โรคคิมูระของต่อมน้ำเหลืองใต้ขากรรไกรล่าง รายงานผู้ป่วยและทบทวนวรรณกรรม
Kimura Disease of Submandibular Lymph Node: A Case Report and Review of Literature

พรภพ รัตนอาภา[1], เอกรัฐ ภัทรธราธิป[2]
[1]โรงพยาบาลภูมิพลอดุลยเดช กรมแพทย์ทหารอากาศ กรุงเทพมหานครฯ
[2]ภาควิชาทันตพยาธิวิทยา คณะทันตแพทยศาสตร์ จุฬาลงกรณ์มหาวิทยาลัย
Pornpop Rattana-arpha[1], Ekarat Phattarataratip[2]
[1]Bhumibol Adulyadej Hospital, Directorate of Medical Services, Royal Thai Air Force, Bangkok
[2]Department of Oral Pathology, Faculty of Dentistry, Chulalongkorn University

ชม. ทันตสาร 2562; 40(2) : 139-146
CM Dent J 2019; 40(2) : 139-146

Received: 24 December, 2018
Revised: 11 February, 2018
Accepted: 22 February, 2018

บทคัดย่อ

โรคคิมูระ (Kimura Disease) เป็นความผิดปกติที่เกี่ยวเนื่องกับภาวการณ์อักเสบเรื้อรังที่ไม่ร้ายแรงและพบได้น้อย สาเหตุเชื่อว่าเกิดจากภูมิคุ้มกันไวเกินโดยไม่ทราบสิ่งกระตุ้น ลักษณะทางคลินิกมักพบก้อนเนื้อใต้ผิวหนังบริเวณศีรษะและคอ ไม่มีอาการเจ็บร่วมกับต่อมน้ำเหลืองบริเวณใกล้เคียงโตหรือเกิดในบริเวณต่อมน้ำลาย การตรวจทางห้องปฏิบัติการพบเม็ดเลือดขาวชนิดอีโอซิโนฟิลในเลือดและเนื้อเยื่อเพิ่มสูงขึ้น ร่วมกับ การเพิ่มขึ้นของอิมมูโนโกลบูลินชนิดอีในเลือด โรคคิมูระมักสร้างความสับสนกับแองจิโอลิมฟอยด์ไฮเพอพลาเซียและอีโอซิโนฟิลเลีย (angiolymphoid hyperplasia with eosinophilia) ซึ่งเป็นรอยโรคอีกชนิดหนึ่ง ที่มีลักษณะทางจุลพยาธิวิทยาทับ

Abstract

Kimura Disease (KD) is a rare benign chronic inflammatory disorder, believed to be resulted from an immune-mediated hypersensitivity reaction to unknown agents. Clinically, it usually manifests as painless subcutaneous nodules in the head or neck region, accompanied by regional lymphadenopathy or salivary gland involvement. Laboratory finding shows peripheral blood and tissue eosinophilia, together with markedly elevated serum immunoglobulin E (IgE) levels. KD is often confused with angiolymphoid hyperplasia with eosinophilia (ALHE), another separate

Corresponding Author:

พรภพ รัตนอาภา
ทันตแพทย์ โรงพยาบาลภูมิพลอดุลยเดช กรมแพทย์ทหารอากาศ
กรุงเทพมหานครฯ 10220

Pornpop Rattana-arpha
Dentist, Bhumibol Adulyadej Hospital,
Directorate of Medical Services, Royal Thai Air Force,
Bangkok 10220, Thailand
E-mail: ***questionnice@gmail.com***

ซ้อนกัน รายงานนี้กล่าวถึง ผู้ป่วยโรคคิมูระบริเวณต่อมน้ำเหลืองใต้ขากรรไกรล่างข้างขวาในชายไทยอายุ 30 ปี พร้อมทั้งการทบทวนวรรณกรรมที่เกี่ยวข้อง

entity with overlapping histopathologic features. A case of KD in a 30-year-old Thai male with right submandibular swelling was presented together with a brief review of literature.

