

Inflammatory cell types in Reinke's edema

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Abstract

Objective: To evaluate the histopathological findings in Reinke's edema and correlate them with otorhinolaryngologic symptoms other than dysphonia.

Methods: Patients diagnosed with Reinke's edema as the source of dysphonia from January to December 2016 were included in this retrospective study. The histopathological findings in Reinke's edema and the correlation with otorhinolaryngologic symptoms other than dysphonia were evaluated.

Results: A total of 13 patients (3 females, 10 males) with Reinke's edema all of whom were smokers underwent surgery. In all of the specimens, the main histopathological finding was edema in the lamina propria of the vocal folds. The dominant inflammatory cell type in the stroma was determined by evaluating the whole microscopic subepithelial zone. The cell types included plasma cells, lymphocytes, monocytes, and neutrophils.

Conclusion: In Reinke's edema the main pathologic finding is the collection of edematous fluid in Reinke's space. As our study was limited by the small number of cases, we could not find any statistically significant correlation between the inflammatory cell types and clinical characteristics of Reinke's edema.

Keywords: Inflammation, Reinke's edema, surgery.

Özet: Reinke ödeminde enflamatuvar hücre tipleri

Amaç: Reinke ödemindeki histopatolojik bulguları değerlendirmek ve disfoni dışındaki otorinolarenolojik semptomlarla ilişkilendirmek.

Yöntem: Bu retrospektif çalışmaya, disfoninin kaynağı olarak Ocak–Aralık 2016 arasında Reinke ödemi tanısı konan hastalar dahil edildi. Reinke ödeminde histopatolojik bulguların disfoni dışındaki otorinolarenolojik semptomlarla korelasyonu değerlendirildi.

Bulgular: Tümü sigara kullanan 3'ü kadın ve 10'u erkek, toplam 13 Reinke ödemli hasta ameliyat edilmişti. Tüm örneklerde esas histopatolojik bulgu ses tellerinin lamina propriasındaki ödem idi. Subepitelial zonun tümüyle mikroskopik değerlendirmesiyle stromadaki başat enflamatuvar hücre tipi belirlendi. Hücre tipleri plazma hücreleri, lenfositler, monositler ve nötrofillerden ibaretti.

Sonuç: Reinke ödeminde asıl patolojik bulgu Reinke mesafesinde ödemli sıvı koleksiyonudur. Çalışmamız az sayıda olgu ile sınırlanmış olduğundan, enflamatuvar hücre tipleriyle Reinke ödeminin klinik özellikleri arasında istatistiksel açıdan anlamlı bir korelasyon saptayamadık.

Anahtar sözcükler: Cerrahi, enflamasyon, Reinke ödemi.

Reinke's edema is a benign laryngeal disease that presents as diffuse polypoid degeneration of the vocal folds.^[1] Since then, it has been related to smoking, gastroesophageal reflux, and vocal abuse. It is also known as 'smoker's polyp'.^[2] The main symptom is dysphonia, and diagnosis is based on history and laryngoscopic findings. Treatment varies depending on the severity of the cases. In mild cases, smoking and alcohol prohibition, voice hygiene, voice therapy and antireflux treatment is sufficient. In severe cases, endolaryngeal microsurgery is needed.^[3]

In this study, we aimed to evaluate histopathological findings in Reinke's edema and correlate with otorhinolaryngologic symptoms other than dysphonia.

Materials and Methods

Patients diagnosed with Reinke's edema as the source of dysphonia from January to December 2016 were included in this retrospective study. Demographic data, histopathological findings, superimposed allergic rhinitis or other otorhinolaryngologic pathologies were collected.

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Additional benign laryngeal lesions, patients older than 65 years-old or younger than 18 years-old, systemic diseases such as diabetes or hypertension were excluded. None of our patients presented a history of laryngeal surgery.

Reinke's edema was analyzed histopathologically to find an association between inflammatory cell types and the severity of the edema.

Surgical technique

Our study included 13 patients. All of the patients were operated by the same surgical team under general anesthesia. The operation was performed under a microscope (Zeiss OPMI Sensera; Carl Zeiss AG, Oberkochen, Germany). A similar incision was made for the microflap technique described by Sataloff et al.^[4] Anterior and posterior commissures are avoided for risk of postoperative adhesion formation. The subepithelial region was dissected, and the fluid in the vibratory part of the vocal cord was carefully collected with the help of a blocked aspirator for further histopathological examination. Surplus vocal cord mucosa was resected, and the flap was replaced carefully. In each patient, the surgical procedure was applied in the same session for both vocal cords. Postoperatively strict voice rest and antireflux treatment were given to all the patients.^[5]

Histopathological examination

Specimens were fixed in a 10% formaldehyde solution, embedded in paraffin, cut into 4 µm-thick sections. Sections were stained with hematoxylin and eosin (H&E). The types of inflammatory cells were evaluated by the pathologist.

