• Clinical Research •

Clinical features and prognosis of orbital inflammatory myofibroblastic tumor

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Abstract

● **AIM:** To investigate the clinical features and prognosis of patients with orbital inflammatory myofibroblastic tumor (IMT).

● **METHODS:** This retrospective study collected clinical data from 22 patients diagnosed with orbital IMT based on histopathological examination. The patients were followed up to assess their prognosis. Clinical data from patients, including age, gender, course of disease, past medical history, primary symptoms, ophthalmologic examination findings, general condition, as well as imaging, laboratory, histopathological, and immunohistochemical results from digital records were collected. Orbital magnetic resonance imaging (MRI) and(or) computed tomography (CT) scans were performed to assess bone destruction of the mass, invasion of surrounding tissues, and any inflammatory changes in periorbital areas.

● **RESULTS:** The mean age of patients with orbital IMT was 28.24±3.30y, with a male-to-female ratio of 1.2:1. Main clinical manifestations were proptosis, blurred vision, palpable mass, and pain. Bone destruction and surrounding tissue invasion occurred in 72.73% and 54.55% of cases, respectively. Inflammatory changes in the periorbital site were observed in 77.27% of the patients. Hematoxylin and eosin staining showed proliferation of fibroblasts and myofibroblasts, accompanied by infiltration of lymphocytes and plasma cells. Immunohistochemical staining revealed that smooth muscle actin (SMA) and vimentin were positive in 100% of cases, while anaplastic lymphoma kinase (ALK) showed positivity in 47.37%. The recurrence rate of orbital IMT was 27.27%, and sarcomatous degeneration could occur. There were no significant correlations between recurrence and factors such as age, gender, laterality, duration of the disease, periorbital tissue invasion, bone destruction, periorbital inflammation, tumor size, fever, leukocytosis, or treatment (P>0.05). However, lymphadenopathy and a Ki-67 index of 10% or higher may be risk factors for recurrence (P=0.046; P=0.023).

● **CONCLUSION:** Orbital IMT is a locally invasive disease that may recur or lead to sarcomatoid degeneration, primarily affecting young and middle-aged patients. The presence of lymphadenopathy and a Ki-67 index of 10% or higher may signify a poor prognosis.

● **KEYWORDS:** inflammatory myofibroblastic tumor; orbital disease; clinical features; prognosis
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INTRODUCTION

Inflammatory myofibroblastic tumor (IMT) is a rare mesenchymal tumor primarily composed of spindle-shaped cells that exhibit characteristics of both myofibroblasts and fibroblasts, along with inflammatory cell infiltration[1]. IMT was first recognized in the lungs as a tumor-like lesion characterized by spindle cell proliferation, and it was initially believed to represent a benign, non-neoplastic change[2]. However, an increasing number of case reports along with studies on the pathology, cytogenetics, and invasive behavior of IMT have raised questions about its classification, suggesting that it may be a unique and potentially malignant entity, rather than just a benign inflammatory lesion[3]. In

2002, the World Health Organization (WHO) first categorized IMT as a borderline tumor, labeling it as "fibroblastic and myofibroblastic tumors, intermediate (rarely metastasizing)"[4]. This classification has been upheld by subsequent WHO classifications of soft tissue and bone tumors published in 2013 and 2020[1,5].

IMT can occur in nearly every part of the body, with the most common locations being the abdominopelvic region, lungs, and retroperitoneum[6-9]. Orbital involvement is rare. The wide range of nonspecific symptoms and imaging changes associated with IMT can make diagnosis challenging, and it is often misdiagnosed as benign lesions[10]. This can result in inappropriate treatment and inaccurate assessments of prognosis[11].

Most studies on orbital IMT consist of brief case reports, with only a few case series available and a lack of research on factors related to prognosis. In this report, we describe 22 cases of orbital IMT in our institution and review the existing literature to gain a deeper understanding of its clinical characteristics and prognosis.

PARTICIPANTS AND METHODS

Ethical Approval This study adhered to the principles of the Declaration of Helsinki. The Ethics Committee of Beijing Tongren Hospital, affiliated with the Capital Medical University, ruled that ethics committee approval was not required for this study owing to the retrospective design.

Participants Selection and Literature Search This study focused on patients with orbital involvement of IMT who underwent surgical treatment and had their diagnosis confirmed through histopathological examination at the Department of Ophthalmology and Neurosurgery of Beijing Tongren Hospital, Capital Medical University, from January 2008 to January 2024 (Table 1). Inclusion criteria: 1) clinical data complete; 2) imaging examinations and surgery confirmed orbital involvement; 3) postoperative histopathology and immunohistochemical staining examination confirmed the diagnosis of IMT. Exclusion criteria: 1) patients with other orbital inflammation-related lesions except IMT; 2) patients unable to participate in follow-up appointments, whether in person or *via* telephone. All participants were provided with a comprehensive explanation of the study's purpose and signed an informed consent form. In addition, we conducted a PubMed search using the following MeSH terms: "inflammatory myofibroblastic tumor", "orbit", and "eye". The search parameters include dates from 1900 to the present and only English language articles (Table 1)[12-35].