คำสำคัญ: โรคคิมูระ แองจิโอลิมฟอยด์ไฮเพอพลาเซียและอีโอซิโนฟิลเลีย ต่อมน้ำเหลืองใต้ขากรรไกรล่าง เม็ดเลือดขาวชนิดอีโอซิโนฟิลเพิ่มสูงขึ้น

Keywords: Kimura disease, angiolymphoid hyperplasia with eosinophilia, submandibular lymph node, eosinophilia

Introduction

Kimura disease (KD) was first reported by Kimm and Szeto in 1937 as 'eosinophilic hyperplastic lymphogranuloma'.[1] The disease became widely known in 1948 when it was reported in the Japanese literature by Kimura and Ishikawa and the terminology 'Kimura disease' has since been adopted.[2] KD is prevalent in Asians, with most cases being reported in the Japanese, Chinese, Vietnamese and Filipino.[3] This disease primarily occurs in people between 20 and 50 years of age. Men are affected five to seven times more than women.[4] The head and neck region accounts for more than 90% of all cases. The most common sites include parotid glands, submandibular glands, lymph nodes, and sub-cutaneous tissues of the head and neck area.[5]

KD often presents clinically as a subcutaneous swelling or nodule, partially associated with regional lymphadenopathy. It is associated with peripheral blood and tissue eosinophilia, along with a markedly increased serum IgE concentration.[6,7] Microscopically, KD exhibits characteristic eosinophilic lymphoid granuloma, forming eosinophilic abscesses with vascular proliferation and varying degrees of fibrosis. Such features are also found in the angiolymphoid hyperplasia with eosinophilia (ALHE) or epithelioid hemangioma. ALHE bears the closest resemblance to KD, to the extent that the two were once considered the same entity. Following the first description of ALHE in 1969, the misapprehension that ALHE and KD were identical, or at least two ends of the same disease spectrum, has been perpetuated by the similarities between the two conditions.[8] A study by Rosai *et al*.[9] eventually clarified this misconception, and KD and ALHE were established as two distinct entities. Currently, KD is believed to be a chronic allergic inflammatory process of unknown origin[10], whereas ALHE is considered a benign vascular proliferative disorder.[11]

In this article, we reported a case of KD presenting as an asymptomatic right submandibular swelling, and reviewed the literatures with special emphasis on the differences between KD and ALHE.

Case report

A 30-year-old Thai male presented with a complaint of swelling on the right submandibular region (Figure 1). The swelling had been slowly increasing in size for approximately one year. There was no history of previous infection. The medical history was non-contributory. Extraoral examination showed a 10 mm x 15 mm x 25 mm non-tender, well-circumscribed soft tissue mass. The lesion was movable, firm and had normal skin covering. Intraoral examination was within normal limit with no incidence of tooth related pain or space infection. Panoramic radiograph revealed no underlying bone abnormalities.

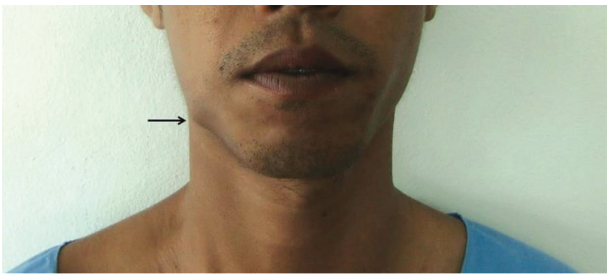
Laboratory investigations showed markedly increased eosinophil count (1.5×10^9 cells/L; normal range 0.0-0.5 $\times$ 10^9 cells/L) and serum IgE levels (1350 IU/ml; normal range 0-87 IU/ml). The renal function studies were normal with no evidence of proteinuria. Allergic disease was not noted.

