and grinded in a mesh (40 mm) using a syringe needle core. Mononuclear cell suspensions were prepared with 10% FBS + RPMI-1640, whose mononuclear cells were purified by means of density gradient centrifugation (Bicoll; Biochrom, Berlin, Germany) and then photographed and observed under an inverted fluorescence microscope. Single-cell suspensions were incubated and determined by the flow cytometry just as previously described for peripheral blood.

Determination of IL-17, IL-10, TGF-β by the ELISA method

Three mL of peripheral venous blood was extracted from each subject under the condition of fasting in the morning and then poured into a dry tube and centrifuged at 2000 r/min for 15 min and stored at -20°C after removal of the supernatant. Mononuclear cell suspensions of adenoids were prepared and stored. The levels of IL-17, IL-10 and TGF-β were determined by the enzyme linked immunosorbent assay (ELISA) with ELISA reagent kit (purchased from the EBIOSCIENCE Corporation). The whole procedure was completed in strict accordance with the kit's instructions.

Determination of Foxp3 and RORγt by RT-PCR

Extraction and identification of Total RNA: Total RNA was isolated from mononuclear cells in the Peripheral blood using TRIzol (TRIzol Reagent ambion company) in accordance with the manufacturer's protocol.

cDNA synthesis

the cDNA mixture was prepared in an RNase-free EP tube (0.2 mL) on ice at 37°C for 15 min and at 85°C for 5 sec and diluted for use by adding 10 μL ddH_2O.

PCR reaction

(1) The designed primer sequence and analyzed conditions are shown in **Table 1.** (2) Amplification detection. The quantification of relative expression was performed by using $2^{-\Delta\Delta Ct}$.

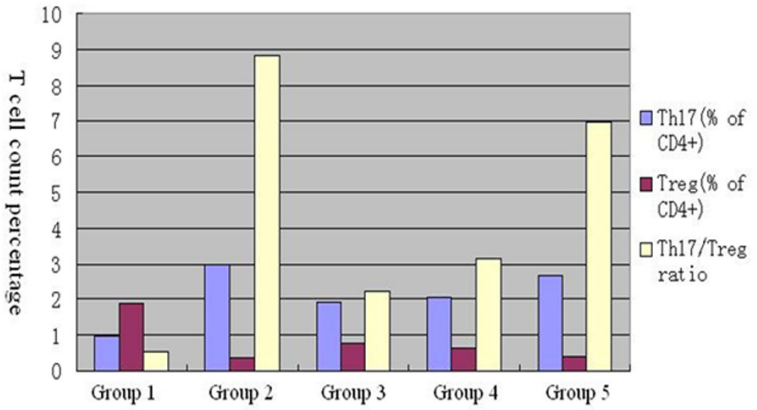

Figure 1. The content of T cell count in peripheral blood.

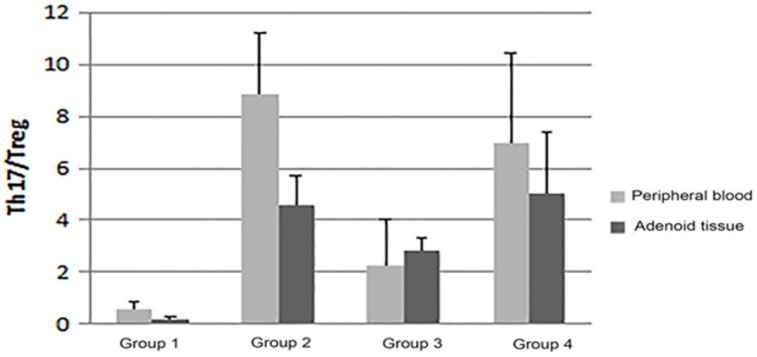

Figure 2. The change of Th17/Treg ratio in peripheral blood and adenoid tissue.

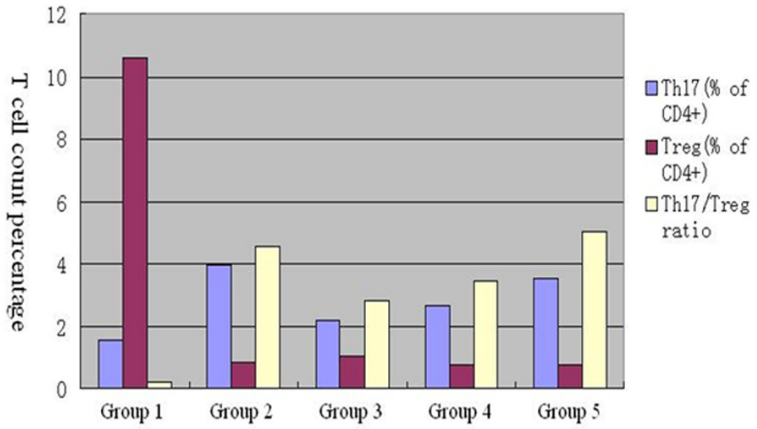

Figure 3. The content of T cell count in adenoid tissue.