Data Collection We collected clinical data from patients, including age, gender, course of disease, past medical history, primary symptoms, ophthalmologic examination findings, general condition, as well as imaging, laboratory, histopathological, and immunohistochemical results from digital records. Orbital magnetic resonance imaging (MRI) and(or) computed tomography (CT) scans were performed to assess bone destruction of the mass, invasion of surrounding tissues, and any inflammatory changes in periorbital areas. We monitored patient treatment and prognosis through outpatient clinics and telephone follow-ups, with the most recent follow-up occurring in March 2024.

Statistical Analysis SPSS 27.0 and GraphPad Prism 10.3.1 were used for data analysis. The Shapiro-Wilk test was conducted to assess the normality of data distribution. Continuous variables that followed a normal distribution were presented as mean±standard deviation (SD), and comparisons between groups were performed using the independent samples *t*-test. For data that did not follow a normal distribution, results were expressed as the median (lower quartile, upper quartile), with differences between groups analyzed using the Mann-Whitney *U* test. Categorical variables were reported as frequency (percentage), with Fisher's exact test employed for statistical analysis. All statistical tests were two-sided, and a *P*-value of less than 0.05 was considered statistically significant.

RESULTS

Patient Characteristics In our institution, a study involving twenty-two patients with orbital IMT was conducted. Among these patients, eight were detailed in the case series by Guo *et al*[36] (specifically Cases 1, 4, 5, 11, 14, 18, 19, and 21). The average age of the patients was 28.24±3.30y, with ages ranging from 28mo to 61y. The cohort included 12 male and 10 female patients, resulting in a male-to-female ratio of 1.2:1. The average age of the male patients was 38.17±3.38y, whereas the average age of female patients was 16.33±3.20y. There was a statistically significant difference in the age of onset for IMT between genders, with male patients generally presenting at an older age compared to female patients (P<0.001).

All orbital lesions demonstrated unilateral involvement, with right orbital (n=15) and left orbital (n=7) distribution. The average time from the onset of symptoms to the diagnosis of IMT patients was 3.00 (1.00, 5.25)mo. One patient (4.55%) had a history of previous surgery (Case 10). She previously underwent excision of an orbital mass at a local hospital, where the pathological result was diagnosed as IMT. She came to our hospital due to a recurrence that occurred one month after her initial surgery. Additionally, three patients (13.64%) reported a history of blunt trauma to the area of the orbital mass prior to the onset of symptoms. The time from trauma to symptom onset varied among these patients, being 2mo, 3mo, and 2y, respectively (Cases 20, 9, and 13). Furthermore, six patients (27.27%) received anti-inflammatory therapy before their surgeries; however, half of them did not experience significant improvement in their symptoms.

Table 1 Clinical details in orbital IMT patients of our study and according to literature review