The initial clinical impression was of benign neoplastic condition in nature. Therefore, the fine needle aspiration biopsy was performed and yielded inconclusive result. Consequently, the lesion was surgically excised under local anesthesia. Under gross examination, the specimen was an oval-shaped, well-circumscribed, firm, gray-tan soft tissue mass (Figure 2). The histopathological examination demonstrated reactive lymphoid tissues with follicular hyperplasia and germinal center formation (Figure 3.1). Notably, the interfollicular infiltrate was rich in eosinophils admixed with lymphocytes and plasma cells (Figure 3.3). Vascular hyperplasia with numerous small venules and thin-walled vessels, lined by flattened endothelial lining was present (Figure 3.2). Also noted are areas of fibrosis. From the histopathologic features as well as the clinical and laboratory findings, the diagnosis of KD was rendered.

The patient's recovery was uneventful. The patient was followed up every 6 months for 2 years with no evidence of recurrence. At the end of the 2-year follow-up, the eosinophil count and serum IgE decreased to 0.7 x 10^9 cells/L and 525 IU/ml, respectively.

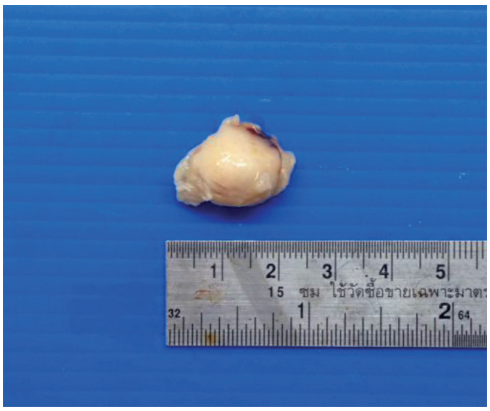
Discussion

KD is a rare chronic inflammatory disease. Its cause remains unclear, however, several etiologic factors have been proposed, including the autoimmunity, allergy, trauma, neoplastic process, parasite infestation and infectious agents.[11,12] It is generally accepted that the immune system plays an important role in the pathogenesis of KD. The findings of increased eosinophils, mast cells, as well as interleukin-5 and IgE levels suggest an abnormal T-cell stimulation in response to the hypersensitivity-typed reaction or type I allergy.[13] It has been speculated that the degree of serum eosinophilia may be correlated with the size of the lesion, which might



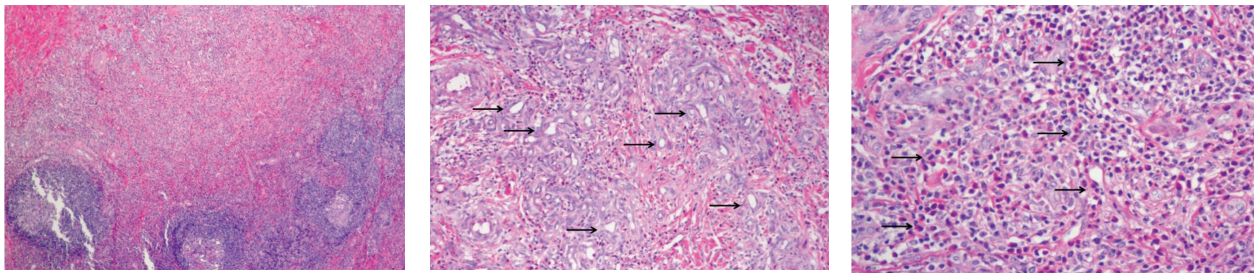
รูปที่ 1 *ลักษณะทางคลินิกผู้ป่วย พบการบวมบริเวณใต้ขากรรไกรล่างข้างขวา (ศรชี้) ไม่มีอาการ ก้อนบวมนี้ค่อย ๆ โตขึ้นมาประมาณ 1 ปี รอยโรคกดแน่นและผิวหนังที่คลุมมีลักษณะปกติ*

Figure 1 *Clinical features of the patient showing asymptomatic swelling in the right submandibular region (arrows). The swelling had been slowly increasing in size for approximately one year. The lesion was firm and had normal skin coverage.*



รูปที่ 2 *ลักษณะทางกายวิภาคของก้อนเนื้อ เป็นเนื้อเยื่ออ่อนสีขาวนวล ขอบเขตชัดเจน รูปกลมรี กดแน่น ขนาด 1.0x2.0x2.5 ซม.*

