Diagnostic Pathology

CASE REPORT

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Warthin tumor concomitant with mantle cell lymphoma: a case report and review of literature

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Abstract

Rationale Warthin tumor (WT) is the second most common benign tumor in salivary gland. It has a slow growth rate and most frequently occurs in the parotid gland. Most patients present with an incidental finding of a painless mass inferior/anterior to the ear. Besides the epithelial component of the tumor, WT is characteristically associated with lymphoid stroma that is considered benign. While there have been a few reports of malignant transformation of the lymphoid components in WT, cases of WT concomitant with mantle cell lymphoma (MCL) are extremely rare. To the best of our knowledge, two cases have been described in the English literature. Herein, we report a case of WT concomitant with MCL in a 70-year-old female patient, and emphasize the importance of careful examination of lymphoid stroma in WT so that concurrent lymphoma is not missed.

Patient concerns A 70-year-old Chinese woman with a 40-year history of cigarette smoking presented with a one year history of a right submaxillary mass with recent enlargement.

Diagnosis Cervical ultrasound (US) and computed tomography (CT) scans of the neck revealed a well-circumscribed mass in the right parotid with a maximum diameter of 3.1 cm. Surgical resection of the mass was performed. Histopathological examination revealed a characteristic double-layer of neoplastic epithelium with prominent lymphoid stroma, suggesting WT. In addition, morphology and immunohistochemistry studies confirmed the coexistence of MCL. Thereafter, the final diagnosis of this case was WT concomitant with MCL.

Interventions The patient was staged as stage I after clinical assessment. Due to the slow growth of parotid lesions, close observation was decided with periodic clinical and radiological monitoring.

Outcomes Currently, the patient demonstrates a stable disease by clinical evaluation.

Lessons To the best of our knowledge, reported cases of WT concomitant with MCL are very rare. This case highlights the importance of a comprehensive assessment of the lymphoid stroma of WT to avoid missed diagnosis of a lymphoma component in a collision tumor.

Keywords Parotid gland, Lymph node, Warthin tumor, Mantle cell lymphoma, Hodgkin lymphoma, Non-Hodgkin lymphoma

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Introduction

Warthin tumor (WT), also known as adenomatoma or lymphopapillary cystadenoma, is the second most common benign tumor of the salivary gland [1, 2], accounting for about 17% of total salivary gland benign tumors, second only to pleomorphic adenoma of the parotid gland [3]. The tumor was first reported by Hildebrand in 1895, and was first described by Warthin in 1929 and named lymphomatous papillary cystadenoma. In 1991, the World Health Organization (WHO) proposed the name Warthin tumor in the new classification of salivary gland tumors, which is still used today [4]. The main feature of WT is that its epithelium is mainly composed of a bilayer of eosinophilic columnar cells embedded in the lymphoid matrix or lymphoid stroma. The incidence of malignancy in WT is exceedingly low, and in recent years there have been reports of malignant transformation of epithelial components. However, lymphomas involving stromal components are extremely rare. Of the cases with lymphomatous involvement reported in the literature, most cases demonstrate a widely disseminated disease. WT with localized mantle cell lymphoma (MCL) is limited to sporadic case reports. Herein, we report a rare collision tumor of WT with MCL. To the best of our knowledge, there have been only two cases of WT with MCL reported in English literature [5]. Since lymphoma cells could potentially be confused with benign stromal components within the tumor, the case highlights the importance of careful examination the lymphoid stroma within WT to avoid diagnostic error.