Cases	Age	Sex	Chief complaint	Course	Involved site	Bone destruction	Periorbital inflammation changes	ALK	Treatment	Prognosis
1	26y	F	Blurred vision	3mo	Rt: intraconal space	No	No	Positive	S+G	No recurrence
2	27y	F	Orbital swelling and pain	1mo	Rt: lacrimal gland, greater wings of the sphenoid bone, temporal fossa, anterior skull base	Yes	No	NA	S+G	No recurrence
3	19y	F	Orbital swelling and pain, superotemporal mass	40d	Rt: lacrimal gland	Yes	Thickening of soft tissue of nasopharynx roof and posterior wall	Negative	S+G	No recurrence
4	10y	F	Proptosis, strabismus	1mo	Rt: intraconal space	No	sphenoid sinusitis (B), ethmoid sinusitis (Rt), mucosal thickening of frontal sinus (Rt)	Positive	S+G	Recurrence after 4mo
5	50y	M	Eyelid numbness, blurred vision	20d	Rt: lacrimal gland	Yes	maxillary sinusitis (Lt)	Positive	S+G	No recurrence
6	47y	M	Eye pain, facial swelling, numbness	2mo	Lt: extraconal space, posterior sinus fat space, pterygopalatine fossa, infratemporal fossa, temporal fossa, temporalis muscle, hard jaw, inferior alveolar bone	Yes	mucosal thickening of maxillary sinus (B), mucosal thickening of sphenoid sinus (Rt), middle and lower turbinate hypertrophy (Rt)	Negative	S+R+G	No recurrence
7	54y	M	Orbital swelling and pain, blurred vision	3mo	Rt: diffuse changes in orbit, the wall of eyeball, posterior sinus fat space, pterygopalatine fossa	Yes	maxillary sinusitis (Rt), ethmoid sinusitis (B)	Negative	S+G	No recurrence
8	5y	F	Proptosis	50d	Lt: extraconal space, cavernous sinus, sphenoid sinus, anterior skull base, nasal cavity	Yes	frontal sinusitis (Lt), maxillary sinusitis (Lt), ethmoid sinusitis (B), mucosal thickening of maxillary sinus (Rt)	NA	S+G	No recurrence
9	28y	M	Proptosis, eye pain	8mo	Rt: extraconal space, pterygopalatine fossa, skull base meninges	Yes	mucosal thickening of frontal sinus, ethmoid sinus and maxillary sinus (Rt)	Negative	S+G	No recurrence
10	23y	F	Proptosis, nasal obstruction	20d	Rt: extraconal space, nasal cavity, paranasal sinus, anterior skull base	Yes	maxillary sinusitis (B), sphenoid sinusitis (B), frontal sinusitis (B), ethmoid sinusitis (B), otitis media (B)	Negative	S+C+G	Recurrence after 10mo. Sarcomatous degeneration were found after resection of recurrence
11	29y	M	Proptosis, diplopia, eye pain	1mo	Rt: intraconal space	No	No	Positive	S+G	No recurrence
12	31y	F	Eye pain, blurred vision	1mo	Rt: extraconal space, ethmoid sinus, maxillary sinus, pterygopalatine fossa, infratemporal fossa	Yes	frontal sinusitis (Rt), ethmoid sinusitis (B)	Negative	S+G	No recurrence
13	26y	M	Increased eye secretions	2wk	Rt: extraconal space, ethmoid sinus	Yes	maxillary sinusitis (Rt), frontal sinusitis (Rt), lower turbinate hypertrophy (B)	Positive	S+G	No recurrence
14	9y	F	Eyelid mass	3mo	Lt: lacrimal sac, nasolacrimal duct, nasal cavity, maxillary sinus, ethmoid sinus	Yes	maxillary sinusitis (Lt)	Positive	S+G+R	Recurrence after 3mo. Sarcomatous degeneration were found after resection of recurrence
15	11y	F	Diplopia, blurred vision	18mo	Rt: extraconal space, orbital apex area, infratemporal fossa, pterygopalatine fossa, maxillary sinus, masticatory muscle space, partial parapharyngeal space, cavernous sinus, anterior skull base	Yes	ethmoid sinusitis (Rt), mucosal thickening of frontal sinus and sphenoid sinus (Rt), submucosal cyst of maxillary sinus (B), mastoiditis (B), adenoidal hypertrophy	Negative	S+G	Recurrence after 8mo
16	61y	M	Inner canthus mass	6mo	Lt: subcutaneous tissue	Yes	maxillary sinusitis (Lt)	Negative	S+G	No recurrence
17	29y	M	Inner canthus mass	7mo	Lt: lacrimal sac	Yes	ethmoid sinusitis (Lt), mucosal thickening of maxillary sinus (B)	Negative	S+G+R	No recurrence
18	31y	M	Orbital swelling, proptosis, diplopia	5mo	Lt: extraconal space, pterygopalatine fossa, cavernous sinus, maxillary sinus, posterior sinus fat space, foramen lacerum, pterygoid canal, foramen rotundum, anterior cistern of posterior cranial fossa	Yes	sphenoid sinusitis (B), maxillary sinusitis (Lt), mucosal thickening of ethmoid sinus (B)	Positive	S+G	Recurrence after 1mo
19	28mo	F	Proptosis	4mo	Rt: extraconal space, greater wings of the sphenoid bone, zygomatic arch, middle cranial fossa, cavernous sinus, infratemporal fossa, pterygopalatine fossa	Yes	ethmoid sinusitis (B), maxillary sinusitis (B), mastoiditis (B), adenoidal hypertrophy	Positive	S+G+C	No recurrence
20	34y	M	Inner canthus mass	4mo	Rt: subcutaneous tissue	No	No	Negative	S+G	No recurrence
21	34y	M	Eyebrow arch mass	8mo	Lt: subcutaneous tissue	No	No	Positive	S+G	No recurrence
22	35y	M	Inner canthus mass	3mo	Rt: subcutaneous tissue	No	mucosal thickening of ethmoid sinus (B)	NA	S+G	Recurrence after 3mo
Sa *et al*[12] 2005	10y	M	A mass-like lesion of the supranasal conjunctiva; limitations of extraocular movement, diplopia	1y; 2wk	Rt: ocular surface, anterior orbit	No	NA	Negative	S(biopsy)+G+R	No recurrence (2y)
McKinney *et al*[13] 2006	50y	M	Orbital pain, headache; progressively worsening vision	Several months; 9d	Lt: infraorbital mass, pterygopalatine fossa, foramen rotundum, pachymeninx, meckel cave, foramen ovale	Yes	NA	NA	S(biopsy)+ G	NA

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Table 1 Clinical details in orbital IMT patients of our study and according to literature review (continued)