Statistical methods

The statistical data of each group were tested for normal distribution. All the data were expressed as mean ± standard deviation. The differences in the content of RORγt and Foxp3 the Th17 to Treg ratios and IL-17, IL-10 and TGF-β levels between groups were compared using the independent-samples test while the differences among multiple groups were compared using the one-way ANOVA analysis.

All the statistical data were analyzed by SPSS 17.0, and $P<0.05$ was considered as statistically different.

Results

Basic information of the cases

All the patients included in this study were preschoolers aged 3-6 years, and the differences in age and weight among the groups were not statistically significant ($P > 0.05$), as shown in **Table 2**.

Determination of T cell counts in the peripheral blood and adenoid tissues

The Th17 to Treg ratios in the peripheral blood increased more significantly inGroup 2 than in Group 1 (8.82±2.36 vs.8.82±2.36), and the difference was statistically significant ($P<0.05$); the ratios of the Group 3 (2.22±1.8) and those of Group 4 (3.15±1.7) reduced significantly compared to those of Group 1 and there were statistically significant differences ($P<0.05$). The ratios of Group 5 (6.98±3.47) increased significantly compared to those of Group 5, and the difference was statistically significant ($P<0.05$); and also increased significantly compared to those of Group 3 and Group 4, and there were statistically significant differences ($P<0.05$); but no statistically significant differences compared to those of Group 2, as seen in **Figures 1** and **2**.

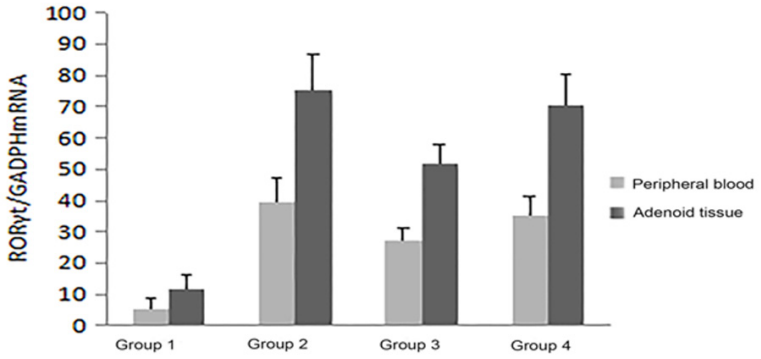

Figure 4. Results of determination of RORγt mRNA expression levels in peripheral blood and adenoid tissue.

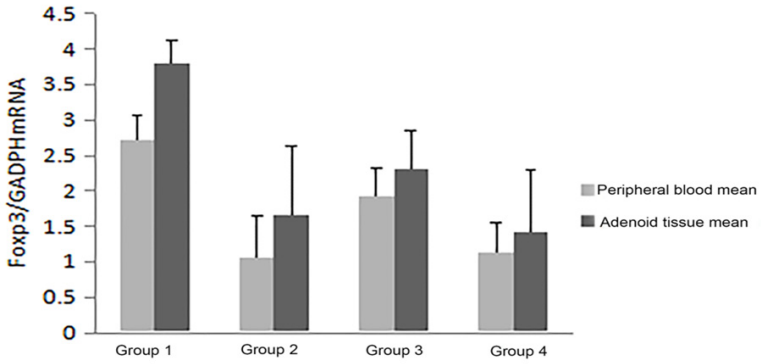

Figure 5. Results of determination of Foxp3 mRNA expression levels in peripheral blood and adenoid tissue.