Case presentation

A 70-year-old Chinese woman with a 40-year history of cigarette smoking presented to our hospital with a one year history of the right submaxillary mass. The patient reported significant enlargement of the mass after tooth extraction. Physical examination showed a painless mass in the area of the right parotid without change of skin color or palpable enlargement of regional lymph nodes. The patient denied fever, night sweats and unintentional weight loss. The ultrasonography showed scattered lymph node echoes in bilateral cervical areas, about 13×4 mm on the left and 18×5 mm on the right. The hypoechoic mass in the right neck, measuring 25×12 mm, showed strong cord-like echo, clear boundaries, regular shape, and enhanced blood flow signal. Neck enhanced CT scan showed that the size of the right parotid gland tumor was 2.5 cm (anterior and posterior) × 1.2 cm (transverse) × 3.1 cm (cranial). There was no pathological cervical lymphadenopathy; facial nerve function was intact, and the House-Brackmann score was 1 (out of 6). The patient subsequently underwent right superficial parotid gland resection and right sternocleidomastoid myocutaneous flap reconstruction. Facial nerve function returned to normal after surgery, and the complete blood count was within normal range.

Gross examination of the right superficial parotid gland specimen showed a brown red oval specimen with lobed surface. Sectioning demonstrated a well-defined tan mass with uniform "fish" appearance, measuring 3.5×3.0×2.5 cm. No tissue necrosis was identified.

Microscopic examination showed an intraparenchymal lesion at low magnification that was composed of epithelial components and lymphoid stromal tissue. The epithelioid tissue had a cystic adenoid structure and formed irregular large glandular tubes and sacs that protrude into the lumen in a papillary growth pattern. Lymphocytes in the stroma were diffusely distributed with markedly expanded stromal cores (Fig. 1A). At a high magnification, the epithelium was composed of a bilayer with eosinophilic columnar cells in the inner layer and basal cells in the outer layer, both of which were compressed by significantly expanded lymphoid stroma. The lymphoid stroma consisted of small to medium-sized lymphocytes with clumped chromatin, irregular nuclear contours, indistinct nucleolus, and scant cytoplasm. Hyalinized vascular walls were noted, and scattered there was an increase in epithelioid histiocytes among the stromal lymphoid proliferation (Fig. 1B). Immunohistochemical analysis showed that the epithelial component was positive for CK and the stromal lymphoid component was positive for CD20, CD43, Cyclin D1, PAX5, SOX11and CD5 (weakly positive) (Fig. 2), and negative for CD3, CD23. The Ki67 proliferation index was estimated to be 70% within stromal lymphoid component and was very low within epithelial component (Fig. 2H). The histopathologic findings supported the diagnosis of WT concomitant with stromal MCL.

Discussion

WT is the second most common benign tumor of the salivary glands [1, 2]. It occurs almost exclusively in the parotid gland, with occasional involvement of regional lymph nodes. It is characterized by multifocal intraglandular and bilateral parotid involvement [1, 6]. Its occurrence in other salivary glands is rare. It is relatively common in men aged 50 to 60 [7]. Both epithelial and lymphoid stromal components of WT are considered benign, but can undergo malignant transformation, with a rate of transformation lower than 1%. The underlying mechanism of malignant transformation is not fully understood [8].

The concurrent detection of malignancy in WT was initially documented in 1954 [9]. In recent years, there have been reports of WT complicated with cancer, however, WT concomitant with lymphoma is rare. An extensive review of the literature revealed 35 reports of lymphoma concomitant with WT (Table 1). These