Cases	Age	Sex	Chief complaint	Course	Involved site	Bone destruction	Periorbital inflammation changes	ALK	Treatment	Prognosis
Polito *et al*[14] 2007	17y	M	Recurrent painful swelling, down-dislocation of the globe	1y	Lt: intraconal space	No	NA	Positive	S	No recurrence (28mo)
Ahmad *et al*[15] 2007; Habib *et al*[16] 2017	7y	M	Upper eyelid swelling, papilledema	NA	Rt: intraconal space	NA	NA	NA	S (biopsy)+G	No recurrence (14y)
Chow *et al*[17] 2010	31y	F	Headaches, jaw pain, trismus, intermittent lower eyelid swelling, diplopia	Over 5mo	Rt: infratemporal and pterygopalatine fossa, extraconal space	Yes	NA	Negative	S+G+R	No recurrence (10mo)
Tawfik and Raslan[18] 2013	8mo	M	upper eyelid swelling	5wk	Lt: extraconal space	Yes	NA	NA	S	No recurrence (2y)
Mudhar *et al*[19] 2013	14y	M	Orbital swelling, loss of vision	2y	Rt: NA	NA	NA	Positive	S	NA
Dutta *et al*[20] 2014	11y	M	Progressive decreasing vision	3mo	Rt: extraconal space, greater wing of the sphenoid bone	Yes	NA	Negative	S (biopsy)+ G	No recurrence (NA)
Lauwers *et al*[21] 2014	71y	M	Progressive proptosis, eyelid edema	NA	Lt: extraconal space, ethmoid sinus, middle cranial fossa	Yes	NA	Negative	S (biopsy)+ G	No recurrence (1y)
Shah *et al*[22] 2015	18mo	M	Progressive painless mass	16mo	Lt: ocular surface, anterior orbit	NA	NA	NA	S	No recurrence (1y)
Cramer *et al*[23] 2015	21y	M	Eyelid red bump	2mo	Rt: ocular surface, anterior orbit	NA	NA	Positive	S	Recurrence after 10mo
Oguz *et al*[24] 2015	7y	F	Pain	NA	Rt: intraconal and retrobulbar space	No	NA	Positive	S+G	NA
	12y	F	Eyelid swelling	NA	Rt: extraconal space, intraconal space	No	NA	Positive	S+G	NA
	11y	M	Diplopia	NA	Lt: intraconal and retrobulbar space	No	NA	Positive	S+G	NA
Kiratli *et al*[25] 2016	7y	F	Painless mass on the nasal part	Many months	Rt: ocular surface, the wall of eyeball, anterior orbit	NA	NA	Positive	S+T (crizotinib)	Recurrence after 15mo
Boudhas *et al*[26] 2017	24y	M	Progressive and indolent mass in the super-external area	4mo	Lt: lacrimal gland	No	NA	Positive	S	No recurrence (7mo)
Callaway *et al*[27] 2018	2y	F	Eyelid mass	Over 6mo	Rt: subcutaneous tissue	Yes	No	Negative	S	NA
Dermarkarian *et al*[28] 2020	8mo	F	Proptosis	2mo	Lt: extraconal space	NA	NA	Positive	S+T (crizotinib)	No recurrence (5mo)
Singh S *et al*[29] 2020	3y	F	Eye pain, redness, irritation, mild proptosis	NA	Rt: extraconal space, maxillary sinus, lamina papyracea	Yes	NA	Negative	S+G	NA
Singh M *et al*[30] 2020	63y	M	Painless, progressive reddish mass	1y	Rt: ocular surface, anterior orbit	NA	NA	Negative	S+G+ topical INFα-2b+ topical antibiotic-steroid	No recurrence (14mo)
Gupta *et al*[31] 2022	5mo	F	Upper and lower eyelid erythema and edema	1wk	Rt: intraconal space	NA	NA	Positive	S (biopsy)+T (crizotinib)	No recurrence (12mo)
Nishadham *et al*[32] 2023	38y	F	Proptosis, blurred vision, papilledema	12mo	Rt: superior aspect of orbit, intraconal space	NA	NA	Negative	S	No recurrence (46mo)
	18y	F	Orbital swelling, headache	3mo	Rt: lateral margin of orbit	NA	NA	Negative	S	NA
	45y	F	Eye proptosis, blurred vision, diplopia	3mo	Rt: superolateral quadrant of orbit, intraconal space	NA	NA	Negative	S	NA
	28y	M	Diplopia, proptosis, headache	2y	Rt: NA	Yes	NA	Negative	S	NA
Zhu *et al*[33] 2023	38y	M	Ocular hypertension	2wk	Rt: inferior orbit, extraconal space	NA	NA	Positive	S	No recurrence (7mo)
Kuga *et al*[34] 2024	47y	F	Diplopia, proptosis	NA	Rt: extraconal space	NA	NA	NA	S	NA
Bair *et al*[35] 2025	33y	F	Upper eyelid swelling, proptosis, and restricted extraocular motility	NA	Rt: lacrimal gland	No	NA	Positive	S	NA

IMT: Inflammatory myofibroblastic tumor; M: Male; F: Female; Rt: Right involvement; Lt: Left involvement; B: Bilateral involvement; S: Surgery; G: Glucocorticoids; R: Radiotherapy; C: Chemotherapy; T: Targeted therapy; NA: Not available.

