called Mikulicz's disease or chronic sclerosing dacryoadenitis.[82–86] Clinically, the lacrimal glands are involved, and bilateral lacrimal gland swelling is frequently observed.[80] Though some patients do not show obvious lacrimal gland involvement clinically, lacrimal gland component was frequently detected histologically. This suggests that accessory lacrimal glands may be involved.

Mikulicz's disease is a unique condition that refers to bilateral, painless and symmetrical swelling of the lacrimal, parotid and submandibular glands. Although Mikulicz's disease has been considered a subtype of Sjögren syndrome, there are several differences between the two diseases. Patients with Mikulicz's disease lack anti-SS-A and anti-SS-B antibodies, but frequently have elevated serum IgG4 levels.[34,82–84] Infiltration of many IgG4-positive plasma cells into the lacrimal and salivary glands has been detected in Mikulicz's disease. Additionally, Mikulicz's disease has good responsiveness to steroids, and reversible of lacrimal and salivary gland function. Thus, it is important to distinguish Mikulicz's disease from Sjögren syndrome.[34,82–84]

The ocular adnexal IgG4-related disease is histologically uniform: marked lymphoplasmacytic infiltration and lymphoid follicles, admixed with dense fibrosis, and infiltration of many IgG4-positive plasma cells.[80] These findings are similar to those of previous reports of IgG4-related disease of other organs. The ocular adnexal IgG4-related diseases often are associated with ones of the salivary glands.[80]

As referred to here, obliterative phlebitis has been identified as a histological feature of IgG4-related diseases since Kawaguchi *et al.* reported on the histopathology of sclerosing pancreatitis in 1991,[9] and it has been easily and characteristically found in sclerosing pancreatitis and sclerosing sialadenitis. But obliterative phlebitis is usually not detected in ocular adnexal IgG4-related disease.[80] Therefore, we suggest that obliterative phlebitis may be organ specific, but not a common feature of IgG4-related diseases.

Interestingly, although serum IgG4 levels are often evaluated after treatment, it remains elevated even in remission.[80] This may be due to residual IgG4-secreting plasma cells located subclinically elsewhere.

Ocular adnexal IgG4-related disease and mucosa-associated lymphoid tissue lymphoma

Little is known about lymphomagenesis in the context of IgG4-related disease.[80,85,87] We recently first reported ocular adnexal mucosa-associated lymphoid tissue (MALT) lymphomas arising from IgG4-related disease, occurring in the same organ.[80]

MALT lymphoma is an extranodal lymphoma consisting of morphologically heterogeneous small B-cells including marginal zone cells.[88,89] The infiltrate is in the marginal zone of reactive B-cell follicles and extends into the interfollicular region. In epithelial tissues, the neoplastic cells typically infiltrate the epithelium, forming lymphoepithelial lesion. The presence of lymphoepithelial lesion is important when making a diagnosis of MALT lymphoma.[88,89]

In many cases of MALT lymphoma, there is a history of chronic inflammatory, often autoimmune disorders that result in accumulation of extranodal lymphoid tissue. These include *Helicobacter pylori*-associated chronic gastritis, Sjögren syndrome or Hashimoto thyroiditis.[88,89] Thus, we considered that patients with ocular adnexal IgG4-related disease may be at an increased risk of developing ocular adnexal MALT lymphoma. Another study has also described ocular adnexal lymphomas arising from IgG4-related disease.[85] Takahashi *et al.* reported that three patients with IgG4-related disease with or without autoimmune pancreatitis later developed B-cell non-Hodgkin lymphoma (two of whom developed diffuse large B-cell lymphoma).[87] In addition, Ochoa *et al.* reported on marginal zone B-cell lymphoma of the salivary gland arising in Küttner tumor.[90] It has previously been noted that autoimmune pancreatitis and Küttner tumor were considered to be IgG4-related disease. Therefore, these reports suggest that IgG4-related disease may be a risk factor for malignant lymphoma.