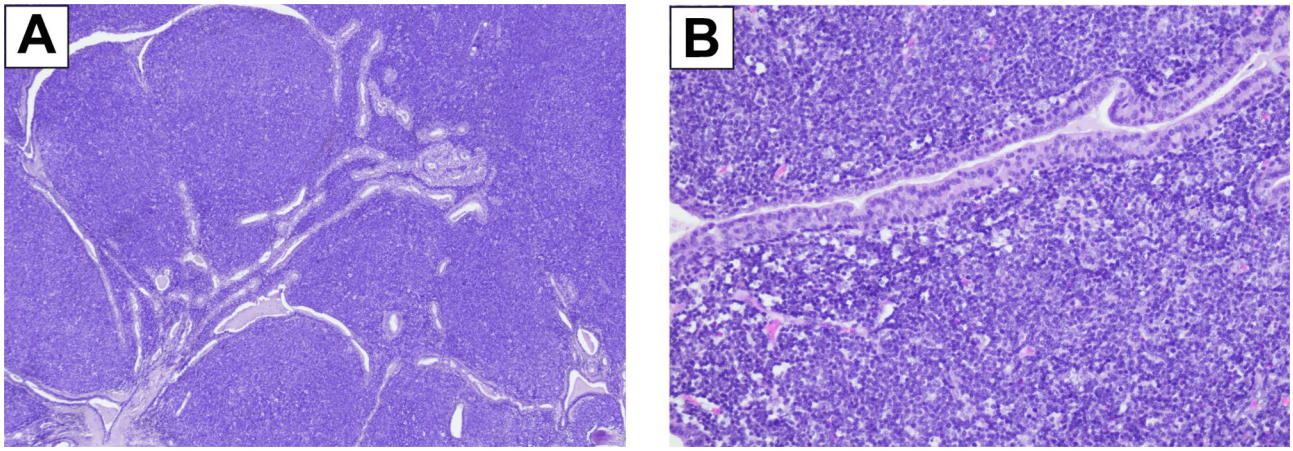

Fig. 1 (**A**) The lesion is composed of epithelial components and lymphoid stroma. Note the cystic adenoid structure of epithelial tissue that protrudes into the lumen in a papillary pattern and marked expansion of stromal component by diffuse proliferation of lymphocytes (H&E stain, 100×). (**B**) A high magnification demonstrates the epithelial component consists of two layers of cells, with eosinophilic columnar cells in the inner layer and basal cells in the outer layer. The stroma is composed of small to medium-sized lymphocytes without appreciable lymphoid follicles. Note the hyaline vascular change in blood vessels and scattered epithelioid histiocytes in lymphoid stroma, the two histologic features characteristic of mantle cell lymphoma (400×)

include thirty-nine cases (including the present case) of concurrent WT and lymphoma. Of these, 33 (84.6%) are Non-Hodgkin lymphoma (NHL) and 6 (15.4%) are Hodgkin lymphoma (HL) The cases of WT concomitant with MCL appear extremely rare. Including the present case, MCL comprises only 3 (9%) of 33 total reported NHL cases [10].

Of two reported cases with concurrent WT and MCL, one described MCL occurring within the parotid gland in patients with chronic parotitis, secondary to an adjacent small WT, but this lymphoma did not occur within the lymphoid stroma of the tumor as seen in the present case [17]. Furthermore, the diagnosis of that case was based on morphology alone without immunophenotypic support or evidence of t(11; 14) (q13; q32) to confirm it [34].

There is a strong correlation between cigarette smoking and WT according to the literature [1]. Most scholars believe WT arises from intraparotid lymph nodes or ectopic salivary gland tissue remnants in regional lymph nodes. It has been proposed that benzene and/or other substances in tobacco tar can be dissolved in saliva and get carried retrograde to the glandular parenchyma via the parotid duct, thus acting on the parotid duct epithelium and stromal lymphoid tissue causing metaplasia and neoplastic transformation of both epithelial and stromal lymphoid components. According to the report, 89.1% of the patients with WT had a history of cigarette smoking and almost 90% were found in males [39]. In addition, autoimmune diseases and radiation exposure increase the risk of WT [1, 2, 6]. All these factors have also been associated with lymphomagenesis.

Summary of literature demonstrates a history of rapid growth of a parotid mass, as was seen in the present case. Therefore, in cases with a sudden increase in size of a parotid lesion, the possibility of a malignant tumor should be raised and subsequent diagnostic procedures should be considered to confirm or rule out the diagnosis.

WT itself is a slow-growing benign tumor. Due to its unique morphological features, the histopathologic diagnosis is not difficult, and surgical resection is generally required for therapeutic purposes. WT concomitant with MCL, however, is quite different in terms of clinical presentation, treatment and prognosis. Therefore, it is critical to carefully evaluate lymphoid stroma, particularly in cases with stromal expansion, to rule out the possibility of lymphomatous involvement.

