what's your diagnosis?

# Asian woman with difficulty in swallowing

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**Citation:** Tanrivermis Sayit A, Elmali M, Yildirim U, Gun S, Koyuncu M. Asian woman with difficulty in swallowing. Ann Saudi Med 2020; 40(2): 159-162. DOI: 10.5144/0256-4947.2020.159

**Received:** May 21, 2019

**Accepted:** September 20, 2019

**Published:** April 2, 2020



**Funding:** None.

A 56-year old Turkish woman was admitted to the ear, nose, and throat clinic with a 6-month history of difficulty in swallowing, especially solid food. She had no significant medical or surgical history except for a 30-pack-per-year history of smoking. Her complete blood count showed eosinophilia (eosinophils; 8.4%), and serological examination showed elevated serum immunoglobulin E (IgE) with a value of 1920 IU/mL. On flexible fiberoptic endoscopic examination, thickening and coarseness of the epiglottis was seen. Other laryngeal structures were normal. There was no history of allergies or drug intake. Contrast-enhanced computed tomography (CT) of the neck was performed (**Figure 1**). CT revealed a thickening of the epiglottis and multiple cervical lymph nodes, each measuring up to 1 cm across (**Figure 1**). The signal intensity of the epiglottis was low on T1-weighted images (WIs) (**Figure 2a**) and high on T2-WIs (**Figure 2b**). Diffuse contrast enhancement was seen after intravenous gadolinium injection (**Figure 2c**).

## What is your diagnosis?

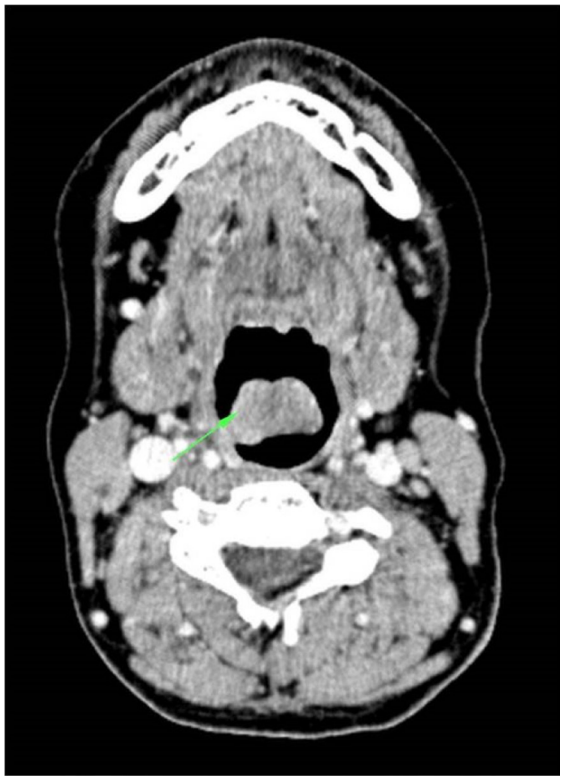

**Figure 1.** Contrast-enhanced computed tomography (CT) shows a mass-like thickening (arrow) of the epiglottis. There are no enlarged lymph nodes.

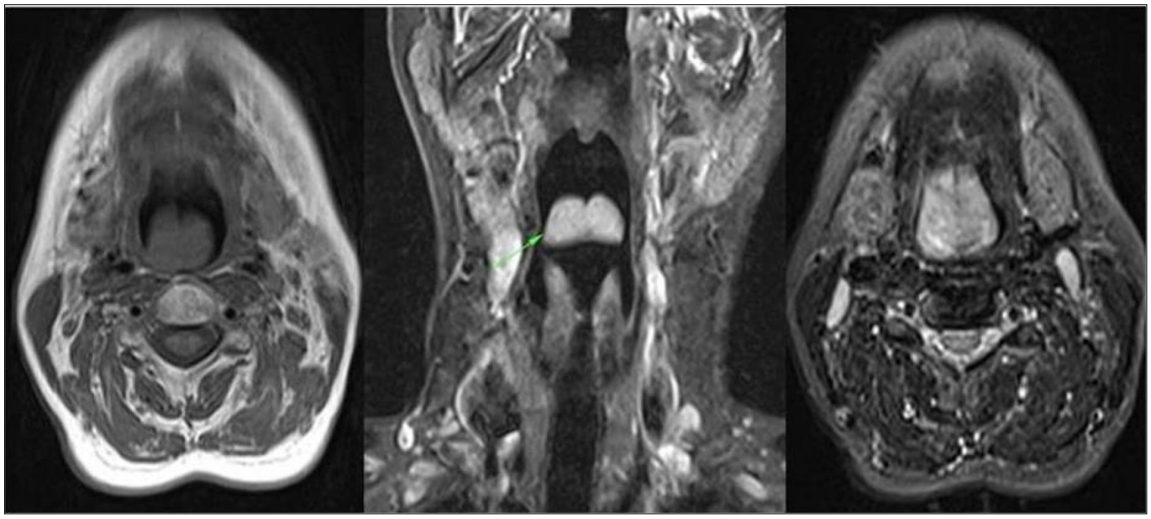

**Figure 2.** The (a) axial T1 weighted TSE (TR/TE: 742/8) image and (b) Coronal fat-saturated T2 weighted image (WI; TR/TE: 4000/42) show a thickened epiglottis (arrow). The epiglottis is hypointense on T1-WI and hyperintense on fat-suppressed T2-WI. (c) The gadolinium-enhanced fat-suppressed axial T1-WI (TR/TE: 4300/62) shows non-homogeneous diffuse enhancement of the epiglottis.