We experienced seven patients with the ocular adnexal MALT lymphomas arising from IgG4-related disease (IgG4-related ocular adnexal MALT lymphoma), occurring in the same organ. Six patients had localized disease (clinical stage IE or IIE; unpubl. data, 2009). Histologically, in this series of patients there was dense fibrosis subdividing the lacrimal gland, and marked lymphoid cell infiltration with lymphoid follicles. These histological findings were consistent with previous reports of IgG4-related disease. However, some infiltrated lymphoid cells showed centrocyte-like features, and Dutcher bodies were found in some of the cases (Fig. 6) in addition to histological finding of IgG4-related disease. All cases had immunoglobulin light chain restriction, and immunoglobulin heavy chain gene rearrangement on polymerase chain reaction and/or Southern blot hybridization. Interestingly, lymphoepithelial lesion was not found in any cases. Lymphoepithelial lesions usually are not found in ocular adnexal MALT lymphomas (especially in the lacrimal gland region).[90] Another report also noted that lymphoepithelial lesion was not found in ocular adnexal IgG4-related MALT lymphoma. It remains unclear whether the absence of lymphoepithelial lesion indicates biological differences in the lacrimal gland, or whether the epithelium may have been destroyed due to IgG4-related chronic inflammation.

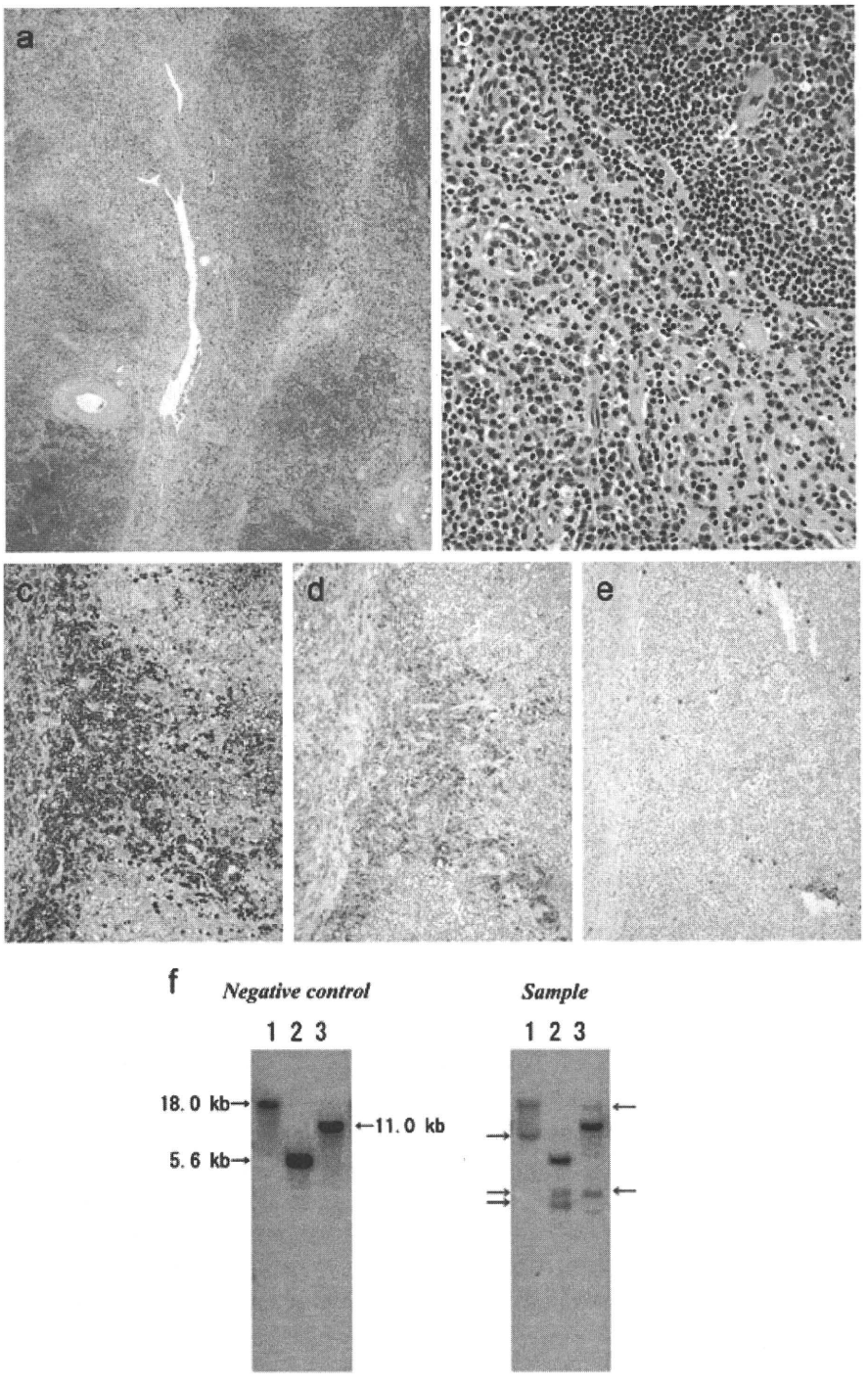

Figure 7 Ocular adnexal IgG4-producing mucosa-associated lymphoid tissue lymphoma. (**a**,**b**) Dense fibrosis and marked lymphoplasmacytic infiltration in the lacrimal gland. Histologically, this is compatible with previous reports of IgG4-related sclerosing disease. Immunostain for (**c**) IgG4, (**d**) kappa-light chain and (**e**) lambda-light chain. Most of the IgG4-positive cells exhibit kappa-light chain restriction. (**f**) Immunoglobulin heavy chain gene rearrangement was detected on Southern blot hybridization.

There have been many reports on ocular adnexal IgG4-related lymphomas at the annual meetings of the Japanese Society, but in IgG4-related disease of other sites, there is rare or absent IgG4-related MALT lymphoma. In the orbital region, the most common tumor is malignant lymphoma, especially MALT lymphoma.[91] In contrast, submandibular gland and pancreas have a low incidence of MALT lymphoma. Therefore IgG4-related MALT lymphoma may occur more easily in the ocular adnexa.