Theoretically, because the lymphoid stroma may be part of lymphoid tissue throughout the body, existing lymphomas may involve a separate WT via systemic dissemination. Of the reported cases of lymphoma within WT, most patients already had a diagnosis of lymphoma or demonstrated significant lymphadenopathy or systemic involvement by clinical staging subsequently to the diagnosis of WT. While MCL in our case appeared to be confined to the stroma of WT without involving adjacent normal parotid glandular parenchyma tissue, clinical staging showed evidence of lymphomatous involvement in multiple lymph nodes, in keeping with majority of the reported cases.

Mantle cell lymphoma (MCL) is a mature B-cell lymphoma, with overexpression of cyclin D1 driven by *CCND1::IGH* fusion [5]. Microscopically, tumor cells were small to medium-sized lymphocytes with scant cytoplasm, irregular nuclear contours, condensed chromatin and inconspicuous nucleolus. In general, MCL is considered a highly invasive and incurable disease. The median survival time of patients is 3–5 years [34]. A high

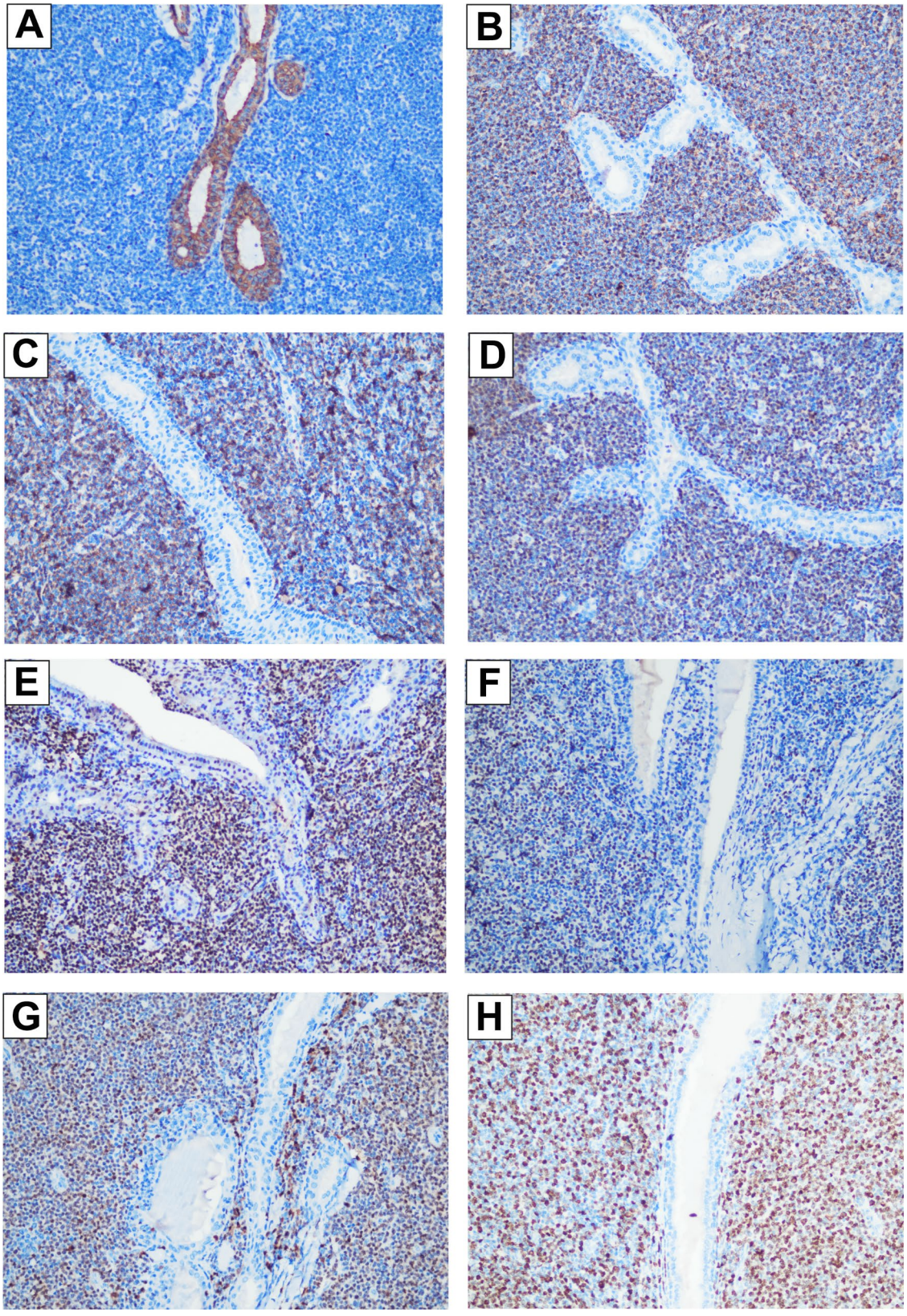

Fig. 2 (**A**) CK stain highlights the epithelial component in WT (100×). CK = Cytokeratin. (**B**) CD20 stain is weakly positive in stromal lymphocytes (100×). (**C-G**) CD43 stain, Pax5 stain, Cyclin D1 stain, SOX11stain, and Mum1 stain show positive expression of each antigen in stromal lymphocytes (100×). (**H**) Ki-67 proliferation index is about 70% within stromal lymphocytes (100×)

Table 1 Summary of malignant lymphoma concomitant with Warthin tumor. (CHL, classical Hodgkin lymphoma; NLPHL, nodular lymphocyte predominant Hodgkin lymphoma; DLBCL, diffuse large B cell lymphoma; SLL/CLL, small lymphocytic lymphoma/chronic lymphocytic leukemia)

Hodgkin lymphoma (*n* = 6, 15.8%)	**Frequency (*n*)**
CHL, mixed cellularity	2 [11, 12]
CHL, lymphocyte rich	1 [13]
CHL (Not distinguished)	2 [14, 15]
NLPHL	1 [16]
Non-Hodgkin lymphoma (*n* = 32, 84.2%)	
Follicular lymphoma	14 [9, 17–26]
In situ follicular neoplasia	1 [10]
DLBCL	6 [22, 27–31]