Clinical Symptoms The primary ocular manifestations observed were proptosis in 12 cases (54.55%), blurred vision in 10 (45.45%), a palpable mass in 7 (31.82%), and pain in 7 (31.82%). Additional secondary manifestations included orbital swelling in 6 cases (27.27%), eye movement disorders in 5 (22.73%), headaches in 5 (22.73%), and diplopia in 4 (18.18%). Furthermore, alongside the ocular symptoms, 6 cases (27.27%) experienced intermittent fever, while 4 cases (18.18%) had lymphadenopathy during the disease.

Imaging Findings All patients with IMT underwent preoperative MRI or CT scans, which revealed a solid mass in the orbit. The tumors measured an average size of 2.7 (1.33, 3.9) cm. In orbital MRI scans, the lesions appeared hypointense to isointense compared to the muscle on T1-weighted images and isointense to hyperintense relative to the muscle on T2-weighted images. Additionally, there was intense or heterogeneous enhancement noted on post-contrast T1-weighted images.

The tumor was confined to the orbit in 10 cases (45.45%), while it involved the periorbital region in 12 cases (54.55%). The commonly affected sites included the greater wings of the sphenoid bone, the anterior skull base, the pterygopalatine fossa, the infratemporal fossa, the temporal fossa, the temporalis muscle, the cavernous sinus, the paranasal sinus, and the nasal cavity. Bone destruction was observed in 16 cases (72.73%), and peripheral inflammatory changes were noted in 17 cases (77.27%). These inflammatory changes could affect various areas, including the maxillary sinus, ethmoid sinus, sphenoid sinus, frontal sinus, turbinates, and the middle ear mastoid. Four patients (18.18%) were found to have enlargement of cervical lymph nodes on ultrasound or CT examination, and one patient also had enlargement of parotid lymph nodes. This represents an inflammatory change according to imaging findings.

Laboratory Test and Pathological Findings In five cases (22.73%), leukocyte levels were elevated; three cases (13.64%) displayed microcytic hypochromic anemia, and seven cases (31.82%) exhibited thrombocytosis, of which three patients had levels ≥450×10^9/L. C-reactive protein was detected in 16 patients, with six patients (37.5%) showing elevated levels.

Intraoperative observations revealed that the tumor appeared greyish-red or greyish-white, with ill-defined borders with the surrounding tissues. The tumor exhibited a fragile, rigid, or hard texture. Microscopic examination showed proliferation of fibroblasts and myofibroblasts, accompanied by infiltration of lymphocytes and plasma cells. Areas of fibrosis and scar formation were also noted, along with occasional tissue necrosis. Immunohistochemical analysis revealed positive expression of smooth muscle actin (SMA) in 19 cases, with expression unknown in 3 cases. Vimentin showed positive expression in 10 cases and was unknown in 12 cases. Anaplastic lymphoma kinase (ALK) was positive in 9 cases, negative in 10, and unknown in 3 cases. The expression level of Ki-67 was measured at 7.5% (5%, 20%).

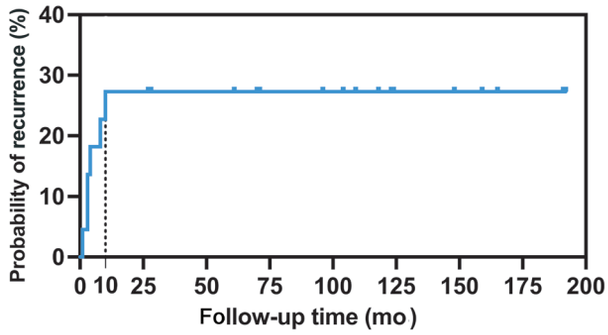

Figure 1 Recurrence rate in 22 orbital IMT patients IMT: Inflammatory myofibroblastic tumor.

Treatment and Follow-Up Surgery, along with a three-day postoperative glucocorticoid therapy, was conducted on 17 patients, out of which 4 (23.53%) experienced a relapse. Additionally, three patients received surgery combined with postoperative glucocorticoids and radiotherapy, and one of these patients (33.33%) relapsed. Additionally, two patients underwent surgery and received postoperative glucocorticoids and chemotherapy, with one patient (50%) experiencing a relapse. The average follow-up period was 106.23±10.52mo, during which six patients had a recurrence, resulting in an overall recurrence rate of 27.27% (Figure 1). The average time until recurrence was 4.83±1.40mo, with a range of 1 to 10mo. Sarcomatous degeneration was observed in two patients following their recurrence (Cases 10 and 14). Univariate analysis revealed no statistically significant differences between the recurrence group and the non-recurrence group in terms of age, gender, laterality, course of disease, periorbital tissue invasion, bone destruction, periorbital inflammation, tumor size, fever, leukocytosis, or treatment. However, lymphadenectasis and a Ki-67 index of 10% or higher may indicate a higher risk of recurrence, with *P*-values of 0.046 and 0.023, respectively (Figure 1, Table 2). No distant metastases were observed during the follow-up period.