Results

A total of 13 patients (3 females, 10 males) with Reinke's edema all of whom were smokers underwent surgery. The ages of the patients ranged between 28 and 59 years with a mean age of 40.

In all of the specimens, the main histopathological finding was edema in the lamina propria of the vocal folds. The inflammatory cells were evaluated in the edematous stroma. The dominant inflammatory cell type in the stroma was determined by evaluating the whole microscopic subepithelial zone. The cell types include plasma cells, lymphocytes, monocytes, and neutrophils (Table 1). As our study was limited by the small number of cases, we could not perform correlation analysis between the cell types and Reinke's edema.

In these 13 patients, 4 of them had a history of allergic rhinitis. This made us think if Reinke's edema had any relation with allergy or not. But the other nine patients did not have any rhinologic findings regarding allergic rhinitis or systemic allergy.

Although four of the patients had allergic rhinitis, the cell types and Reinke's edema was not correlated statistically in this group. However, half of the allergic patients that had Reinke's edema had lymphocytes as dominant cell type in histopathological specimens.

Discussion

Reinke's edema is a benign vocal fold pathology that is characterized by excessive extracellular matrix production. Although the main risk factor is smoking, Reinke's edema is not considered to be a precancerous lesion.^[6]

Morphological changes in vocal cords are the results of vascular congestion and edema in the superficial layer of lamina propria which is known as Reinke's space. Yonekawa et al. classified Reinke's edema lesions in three grades regarding severity. The contact of the anterior one-third is Grade 1, anterior two-thirds is Grade 2, and the contact of

Table 1. The dominant cell type in the stroma determined by evaluating the whole microscopic subepithelial zone.

Age (years)	Gender	Smoking	Histopathological findings / dominant cell types	Allergy-related findings / symptoms
43	F	+	Plasma cells	+
28	M	+	Edema / few inflammatory cells	-
37	M	+	Edema / few inflammatory cells	-
34	M	+	Lymphocytes	+
52	M	+	Neutrophils	-
59	M	+	Edema / few inflammatory cells	-
41	M	+	Monocytes	+
45	F	+	Edema / few inflammatory cells	-
38	M	+	Neutrophils	-
50	M	+	Lymphocytes	+
32	M	+	Edema / few inflammatory cells	-
35	M	+	Neutrophils	-
33	F	+	Edema / few inflammatory cell	-

the entire vocal cords represents Grade 3.^[7] However, this grading description was not validated.^[8] Hellquist et al. defined Reinke's edema patients in 2 groups histologically: with or without dysplasia.^[9] In 2017, Tan et al. classified the patients with Reinke's edema in 4 grades. Grade 1: minimal polypoid degeneration of the vocal fold up to 25% of the glottic airway. Grade 2: lesion occupying 50% to 75% of the glottic airway, grade 4 is the obstructive lesion occupying more than 75% of the glottic airway.^[8] Unfortunately, a limited number of the patients prevented us defining a new classification. Treatment varies depending on the severity of the cases. In mild cases, smoking and alcohol prohibited, voice hygiene, voice therapy and antireflux treatment are sufficient. In severe cases, endolaryngeal microsurgery is needed.^[10] We used the microflap technique described by Sataloff et al.^[4] in our patients. We made the incision to the mucosa by avoiding the anterior and posterior commissures for risk of postoperative adhesion formation. We dissected the subepithelial region, and carefully collected the fluid in the vibratory part of the vocal cord with the help of a blocked aspirator for further histopathological examination. We resected surplus vocal cord mucosa, and carefully replaced the flap. In each patient, we applied the surgical procedure in the same session for both vocal cords. We administered postoperatively strict voice rest and antireflux treatment to all the patients.^[5] Complications are very rare including scarring of the vocal cords after microsurgery, voice fatigue or strain.^[10] None of our patients had complications.

Histopathologically, Reinke's edema is characterized by different types of epithelial lesions; however, a high percent of patients do not exhibit any alterations. Tillman et al. did not find any epithelial lesions except for light parakeratosis in a study with 60 patients.^[11] In our study, we reported no epithelial lesion. In all of the cases, the main histological alteration in H&E stained sections was edema in the lamina propria of the vocal folds. The inflammatory cell types included plasma cells, lymphocytes, monocytes, and neutrophils. However, due to the limited small number of cases, we could not find a correlation between the cell types and Reinke's edema.

Conclusion

In Reinke's edema, the main pathological finding is the collection of edematous fluid in Reinke's space. However, a limited number of studies have been carried out on this edematous fluid. As our study was limited by the small number of cases, we could not find any statistically significant correlation between the inflammatory cell types and clinical characteristics of Reinke's edema. Further studies are needed for understanding the role of inflammatory cells in the pathogenesis of Reinke's edema.

Conflict of Interest: No conflicts declared.

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