IgG4-PRODUCING LYMPHOMA

Little is known about IgG4-producing lymphoma.[85,92] We recently reported the first case of IgG4-producing marginal zone B-cell lymphoma of the lymph node.[92] The IgG4-positive tumor cells were lambda light-chain-restricted and CD138 partially positive, although the expression was fainter than that of the non-neoplastic cells. Additionally, the tumor cells were partially positive for CD20, which is normally

negative in non-neoplastic plasma cells, and had elevation of serum IgG4 level.[92] Therefore that case indicates that not only can malignant lymphomas occur in the setting of IgG4-related diseases, but that IgG4-producing cells can also be neoplastic.

Moreover, we encountered a case of ocular adnexal IgG4-producing MALT lymphoma (Fig. 7). The histology was compatible with ocular adnexal IgG4-related disease (Fig. 7a,b), and there was elevation of serum IgG4 level, serum IgG4/IgG ratio, and IgG4/IgG-positive cell ratio (≥50%). The lesion exhibited immunoglobulin light chain restriction of IgG4-positive cells (Fig. 7c–e) and immunoglobulin heavy chain gene rearrangement (Fig. 7f). Previously, Cheuk *et al.* also reported on ocular adnexal IgG4-producing lymphoma.[85] They concluded that it remains unclear whether ocular adnexal IgG4-producing MALT lymphoma arises from pre-existing IgG4-related disease, or de novo IgG4-postive MALT lymphoma. We suggest that it may clonal expansion of IgG4-positive cells occurring against a background of IgG4-related chronic inflammation. This is because the case showed marked lymphoplasmacytic infiltration and lymphoid follicles, admixed with dense fibrosis, and also detected elevation of serum IgG4 level. These findings are compatible with IgG4-related disease.

Clinicopathological features of IgG4-producing lymphoma should be clarified in the future by accumulation and evaluation of such cases.

CONCLUSION

IgG4-related diseases are a new clinicopathological systemic entity, but the pathogenesis and etiology remain unclear. IgG4-related diseases have a good response to steroids. Accordingly, accurate pathological diagnosis is very important.

ACKNOWLEDGMENTS

This work was supported in part by grants from Intractable Diseases, the Health and Labour Sciences Research Grants from Ministry of Health, Labor and Welfare (H21-112).

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