A Case Report (Case 20) A 34-year-old man presented to the ophthalmology clinic with a progressively enlarging mass at the right medial canthus, persistent for 4mo. He denied symptoms of visual impairment, ocular redness, or photophobia. His medical history included blunt trauma to the nasal aspect of the right upper eyelid, initially causing dizziness and localized pain; this resolved spontaneously without intervention, but a mass emerged at the injury site 2mo later and gradually expanded. Ophthalmic examination revealed best-corrected visual acuity (BCVA) of 1.0 bilaterally and intraocular pressure within normal limits (15 mm Hg

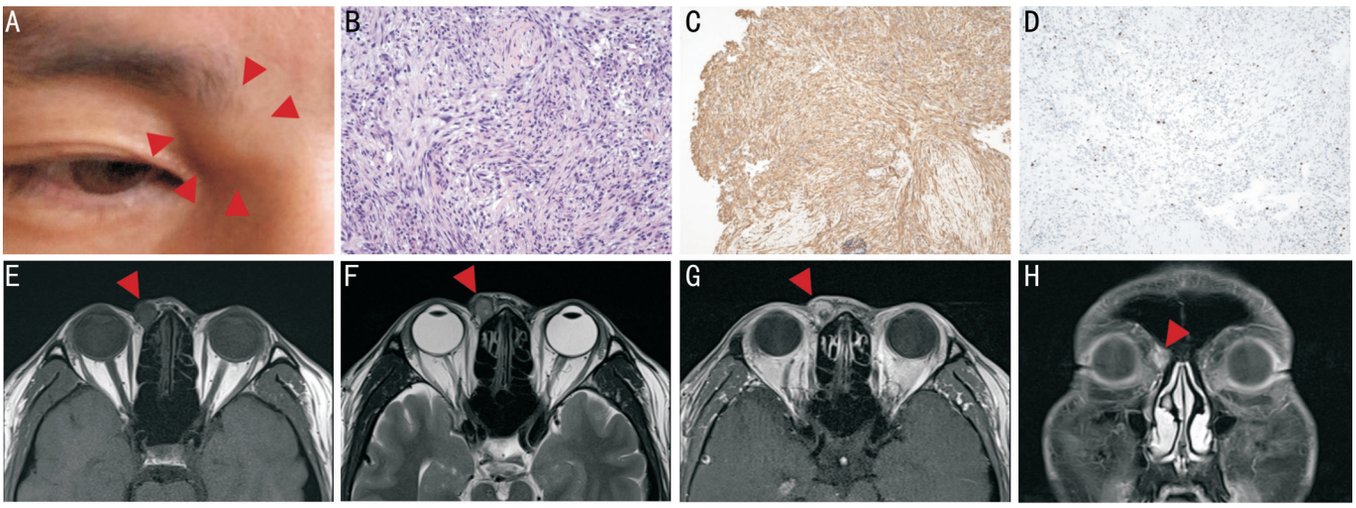

Figure 2 Clinical manifestations of Case 20 A: The clinical picture of Case 20. B-D: Histopathological and immunohistochemical images. B: The histopathological finding (HE ×200). C: A positive expression of smooth muscle actin (IHC ×100). D: A Ki-67 positive expression of approximately 10% (IHC ×100). E-H: The lesion in the right inner canthus as observed on MRI. T1WI signal was isointense (E), the T2WI signal was slightly hyperintense (F), and the lesion showed heterogeneous enhancement after enhancement (G). H: A coronal view of the mass location. HE: Hematoxylin and eosin staining; IHC: Immunohistochemistry; MRI: Magnetic resonance imaging.

OD, 16 mm Hg OS). A firm, poorly mobile, smooth-surfaced mass with tenderness was palpated at the nasal region of the right upper eyelid, measuring approximately 1×1 cm^2. Orbital MRI identified a round subcutaneous nodule adjacent to the right medial canthus, exhibiting intermediate signal intensity on T1-weighted images, slightly hyperintense signal on T2-weighted images, and ring enhancement post-contrast. The patient underwent a complete tumor excision under general anesthesia. Histopathological analysis demonstrated spindle-shaped cells arranged in bundles within a mucoid-to-collagenous matrix, accompanied by various inflammatory components, with immunohistochemistry positive for SMA and β-catenin but negative for ALK, MSA, CD34, STAT6, Desmin, EMA, SOX10, CD117, Olig-2, and S-100; a Ki-67 index of 10% confirmed the diagnosis of IMT. Postoperatively, he received oral methylprednisolone (24 mg daily for 3d) and remained recurrence-free throughout a 28-month follow-up period (Figure 2).