Figure 2 *Gross examination showing a 1.0x2.0x2.5 cm. oval-shaped, well-circumscribed, firm, off-white soft tissue mass.*



3.1 3.2 3.3

รูปที่ 3 *ลักษณะทางจุลพยาธิวิทยา แสดง*

(3.1) เนื้อเยื่อของน้ำเหลืองและสภาวะการแบ่งเซลล์ที่มากขึ้นของเนื้อเยื่อต่อมน้ำเหลืองส่วนฟอลลิเคิล (ย้อมสีฮีมาท็อกไซลินและอีโอซิน ที่กำลังขยาย 40 เท่า)

(3.2) เส้นเลือดขนาดเล็กจำนวนมากมาย (ศรชี้) ซึ่งมีผนังบางและเซลล์ที่บุผนังเส้นเลือดแบนเรียบ (ย้อมสีฮีมาท็อกไซลินและอีโอซิน ที่กำลังขยาย 100 เท่า)

(3.3) การแทรกซึมของเม็ดเลือดขาวชนิดอีโอซิโนฟิลจำนวนมากและรวมตัวกันเป็นฝีหนองขนาดเล็กของเม็ดเลือดขาวชนิดอีโอซิโนฟิล (ศรชี้)

(ย้อมสีฮีมาท็อกไซลินและอีโอซิน ที่กำลังขยาย 400 เท่า)

Figure 3 *Histopathologic examination showing*

(3.1) Lymphoid tissue with reactive follicular hyperplasia (H&E stain, ×40)

(3.2) Numerous thin-walled vessels (arrows) with flattened endothelial lining (H&E stain, ×100)

(3.3) Intense eosinophilic infiltration with formation of eosinophilic microabscesses (arrows)

(H&E stain, ×400)

be useful for monitoring the disease activity.[14] In addition, many synonymous terms have been used to describe KD, including subcutaneous eosinophiliclymphoid granuloma, eosinophilic lymphoid follicular hyperplasia, and eosinophilic folliculosis of the skin.[15]

Following its first description by Kimura *et al*[2], Wells and Whimster in 1969 reported ALHE as a KD related entity.[7] The rationale was based on the similarities between both diseases, including the predilection for the head and neck region and some overlapping histopathologic features (abundant eosinophils and lymphoid hyperplasia). Since then, both terms were used synonymously for over a decade. However, with more case reports and data from subsequent studies, it has now been accepted that KD and ALHE are two separate entities with distinct clinical and histological features.[16-18]

The clinical features of KD and ALHE are compared in Table 1.[11-14,18-22] It appears that KD is more prevalent in Chinese and Japanese patients, especially in young men. The peak incidence is in the third decade of life (age range, 2-59 years). ALHE occurs in all races and usually affects middle-aged women. Both conditions frequently involve the head and neck region. However, KD usually presents as deep, painless, subcutaneous nodules with associated lymphadenopathy, whereas ALHE often are superficial reddish papules or small nodules without lymphadenopathy. KD can involve lymph nodes and/or major salivary glands, whereas ALHE tends to have a predilection for the periauricular area and scalp.

Characteristically, KD is accompanied by peripheral blood eosinophilia and elevated serum IgE level, while in ALHE the blood eosinophilia

ตารางที่ 1 *เปรียบเทียบลักษณะทางคลินิกระหว่างโรคคิมูระกับแองจิโอลิมฟอยด์ไฮเพอพลาเซียและอีโอซิโนฟิลเลีย*

Table 1 *A comparison of the clinical features between Kimura disease and with angiolymphoid hyperplasia with eosinophilia.*