# Diagnosis: Kimura's Disease

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**Accepted:** September 20, 2019

**Published:** April 2, 2020



**Funding:** None.

Direct laryngoscopy (DL) was performed under general anesthesia to exclude tumoral pathologies. DL revealed that the epiglottis was lobulated and diffuse hypertrophic (**Figure 3**). Mucosa of the epiglottis and larynx were normal. Submucosal punch biopsies were taken from the right and left halves of the epiglottis with separate mucosal incisions. Histopathologic examination revealed vaguely nodular dense lymphoid aggregates with the formation of follicles and germinal centers beneath the intact stratified squamous epithelium of the epiglottis, accompanied by vascular proliferation and numerous eosinophilic infiltrates within and around the lymphoid follicles, as well as stromal sclerosis (**Figure 4a, b**). The histopathologic findings and the presence of peripheral eosinophilia were considered compatible with KD.

Kimura's disease is a benign reactive chronic inflammatory disorder of unknown etiology that is predominantly seen in Asian males during the second and fifth decade of life. It is characterized by a triad of subcutaneous masses in the head and neck region, peripheral eosinophilia, and elevated serum IgE levels.[1] It is most commonly seen in the head and neck region as a hard, painless, single or multiple subcutaneous lesion or as a mass in major salivary glands. Involvement of the epiglottis is very rare. It can also be seen in the axilla, extremities, groin, and trunk.[2,3] It is often accompanied by regional lymphadenopathy with an incidence of 42–100%.[4] Involvement of the epiglottis is very rare; only a limited number of cases are reported in the literature.[2,3]

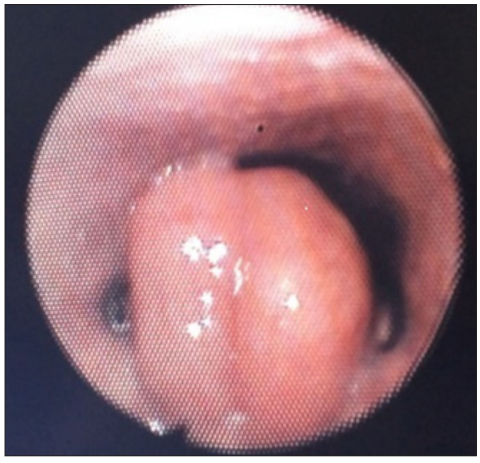

**Figure 3.** Swelling of the epiglottis with a normal mucosal surface on fiberoptic endoscopic evaluation of swallowing.

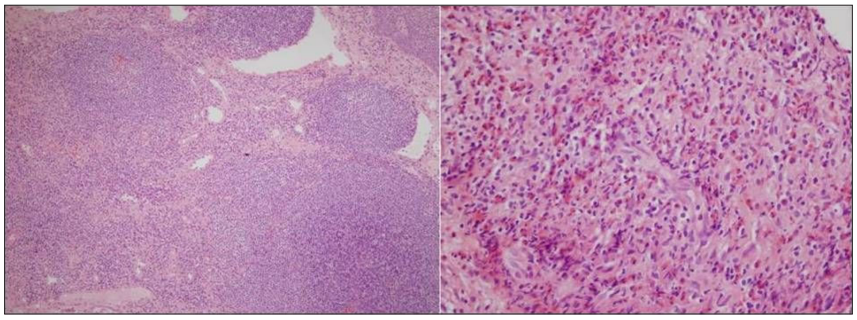

**Figure 4.** (a) Lymphoid follicles with reactive germinal centers, eosinophils, and proliferating vascular structures in the submucosal area (HE ×100). (b) Eosinophilic infiltration and endothelial hyperplasia (HE ×400).

# what's your diagnosis?

Diagnostic imaging methods such as ultrasonography, CT, and magnetic resonance imaging (MRI) can be used for the diagnosis. On an unenhanced CT, the lesion can usually be seen as a homogeneous isodense or hyperdense mass without necrosis or calcification. Cervical lymphadenopathy can be seen.[1] On an MRI, the lesion can be seen with low or high signal intensity on T1- and T2-WIs. The contrast enhancement pattern varies with the fibrosis and vascular distribution in the lesion and the enhancement may be mild or high, homogeneous or heterogeneous. Imaging findings are useful for evaluating the lesion, but they are non-specific and variable, so the definitive diagnosis is made histopathologically.[1]

The primary treatment modality is surgical resection. Additional treatment methods include steroid treatment, chemotherapy, radiotherapy, and antihistamine treatment.[2,5]

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