Table 2 Univariate analysis of factors affecting recurrence in 22 orbital IMT patients

Factors	Recurrence		*P*
	Yes (*n*=6)	No (*n*=16)	
Age (y)	19.83±11.46	31.40±15.89	0.121[a]
Gender			0.348[b]
Male	2	10	
Female	4	6	
Laterality			1.000[b]
Right	4	11	
Left	2	5	
Course of disease (mo)	3.00 (0.92, 8.25)	2.50 (1.00, 5.50)	0.824[c]
Periorbital tissue invasion	4	8	0.646[b]
Bone destruction	4	12	1.000[b]
Periorbital inflammation	6	11	0.266[b]
Tumor size (cm)	3.91±2.34	2.48±1.33	0.104[a]
Leukocytosis	3	2	0.100[b]
Fever	1	5	0.634[b]
Lymphadenectasis	3	1	0.046[b]
Ki-67 index			0.023[b]
<10%	1	12	
≥10%	5	4	
Treatment			0.706[b]
S+G	4	13	
S+R+G	1	2	
S+C+G	1	1	
Follow-up time (mo)	91.83±43.51	111.63±51.58	0.415[a]

S: Surgery; G: Glucocorticoids; R: Radiotherapy; C: Chemotherapy. [a]Independent samples *t*-test; [b]Fisher's exact test; [c]Mann-Whitney *U* test.

DISCUSSION

Orbital IMT is a rare condition, accounting for only 3% of cases in previous studies[37]. The exact cause and mechanisms behind IMT remain unknown. However, some cases suggest a possible link to infection, surgery, or trauma, indicating that it may result from abnormal healing processes triggered by injury[3]. In our study, we observed leukocytosis, elevated levels of C-reactive protein, and fever in affected patients, three of whom had a history of blunt trauma to the orbit. Additionally, around 77.27% of patients showed inflammatory changes in the periorbital sites, and nearly 20% experienced lymphadenopathy. These findings suggest that inflammation may play a role in the development of IMT, although the molecular mechanisms behind it still require further investigation. Furthermore, the occurrence of IMT is associated with ALK gene rearrangements due to chromosomal translocations[38]. Current knowledge identifies various gene fusion partners for ALK in IMT, including TPM3, TPM4,

CLTC, ATIC, TFG, CARS, RANBP2, SEC31L1, PPFIBP1, DCTN1, FN1, EML4, LMNA, NRP2, TNS1, HNRNPA1, and PRKAR1A[38-43]. Approximately 50% of IMT patients test positive for ALK[8]. In addition to gene rearrangements, the presence of multiple copies of the ALK gene may also contribute to the development of IMT[44]. ROS1, PDGFRβ, RET, and NTRK3 gene rearrangements can be detected in ALK-negative IMT[3,45-46].

IMT can occur at any age, but most patients are diagnosed before the age of 40[11]. A total of 28 cases of orbital IMT have been reported, with a median age of 15.5 (7, 36.75)y (ranging from 5mo to 71y) and 82.14% of the patients being younger than 40. In our study, the average age of patients with orbital IMT was 28.24±3.30y, with 18 patients under the age of 40, accounting for 81.82% of the cohort, which aligns with previous findings. Upon reviewing the literature, we found that there were 15 male and 13 female patients, indicating a slight predominance of males over females, consistent with our study results. Interestingly, we found that the average age of onset for male patients was greater than that for female patients in our study. However, this conclusion requires validation through larger studies due to the small sample size.

All patients exhibited unilateral involvement of the orbit. Tumors can appear in the anterior part of the orbit, including the lacrimal fossa, lacrimal sac, and subcutaneous tissue. They may also be found in the internal extraconal and intraconal spaces. Tumors situated in the anterior orbit are often palpable and noticeable, but they are frequently misdiagnosed as benign lesions, such as a chalazion, which can delay diagnosis and treatment. Symptoms of orbital tumors typically include exophthalmos, swelling of the orbit, and pain due to the mass effect. Compression of the optic nerve may lead to blurred vision. Additionally, the presence of these tumors, particularly with unclear boundaries and adhesion to the muscles, can restrict eye movement, resulting in diplopia for some patients.

Orbital CT and MRI findings can vary based on the relative amounts of fibrous components and cellular infiltrates present in the affected tissue[47]. As the course of IMT progresses, the structure and composition of the lesion tissue continually change, leading to diverse imaging results that often lack specificity. In our study, we found that the incidence of bone destruction and invasion of surrounding tissues was 72.73% and 54.55%, respectively. Additionally, 77.27% of patients exhibited inflammatory changes in the periorbital region, as observed through imaging. These findings highlight the invasive nature of IMT and provide important clues for its clinical diagnosis[48]. Furthermore, the occurrence of inflammatory changes in the periorbital region has not been documented in existing literature, indicating a need for further observation and confirmation through multicenter studies.

Inflammatory markers are often elevated in IMT patients, including leukocytosis, increased levels of C-reactive protein, anemia, and thrombocytosis[49]. In our study, we also observed abnormalities in the inflammatory index among some patients. This finding suggests that clinicians should closely monitor laboratory test results and consider the possibility of an IMT diagnosis in cases of orbital masses accompanied by an elevated inflammatory index.