Characteristics	Kimura disease	Angiolymphoid hyperplasia with eosinophilia
Sex	Male predominance	Female predominance
Age Range Peak incidence	 2-59 years 3rd decade of life	 20-50 years Middle-aged
Race	Asians	All races
Location	Head and neck (esp. parotid glands, submandibular glands, lymph nodes, and subcutaneous tissue)	Head and neck (esp. around ears)
Presentation	Localized subcutaneous mass	Dermal papules or nodules
Number	Single or multiple	Usually multiple
Average size	3 cm	1 cm
Lymph node involvement	Common	Uncommon
Salivary gland involvement	Common	Uncommon
Peripheral eosinophilia	Common (70%)	Rare (<20%)
Serum IgE level	Elevated	Normal
Nephropathy	Present in 10-60% Proteinuria (12-16%)	Rare
Recurrence rate	30-40%	15-40%

is noted in less than 10% of cases. In addition, KD patients often had co-existing renal diseases, particularly the nephrotic syndrome, with incidence rate ranging from 10% to 70%. Interestingly, both lesions may coexist in a patient, and patients with ALHE may develop KD later in their clinical course or vice versa.[11] Several reports have described the findings of KD in computed tomography (CT), magnetic resonance (MR) imaging and ultrasonography. However, all these modalities only provide non-specific picture and the diagnosis is not possible solely on basis of radiography.[23] The role of imaging is to provide the extent and dimensions of the tumor to aid in its resection. Nevertheless, most KD lesions (98%) showed ill-defined borders on CT and MR images.[15]

In the present case, fine needle aspiration cytology was used as an initial investigation, but the finding was inconclusive. The notable feature in the cytologic smears of KD was the presence of a significant number of eosinophils in a background of lymphoid cells. These features were rather not specific and the presumptive cytologic diagnosis may include reactive lymphoid hyperplasia, KD as well as malignant lymphoma.[24]

The histological features of KD and ALHE are compared in Table 2.[8,11,16,18,19,25,26] The microscopic features of KD consists of lymphoid proliferation with prominent germinal centers and eosinophilic infiltrates, which may lead to folliculolysis and eosinophil aggregation forming microabscesses. In contrast, in ALHE, the lym-

phoid infiltration is generally mild to moderate with varying presence of eosinophils and lymphoid follicles. Folliculolysis and eosinophilic abscesses are usually absent. The unique characteristic of ALHE is the florid proliferation of blood vessels with a characteristic plump, low cuboidal, epithelioid or histiocytoid endothelial lining. These endothelial cells often have abundant, acidophilic cytoplasm with vacuolization, vesicular nuclei and display a cobblestone like arrangement. On the contrary, the blood vessels in KD are thin-walled with flattened endothelial cells. Furthermore, stromal fibrosis is a frequent feature in KD and usually absent in ALHE.

KD may be classified histopathologically according to Hui et al. into 3 groups, i.e. constant, frequent and rare types.[21] The first group, the constant features include a preserved nodal architecture, florid germinal center hyperplasia, eosinophilic infiltration and postcapillary venule proliferation. The second group, frequent features comprise sclerosis, polykaryocytes, vascularization of the germinal centers, proteinaceous deposits in the germinal centers, necrosis of the germinal centers and eosinophilic abscesses. The last group, the solitary rare feature is the progressive transformation of the germinal centers. The feature of our case is consistent with constant-type in Hui classification.

There is no current consensus on the management of KD. Many treatment options have been used, including surgical excision, systemic corticosteroid (prednisolone), cytotoxic agents, retinoids, antihistamine (cetirizine), immunosuppressants (cyclosporine, tacrolimus), monoclonal antibodies (imatinib), chemotherapy medication (vinblastine),

ตารางที่ 2 *เปรียบเทียบลักษณะทางพยาธิวิทยาระหว่างโรคคิมูระกับแองจิโอลิมฟอยด์ไฮเพอพลาเซียและอีโอซิโนฟิลเลีย*

Table 2 *A comparison of the histological features between Kimura disease and angiolymphoid hyperplasia with eosinophilia.*