SLL/CLL	4 [8, 10, 32, 33]
Mantle cell lymphoma	2 [17, 34]
MALT-type lymphoma	1 [35]
Peripheral T cell lymphoma	1 [36]
T cell-lymphoblastic lymphoma	2 [37, 38]
Unclassified	1 [20]

proportion of Ki67-positive cells has been reported as a poor prognostic indicator. In the current case, the Ki67 proliferation index was estimated at 70%, predicting aggressiveness of the lymphoma [34].

Based on the previous types of WT combined with lymphoma, our case should first be differentiated from WT combined with Hodgkin's lymphoma [40]. Hodgkin's lymphoma is characterized by CD30 positive RS cells, which our case lacks [11–16]. Nodular MCL also needs to be differentiated from nodular FL. MCL cells generally have a single cell morphology, lack central mother cells, have local interstitial sclerosis or small vessel wall with hyaline degeneration. Tumor cells express CD5, Cyclin D1 and SOX11, but negative for CD10 and BCL6 [17, 34]. While FL contains a small number of central mother cells, and nodules are often relatively well-defined, exhibiting follicular central cell phenotype which expressing CD10, BCL2, and BCL6 [9, 17–26]. The polymorphic subtype of MCL also needs to be differentiated from DLBCL. In these kinds of cases, the nuclear contour of tumor cells are irregular, with a disproportionate amount of large nuclei and small nucleoli. Tumor cells are always positive for CD5, Cyclin D1, SOX11, and it has t (11; 14) chromosome translocation. While very few DLBCLs express CD5, and only 1% of diffuse large B-cell lymphomas can express Cyclin D1 [22, 27–31]. Some MCL tumor cells may have marginal cell like morphology. But MCL expresses CD5, Cyclin D1, and SOX11 while MALT-type lymphoma often accompanies plasma cell differentiation, which is rare in MCL [35]. A few CLL/SLL cells have irregular nuclear shapes which are similar with MCL, and may also exhibit follicular growth patterns; But LEF1 and CD23 are usually positive in CLL/SLL, which are usually negative in MCL [8, 10, 32, 33]. The expression of Cyclin D1 and SOX11 also contributes to the diagnosis of MCL [41].