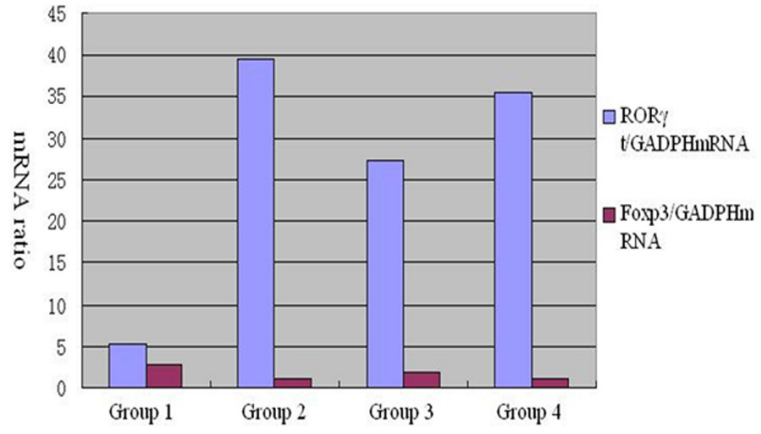

Figure 6. The expression of RORγt and Foxp3 in peripheral blood.

The Th17 to Treg ratios in the adenoid tissues increased more significantly in Group 2 than in Group 1 (4.56±1.15 vs. 0.20±0.07), and the difference was statistically significant (P<0.05); the ratios of Group 3 (2.81±0.49) and those of Group 4 (3.45±1.9) reduced significantly compared to those of Group 2 and there were statistically significant differences (P<0.05). The ratios of Group 3 (5.02±2.38) increased significantly compared with those of Group 1, and the difference was statistically significant (P<0.05); and also increased significantly compared with those of Group 3 and of Group 4, and there were statistically significant differences (P<0.05); but no statistically significant differences compared with those of Group 2, as shown in **Figures 2** and **3**.

Analysis on the determined results of Foxp3 mRNA and RORγt mRNA expression levels

The RORγt mRNA levels in the peripheral blood of Group 2 (39.53±7.73) increased significantly compared with those in Group 1 (5.37±3.19). However, the RORγt mRNA levels in Group 3 (27.31±3.65) were between those of Group 1 and of Group 2 and the difference was statistically significant. The RORγt mRNA levels were higher in Group 5 than in Group 4, suggesting the elevated transforming regulator Th17 cells while the RORγt mRNA levels were lower in Group 5 than in Group 3, indicating the significant reduction in the number of the Treg cells but no significant changes compared to those of Group 2. The determined results of the adenoid tissue were also consistent with this conclusion, as shown in **Figures 4**-**7**.

Changes of cytokines IL-17, IL-10 and TGF- levels in each group

The results showed that for the peripheral blood and the adenoid tissue samples, there were significant changes in the TGF-β levels in

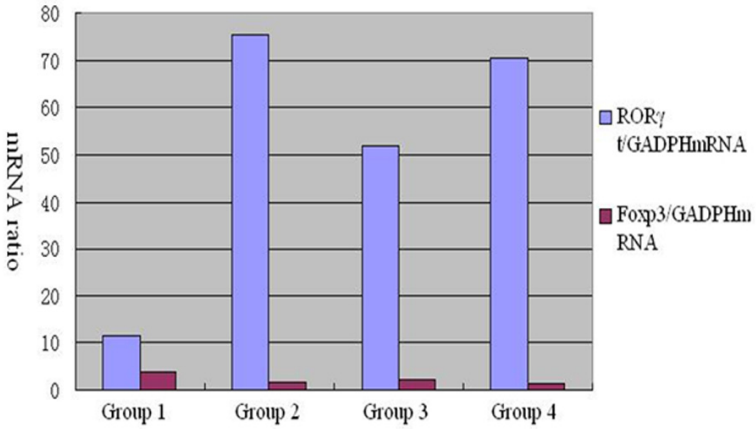

Figure 7. The expression of RORγt and Foxp3 in adenoid tissue.

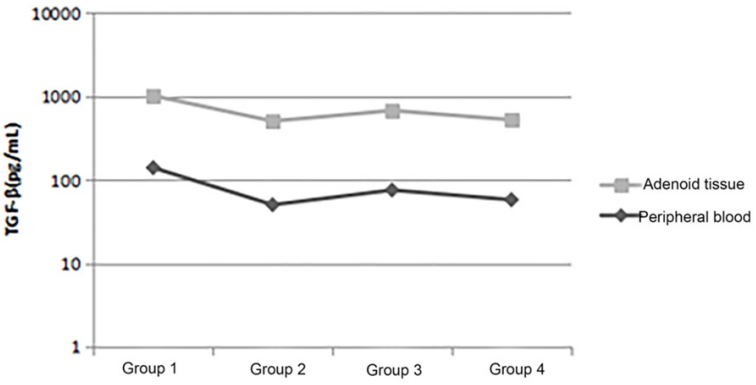

Figure 8. TGF-β levels in the peripheral blood and adenoid tissues. It shows TGF-β concentrations in each group. All the data are expressed as mean ± standard deviation.