Characteristics	Kimura disease	Angiolymphoid hyperplasia with eosinophilia
Depth	Deep-seated	Superficial
Infiltrate	Multiple lymphoid follicles with abundant eosinophils	Few lymphoid follicles with plasma cells, lymphocyte and sparse to abundant eosinophils
Eosinophilic abscesses	Present	Absent
Vascular proliferation and endothelium	Some degree of vascular proliferation	Florid vascular proliferation
	Absence of irregular and dilated blood vessels	Dilated blood vessels, some of them with irregular shape
	Absence of smooth muscles in vessel wall	Present of smooth muscles in vessel wall
	Moderate proliferation of flat endothelium cells	Copious proliferation of epithlioid/ histiocytic endothelium cells
	None protuberant endothelial cells in vascular lumen	Enlarged and protuberant endothelium cells.
	Absence of cytoplasmic vacuoles in endothelial cell	Presence of one or more cytoplasmic vacuoles in endothelial cells
Fibrosis	Typical	Not typical
Edema	Often marked	Minimal

pentoxifylline, leukotriene-receptor antagonist (pranukast), trans-retinoic acid, retinoids, interferon Inducer (imiquimode) and radiotherapy.[7,11,27-32] As in our case, the complete surgical excision was the first line of treatment and preferable as it was both therapeutic and diagnostic. Systemic steroids may be indicated in cases with frequent relapses or those complicated by nephrotic syndrome. Radiation may be considered in KD lesions which are refractory tosurgical and medical therapy, or when surgery is not feasible.[30] Recurrence is more common in patients with eosinophil counts >50% and serum IgE >10,000 IU/ml.[21] No malignant change has been reported[7,10].

Conclusion

KD is a rare entity. Due to the distinctive clinical and histological features of KD and ALHE, the two entities should be distinguished. Treatment of KD can be challenging. Although, the prognosis of KD is generally good, recurrence is common after treatment. As a result, KD patients should be followed rigorously for a long term.

References

1. Kimm HT, Szeto C. Eosinophilic hyperplastic lymphogranuloma, comparison with Mikulicz's disease. *Chin Med J* 1937; 23: 699-700.
2. Kimura T, Yoshimura S, Ishikawa E. Unusual granulation combined with hyperplastic changes of lymphatic tissue [in Japanese]. *Trans Soc Pathol Jpn*. 1948; 37: 179-180.
3. Sud K, Saha T, Das A, et al. Kimura's disease and minimal-change nephrotic syndrome. *Nephrol Dial Transplant* 1996; 11:1349-1351.
4. Miya T, Yamaki M, Furuya T, Watanabe T, Yokoe H, Tazawa H. A case of eosinophilic granuloma of the soft tissue (Kimura's disease) arising in the palate. *Jpn J Oral Maxillofac Surg* 1999; 45: 527-529.
5. Ishii M. Kimura's disease: a review of 429 cases and 4 new cases. Oto- *RhinoLaryngology (Tokyo)* 1982; 25: 407-416.
6. Terakado N, Sasaki A, Takebayashi T, Matsumura T, Kojou T: A case of Kimura's disease of the hard palate. *Int J Oral Maxillofac Surg* 2002; 31: 222-224.
7. Meningaud JP, Pitak-Arnnop P, Fouret P, Bertrand JC. Kimura's disease of the parotid region: report of 2 cases and review of the literature. *J Oral Maxillofac Surg* 2007; 65: 134-140.
8. Seregard S. Angiolymphoid hyperplasia with eosinophilia should not be confused with Kimura's disease. *Acta Ophthalmol Scand* 2001; 79: 91-93.
9. Rosai J, Gold J, Landy R. The histiocytoid hemangiomas. A unifying concept embracing several previously described entities of skin, soft tissue, large vessels, bone, and heart. *Hum Pathol* 1979; 10: 707-730.
10. Sun QF, Xu DZ, Pan SH, et al. Kimura disease: review of the literature. *Intern Med J* 2008; 38: 668-672.
11. Chong WS, Thomas A, Goh CL. Kimura's disease and angiolymphoid hyperplasia with eosinophilia: two disease entities in the same patient: case report and review of the literature. *Int J Dermatol* 2006; 45: 139-145.
12. Tseng CF, Lin HC, Huang SC, Su CY. Kimura's disease presenting as bilateral parotid masses. *Eur Arch Otorhinolaryngol* 2005; 262: 8-10.
13. Rajpoot DK, Pahl M, Clark J. Nephrotic syndrome associated with Kimura disease. *Pediatr Nephrol* 2000; 14(6): 486-488.
14. Dik VK, van der Wiel BA, Vasmel WL. Kimura's disease of the parotid glands and multiple cervical lymph nodes. *Neth J Med* 2010; 68: 17-57.