Patients with MCL require a comprehensive clinical staging, as most patients have developed the tumor with advanced clinical stages (Ann Arbor III/IV) at the time of diagnosis, accompanied by enlargement of multiple lymph nodes, often involving the spleen, liver, and bone marrow [5]. While the patient in this case was assigned to stage I according to Ann Arbor system, this seems somewhat contradictory. We speculate that the possible reason is that WT itself is a benign tumor with a complete capsule and abundant epithelial and basement membrane components inside, which may limit the invasiveness of tumor cells to some extent. Nonetheless, given the slow growth of parotid lesions, the case was judged as low to intermediate risk, and close observation was decided for the initial management with next PET-CT examination scheduled in 6 months. Because cases of WT combined with mantle cell lymphoma are very rare, data on their treatment and prognosis require case accumulation and long-term follow-up.

In the examination and diagnosis of patients with WT concomitant with MCL, CT has been preferred as the initial method because it is easily accessible and provides the location, size, and structures involved in the tumor [31]. Magnetic resonance imaging (MRI) may also be useful in the assessment of parotid tumors [1]. A definitive diagnosis relies primarily on histopathological assessment of tissue biopsy, including fine needle aspiration (FNA), core needle biopsy (CNB), and excisional biopsy. FNA plays an important role in the assessment of salivary gland tumors [42]. However, since the diagnostic value of FNA may be affected by insufficient cells and a high false negative rate, CNB is considered to be more superior to FNA in the detection of malignant tumors. However, it remain risky to overlook lymphomatous component within the stroma of WT [43]. We believe that surgical excision with histopathological examination is necessary for a definitive diagnosis of WT and exclusion of lymphomatous involvement.

In conclusion, collision tumors characterized by WT concomitant with MCL are very rare, with only 2 cases reported in the English literature to date. WT is a slow-growing benign tumor that can be surgically removed. WT concomitant with MCL, however, is quite different in terms of treatment and prognosis. Due to a distinct treatment approach, overlooking the diagnosis of mantle cell lymphoma would lead to delayed treatment and dismal clinical outcome. Pathologists should carefully and thoroughly examine the stromal lymphoid component of WT to identify potential lymphoma hidden in WT so that adequate management can be delivered timely.

Abbreviations

WT	Warthin tumor
MCL	Mantle cell lymphoma
MRI	Magnetic resonance imaging
US	Ultrasound
CT	Computed tomography
PET/CT	Positron emission tomography/computed tomography
HL	Hodgkin lymphoma
NHL	Non-Hodgkin lymphoma
WHO	World Health Organization
MRI	Magnetic resonance imaging

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Author contributions
Conceptualization: Hai-Chao Tong, Wan-Lin Zhang, Data curation: Ying-Chun Li, Formal analysis: Hai-Chao Tong, Shuang Ma, Funding acquisition: Lian-He Yang, Investigation: Ying-Chun Li, Shuang Ma, Methodology: Hong-Tao Xu, Project administration: Lian-He Yang, Resources: Le-Yao Li, Software: Le-Yao Li, Supervision: Endi Wang, Validation: Hong-Tao Xu, Visualization: Wan-Lin Zhang, Writing-original draft: Hai-Chao Tong, Writing-review and editing: Lian-He Yang, Tyler Wildes.

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Data availability
No datasets were generated or analysed during the current study.

Declarations

Ethical approval and consent to participate
The ethical approval and documentation for a case report was waived with approval of the Institutional Review Board at China Medical University.

Consent for publication
Written informed consent was obtained from the patient for the publication of this case report and any accompanying images. A copy of the written consent is available for review on request by the Editor-in-Chief of this journal.

Competing interests
The authors declare no competing interests.

Conflict of interest
None.

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