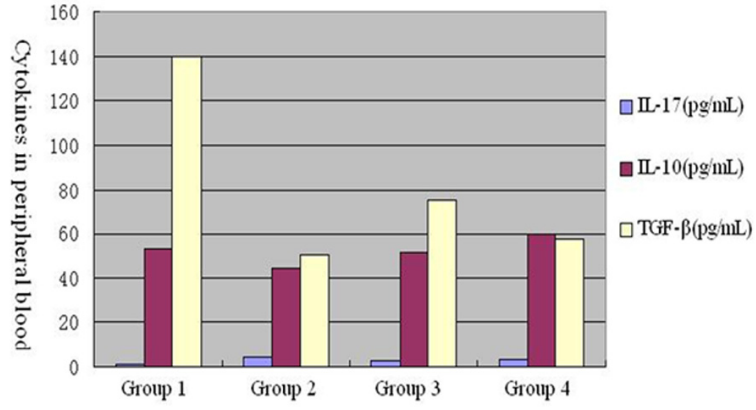

Figure 9. The expression of cytokines in peripheral blood.

the four groups (as shown in **Figures 8**-**10**). The cytokine secretions (IL-10 and IL-17) in Group 2 were different from those of Group 1 but not significantly different from those of Group 3. That is to say, it might be not evident associated with OME complications (**Table 2**).

Analysis on the correlation of clinical characteristics and AH with complications

To determine the correlation of clinical data and AH with complications, we conducted the multivariate *Logistic* regression analysis, and the results showed that the Th17/ Treg ratios were positive related to AH with complications, but the patients's age, gender, family history, the serum cytokines levels of IL-17, IL-10 and TGF-β were not related to the diseases (**Table 3**).

Discussion

The Th17/Treg ratio plays an important role in the pathogenesis of AH in children. Our study found that compared with Group 1, the children with AH had increased Th17 cells in the peripheral blood and local adenoid tissues, which elevated effector IL-17 and RORγt expression levels but reduced Treg proportion but reduced FoxP3 levels and effectors IL-10, TGF-β levels. The Th17/Treg ratios increased ($P<0.05$). The results are consistent with previous studies [1]. Meanwhile, we also found that children suffering from AH complicated with AR showed higher Th17/Treg ratios, and the ratio errors tended to be more obvious. (The difference was statistically significant). AH is one of the main reasons for obstructive sleep apnea-hypopnea syndrome (OSAHS) in children. In this study, Degrees III and IV AH children tested by PSG were consistent with diagnostic symptoms of

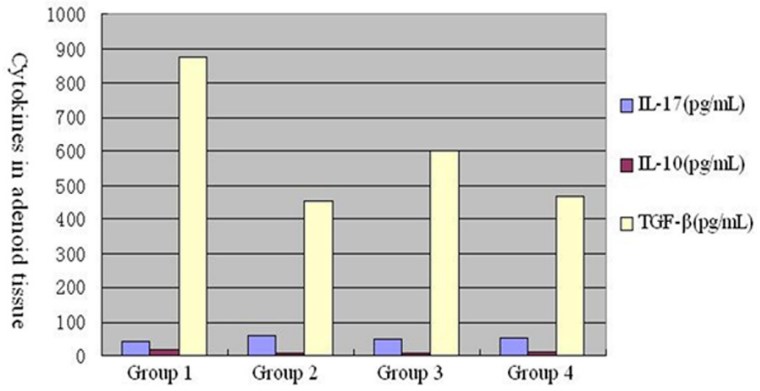

Figure 10. The expression of cytokines in adenoid tissue.

OSAHS. Some studies [5] found that OSAHS might be the trigger factors for the development of autoimmunity. The Th17/Treg ratio imbalance caused by sleep disorders will lead to immune tolerance and adaptive disorders, resulting in a continuous, low-grade systemic immune responses, such as autoimmune diseases or allergic diseases.