Histopathological examination is the primary criterion for a definitive diagnosis. The key pathological features include spindle-shaped cells arranged in bundles within a mucoid-to-collagenous matrix, accompanied by various inflammatory components such as lymphocytes, plasma cells, and occasionally eosinophils and neutrophils[3]. In our study, both Vimentin and SMA were expressed positively in the spindle-shaped cells. Vimentin is typically strong and diffuse in the cytoplasm of the spindle-shaped cells, and SMA is 80%-90% positive in previous studies[11]. The positive rate of ALK expression in patients with IMT is approximately 50%[50-51]. However, ALK expression can vary significantly among patients, limiting its diagnostic utility[52]. Among the 22 cases of orbital IMT we studied, 47.37% exhibited positive ALK expression. In previously reported cases, 12 were ALK-positive, 11 were negative, and 5 were unknown, resulting in an ALK positivity rate of 42.86%, which aligns with our findings. At the same time, molecular detection plays a vital role in the diagnosis of IMT and may provide potential therapeutic targets for tumors[53-54].

Surgical resection of the tumor is the primary treatment for orbital IMT[3]. In cases where the tumor cannot be completely removed through surgery, a combination of surgery and adjuvant therapies can be utilized. These therapies may include glucocorticoid therapy, chemotherapy, low-dose radiotherapy, cryotherapy, and targeted therapy. Glucocorticoids have shown effectiveness in previously reported cases of orbital IMT. For instance, a 7-year-old boy with orbital IMT received just corticosteroids, presenting a stable mass documented by MRI over 12y, and the mass was reduced after 14y[16]. Similarly, an 11-year-old child with IMT diagnosed through biopsy had a favorable prognosis after receiving glucocorticoid therapy alone, with no recurrence reported during the follow-up period[20]. However, responses to glucocorticoid treatment can vary significantly among patients with orbital IMT, which may be attributed to the differing proportions of inflammatory and fibrotic components within the tumor[19]. Radiotherapy can be considered for patients whose IMT involves areas of the orbit that cannot be entirely resected[17]. Although chemotherapy had not been used for previous orbital IMT cases, our study included two patients who underwent postoperative chemotherapy. One of these patients experienced a relapse,

indicating that the efficacy of chemotherapy for this condition requires further investigation. In a reported case, cryotherapy was administered to a patient who experienced a recurrence of IMT. A 21-year-old patient relapsed 10mo after an initial surgical resection, underwent a second surgical procedure, and received cryotherapy as an additional treatment. Following this, no recurrence was observed for 10mo[23]. Recent advancements in IMT cytogenetics have led to significant progress in targeted therapy[53,55]. Crizotinib, a first-generation ALK Tyrosine Kinase Inhibitor (ALK-TKI), works by disrupting tumor mutational signaling pathways, thereby inhibiting sustained tumor growth[49]. Both crizotinib and second- or third-generation ALK-TKIs are viable treatment options for IMT patients with gene rearrangements. However, due to the rarity of orbital IMT, there are limited clinical trials and case studies focusing on targeted therapy for ALK-positive IMT. To date, only three cases of crizotinib treatment for IMT have been reported[25,28,31]. In one case, a patient was treated with crizotinib alone, while the other two received crizotinib after surgical resection. All patients displayed good prognoses. Notably, one individual experienced complete regression while on crizotinib one year post-surgery but relapsed 3mo after discontinuing the medication. After increasing the crizotinib dosage, this patient achieved complete remission again within 6mo, with no recurrence or metastasis noted during a 14-month follow-up[25]. For patients with gene rearrangements, targeted therapy offers a promising option as an effective adjuvant treatment.

The overall prognosis for IMT is favorable[51,56]. Previous studies reported a recurrence rate of 24%, while our study found a recurrence rate of 27.27%, consistent with earlier research[47]. In past 28 cases, recurrences occurred at intervals of 10 and 15mo. In our study, recurrences were identified within one year. Notably, two patients exhibited sarcomatous degeneration during their recurrence, highlighting the importance of rigorous follow-up after treatment. We conducted a univariate analysis of prognostic factors. Variables such as age, gender, laterality, duration of disease, invasion of periorbital tissue, bone destruction, periorbital inflammation, tumor size, fever, leukocytosis, and treatment type showed no statistical differences between the recurrent and non-recurrent groups. However, patients presenting with lymphadenopathy and a Ki-67 index of 10% or higher may be at a greater risk for recurrence. Four patients with lymphadenopathy did not undergo lymph node biopsy, but imaging findings suggested that an inflammatory reaction was more probable. This indicates the importance of careful physical examinations, as lymph node enlargement may signal a worse prognosis.

The limitations of this study include the small number of patients and the retrospective design, which may lead to discrepancies in data collection, so our findings still need to be verified by multi-center and large-sample IMT clinical studies.

In conclusion, the incidence of orbital IMT is low. These tumors typically occur in young and middle-aged patients, with male patients generally experiencing onset at an older age than female patients. The clinical manifestations of IMT are non-specific, often leading to misdiagnosis as benign lesions. IMT is prone to bone destruction and invasion of adjacent sites, and inflammatory changes in periorbital regions can be observed in imaging examinations. Orbital IMT is considered a locally invasive disease with the potential for recurrence and sarcomatous degeneration. Overall, the prognosis is favorable; however, IMT accompanied by lymphadenopathy and a Ki-67 index of 10% or higher may indicate a greater risk of recurrence.

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