15. Park SW, Kim JH, Sung KJ, Lee JH, Park IS. Kimura Disease: CT and MR imaging findings. *AJNR Am J Neuroradiol* 2012; 33: 784-788.
16. Googe PB, Harris NL, Mihm MC, Jr. Kimura's disease and angiolymphoid hyperplasia with eosinophilia: two distinct histopathological entities. *J Cutan Pathol* 1987; 14: 263-271.
17. Rosai J. Angiolymphoid hyperplasia with eosinophilia of the skin. Its nosological position in the spectrum of histiocytoid hemangioma. *Am J Dermatopathol* 1982; 4: 175-184.
18. Chun SI, Ji HG. Kimura's disease and angiolymphoid hyperplasia with eosinophilia: clinical and histopathologic differences. *J Am Acad Dermatol* 1992; 27: 954-958.
19. Kristina B, Sabrina R, Axel T, Eva-Bettina B, Matthias G, Andreas K. Angiolymphoid hyperplasia with eosinophilia and Kimura's disease – a clinical and histopathological comparison. *J German Soc Dermatol* 2014; 224-228.
20. Goecmen H, Oguz H, Astarci M, Samim E. Kimura's disease: case report and brief review of the literature. *J Otolaryngol* 2006; 35(5): 358-360
21. Hui PK, Chan JK, Ng CS, Kung IT, Gwi E. Lymphadenopathy of Kimura's disease. *Am J Surg Pathol* 1989; 13: 177-186.
22. Briggs PL. Kimura disease is not angiolymphoid hyperplasia with eosinophilia: clinical and pathological correlation with literature review and definition of diagnostic criteria. *An Bras Dermatol* 2006; (2): 167-173.
23. Zhang R, Ban XH, Mo YX, et al. Kimura's disease: the CT and MRI characteristics in fifteen cases. *Eur J Radiol* 2011; 80(2): 489-497.
24. Chow LT, Yuen RW, Tsui WM, Ma TK, Chow WH, Chan SK. Cytologic features of Kimura's disease in fine-needle aspirates. A study of eight cases. *Am J Clin Pathol* 1994; 102(3): 316-321.
25. Motoi M, Wahid S, Horie Y, Akagi T. Kimura's disease: clinical, histological, and immunohistochemical studies. *Acta Med Okayama* 1992; 46: 449-455.
26. Armstrong WB, Allison G, Pena F, Kim JK. Kimura's disease: Two case reports and a literature review. *Ann Otol Rhinol Laryngol* 1998; 107: 1066-1071.
27. Hongcharu W, Baldassano M, Taylor CR. Kimura's disease with oral ulcers: response to pentoxifylline. *J Am Acad Dermatol* 2000; 43: 905-907.
28. Day TA, Abreo F, Hoajsoe DK, Aarstad RF, Stucker FJ. Treatment of Kimura's disease: a therapeutic enigma. *Otolaryngol Head Neck Surg* 1995; 112: 333-337.
29. Olsen TG, Helwig EB. Angiolymphoid hyperplasia with eosinophilia. A clinicpathologic study of 116 patients. *J Am Acad Dermatol* 1985; 12: 781-796.
30. Kim GE, Kim WC, Yang WI, et al. Radiation treatment in patients with recurrent Kimura's disease. *Int J Radiat Oncol Biol Phys* 1997; 38: 607-612.
31. Massa MC, Fretzin DF, Chowdhury L, Sweet DL. Angiolymphoid hyperplasia demonstrating extensive skin and mucosal lesions controlled with vinblastine therapy. *J Am Acad Dermatol* 1984; 11: 333-339.
32. Da-long S, Wei R, Bing G, Yun-yan Z, Xiang-Zhen L, Xin L. Tacrolimus on Kimura's disease: a case report. *Oral Surg Oral Med Oral Pathol Oral Radiol* 2014; 117: e74-e78.