Studies have confirmed that [6], the positive correlation between the serum cytokine IL-17 expression levels and the clinical severity of the patients with AR showed that Th17 cells play a positive regulatory role in the onset of AR, and regulators T and Th17 cells play a very pivotal role in the incidence of AR. We found that the Th17/Treg ratios in the peripheral blood of the patients with OSAHS complicated with AR were higher than those of Group 1 and Group 2, and the difference was statistically significant. Thus we argue that the imbalance in Th17/Treg ratios caused by AR and OSAHS promote and expand with each other, leading to more significant clinical symptoms. Initial onset of either AR or OSAHS may cause the changes in Treg and Th17 subsets of T cells and the imbalance in Th17/Treg ratios, leading to the onset and development of other related diseases.

There are a variety of theories about the etiology and mechanisms of OME, such as microbial infection, Eustachian tube dysfunction and obstruction and AH obstruction etc., but none of the theories can explain all the cases. The theories about middle-ear mucosal immunological abnormalities have attracted much attention in recent years. With the appearance of the concept of "consistency of upper and lower respiratory tract inflammation", the occurrence and development of immunoreaction is being paid more and more attention [8]. Inherent autoimmunity has a protective effect on the middle ear. The inflammation in the middle ear destroys the protective reaction, leading to the immune imbalance in the middle ear [9, 10]. There are specific regulatory T cells in the normal immune system. The dysfunction of these cells may contribute to the pathogenesis of allergic diseases. The studies on the changes of T cell subsets in the pathogenesis of OME confirmed the abnormality of T cell subsets. Some researchers in China [11, 12] found that the $CD4^+/CD8^+$ ratios were significantly higher in the adenoid tissues and peripheral blood of the children with OME. Yongchen Lin studied 50 cases of SOM patients, and found that their $CD8^+$ levels were significantly lower but their $CD4^+/CD8^+$ levels higher than those of the control group. The primary lymphocytes in the middle-ear effusions of the SOM patients were $CD3^+$ T lymphocytes and the $CD4^+/CD8^+$ ratios were significantly higher than those in the peripheral blood. These findings suggested that $CD4^+$ T cell abnormalities played a vital role in the pathogenesis of otitis media. We know that Treg as T cell subsets with mature functions, suggested by a large number of studies, can suppress the immune response and regulate the progression of chronic inflammation by cell contacts and secretions of IL-10 and TGF-β1, and are related to the onset and development of chronic inflammatory diseases in the upper respiratory tract [15-17]. Treg has attracted much attention in the studies on OA in recent years, and studies have shown that the proportion of $Foxp3^+CD4^+CD25^+$ T/$CD4^+$ T cells in the peripheral blood in patients with chronic OME was significantly higher than that in the patients with acute OME and that in the normal controls, suggesting that the changes in the proportion of $Foxp3^+CD4^+CD25^+$ T/$CD4^+$ T cells may be associated with persistent inflammation in the patients with chronic OME [18].

There are many children suffering from AH complicated with OME and AR. Analyses revealed that the mechanism of AR to promote the occurrence of SOM might be implicated in

Table 3. Multivariate *Logistic* regression analysis of Clinical characteristics to adenoid hypertrophy with complications

	regression coefficient (β)	*standard error (SE)*	*odds ratio (OR)*	*Wald value*	*95% CI*	*P*
Age	1.079	1.427	2.943	3.418	2.267~4.923	0.089
Gender	0.316	1.154	1.371	0.784	0.436~1.109	0.136
Family history of AH	0.927	1.653	2.528	6.576	4.953~8.138	0.089
Th17 (% of $CD4^+$)	0.559	0.847	1.749	4.728	2.545~5.923	0.095
Treg (% of $CD4^+$)	-0.014	0.586	0.986	0.937	0.406~1.452	0.075
Th17/Treg ratio	1.271	0.983	3.563	8.412	3.247~13.472	0.026
IL-17	0.640	2.348	1.896	9.326	8.014~11.241	0.076
IL-10	1.010	1.285	2.746	7.021	3.206~8.954	0.075
TGF-β	0.980	1.076	2.664	10.154	8.106~12.374	0.086

mechanical obstruction and immune effects. AR might result in nasal mucosa edema, cause swelling and congestion of Eustachian tube mucosa and the formation of middle ear pressures, leading to the production of middle-ear effusions [1]. On the other hand, lymphoid system associated with mucosa in middle-ear mucosa epidermis is the main interface for the middle-ear system to defend against the pathogens. A variety of pathological factors can promote the migration and proliferation of immune cells to involve in the immune responses [19]. The attenuated immune suppression of T cells is the core factor in the pathogenesis of AR. Nasal mucosais is also a lymphoid system associated with the upper airway mucosa, so the association between nasal mucosa and the immune system of the mucosal epitheliums in the middle ear might be an important reason for the onset and development of OM. Ratner et. [20] found that compared with the normal control group, AR patients had the odds of having abnormal tympanogram (Type B and Type C), suggesting that such patients tend to suffer from eustachian tube dysfunction. Hurst [21] treated 89 Patients having refractory OME complicated with AR (127 ears). After specific desensitization treatment, 85% of them were cured and 55% significantly improved (efficacy of 90.5%). These results suggest that the AR is closely correlated to OM and studies on the abnormal relationship between the two diseases in Treg cells and the pathogenesis are helpful to reveal the impact of immune factors on AH and concurrent AR and OM.

This study investigated the changes of Th17/Treg ratios in the children suffering from AH complicated with OME and in children suffering from AH complicated with OME and AR. We found the Th17/Treg ratios in both peripheral blood and local adenoid tissues were higher in the children suffering from AH complicated with OME, which means decreased Treg cells compared to the controls. This seems inconsistent with the elevated Treg levels in the peripheral blood of the patients with OME in the previous studies. This might be attributed to the complications of AH. AH leads to the increase in Th17 cells but decrease in Treg cells. The Th17/Treg imbalance might have influenced the functions of the lymphoid tissues in the middle-ear mucosa via some mechanism, resulting in local Treg disorders in the middle-ear mucosa and abnormalities. According to our assumptions, the Th17 levels in middle-ear mucosa were also elevated, but due to the persistent inflammation, chronic lesions might cause the changes in the regulation of T cell subsets, especially the activation of inflammatory factors in the middle-ear effusion in a complicated cytokine environment contributes to the gradual elevation of Treg cells, excessive immunosuppression and persistent inflammation, resulting in the presence of intractable chronic effusions. Acute adenoid infection has not been standardized, so whether the infected adenoid hypertrophy caused by the decrease in Treg levels can influence the elevation of the Treg levels in the middle ear remains unknown.

RORγt, the specific transcription factor Th17 cells, plays a key role in the cellular differentiation of Th17 cells and in promoting their secretions. Foxp3, the key transcription factor for regulating the differentiation of Treg cells, is indispensable in activity inhibition, phenotype

stabilization and peripheral survival of Treg cells. Studies showed that TGF-β and IL-6 initiate Th17 cellular differentiation through RORγt Signal Transduction. Our study indicated the rise of RORγt and the decrease of Foxp3 expression levels in the patients suffering from AH with various complications, suggesting the rise of expression levels may correspondingly strengthen secretions of Th17 cells, which improves the inflammatory reaction of the medicated immune diseases and participation in the onset of the diseases. Foxp3 plays an irreplaceable role in the development and differentiation of Treg cells, Foxp3's gene expression might contribute to the reduction of the number of Treg cells and their dysfunction in the patients suffering from AH with complications.

In addition, we also analyzed the change of Treg cells in AH complicated with AR and OME and found that the down-regulation in the Tregratios but the upregulation in the OSAHS + OME ratios compared to the controls, suggesting the onset of AR might aggravate the down-regulation of Treg ratios. However, the Th17/Treg ratios have changed in Group 5 compared to Group 2. This might be due to the mutual influence between the regulatory mechanism of Treg cells in AR and the regulatory changes of Treg cells in the onset of OME, which may lead to the feedback regulation or suppression, hiding the changes of the Treg cells. It might be attributed to the insufficient cases in our study which in turn led to the errors due to inaccurate controlling factors from the outside, for which the specific reasons or mechanisms remains to be elucidated. Up till now, the initial conclusions indicate that the further specialized studies on the Treg cells may play a pivotal role in the relationship between the functions of the lymphatic system related to the upper airway mucosa. Our findings may bring some insight into the resolution of AH disorders by the systemic immuno-regulation and lay a foundation for the further relevant studies.

Conclusion

The Th17/Treg ratios increased in both the peripheral blood and local adenoid tissues of the patients suffering from AH complicated with OME but decreased in the children with AH alone. There were no significant differences in the deviation of the Th17/Treg ratios between Group 5 and Group 2 but the ratios in Group 4 unregulated, suggesting that the deviation of the Th17/Treg ratios might take effects on the pathogenesis of adenoid hypertrophy with complications in children.

Acknowledgements

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Disclosure of conflict of interest

None.

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